American College of Physicians- Minnesota Chapter Annual Abstract Competition

Poster Session

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Abstracts Submitted for Competition

Medical Students	
Research - Medical Students	
Ronin Joshua Cosiquien Krishna Allam Sai Anoop Dhamera Hani Hassoun Dr. Edgar Jaimes Dr. Abhijat Kitchlu Dr. Heather Landau Ming Shen Veronica Torres Dr. Sabine Karam	*Metabolic Acidosis Independently Predicts Overall Survival in Multiple Myeloma and Captures at Diagnosis Prognostic Information Complementary to R-ISS* Background: The Revised International Staging System (R-ISS) is the standard prognostic framework for newly diagnosed multiple myeloma (MM), yet host-level metabolic derangements contributing to survival heterogeneity within stages remain understudied. Metabolic acidosis is common at MM diagnosis, but its independent prognostic significance after adjustment for renal function and staging has not been established. Methods: In a single-center retrospective cohort of 293 patients with newly diagnosed MM (March 2009–January 2024, University of Minnesota; median follow-up 60.0 months), we evaluated overall survival (OS) from treatment initiation (censored at 5 years). Metabolic acidosis was defined as serum bicarbonate <22 mmol/L; renal impairment was defined per International Myeloma Working Group (IMWG) criteria (creatinine ≥2.0 mg/dL or eGFR ≤40 mL/min/1.73 m²). A multivariable Cox model assessed metabolic acidosis, age, sex, renal impairment, diabetes, R-ISS stage, and IgA heavy chain status. Missing data were addressed by multiple imputations by chained equations (MICE) with Rubin's rules pooling. A 2-factor risk score was compared with R-ISS by Harrell's C-index and Spearman correlation. Results: Among 293 patients (median age 65.0), metabolic acidosis was present in 14.8% and diabetes in 12.0%. Acidosis was independently associated with inferior OS (HR 2.60, 95% CI 1.31–5.16; P = 0.007). This association was not explained by graded renal dysfunction: acidosis remained significant when continuous eGFR replaced binary renal impairment (HR 2.46, 95% CI 1.24–4.79; P = 0.009), and bicarbonate correlated only moderately with eGFR (Pearson r = 0.42). Acidosis also remained significant after ASCT adjustment (HR 2.33, 95% CI 1.19–4.56; P = 0.014).

	Other covariates independently associated with OS included diabetes (HR 2.69, 95% CI 1.51–4.79; P = 0.001), R-ISS stage (HR 1.96 per stage, 95% CI 1.32–2.91; P = 0.001), IgA heavy chain (HR 1.94, 95% CI 1.16–3.25; P = 0.013), and age (HR 1.05 per year, 95% CI 1.03–1.08; P < 0.001). Renal impairment was not independently significant (HR 0.63, 95% CI 0.32–1.25; P = 0.179). Full model discrimination was good (C-index 0.736). 2-factor stratification using acidosis and diabetes (≥1 vs. 0 factors) yielded an unadjusted HR of 2.57 (95% CI 1.57–4.19; P < 0.001). This score was minimally correlated with R-ISS (Spearman ρ = 0.069), and a combined model improved discrimination beyond either alone (C-index 0.658 vs. 0.604 for R-ISS and 0.603 for the 2-factor score individually).
Varun Dabade Arash Kardoust	*Feeling the Feedback: A Medical School Curriculum to Improve Soliciting and Processing of Feedback* Introduction: Medical education is moving towards competency-based assessments, relying on effective, formative feedback. Medical students must solicit and process large amounts of variable feedback without clear guidance, which can lead to feelings of shame, inadequacy, and confusion. Early, clear instruction in soliciting and processing feedback may help promote a growth mindset. Methods: Starting in 2018, our institution began dedicated feedback related sessions in the peri-clerkship curriculum. A session taught to early clerkship students focuses on how to process and solicit feedback during clinical rotations. Starting in 2022, students who participated in this session were voluntarily surveyed to determine the effectiveness of overtly teaching this content. Results: Evaluations using pre- and post-surveys showed improvement of student perception regarding their feedback skills, including confidence recognizing effective versus ineffective feedback as well as their comfort processing constructive feedback. Most impressively was the improvement in students' understanding for how to solicit feedback from peers and superiors. Discussion: Clear instruction and guidance in developing skills surrounding feedback was met with great improvement in self-perception and confidence. Other medical schools and health care professions may benefit from overtly teaching the reception of feedback to support their students as they enter portions of their training dependent on formative assessment.
Anoop Dhamera Krishna Allam Ronin Joshua Cosiquien	*KDIGO Acute Kidney Injury Criteria do not Predict End-Stage Kidney Disease in Newly Diagnosed Multiple Myeloma*

Hani Hassoun, Edgar Jaimes Abhijat Kitchlu Heather Landau Ming Shen Veronica Torres Sabine Karam	Background: Renal impairment occurs in approximately 20% of patients with newly diagnosed multiple myeloma (NDMM) and influences treatment selection and prognosis. The International Myeloma Working Group (IMWG) and Kidney Disease: Improving Global Outcomes (KDIGO) frameworks define renal impairment differently, yet their agreement and comparative ability to predict long-term renal outcomes in NDMM have not been evaluated. Methods: In a retrospective single-center cohort of 294 patients with NDMM, we compared two definitions of renal impairment at treatment initiation: IMWG renal impairment (IMWG-RI; serum creatinine ≥2 mg/dL or eGFR ≤40 mL/min/1.73 m²) and KDIGO acute kidney injury (KDIGO-AKI; creatinine ≥1.5× baseline). The primary endpoint was cumulative incidence of end-stage kidney disease (ESRD; eGFR <15 mL/min/1.73 m²) in a competing-risks framework with death as the competing event, modeled using age-adjusted Fine–Gray subdistribution hazard regression. Definitional agreement was assessed by Cohen's kappa. Discrimination for ESKD was compared across continuous admission eGFR, binary IMWG-RI, and binary KDIGO-AKI using area under the receiver operating characteristic curve (AUC) with DeLong testing. Renal recovery was defined as eGFR >40 mL/min/1.73 m² by end of induction. Multiple imputations (m = 20) addressed missing admission creatinine values. IMWG-RI was present in 49/263 evaluable patients versus KDIGO-AKI in 9/260. IMWG-RI prevalence varied significantly by myeloma subtype (P = 0.033) highest in light-chain (40.0%) and lowest in IgG myeloma (13.7%). Agreement between the two definitions was poor (κ = 0.121, 95% CI −0.104 to 0.345), with significant marginal asymmetry (McNemar P = 1.81×10^{-8}). Results: Among 44 patients classified as IMWG-RI–positive but KDIGO-AKI–negative, 93.2% had baseline eGFR <40 mL/min/1.73 m², indicating pre-existing chronic kidney disease (CKD) rather than acute injury. Patients with IMWG-RI had lower hemoglobin, lower platelets, and higher beta-2-microglobulin. Over a median potential follow-up of 4.99 years, 16 ESRD events and 75 competing deaths occurred among 261 evaluable patients. IMWG-RI was strongly associated with ESKD (age-adjusted Fine–Gray HR 5.04, 95% CI 1.87–13.56; P = 0.0014), with 5-year cumulative incidence of 21.5% versus 3.8% without IMWG-RI. By contrast, KDIGO-AKI identified none of the 16 ESRD cases, compared with 50.0% for IMWG-RI. AUC for ESKD was 0.805 for continuous eGFR, 0.698 for IMWG-RI, and 0.481 for KDIGO-AKI; continuous eGFR outperformed binary IMWG-RI (DeLong P = 0.015). Renal recovery occurred in 27/49 IMWG-RI patients (55.1%). IMWG-RI was not associated with overall survival (age-adjusted HR 1.06, 95% CI 0.60–1.86; P = 0.840). Conclusion: Multiple imputation confirmed the primary finding In newly diagnosed MM, IMWG-RI and KDIGO-AKI show poor agreement and identify fundamentally different populations: IMWG-RI captures patients with pre-existing CKD who carry a 5-fold

	increased risk of ESRD, whereas KDIGO-AKI fails to identify any patient who progresses to ESRD. Continuous admission eGFR provides superior discrimination for long-term renal outcomes and should be incorporated into risk stratification at diagnosis.
Fernando Flores Leon Dr. Shahd Mansour Daniela Polonio Mejia Dr. Mohammad Alomari] Dr. Diem Vu Dr. Moustafa El Khatib Dr. John Zietlow Dr. Stepanie Heller Dr. Mark Sawyer Dr. Ahmad Abutaka Dr. Henry Schiller Dr. Daniel Stephens	*Endoscopic Extraction of a Trans-Cystic Stent and Potential Reduction of Surgical Site Infections After Interval Cholecystectomy* Background: Our aim is to determine whether endoscopic extraction of trans-cystic stents during or prior to interval cholecystectomy decreases the risk of surgical site infection, compared to transabdominal extraction during cholecystectomy. Methods: Single institution, retrospective medical record review. Results: There were 104 patients who underwent cholecystectomy after endoscopic placement of a trans-cystic stent. Any surgical site infection (SSI) was documented in 20 patients (19%), superficial in 13 (13%), and deep or organ-space in 13 (13%). Importantly, 16% of incisions were left open to heal by secondary intention. The stents were extracted transabdominally during surgery in 64 patients (62%) and endoscopically in the remaining 40 (38%). There was no significant difference in the occurrence of any SSI (6 (15%) vs 14 (22%), p= 0.39), superficial SSI (4 (10%) vs 9(14%), p=0.54) or deep/organ space SSI (4 (10%) vs 9 (14%), p=0.54). Intraoperative disruption of the gallbladder occurred in 44 patients (43%) and was not associated with a significant increase in surgical site infections (any SSI in 12 out of 60 patients with intact gallbladder (20%) vs 8 out of 44 patients with intraoperative disruption (18%), p=0.82). Intraoperative drain placement was not associated with a significant reduction in deep/organ-space SSI (10 out of 69 patients without intraoperative drain placement (14%) vs 3 out of 35 patients with intraoperative drain placement (9%), p=0.39). To demonstrate a statistically significant difference in the proportion of SSI occurrences between two methods of trans-cystic stent extraction, with an alpha of 0.05 and a power of 0.8, a sample size of 479 patients per group is required. Conclusion: SSIs are common after interval cholecystectomy following initial endoscopic treatment. We were unable to demonstrate a significant reduction. However, we have identified a trend and established the basis for future studies.
Sakhi Gautam Dr. Ashley Petersen Shurui Pan Katherine Harrison, MPH Dr. Sharon Allen	*Association Between Smoking Status at Enrollment, Social Support, and Happiness in Postpartum Women* Objective: The study evaluates whether perceived social support and happiness measures are associated with smoking status at enrollment among postpartum individuals in a relapse prevention trial. We also

	describe the longitudinal changes in social support and happiness measures over the subsequent 1-year period. Background: Postpartum individuals face a heightened risk for continued smoking or relapse, even among women who remained abstinent during pregnancy. Previous studies have found that improvements in mental health and social support may play a role in smoking cessation. There is limited information about how perceived social support and subjective happiness influence smoking status. Methods: Participants were postpartum treatment-seeking individuals enrolled in the BurPPP (Bupropion for the Prevention of Postpartum Smoking Relapse) study (N=53). At enrollment, the Multidimensional Scale of Perceived Social Support (MSPSS) was used to assess the perceived adequacy of participants' social support networks, and the HPPNSS, a four-item scale in which participants rate their general happiness and how they compare to peers, was used to assess happiness. Two separate logistic regressions were constructed to evaluate the associations between smoking status at enrollment and (1) happiness and (2) perceived social support, adjusting for pre-specified confounders. Results: Self-reported happiness was not significantly associated with smoking status at enrollment ($p = 0.97$; aOR = 1.01, 95% CI: 0.75-1.34). Social support showed no significant relationship with smoking status at enrollment ($p = 0.19$); each one-unit increase in the total MSPSS score was associated with a 5% increase in the odds of smoking at enrollment (aOR = 1.05, 95% CI: 0.98-1.12). Participants who were smoking at enrollment saw their social support decline markedly at week 24, before slowly starting to recover by week 52. Those who were not smoking at enrollment saw their happiness grow slowly and steadily across the whole year. Participants who smoked at baseline hit a peak in happiness at week 24, but then dropped by week 52. Conclusion: Perceived social support and happiness were not significantly associated with smoking status at enrollment among postpartum individuals. Trends over a one-year period suggest these measures change differently over time by enrollment smoking status, highlighting the need for further investigation.
Laura Hankins Dr. Makayla Maher Dr. Apoorv Tiwari Luke Levasseur Dr. Saarah Wernimont Katelyn Tessier	*Prescribing Trends of Continuous Glucose Monitor Use in Pregnancies Complicated by Type 2 Diabetes and Associated Maternal and Neonatal Outcomes* Introduction: The use of continuous glucose monitoring (CGM) in pregnancy has expanded following recent regulatory approvals. However, while there are established benefits in type 1 diabetes, data supporting CGM use in pregnancies complicated by type 2 diabetes (T2DM) remain limited. Understanding CGM adoption and associated outcomes is critical to evaluating its role in this population.

Dr. Megan Kristan	Objective: To evaluate demographic and clinical factors associated with CGM use in pregnant individuals with T2DM and to compare maternal and neonatal outcomes between CGM and self-monitoring of blood glucose (SMBG). Methods: We conducted a retrospective cohort study using an existing database at a large community-academic health system, including births from January 2023 to July 2025. Pregnant individuals with pre-existing T2DM were identified and categorized by glucose monitoring modality: CGM or SMBG. Baseline demographics, glucose metrics, and maternal and neonatal outcomes were compared using Wilcoxon rank-sum tests for continuous variables and chi-square or Fisher's exact tests for categorical variables, with significance defined as $p<0.05$. Results: Among 283 patients included in the prescription cohort, those prescribed CGM were more likely to be white, English-speaking, not require an interpreter, receive care in urban settings, and they had a higher pre-pregnancy BMI. CGM prescription was also associated with endocrinology involvement and greater insulin use, including higher total daily insulin dose at birth. In a subset of 207 patients with documented glucose monitoring data, 111 were monitored using SMBG and 96 using CGM. CGM use was strongly associated with endocrinology involvement (77.1% vs 33.3%, $p<0.001$). CGM patients had more glucose data documented during pregnancy, and higher recorded fasting and postprandial glucose values than those using SMBG. At 28–30 weeks' gestation, CGM patients had significantly higher average fasting and 1-hour postprandial glucose values, while SMBG patients had a significantly greater proportion of fasting values <95 mg/dL and postprandial values within target range (all $p<0.01$). Hemoglobin A1c 3 and 6 months prior to birth was lower in the CGM group than in the SMBG group, although interpretation is limited by differential data availability. There were no statistically significant differences in maternal or neonatal outcomes. Conclusion: Patients differed along key demographics for use of CGMs. While those monitored with CGM had increased glucose data capture and higher recorded fasting and postprandial values, there were no statistically significant differences in perinatal outcomes. Differences in recorded glycemic measures may reflect differences in baseline disease severity, treatment intensity, and data capture rather than glycemic management alone, highlighting the need for prospective studies for standardized CGM implementation.
Rebecca Heinze Dr. Claire Embree	*Delirium Detection Using Point-of-Care Single-Channel EEG: Comparison Across Two Acute Care Populations*

Dr. Mohammed Elgendy Yvonne Larson Tage Runkle Emma Behnken Dr. Gen Shinozaki Dr. Allyson Palmer	Introduction: Delirium is an acute state of confusion commonly characterized by fluctuations in attention, cognition, and consciousness. It is common yet frequently underdiagnosed among hospitalized older adults and is associated with prolonged hospitalization, dementia, increased mortality, and poor patient outcomes. Current diagnostic methods, including electroencephalography (EEG) and bedside cognitive assessments, may be limited by cost, workflow disruption, and subjectivity. We evaluated the diagnostic performance of a point-of-care, single-channel for delirium detection in two acute care populations. Methods: A total of 242 adults aged 60 or older were enrolled, including 91 post-operative patients (mean age 74.7; 50.5% male) and 151 general medicine patients (mean age 75.5; 54.5% male). Participants underwent daily paired 3D-CAM and EEG assessments for up to seven days. EEG recordings were processed using an online application to calculate a score reflecting the ratio of slow- to fast-frequency brain activity (bispectral EEG, or BSEEG, score), with higher scores hypothesized to reflect delirium-associated slowing. Conclusion: Mean BSEEG scores were significantly higher in patients with versus without delirium in both the general medicine (1.492 vs. 1.376, P = 0.003) and post-operative (1.566 vs. 1.417, P = 0.001) cohorts. Using BSEEG score cutoffs of 1.334 (general medicine) and 1.41 (post-operative) to classify delirium, sensitivity and specificity were 84.85% and 44.80%, respectively, in general medicine patients, and 82.35% and 51.66% respectively, in post-operative patients. BSEEG scores increased with age, suggesting reduced diagnostic accuracy in older patients. The BSEEG method identified delirium with sensitivities of 84.85% and 82.35%, and specificities of 44.80% and 51.66% in the general medicine and post-operative populations, respectively. BSEEG scores increased with age, suggesting reduced diagnostic accuracy in older patients. Discussion: These findings demonstrate the potential utility of point-of-care, single-channel EEG as a rapid, objective bedside screening tool for delirium in diverse inpatient settings. BSEEG demonstrated high sensitivity but modest specificity in both general medicine and post-operative populations, and an association between BSEEG scores and age suggest that age-adjusted thresholds may improve diagnostic performance. Future studies should focus on improving specificity and validating age-adjusted thresholds across diverse inpatient populations.
Eashwar Krishna Sam Johnson Sanskar Shah	*Opioid Dependency and Long-Term Opioid Therapy Among Adults with Rheumatic Disease: A Mixed-Methods Analysis* Background: Rheumatic diseases are a major cause of chronic pain and opioids remain a common treatment despite growing recognition of opioid use disorder (OUD) risk. Claims-based studies report chronic

	opioid use in rheumatoid arthritis (RA) patients rising from roughly 12–31% between 2002–2015, with highest risk among patients aged 50–64 and those with Medicaid/Medicare coverage, higher disease activity, or psychiatric comorbidity. However, few studies have attempted to collate the relevant evidence around long-term opioid use along with OUD, an important goal considering that not all patients dependent on opioids may receive a use disorder diagnosis. Methods: We conducted a narrative literature review alongside a cross-sectional analysis of NHANES (2011–2018) among adults ≥20 years with osteoarthritis (OA), RA, or psoriatic arthritis (PsA). Survey-weighted logistic regression identified predictors of long-term (>90-day) opioid use among respondents reporting opioid use. Results: Among 3,811 respondents with rheumatic disease, weighted opioid use prevalence was 17–26% across subtypes, comparable to claims-based estimates (16.8–21.9%). In adjusted models, Medicare coverage (aOR 3.81, 95% CI 1.87–7.76) and age 50–64 (aOR 2.33, 95% CI 1.17–4.66) were independently associated with long-term use, consistent with prior RA-specific findings implicating this same age band and insurance type. A recent coverage gap was associated with lower odds of long-term use (aOR 0.25, 95% CI 0.08–0.79), suggesting continuity of insurance access, rather than insurance status alone, may drive sustained opioid therapy. Prior work estimates OUD diagnosis affects ~4% of rheumatic disease patients, with MOUD uptake (~14%) below national benchmarks (~25%). Conclusion: Insurance continuity and age are consistently associated with long-term opioid use in rheumatic disease. Combined with low medications for opioid use disorder (MOUD) uptake reported in our review of the literature, these findings support increased rheumatologist education around opioid prescribing and OUD treatment access.
Luke Levasseur Dr. Kerry Sheets	*History of AIDS and Subjective Cognitive Concerns Among Older Adults Living with HIV* Introduction: Neurocognitive disorders are common among people living with HIV (PLWH). Even with the modern era of effective antiretroviral treatment (ART), it is estimated that 30-50% of HIV positive patients have some level of neurocognitive cognitive concerns.1 In addition, low CD4 NADIR, advanced disease state, and other comorbidities common in older adults often amplify this risk.2 With the recent advancements of ART increasing life expectancy for PLWH 3, these comorbidities have to potential to display a compounding effect on neurocognition. Our objective was to determine the extent to which a history of AIDS is associated with subjective cognitive concerns among older adults seen in the HIV and Aging Clinic at Hennepin Healthcare (HHS).

	Methods: We abstracted data from the medical records from 133 PLWH aged 50 years and older seen in HHS's HIV and Aging clinic from 2021-2025. The data collected was either at the time of the patient's first visit to the clinic or the nearest clinical documentation of the visit. We specifically collected based on AIDS status (based on <200 CD4 NADIR count at any time or clinical documentation), Mini-cog, MoCA, PHQ-9, and PHQ-2 scores, date of HIV diagnosis, and patient reported changes in cognition. Logistic regression was used to determine associations between a history of AIDS and subjective cognitive concerns. Results: In this cohort of PLWH, a history of AIDS was not significantly associated with subjective cognitive concerns in either unadjusted (OR 0.97 95% CI 0.46-2.0) or adjusted (OR 0.91, 95% CI 0.42-2.0) analyses, but the confidence intervals were wide. Conclusion: Contrary to expectations, a history of AIDS was not associated with subjective memory concerns. Further research with larger sample sizes and objective neurocognitive testing may be needed to fully understand the relationship between advanced HIV disease and cognitive outcomes in this population. Future studies investigating neuroprotective effects of ART in PLWH over the age of 50 may provide insight to the findings in this study.
Pablo Monterroso Cassandra Butterbaugh Jacob Rugloski Dr. Martin Felices Dr. Jeffrey Miller Dr. Darko Bosnakovski Dr. Andrea Espejo Freire Dr. Nicholas Zorko	*Preclinical Evaluation of a B7-H3 Tri-Specific Killer Engager in Ewing Sarcoma and CIC-DUX4 Sarcoma* Objective: Ewing sarcoma and CIC-DUX4 (CDX4) sarcoma are aggressive, fusion-driven round cell sarcomas with limited treatment options. Both are characterized by low neoantigen burden and immune-evasive biology, including impaired antigen presentation through HLA/MHC-I downregulation, which suggests that NK-cell engagers may be active in these tumors. B7-H3 (CD276) is highly expressed across sarcomas, but minimally expressed in normal tissues, and is a promising immunotherapy target. We evaluated the activity of a novel dual camelid nanobody B7-H3 Tri-Specific Killer Engager (TriKE or GTB-5550®), developed at the University of Minnesota, combines a CD16-binding domain, a B7-H3–targeting domain, and an IL-15 linker to activate and redirect NK cell killing against Ewing sarcoma and CIC-DUX4 sarcoma cell lines. Methods: Peripheral blood mononuclear cells from healthy donors were co-cultured with Ewing sarcoma (ES) cell lines (A673, TC71, SK-ES-1) and CDX4 sarcoma cell lines (NCC-CDS-X1, KITRA-SRS). NK-cell activation was assessed by CD107a expression and inflammatory cytokine production (IFN-γ). Controls included PBMCs alone, IL-15, or B7-H3 TriKE. Results: Co-culture with B7-H3 TriKE increased NK-cell activation compared with controls across all ES and CDX4 sarcoma cell lines. Mean CD107a-positive NK cells reached 47.8% in ES models and

	58.3% in CDX4 sarcoma models following TriKE treatment, and was most robust in NCC-CDS-X1 (64.0%), p<0.001 for all lines, 1-way ANOVA. IFN-γ production showed a similar pattern, increasing to 36.1% and 43.2% in ES and CDX4 sarcoma models, respectively, %), p<0.01 for all lines, 1-way ANOVA. TriKE induced robust NK-cell degranulation and cytokine activation across all five fusion-driven sarcoma models. Conclusion: B7-H3–directed TriKE enhances NK-cell activation in fusion driven ES and CDX4 sarcoma models. These findings support further development of B7-H3–targeted NK-cell therapies in translocation associated sarcomas, particularly in sarcomas resistant to conventional T cell activating therapies. Ongoing studies will evaluate direct cytotoxicity in 2D and 3D sarcoma models and further define the role of B7-H3 TriKE in patient-derived sarcoma systems.
Swati Rampalli Hassan Ahad Firdows Mujir	*"How Systems See Us:" Evaluation of a Three-Part Workshop Series on Medical Students' Understanding of AI Bias and Health Equity in Clinical Practice* Background: Artificial intelligence (AI) has become a powerful tool in shaping evidence-based medicine among healthcare professionals, creating opportunities that allow for quick, easy clinical decision making and efficiency across many settings. Despite the growing importance of AI literacy among future physicians, medical education rarely examines the ethical and societal implications of AI, particularly among underrepresented, vulnerable populations in healthcare. We developed and evaluated a three-part educational workshop series, How Systems See Us, designed to strengthen medical student understanding of the ethical and health equity-related implications of AI in clinical settings. Methods: In collaboration with physician facilitators, we developed a three-part workshop curriculum, delivered across individual 90-minute sessions, that consisted of case-based discussions of AI usage in clinical practice, medical education, and healthcare decision-making followed by guided discussion. The first and third session were delivered by hospitalists and the second, a pediatric rheumatologist. During each session, participants simultaneously completed an interactive module developed by the student team with additional case-based scenarios that prompted students to critique whether AI systems adequately represented diverse patient populations. Students were further challenged to recognize sources of error within AI models and consider approaches to improving the equitable and sustainable use of AI for future physicians. Physician facilitators additionally incorporated examples from their own clinical experiences to contextualize potential benefits, limitations, and ethical concerns surrounding AI. Pre/post-session surveys were administered before and after the session and consisted of 10 items derived from pre-existing AI scales validated in medical education settings. Responses were analyzed as independent samples using Mann–Whitney U tests to

	evaluate change in students' perceptions and intended behaviors regarding AI. Results: Across the analyzed cohort, mean overall survey scores increased from 42.6 pre-intervention to 46.6 post-intervention (p=0.009). Students demonstrated a significant improvement in their ability to critically evaluate the credibility of information provided by AI (mean 3.60 vs 4.58, p=0.001) and in their intention to understand underlying rationale for AI-generated output in clinical scenarios (mean 4.07 vs 4.67, p=0.013). The largest improvement was observed in students' ability to critically evaluate AI-generated information. Other items demonstrated directional improvements, including students' willingness to compare AI outputs with their existing knowledge and using AI to assist disadvantaged populations, although these changes did not reach statistical significance. Conclusion: Our three-part workshop series, How Systems See Us, was associated with significant improvements in medical students' understanding regarding ethical implications of AI in healthcare. Integrating clinical cases, active learner participation, and real-world physician perspectives may provide an effective approach for teaching AI ethics beyond technical AI literacy. As AI becomes increasingly integrated into clinical practice, medical education should prepare future physicians to critically evaluate not only the accuracy and utility of AI systems, but also their potential effects on diverse patient populations and existing health disparities.
Amal Suri Dr. Maarya Pasha	*Cardiometabolic Risk Factors Among East African Patients Compared with U.S.-Born and Other Foreign-Born Patients at Hennepin Healthcare* Background: East African immigrants and refugees are a growing U.S. population, yet their cardiometabolic health profile remains incompletely characterized. Prior studies have reported substantial diabetes and cardiovascular risk among East African populations, but population-level comparisons with U.S.-born and other foreign-born patients are limited. We compared the prevalence of selected cardiometabolic risk factors among East African, U.S.-born, and other foreign-born patients receiving primary care. Methods: We conducted a retrospective cross-sectional study of adults aged ≥18 years with a primary care encounter at the Hennepin County Medical Center between January 1, 2023, and December 31, 2025. Patients were categorized as East African (Somalia, Ethiopia, or Kenya), U.S.-born, or born in other countries. Cardiometabolic diagnoses and clinical and laboratory measures were obtained from electronic health records. Prevalence estimates were standardized to the age and sex distribution of the overall study population. Adjusted prevalence differences with 95% confidence intervals (CIs) were calculated, and two-sided Wald tests with Benjamini-Hochberg correction were used to account for multiple comparisons.

	Results: Among 103,683 patients, 7,409 were East African, 60,789 were U.S.-born, and 35,485 were born in other countries. Among East African patients, 64.4% were from Somalia, 23.1% from Ethiopia, and 12.5% from Kenya. Compared with U.S.-born patients, East African patients had a higher adjusted prevalence of type 2 diabetes (20.0% vs. 14.6%; difference, 5.4 percentage points [95% CI, 4.3–6.5]; P<0.001) and high blood glucose (27.8% vs. 21.4%; difference, 6.4 [95% CI, 5.1–7.7]; P<0.001). East African patients had a lower prevalence of overweight/obesity (14.6% vs. 24.7%; difference, −10.1 [95% CI, −11.1 to −9.1]; P<0.001), hypertension (26.0% vs. 33.8%; difference, −7.8 [95% CI, −9.1 to −6.5]; P<0.001), and tobacco use (8.6% vs. 27.0%; difference, −18.5; P<0.001). The prevalence of A1C >8% was higher among East African patients than U.S.-born patients (6.2% vs. 3.6%; difference, 2.6; P<0.001). Compared with other foreign-born patients, East African patients had similar prevalence of type 2 diabetes (20.0% vs. 19.7%; P=0.652) and hypertension (26.0% vs. 27.0%; P=0.153), but lower overweight/obesity (14.6% vs. 19.0%; P<0.001). Conclusions: East African patients in this primary care population had a distinct cardiometabolic profile, characterized by higher prevalence of diabetes and hyperglycemia but lower prevalence of overweight/obesity, hypertension, and tobacco use compared with U.S.-born patients. The higher prevalence of diabetes despite lower overweight/obesity prevalence underscores the importance of diabetes screening and prevention strategies that account for population-specific patterns of cardiometabolic risk. Further research is needed to identify factors underlying these differences and to inform culturally responsive approaches to cardiometabolic disease prevention and management.
Talia Williams	*Posttraumatic Stress Symptoms and Coping Concerns During Pregnancy: A Study in a U.S. Obstetric Population* Introduction: Post-traumatic stress disorder (PTSD) is a psychiatric condition that may develop following exposure to a traumatic or highly stressful event and is characterized by intrusive symptoms, avoidance, negative alterations in mood or cognition, and hyperarousal lasting longer than one month.1 Posttraumatic stress symptoms have been associated with adverse obstetric and perinatal outcomes, including nonviable pregnancies, preterm birth, and low birth weight.4 Despite the impact of PTSD on maternal and fetal health, routine PTSD screening of pregnant people remains rare, in part due to a limited understanding of these impacts.5 This study aimed to characterize PTSD symptom burden and its association with concerns related to emotional and physical coping across the perinatal period to build an understanding of which patients may require more physical and mental health support during the perinatal period.

	Methods: A total of 120 pregnant participants were recruited during in-person visits at a women's health clinic. Participants completed electronic questionnaires, including the PTSD Checklist for DSM-5 (PCL-5)6, mental health history, obstetric history, demographic information, and prior mental health diagnoses. Associations between past or current mental health diagnoses and coping concerns were evaluated using Wilcoxon rank-sum tests. Spearman's rank correlation coefficients with 95% confidence intervals were calculated to assess relationships between PCL-5 scores and coping concerns. Results: The mean participant age was 33 ± 4.9 years, and mean gestational age was 27.8 ± 1.2 weeks. 75% identified as White and 25% identified as Black or another racial identity. Most participants had at least some college education (95%). Past or current mental health diagnosis (MHD) was reported by 60% (n=72) of participants, and 26% of those reported a history of PTSD (n=13). Participants in the MHD group reported significantly greater concerns across nearly all domains of emotional and physical coping, except physical coping in the postpartum period. In the MHD group, 34.7% reported moderate concerns around emotional coping during labor and delivery, whereas 14.6% of those without MHDs reported moderate concern (p=0.014). Mean PCL-5 score was 9.0 ± 10.6. Higher PCL-5 scores were significantly correlated with concerns regarding emotional coping during pregnancy (rho: 0.55, 95% CI: 0.41-0.68), physical coping during pregnancy (0.34, 0.16-0.49), the postpartum period (0.3, 0.11-0.47), as well as labor and delivery (0.23, 0.05-0.41). Conclusion: PTSD symptom burden and MHD were associated with increased concerns regarding emotional and physical coping across the perinatal period. Routine screening for MHD in addition to PTSD screening may help providers identify pregnant individuals at increased risk for worries around their emotional and physical functioning in the perinatal period and make referrals for additional support.
Clinical Vignette - Medical Students	
Fiza Alam Jacob Borgida Dr. Harsh Sharma Dr. Abigail Rice Hannah Cushen	*From Hematoma to Threatened Limb: A Case of Atraumatic Forearm Compartment Syndrome* Introduction: Compartment syndrome occurs when elevated pressure in a closed anatomical space compromises tissue perfusion, leading to neurovascular injury and ischemia. Although most commonly associated with blunt trauma or penetrating injuries and fractures, spontaneous hemorrhage is an important cause in anticoagulated patients. This case report describes the case of an atraumatic forearm compartment syndrome in the setting of anticoagulation, which was detected with early clinical examination and managed with emergent fasciotomy preserving neurovascular function.

	Case Presentation: A 62-year-old woman with past medical history of progressive fibrosing interstitial lung disease, pulmonary embolism, and a recent left-thigh hematoma was receiving unfractionated heparin when she developed progressive right-arm pain and ecchymosis following removal of a peripheral intravenous catheter. At approximately 0800, examination demonstrated circumferential right-arm swelling, marked tenderness, a palpable radial pulse, and 4/5 grip strength; she also reported numbness and tingling of the fingertips. Ultrasound demonstrated a 2.3 x 2.6 centimeters (cm) soft-tissue collection measuring at least 10 cm in length. The initial differential included an expanding hematoma, evolving compartment syndrome, and acute arterial occlusion. Heparin was held because of concern for progressive bleeding, and orthopedic surgery was consulted. At approximately 1000, repeat examination demonstrated worsening pain, swelling, and ecchymosis, prompting computed tomography angiography. At 1100, orthopedic evaluation found a warm, well-perfused hand with soft compartments, intact sensation and motor function, weak but present grip strength, and a palpable radial pulse. Computed tomography angiography demonstrated a new right brachial artery occlusion above the elbow involving the radial, ulnar, and interosseous artery origins. Given concern for an acute arterial thrombus, Vascular surgery was consulted and heparin was resumed. On examination by Vascular at 1200, the radial pulse went from weakened to undetectable by Doppler, and her hand became progressively cool and pale. With rapidly evolving acute limb ischemia and impending irreversible neuromuscular injury, she was taken emergently to the operating room. Exploration identified bleeding from a small brachial artery branch near the previous intravenous site without an intraluminal thrombus. Volar arm fasciotomy immediately restored brachial and distal Doppler signals, confirming compartment syndrome. Following decompression and hemostasis, a palpable radial pulse and distal perfusion were restored. Anticoagulation was resumed post-operatively, and Hematology was involved and advised ongoing low-intensity heparin to mitigate bleeding risk and treat her PE. Discussion: This case illustrates atraumatic forearm compartment syndrome in an anticoagulated patient, initially presenting as an enlarging hematoma with preserved distal perfusion. Although compartment syndrome is most often associated with trauma, anticoagulation-associated bleeding should be considered in patients with escalating extremity pain, swelling, and paresthesias. A palpable pulse, warm hand, and soft compartments may be falsely reassuring early in the disease course. In this patient, serial examinations demonstrated rapid progression to coolness, pallor, and loss of Doppler signals, prompting emergent decompression before irreversible ischemic injury occurred. In this case, urgent serial reassessment and multidisciplinary escalation despite initially reassuring examination findings were critical to timely diagnosis and restoration of distal perfusion.

Colleen Bartman Apoorva Komaragiri Dr. Danica Balsiger Dr. Ayley Seveson	*Blood Tells the Story: Pernicious Anemia Presenting with Profound Vitamin B12 Deficiency and Pancytopenia* Introduction: Vitamin B12 deficiency can arise from malabsorption, dietary insufficiency, toxin or infectious exposure, medications, as well as autoimmune conditions. Pernicious anemia (PA) is a rare autoimmune condition leading to severe vitamin B12 deficiency. It can affect all ages but has a 0.1% prevalence in the general population and a 1.9% prevalence in populations over the age of 60. It often escapes timely recognition due to its insidious course, nonspecific symptomatology, and relative rarity leading to frequently delayed diagnosis while evaluating for more common causes of anemia and B12 deficiency. Case Presentation: In this case, a 55-year-old male presented with fatigue, progressive exertional dizziness and dyspnea, intermittent headaches, bilateral lower extremity paresthesias, and hematochezia. He had severe pancytopenia (WBC 2.2 10e3/uL, hemoglobin 5.2 g/dL, platelets 102 10e3/uL) with macrocytic anemia (MCV 117) requiring transfusion, laboratory studies concerning for hemolytic anemia, and numerous dacrocytes in peripheral blood. There was initially concern for malignancy (including myelodysplasia) or malabsorption (including celiac disease, pancreatic insufficiency). However, extensive lab evaluation showing severe vitamin B12 deficiency and positive anti-parietal cell and anti-intrinsic factor antibodies, and endoscopy showing atrophic gastritis, ultimately supported diagnosis of PA. All of his signs and symptoms improved with subcutaneous supplementation of vitamin B12. Conclusion: This case highlights that severe vitamin B12 deficiency can mimic hemolytic, thrombotic, and neoplastic disease, and that severe B12 deficiency from PA should be considered in any patient with unexplained pancytopenia. Discussion: Recognizing pernicious anemia enables prompt and therapeutic management, providing complete hematologic recovery for patients.
Apoorva Komaragiri Colleen Bartman Dr. Danica Balsiger Dr. Hayley Severson	*An Adult Case of Febrile Infection-Related Epilepsy Syndrome with New-Onset Refractory Status Epilepticus* Background: New-onset refractory status epilepticus (NORSE) and its subtype, febrile infection-related epilepsy syndrome (FIRES), is a rare and devastating clinical presentation characterized by refractory status epilepticus following recent febrile illness occurring mainly in previously healthy pediatric patients. Case Presentation: Here, we present young adult-onset NORSE FIRES following a febrile infection in an individual without history of seizures. Factors predisposing to development of NORSE have not been clearly identified, and the prodromal symptoms may be vague

	and nonspecific, including confusion, fatigue, upper respiratory tract infection, gastroenteritis, and fever. A growing body of evidence does implicate postinfectious cytokine-mediated neuroinflammation as a broad mechanism of NORSE FIRES, including in cryptogenic cases. In this case, we explore the multidisciplinary management of the patient's very prolonged and complicated hospital course, as NORSE is particularly challenging to control with typical anti-epileptic medications and has high risk of side effects due to multi-drug therapy. It is imperative to remain aware of the impacts of treatments to best treat this complex disease process. Conclusion: The mortality in adult cases of NORSE/FIRES is 16-27%, which further underscores the importance of preparedness for teams to manage changing treatments throughout lengthy hospitalizations. This case truly highlights the challenges in approaching treatment of NORSE FIRES and emphasizes the need for early recognition, timely intervention, and further research into targeted therapies.
Priya Bhatt Dr. Allison Reinhardt	*Fibromyalgia Across the Decades: Navigating Evolving Diagnostics and Treatments* Background: Fibromyalgia (FM) is a chronic pain disorder characterized by widespread musculoskeletal pain often accompanied by symptoms such as fatigue, sleep disturbance, and activity limitations that significantly impact patients' lives. The estimated prevalence of FM in the general population of Olmsted County, MN is 6.4%. FM management requires an individualized approach with nonpharmacologic and pharmacologic approaches to address specific disease manifestations and/or comorbidities. Case Presentation: A 68-year-old woman presented with a longstanding history of FM for which she sought reevaluation and management according to current clinical practice guidelines. She was initially diagnosed in her 30s using tender point examination after years of episodic muscle pain. Over the decades, she continued to have migratory flares. She has tried multiple treatment modalities including trigger point injections for focal pain, acupuncture, massage therapy, physical therapy, and low-impact water aerobics. She reported anxiety that worsened during FM flares, with a cycle of anxiety about pain and pain increasing anxiety. At the time of presentation, the patient was taking low-dose naltrexone 6 mg nightly, venlafaxine, and citalopram. The current 2016 American College of Rheumatology (ACR) diagnostic criteria state FM is diagnosed if patients meet all three criteria: 1) widespread pain index (WPI) > 7 and symptom severity score (SSS) > 5 OR WPI 4-6 and SSS > 9, 2) generalized pain: met if pain is experienced in 4/5 of specified regions, and 3) symptoms present for > 3 months. If these criteria are met, diagnosis is made irrespective of whether other diagnoses contribute to these symptoms.

	Current guidelines indicate that nonpharmacologic interventions should be pursued first, including patient education, cognitive/psychological therapy, exercise (preferably low-impact aerobic exercise), diet/supplements including extra-virgin olive oil and ancient grains, and adjunctive modalities such as acupuncture. If symptoms persist, pharmacologic interventions may be pursued based on prominent symptoms. Duloxetine and milnacipran (SNRIs) may be useful in addressing depression and fatigue. Amitriptyline may address sleep disturbances and nortriptyline may address fatigue (TCAs). Pregabalin and gabapentin (antiepileptics) may address sleep disturbances and anxiety. Low-dose naltrexone is increasingly being utilized off-label for FM patients struggling with fatigue, brain fog, and pain. For our patient, we recommended that she continue low-dose naltrexone. We hoped to optimize her medication strategy through collaboration with psychiatry. We recommended transitioning current dual SSRI/SNRI therapy to FM-indicated duloxetine or milnacipran. The patient hoped to avoid gabapentin due to a family member's poor experience. We referred her to our 3-week intensive outpatient Pain Rehabilitation Center (PRC) program, which utilizes a combination of physical therapy, occupational therapy, and cognitive behavioral therapy. If an intensive outpatient pain rehabilitation program is not feasible, patients are referred to Lin Health, a telehealth company that offers similar services. Finally, we advised her to continue her current exercise, acupuncture, and chiropractic regimens as adjunctive therapies. Conclusion: FM is a prevalent condition with significant disease burden. It is reasonable to do periodic assessment of FM patients, especially as diagnostic criteria are updated and treatment strategy evolves. Management should be highly individualized to see greatest effects and should include pharmacologic and nonpharmacologic strategies.
Kacey Bui Dr. Mahima Devarajan Dr. Ellen Overson	*Gastrointestinal-Predominant Henoch-Schönlein Purpura: A Diagnostic Challenge* Introduction: Henoch-Schönlein purpura (HSP), also referred to as IgA vasculitis, is the most prevalent form of systemic vasculitis in childhood. The disease is characterized by the deposition of IgA-containing immune complexes within small blood vessels, leading to inflammation and the classical presentation of a palpable purpura, arthralgias, abdominal pain, and renal involvement. However, nonspecific symptoms, particularly severe abdominal pain or arthralgias without the pathognomonic rash, can often mimic other acute abdominal or rheumatologic pathologies. Here we report the case of a 3-year-old male with symptoms suggestive of HSP with an atypical presentation, and describe the key clinical and laboratory

	features that clinicians can employ to distinguish between an atypical presentation of IgA vasculitis and inflammatory bowel disease (IBD). Case Presentation: An otherwise healthy 3-year-old boy presented to the ED with abdominal pain that had been present for about 1 week. About 2 weeks prior to admission, he had influenza and recovered without issue. Ten days prior to admission he had ankle swelling that resolved spontaneously the same day. As this was his third presentation for abdominal pain unresponsive to constipation treatment and apparent dehydration, a more thorough work-up was pursued. Labs showed leukocytosis with neutrophil predominance, thrombocytosis, and elevated CRP. The appendix was not identifiable by abdominal ultrasound; thus, a CT of the abdomen was obtained to rule out appendicitis and the patient was found to have thickening of the distal ileum concerning for IBD. The case was discussed with a Pediatric Gastroenterologist who recommended esophagogastroduodenoscopy and colonoscopy which were performed the next day. These were grossly normal aside from a mildly edematous terminal ileum. Biopsies of the terminal ileum and colon showed patchy lamina propria with extravasated blood. The patient was discharged, but returned one day later with continuing abdominal pain and new onset non-blanching petechial rash on the bilateral elbows and ankle with accompanying swelling. Vitals were within normal limits, and CBC, BMP, and UA were unremarkable. While hospitalized, his rash progressed to the buttocks. After discussion with Pediatric Gastroenterology and Pediatric Rheumatology, there was rising suspicion for HSP given biopsy results and ongoing symptoms (abdominal pain, arthralgia, and non-thrombocytopenic petechial rash), despite lack of renal involvement and classical rash presentation. Conclusion: Overall, this report demonstrates how IgA vasculitis can present in a non-classical pattern and may mimic other pathologies, including IBD. The overall prevalence of GI tract involvement in IgA vasculitis is not currently known; however, the terminal ileum is one of the most commonly and severely affected sites if the GI tract. While a rash is often the presenting symptom for this condition, 30-40% of cases have abdominal pain as the presenting symptom and 15-25% have joint symptoms prior to onset of rash or abdominal symptoms (Reid-Adam 2014). This presentation indicates clinical suspicion for HSP should be high in patients with IBD-like symptoms and should be considered even in the absence of the classical petechial rash.
Sophia Clark Dr. Abby Sigurdson Dr. Amy Holbrook	*A Royal Pain in the Neck: A Case of Crowned Dens Syndrome* Introduction: Crowned Dens Syndrome (CDS) is caused by calcium pyrophosphate crystal deposition in the ligaments surrounding the odontoid process of the C2 vertebra. It is an under diagnosed cause of severe neck pain in elderly patients. Case Description: A 78-year-old woman presented to the Emergency Department with right sided neck pain radiating to her right trapezius.

	A CT of the cervical spine showed no acute fracture and moderate multilevel cervical spondylosis. She was discharged with ccetaminophen, cyclobenzaprine, lidocaine patches, and a physical therapy referral. She was seen in clinic two days later with continued pain that was not responsive to the medication regimen, and physical therapy was again encouraged. Four days later, she presented to the Emergency Department with worsening, and now bilateral, neck pain. Her physical exam was notable for significantly limited neck range of motion and tenderness to bilateral paraspinal muscles in the cervical region extending to the right trapezius. Her strength and sensation were grossly intact. Laboratory testing was remarkable for an elevated white blood cell count to 15.5 and CRP to 11.0. Cervical MRI demonstrated cervical spondylosis without compression fractures, and mild spinal canal stenosis at C2-3 through T2-3. A bilateral temporal duplex ultrasound was negative for potential giant cell arteritis. Upon thorough review of the CT neck imaging, she was noted to have a curvilinear band of calcification encircling the odontoid process, and the presumed diagnosis of CDS was made. She was started on renally dosed colchicine and prednisone with dramatic improvement of her neck pain. She was discharged with two weeks of colchicine and a prednisone taper with complete resolution of presenting pain. Discussion: In elderly patients with severe, irretractable neck pain and elevated inflammatory markers, CDS is an often-overlooked diagnosis. The differential diagnosis may include musculoskeletal strains, cervical spondylosis, and inflammatory or infectious conditions such as giant cell arteritis or meningitis. CDS is reasonable to consider when patients present with elevated inflammatory markers in the setting of neck pain. Characteristic findings on CT imaging can be an important diagnostic signal that is easily missed. Treatment with colchicine and steroids improves symptoms quickly; though, risk for relapsing symptoms is high.
Mark Delisi Dr. Ellen Overson	*A Wolf in Two Sheep's Clothing: Mycoplasma pneumoniae Hemolysis Mimicking HLH and Primary AIHA in a 12-Year-Old* Introduction: Mycoplasma pneumoniae (MP) is a common cause of community-acquired pneumonia in children and young adults but can also cause extrapulmonary disease. In rare cases, infection has been associated with complement-mediated cold agglutinin syndrome (CAS), primary/warm autoimmune hemolytic anemia (AIHA), and, less commonly, hemophagocytic lymphohistiocytosis (HLH). Because these conditions can present with overlapping clinical and laboratory findings, differentiating between them is important to avoid unnecessary immunosuppression. Case Presentation: A 12-year-old patient presented with three-day history of fever up to 104°F, jaundice, abdominal pain, and dark urine. Exam was notable for scleral icterus, tachycardia (HR 121), and splenomegaly without cervical lymphadenopathy or rash. Initial labs were consistent with hemolytic anemia, an undetectable haptoglobin

	(<10), elevated LDH (658), indirect hyperbilirubinemia (total 4.6 mg/dL, direct 0.54 mg/dL), and reticulocytosis (3.18%). A direct antiglobulin test was C3d-positive with weak/negative IgG, supporting a cold-antibody AIHA pattern. Her fever, splenomegaly, cytopenias (anemia, evolving leukopenia and mild thrombocytopenia), elevated ferritin (880) and other elevated cytokines also raised concern for HLH. The fact that her ferritin was severely elevated and that her fibrinogen (259) and triglycerides (127) were normal placed HLH lower on the differential. Given that both cold-antibody AIHA and HLH can both be triggered by other conditions, a broad infectious, rheumatologic and oncologic work-up was pursued with multiple negative studies including CMV, EBV, hepatitis panel, tickborne panel, respiratory viral panel, flow cytometry, ANA, dsDNA. On the second day of admission, the patient developed a maculopapular rash and cough. Given that MP can present with an urticaria-like eruption and with hematologic abnormalities (although rare), she was empirically started on treatment with doxycycline. Her lab abnormalities improved, and she was discharged with pending testing for Mycoplasma. Post-discharge serology showed rising IgG with IgM seroconversion on paired sera confirming acute MP infection. Discussion: This case highlighted how complex the diagnostic process can be in the setting of fever, splenomegaly, cytopenias and elevated inflammatory markers. It further highlighted how despite high suspicion for diagnoses such as AIHA or HLH, the diagnostic process has still only just begun due to the multitude of secondary triggers of each condition. In this case, the development of rash and cough helped prompt further consideration of Mycoplasma pneumoniae which fortunately is easily treatable. Recognizing and treating for MP also helped avoid unnecessary immunosuppression. Conclusion: This case highlights the importance of keeping infectious etiologies such as Mycoplasma pneumoniae on the differential when meeting a pediatric patient with fever, splenomegaly, hemolytic anemia, and elevated inflammatory markers. Considering empiric treatment with doxycycline and obtaining confirmatory testing can help avoid immunosuppression and expedite recovery.
Ming Shen Ronin Joshua S. Cosiquien Dr. Aditya Chandorkar Dr. Sashi Nair	*The Minnesota Patient: A Possible Path to HIV Remission* Introduction: Human Immunodeficiency Virus (HIV) is a retrovirus that targets CD4+ T-helper cells. This renders the viral host in an immunocompromised state, which increases the likelihood of opportunistic infections, and if left untreated, can progress to Acquired Immunodeficiency Syndrome (AIDS). While current therapies for HIV target various parts of the virus-cell integration cycle and viral replication, allogeneic stem cell transplantation (ASCT), particularly with CCR5-deficient cells that block integration of CCR5-tropic HIV strains, may be a possible cure.

	Case Presentation: We describe a 50-year-old patient who acquired HIV and, nearly 30 years later after receiving ASCT and numerous chemotherapies for myelofibrosis, achieved 8 months of sustained antiviral-free remission after analytical treatment interruption. This was verified by HIV1 antibody testing, HIV RNA Quantitative testing, Karius testing, and HIV1 proviral sequencing. Conclusion: This was reminiscent of previous patients deemed as cured after receiving ASCT, 7 out of 8 of whom harbored the CCR5Δ32 mutation in the transplanted cells. However, this mutation is not considered a definitive cure, as several patients who have CCR5Δ32 cells experienced viral relapse. Further investigation into this phenomenon and how the interplay of viral-host genetics, chemotherapy, and stem cell transplantation may facilitate HIV suppression and elimination could serve as the key to discovering a definitive cure for HIV.
Ella Dockendorf Dr. Mithun Suresh	*A Case of Membranous Nephropathy in Healthy Male with no Previous Medical History* Case Presentation: 47-year-old man with no known medical comorbidities who initially presented to outside ED with a complaint of progressively worsening bilateral lower extremity edema, shortness of breath, and pleuritic chest pain for 1 week duration. Upon initial evaluation, physical exam was notable for significant edema in bilateral lower extremities up to the waist. Labs were notable for elevated creatinine, low albumin, and elevated d-dimer. Urinalysis was notable for significant proteinuria and microscopic hematuria. Chest x-ray was notable for a small pleural effusion on the left side. CT chest was notable for small bilateral pulmonary emboli without right heart strain and moderate pleural effusion on the left side and mild pleural effusion on the right side. CT abdomen pelvis revealed small volume ascites with diffuse subcutaneous edema. Bilateral Lower extremity doppler US was negative for DVT. Echocardiogram showed EF of 60% normal LV and RV function, normal IVC size and greater than 50% collapse with respiration, RA pressure normal at 3 mmHg. Following admission, the patient was initiated on a heparin drip. Nephrology was consulted, as there was concern for nephrotic syndrome given the significant proteinuria. He was initiated on a diuretic infusion along intermittent albumin. After adequate IV diuresing and improvement of anasarca, he was changed to oral lasix. For his proteinuria patient was also started on low-dose lisinopril. Patient underwent renal biopsy, and the results demonstrated membranous nephropathy. He notably also had a positive PLA2R Ab blood test as well. Infectious workup was negative for HIV and viral hepatitis. Upon resolution of his symptoms and stabilization of his renal function, he was discharged with plans to initiate rituximab as an outpatient.

Ethan Greene Dr. Mithun Suresh	*An Unusual Cause of Ascites: Intraperitoneal Tuberculosis* Case Presentation: 23-year-old female presenting for evaluation of abdominal pain, fevers for about 1 week prior to presentation. Medical issues include no known medical history aside from a positive quantiferon-gold test about 9 months prior in the context of screening for employment. The patient recently immigrated from Nigeria. About 1 week prior to presentation, she developed progressive abdominal pain, distention, and fevers. She initially presented to urgent care, where CT abdomen and pelvis demonstrated large volume pelvic ascites of unclear etiology. She was then transferred to the nearby ER, and initiated on antibiotics due to concerns for peritonitis and admitted for further evaluation and management. Ascitic fluid was sampled showing a lymphocytic predominant ascitic fluid with markedly elevated inflammatory markers. Cytology testing was negative for malignancy. Infectious diseases was consulted to help assist with infectious workup with negative AFB smears on peritoneal fluid samples and a negative MTB PCR from peritoneal fluid. CA125 was markedly elevated, though pelvic imaging showed no discrete ovarian or adnexal mass. With persisting fevers, the lymphocytic exudative ascites, and epidemiologic risk factors, there was primary concern for intraperitoneal tuberculosis. Other diagnoses included inflammatory peritonitis versus primary peritoneal/gynecological malignancy. The decision was made to pursue tissue diagnosis with the patient undergoing a laparoscopic peritoneal biopsy. Operative findings indicated the presence of a significant volume of serous ascites, dense adhesions of the small bowel and colon to the abdominal wall in the presence of numerous tiny peritoneal surface nodules of the abdominal wall and liver capsule. A sample of the ascitic fluid and peritoneal biopsies were sent for pathology, cytology, and mycobacterial studies. With overall suspicion and concern for peritoneal tuberculosis, empiric treatment with the initiation of RIPE therapy plus pyridoxine commenced (pyrazinamide, ethambutol, rifampin and INH as well as vitamin B6). Care was coordinated with the Minnesota Department of Health per protocol. She was subsequently discharged from the hospital once she had recovered from her surgery and her medications could be ordered. She had regular follow up appointments with ID, and during follow-up, the peritoneal fluid that was obtained during her diagnostic laparoscopy and peritoneal biopsies tested positive for mycobacterium tuberculosis. In addition, the biopsy results showed benign peritoneal tissue with chronic inflammation and non-necrotizing granulomas. She continues to follow with ID, with plans for a prolonged course of treatment for intraperitoneal tuberculosis
Lucy Greenhagen Dr. Mithun Suresh	*An Interesting Case of a Rash, Sinusitis, and Renal Failure* Case Presentation: 39-year-old female with hypertension, presented for worsening rash and AKI. She first developed upper respiratory

	symptoms about 6 months prior to presentation. This included sinus congestion, sore throat, nonproductive cough, and hoarse voice. Over the 6 months, her symptoms would wax and wane. She had tried multiple courses of antibiotics and prednisone. There may have been slight improvement in her symptoms with these medications, but she never fully recovered. About 1 week prior to admission, she developed some swelling in her neck and face along with increasing sinus congestion. She was initiated on a course of augmentin (which she had taken in the previous 6 months). She did not see much improvement in her symptoms. Then approximately 4 days later, she developed a rash which started on her forearms, then continued to spread throughout her extremities and trunk. The rash was associated with a burning and tingling sensation and swelling of her hands and feet. She tried a variety of medications including Benadryl, ibuprofen, and loratadine. Despite this, the rash continued to progress. No open sores or blistering has been noted. No myalgias or arthralgias, no definitive fever although she has had the night sweats some mild nausea. The other primary symptom she noticed was decreased urination, but no burning or other changes to her urine appearance such as blood, foam, or cloudiness. She presented to an outside hospital. There work up was notable for acute kidney injury, non-anion gap metabolic acidosis, and leukocytosis. She was then transferred to a tertiary referral center. Following transfer, she was seen by the nephrology team in consultation. Further work-up was notable for positive MPO, but negative p-ANCA and c-ANCA, along with weakly positive ANA. With this work-up, She was initiated on high dose steroids. Within 24-48 hours, she saw significant improvement in her symptoms and renal function. Further work-up included CT imaging of her sinuses, which yielded opacification and air-fluid levels within the maxillary, sphenoid, frontal, and ethmoid sinuses consistent with pansinusitis. With this constellation of symptoms and signs, along with improvement after starting steroids, overall suspicion was for a diagnosis of churg-strauss syndrome, or eosinophilic granulomatosis with polyangiitis. Treatment continued with steroids for the remainder of her hospitalization. A skin biopsy was performed, but the results were still pending on discharge. The patient ultimately discharged on a steroid taper, and during follow-up appointments, her symptoms had completely resolved along with improvement in her renal function. Plan was for further follow-up with ENT and rheumatology after discharge.
Katelyn Kim Dr. Roy Kao	*Successful Treatment of Acquired Amegakaryocytic Thrombocytopenia Purpura and Pure Red Cell Aplasia with Bortezomib and Daratumumab in a Patient with a History of Systemic Lupus Erythematosus* Background: Acquired amegakaryocytic thrombocytopenia purpura (AATP) is a rare disorder that can present as a hematologic complication in systemic lupus erythematosus (SLE), as well as in

	other lymphoproliferative, autoimmune disorders. The pathology underlying this disorder has yet to be elucidated. Mechanisms involving the immune-mediated suppression of megakaryocyte (MK) maturation are likely implicated, potentially stemming from intrinsic defects in megakaryocytopoiesis or autoimmune-mediated inhibition of platelet precursors. There is limited research on treatment approaches, with few cases documenting the use of bone marrow transplantation (BMT) with varying success. Case Presentation: We report a patient with a history of SLE who was diagnosed with AATP and pure red cell aplasia. She was refractory to multiple lines of therapy, including dexamethasone, intravenous immunoglobulin (IVIG), romiplostim, rituximab, eltrombopag, daratumumab, and fostamatinib and subsequently underwent an allogeneic BMT from a 7/8 unrelated donor. Neutrophil engraftment was achieved by day +19 and 100% donor chimerism was established by day +30. However, she remained red cell and platelet transfusion dependent. Positive antinuclear antibodies and anti-A1 titers (1:32) prompted administration of bortezomib and daratumumab with resultant transfusion independence in all lines. The patient is now 2 years 11 months post-transplant and remains transfusion independent with full donor hematopoiesis. Conclusion: Further research is needed to determine the pathogenesis of AAMT. Our case suggests the likelihood of an autoimmune pathology within the context of SLE, positing that residual plasma cells producing autoantibodies persisted despite immunosuppressive therapies and BMT. Consequently, eradication of these cells post-BMT led quickly to transfusion independence.
Maria Landherr Dr. Claire Bourgeois	*Making Meaning from the Mass: A Case Report of Cardiobacterium Hominis Infective Endocarditis* Case Presentation: A 34-year-old male with past medical history of viral myocarditis one month prior, hyperlipidemia, heroin use (in sustained remission, no IV use), and anxiety presented to the emergency department with abdominal pain. Initial labs showed WBC 11.48 k/μL and CRP 142 mg/L, with normal LFTs, lipase, and lactic acid. CT abdomen/pelvis showed a soft tissue mass encasing and occluding the superior mesenteric artery (SMA) without bowel ischemia and with associated thrombus of the superior mesenteric vein (SMV). Endoscopic biopsy was completed on hospital day 2 without visualization of a discrete abdominal mass; samples from the SMV were submitted for pathology and culture. The initial differential on multidisciplinary evaluation included malignancy, such as a mesenteric neoplasm, or an inflammatory process, such as sclerosing mesenteritis. A TTE obtained given recent history of viral myocarditis showed EF 55-60% with a possible mobile echodensity of the noncoronary cusp of the native bicuspid aortic valve

	with excursion into and out of the outflow tract. This prompted evaluation with blood cultures and TEE. Surgical oncology favored a benign etiology of the intra-abdominal lesion based on radiographic appearance with immediate vessel cut-off and absence of vasculature within the lesion. On hospital day 5, two peripheral blood cultures grew gram-negative bacilli, and the patient was started on ceftriaxone. TEE the same day showed a calcific nodule adherent to the bicuspid valve leaflet tip. Blood cultures subsequently identified Cardiobacterium hominis, later isolated from fine-needle aspirate from the SMV. Further workup for evaluation of source and metastatic disease was extensive and included MRI abdomen, carotid ultrasound, CTA head and neck, MRI brain, and CT dental. Pertinent findings included bilateral renal infarcts and small acute infarcts in the right frontal lobe, with dental imaging reassuring against caries and periapical disease. Discussion: This case demonstrates the diagnostic challenge of infective endocarditis (IE) when initial symptoms result from metastatic spread. While initial abdominal imaging suggested a primary mesenteric pathology, positive blood cultures and aortic valve vegetation on TEE satisfied Duke's criteria (1) and confirmed C. hominis aortic valve endocarditis. C. hominis has low virulence as normal oropharyngeal flora and accounts for ~0.18% of all infective endocarditis cases (2) with embolic phenomena in up to 27% of cases (3). C. hominis predominantly affects the aortic valve (4) and is rarely implicated in endocarditis secondary to IVDU (5). A bicuspid aortic valve is an independent risk factor for development of IE, conferring intermediate risk (6), with a lifetime risk of approximately 6% (7). Whether recent viral myocarditis could predispose to IE is unclear and not supported by the literature. Cardiac MRI obtained at the time of the patient's diagnosis of viral myocarditis ruled out endocardial involvement. Additionally, myocarditis more commonly occurs subsequent to infective endocarditis, rather than preceding it (3). Initial surgical consultation favored benign etiologies, including perivascular inflammation and septic thrombosis attributed to IVDU. However, the patient was in sustained remission without any history of IV use. This focus risked overlooking alternative diagnoses rather than maintaining a broad differential.
Alexandra Poeschla Dr. Thomas Reimann	*From Vision Changes and Imbalance to Rapidly Progressive Dementia: Diagnostic Challenge of Creutzfeldt-Jakob Disease* Introduction: Creutzfeldt-Jakob disease (CJD) is a rare, progressive and fatal prion disease that is characterized by rapid neurologic, behavioral, and cognitive decline. Its heterogeneous presentation can make it difficult to diagnose. With an estimated annual incidence of 1-2 cases per million worldwide, CJD may not be initially considered

	among more common etiologies. We present a case of CJD initially attributed to worsening baseline visual impairment and multifactorial imbalance. Case Presentation: A woman in her late 60s with chronic visual impairment, sensorineural hearing loss and atrial fibrillation presented to her primary care physician with worsening vision, dizziness, and balance. Initial laboratory testing and ECG was unrevealing, and she was referred to cardiology and ophthalmology. Over the following two months, her symptoms progressed. Further evaluation including head CT and cardiac testing were unremarkable except for brief runs of nonsustained ventricular tachycardia. Two months later, she presented to the emergency department with her son, with complaints of worsening unsteadiness, confusion, memory issues, and visual hallucinations. Brain MRI demonstrated chronic small vessel ischemic disease. Her symptoms were thought to be multifactorial, including chronic small vessel disease, multisensory imbalance of aging, and inner ear pathology. She was discharged to a short-term rehabilitation facility with plans for vestibular physical therapy. Two months after this, the patient presented to the emergency department with worsening dizziness, vision, cognition, and weakness despite physical therapy. She was oriented only to self and had inattentiveness, delusional thought content, and impaired memory. Physical examination revealed suprapubic abdominal tenderness and generalized weakness. CT imaging of the head, chest, abdomen, and pelvis was unremarkable aside from findings consistent with cystitis; she was subsequently started on ceftriaxone for this. The rapid progression of her dementia and physical debility prompted further workup for neurodegenerative conditions including Dementia with Lewy Bodies, CJD, autoimmune or paraneoplastic encephalitis, toxic-metabolic encephalopathy related to cystitis, and vitamin deficiency. Extensive laboratory workup was unrevealing. Brain MRI showed symmetric areas of intermediate restricted diffusion and signal change in the bilateral basal ganglia, raising concern for hypoxic ischemic injury, toxic/metabolic injury, and CJD. Given her presenting symptoms, there was a high clinical suspicion of CJD, so lumbar puncture was performed for CSF analysis. Further investigation included EEG, which showed no epileptiform changes, and a course of high-dose steroids for possible autoimmune or paraneoplastic encephalitis, which produced no improvement. During her hospitalization, she was intermittently agitated with fluctuating alertness. Two weeks after CSF was collected, prion testing RT-QuIC was found to be positive, confirming CJD. She transitioned to comfort-focused care and died shortly thereafter.

	Conclusion: This case highlights the diagnostic challenge of CJD, which may not initially be considered due to its rarity and heterogeneous presentation. This patient's presenting symptoms of progressive vision loss and discoordination were nonspecific, with many potential etiologies more common than CJD. Rapidly progressive dementia accompanied by hallucinations, ataxia, and other neurologic abnormalities should raise suspicion for CJD, particularly when evaluation for more common etiologies is unrevealing. Early recognition can facilitate timely diagnostic testing and goals of care discussions.
Annika Pokorny Dr. Fizza Zulfiqar Dr. Daniel Mueller Dr. Amy Beckmna Dr. Darrell Horton	*The Great Mimicker: An Unusual Case of Lymphadenopathy* Introduction: Kikuchi-Fujimoto disease, also referred to as histiocytic necrotizing lymphadenitis, is a rare, self-limited inflammatory disorder that results in benign lymphadenopathy and constitutional symptoms over the course of weeks to months. This disease primarily occurs in pediatric populations, though there is increasing recognition of Kikuchi-Fujimoto disease in young adults. It is often a diagnosis of exclusion, as many other processes, including infectious, inflammatory, and malignant, may present similarly. This case explores the wide diagnostic and interdisciplinary approach needed to fully evaluate subacute lymphadenopathy. Case Presentation: A 25-year-old female at seven weeks gestation presented to the emergency department with a two-month history of tender cervical lymphadenopathy and one month of progressive fatigue, malaise, arthralgias, unintentional weight loss, and recurring fevers. Prior to admission, she was evaluated in the outpatient and emergency department settings, ultimately hospitalized twice. She was initially diagnosed with tonsillitis and given antibiotics without symptomatic improvement. A fine needle aspiration of the lymph node was normal. At presentation, physical examination showed right-sided posterior cervical lymphadenopathy, an ulcerative lesion on the underside of the tongue, and subtle bilateral soft tissue swelling of the wrists. There were no rashes or other skin changes. Laboratory testing revealed anemia, lymphopenia, neutropenia, and elevated C-reactive protein, though rheumatologic workup, including ANA, RF, anti-CCP, C3, C4, dsDNA, and ACE, was normal. Extensive infectious disease testing, including EBV, CMV, HSV, TB, syphilis, Histoplasma, and Bartonella henselae, was negative. Imaging of the neck demonstrated stable, conglomerated right cervical lymphadenopathy and a multinodular thyroid gland, while the chest was normal without mediastinal or hilar lymphadenopathy. She underwent excisional biopsy of a right cervical lymph node, which revealed necrotizing lymphadenitis without morphologic or immunophenotypic evidence of a lymphoproliferative disorder. Given the histopathologic findings, clinical course, and exclusion of other processes, she was diagnosed with Kikuchi-Fujimoto disease and ultimately chose to forgo glucocorticoid treatment in the setting of pregnancy.

	Conclusion: This case demonstrates the importance of a broad differential, including infectious, autoimmune, autoinflammatory, and malignant processes, to guide diagnostic evaluation when encountering a patient with subacute, unremitting lymphadenopathy with constitutional symptoms. The diagnosis of Kikuchi-Fujimoto syndrome relies on excisional biopsy. However, necrotizing lymphadenitis is non-specific, and immunohistochemistry may be more helpful in a clear diagnosis, showing few B cells, eosinophils, and neutrophils, and the presence of myeloperoxidase, CD68, and CD8-positive T cells. Several other processes may present with necrotizing lymphadenitis, including lymphoproliferative malignancies, systemic lupus erythematosus lymphadenitis, macrophage activation syndrome, granulomatous diseases including sarcoidosis, and viral infection, making histopathologic distinction important. Thorough laboratory evaluation is paramount, and the lack of infectious or other rheumatologic process ultimately led to the diagnosis of Kikuchi-Fujimoto disease. Lymphadenopathy in young adults warrants clinical evaluation and workup, and this case explores an unusual cause of a common presentation.
Nicholas Sanford Dr. Patrick Osborn	*Fasciotomy for Acute Compartment Syndrome Secondary to Extravasation of Intraosseous Fluids* Introduction: Intraosseous access for fluids is a useful option when rapid access in critically ill or injured patients is needed; it has a success rate of 94% and can be established in less than a minute with complications seen in less than 1% of insertions. Extravasation of fluid is a known, but rare complication, and acute compartment syndrome (ACS) is a serious complication of extravasation. The earliest symptom of ACS is pain out of proportion. If that is missed then pallor, pulselessness, paresthesia, poikilothermia, and paralysis can denote late compartment syndrome. Case Presentation: In this case, a 57-year-old male with past medical history of peripheral vascular disease and DVT presented to the ED for three days of chest pain. He was admitted to the CICU with complete AV block and received IO fluids in his left tibia. Later that morning his left lower leg appeared dusky and after consulting with orthopedic personnel was determined to have ACS due to extravasation of IO fluids into the compartments of the lower leg. The patient was taken to the OR where an orthopedic surgeon performed a fasciotomy, relieving pressure in all four compartments. Appreciable pulse was returned to the dorsalis pedis artery, but not the posterior tibial artery. Vascular surgery was consulted and a thrombectomy was performed in the left popliteal artery and the left posterior tibial artery, however, no appreciable thrombus was retrieved. After the thrombectomy a dopplerable pulse was eventually found at the level of the posterior tibial artery. Over the next couple of days pulses in the left lower extremity disappeared and an angiogram was performed which showed high resistance flow and poor filling of the anterior and posterior tibial

	vessels. The patient's condition continued to deteriorate and four days later he required an above-the-knee amputation due to ischemic injury. Conclusion: While IO access is viable for emergent treatment, IV fluids should be established as soon as possible and IO fluids removed to prevent complications. Continuous monitoring of patients on IO fluids is also essential when they are intubated or sedated since they will not be able to report the initial symptom of pain out of proportion.
Lauren Voll Dr. Mithun Suresh	*Post-Streptococcal Glomerulonephritis: A Rare Complication to a Common Condition* Case Presentation: 43-year-old male with pertinent history of hypertension, obesity, OSA, type 2 diabetes presented with nausea, vomiting, back pain, malaise and fatigue. Symptoms started about 10 days prior to admission. They have occurring on and off over the preceding 10 days. Since his symptoms were not resolving, he presented to his local clinic, and labs were notable for acute kidney injury, normal albumin, normal total protein. Urinalysis was notable for high specific gravity, large amount of gross blood and protein, but no evidence of microscopic hematuria. Following admission, he was initiated on IV fluids, and nephrology was consulted. Urine culture grew Klebsiella pneumoniae, and CT imaging demonstrated bilateral perinephric and periureteral edema suggestive of possible bilateral ascending urinary tract infection/pyelonephritis. Accordingly, he was initiated on antibiotics. Despite this, renal function continued to worsen, and he became more and more oliguric and hypervolemic. Accordingly, dialysis was initiated. During this time, broad serological work-up was underway, the notable findings were significantly elevated Streptolysin O/ASO Ab Titer and Anti-DNase B Titer. Upon further questioning, the patient reported possibly having a sore throat at some point during the past 1 month. Accordingly, concern was for possibly concurrent glomerulonephritis related to post-streptococcal glomerulonephritis. Prednisone was started for a possible component of crescentic disease. He continued on dialysis and a prednisone course while hospitalized, and ultimately discharged on prednisone and still needing dialysis. He finished prednisone about 1 week after discharging, and then was able to stop dialysis after discharge with resolution of renal failure.
Whitney Wenner Dr. Mithun Suresh	*A Difficult Case of Pembrolizumab Induced Myositis* Case Presentation: 85-year-old female who presented with fatigue, anorexia, weight loss, dysphagia, ptosis, and diplopia. Her medical history included hypertension, GERD, history of Barrett's esophagus, type 2 diabetes mellitus, dyslipidemia, chronic obstructive pulmonary disease, CKD stage III, OSA, osteopenia, recurrent Merkel cell carcinoma status post 4 cycles of pembrolizumab. Three days prior to presentation to the hospital, she was seen in the oncology clinic with these symptoms. CK was checked, and was elevated, so concern was for statin induced myositis. Despite stopping atorvastatin, her

	symptoms remained, so three days later, she presented to the hospital, and was then admitted for further work-up and management. CK still remained elevated. She was seen by oncology and neurology. Concern was for pembrolizumab immune-mediated toxicity, and she was initiated on high dose steroids. Concurrent testing was performed for autoimmune diseases, including myasthenia gravis and lambert-eaton syndrome. Accordingly, ACh Receptor (Muscle) Binding Ab, P/Q-Type Calcium Channel Ab, MuSK Autoantibody testing did not detect any antibodies. Despite 4 days of high dose steroids, she had minimal improvement in her symptoms, and she was subsequently started on plasma exchange. At the time of initiating plasma exchange, she had increased swallowing difficulty and ptosis, along with slurring of her speech. Concurrently, she also developed significant acute kidney injury and oliguria prompting consultation with nephrology. Plasma exchange was initiated for a total of 5 sessions over the next 8 days. One round of dialysis was also completed as well. During this time, her renal failure resolved and she was able to stop dialysis. She had some improvement in her symptoms of ptosis, dysphagia, and generalized weakness, but they were still not completely resolved. While she was getting plasma exchange, GI was consulted due to the dysphagia and EGD was performed, which revealed no acute findings. Accordingly, the dysphagia was thought to be a side effect of pembrolizumab. Over the next several days, she had minimal improvement in her appetite, energy level, and symptoms. Accordingly, goals of care discussion was held with patient and family regarding prognosis given prolonged hospitalization in the setting of ongoing symptomatology, poor oral intake, and recurrent malignancy, and they elected to transition to hospice. Accordingly, the patient was discharged on hospice. Conclusion: This was an interesting case of pembrolizumab induced myositis. With the growing use of immunotherapy, knowledge and awareness of the potential side effects of these medications is critical.
Jenna Zarzaur Dr. Nick Dabiran Dr. Benji Mathews	*The Tip of the Iceberg: Dialysis-Requiring Tip-Variant FSGS* Background: Primary focal segmental glomerulosclerosis (FSGS) is a pattern of glomerular injury commonly manifesting as nephrotic syndrome and renal insufficiency. Tip-variant lesions typically carry the most favorable prognosis among FSGS variants. We report on a patient with tip-variant primary FSGS with poorly preserved renal baseline who required inpatient hemodialysis support, thoracentesis, and paracentesis while awaiting treatment response to steroids. Case Presentation: A 42-year-old male was hospitalized for several weeks of progressive shortness of breath, marked full-body anasarca, and fatigue. His workup was notable for hypertension, elevated creatinine (3.06 mg/dL), and nephrotic-range proteinuria (>2000 mg/dL). Unfortunately, due to socioeconomic barriers he deferred further management and was discharged without established follow-up

	care. He was admitted one year later for symptom progression. Physical examination was notable for massive ascites with fluid wave, 2+ pitting edema in all extremities, decreased bilateral breath sounds, and skin pallor. Laboratory investigations revealed progression of his renal disease with creatinine of 4.27 mg/dL, urine protein of 474 mg/dL, hypoproteinemia, BNP of 34,000 pg/mL, anemia of chronic disease, and dyslipidemia. Renal biopsy revealed tip-variant sclerosis with substantial acute tubular injury, arteriolar hyalinosis, and 40% interstitial fibrosis. A comprehensive workup for infectious and autoimmune causes was unremarkable and the patient was diagnosed with tip-variant primary FSGS. Following diagnosis, he was promptly initiated on high-dose prednisone and bumetanide. He required significant assistance with ADLs and mobility due his edema, physical deconditioning, and muscle wasting. Renal function initially declined despite steroid administration with worsening hypervolemia requiring frequent thoracentesis and paracentesis for bilateral pleural effusions and ascites, respectively. Given his severe volume overload, inadequate response to diuresis, and increasing oxygen requirements, the patient ultimately underwent tunneled catheter placement and received several sessions of hemodialysis while awaiting treatment response to steroids. Several weeks later, his renal function began to steadily improve. He discontinued dialysis and was discharged on oral diuretics and a long-term course of steroids to be managed by outpatient Nephrology. Discussion: Tip-variant lesions are associated with a favorable prognosis when compared to other primary FSGS subtypes. Most patients improve substantially with high-dose steroids, present with lower baseline creatinine levels, and carry relatively low risk of progression to end-stage renal disease. Despite this patient's diagnosis of steroid-responsive glomerulosclerosis, meaningful symptom improvement required several weeks of treatment to manifest clinically. Due to lack of treatment for one year prior to admission, his severe nephrotic complications necessitated hemodialysis and drainage interventions for management of volume status and increased oxygen needs until renal function improved. Conclusion: Sequelae of nephrotic syndrome in patients with tip-variant primary FSGS may require significant escalation of care despite appropriate treatment of the underlying disease. This scenario becomes more likely in the setting of delayed management. This patient's degree of hypervolemia with inadequate diuresis response, increasing oxygen needs, and declining renal function emphasizes the need for early recognition and treatment of nephrotic disease. This case highlights the importance of managing acute nephrotic complications in parallel with standard treatment for tip-variant primary FSGS.
Quality Improvement – Early Career Physicians	

Spencer Bonnerup	*Staying Alive: A Simulation Based Curriculum to Increase Resident Confidence & Skill in Running Codes* Introduction: Code blue and rapid response situations can be some of the most daunting clinical scenarios that physicians may encounter. These scenarios can be even more daunting for physicians in training due to decreased experience in these time sensitive situations. Each year approximately 300,000 adults have an in-hospital cardiac arrest, of whom approximately 25 % achieve return-of-spontaneous-circulation.1 From my own experience as an intern, I recall trepidation in ordering adenosine for an otherwise stable patient in supraventricular tachycardia without the assent of an attending physician. This project's aim was to improve resident confidence and skills in responding to these time sensitive patient scenarios. Methods: To this end we developed a curriculum to be used in the high-fidelity simulation centers at our home institutions.2-4 These scenarios are designed to give the learner experience managing patient presentations that they may encounter in clinical practice in a controlled environment giving them the skills necessary to manage these emergencies. These sessions are also designed to boost the resident's confidence in their ability to manage codes and rapid response situations during training. Anonymous surveys were given to the residents before and after the training sessions to gauge their perceptions in their skills in managing these clinical scenarios as well as solicit summative feedback. These simulations were supervised by attending physician(s) to provide real-time feedback to the learners about the simulation following guidelines and attending experience.5 Conclusions: Based on collected survey data, resident confidence overall improved with simulation center training. Pre-simulation center confidence (out of 10): 5.06, post-simulation center confidence: 5.68, p=0.167. While not significant, the educational value of resident exposure to time critical patient scenarios in a controlled environment are invaluable and difficult to quantify. Resident feedback on the cases and the simulation sessions themselves were obtained and indicated positive resident experiences including resident desire for more sessions and fidelity to real-life scenarios. References: 1Holmberg MJ, Ross CE, Fitzmaurice GM, et al. Annual Incidence of Adult and Pediatric In- Hospital Cardiac Arrest in the United States. Circ Cardiovasc Qual Outcomes. 2019;12(7):e005580. Published 2019 Jul 9. 2Bonnerup S. Composite Code Scenarios [Internet]. 2025 [cited 24 July 2025]. Available from: https://drive.google.com/file/d/1l4wFXXcM5xRsbx-Uglbzew29O0vobuKH/view?usp=drive_link

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Transitional Medical Graduates	
Research - Transitional Medical Graduates	
Nouran Keshk Jeremiah Aakre Dr. Ron Petersen Dr. Sara Aboelmaaty Dr. Hunter Sylvester Dr. Michael Griswold Dr. Gwen Windham Dr. Maria Vassilaki	*Loneliness, Social Engagement and Cognitive Function in Community-Dwelling Older Adults* Introduction: Loneliness and social isolation have been associated with cognitive impairment in older adults and remain important public health concerns. Further research is needed, particularly in community-based populations with detailed cognitive assessments. The Mayo Clinic Study of Aging (MCSA) is a population-based study of cognitive aging and impairment that began in Rochester, Minnesota, in 2004 and expanded to Jackson, Mississippi, in 2022. Loneliness and social engagement were assessed using the NACC Social Determinants of Health questionnaire (loneliness scored 1-5, with higher scores indicating greater loneliness; social engagement scored 1-6, with higher scores indicating greater engagement). Consistent subjective cognitive decline (cSCD) was defined as having at least one Everyday Cognition Scale item rated ""consistently a little worse"" compared with 10 years earlier. Linear and logistic regression models adjusted for age, sex, and education examined cross-sectional associations of loneliness and social engagement with cSCD, mild cognitive impairment (MCI), and cognitive performance. Methods: The analysis included 2,179 participants without dementia aged ≥50 years (mean [SD] age, 73.2 [10.4] years; 50.6% make), including 137 participants with MCI. Greater loneliness was associated with higher odds of cSCD (OR 1.92, 95% CI 1.69-2.19 per unit increase) and MCI (OR 1.46, 95% CI 1.15-1.85), as well as lower cognitive scores across memory, executive, language, and visuospatial domains. Higher social engagement was associated with lower odds of cSCD (OR 0.85, 95% CI 0.74-0.97) and MCI (OR 0.79, 95% CI 0.61-1.01). Conclusion: Greater loneliness and lower social engagement were associated with early cognitive changes in older adults. Although the cross-sectional design does not establish the direction of theses associations, the findings support the clinical and public health

	relevance of loneliness and social engagement in the growing older adult population.
Mounika Pusa Dr. Roslin Jose George Hannah Langenfeld, MPH Dr. Juna Musa Dr. Elena Myasoedova Dr. John Davis Dr. Cynthia Crowson	*Geographic Disparities in the Initiation of Glucocorticoid, DMARDs and Biologic Agents Among Patients with Rheumatoid Arthritis* Background: Early and sustained treatment with disease-modifying antirheumatic drugs (DMARDs) and biologic therapies is central to RA management. In addition, glucocorticoids (GCs) are generally recommended as short-term bridging therapy because of their well-recognized adverse effects. Access to these therapies depends on timely rheumatology care. However, patients in rural and micropolitan areas often face barriers like workforce shortage, transportation challenges, and limited specialty access. There is limited data evaluating differences in the utilization of RA therapies across geographic areas. Thus, we examined GCs, DMARDs and biologic initiation across the rural-urban spectrum. Methods: We identified a population-based inception cohort of patients with RA diagnosed during 2010-2019 and followed them longitudinally through their medical records until death, migration or 12/31/2024. All patients fulfilled 1987 and/or 2010 classification criteria for RA. Medication start and stop dates for GCs, DMARDs and biologic agents were collected. Regional measures included the Area Deprivation Index (ADI) and Social Vulnerability Index (SVI), categorized as low (≤25%), moderate (26-74%) and high (≥75%). Rurality was defined using RUCA codes as urban (1–3), micropolitan (4–6), and rural (7–10). Cox proportional hazards models adjusted for age, sex, and year of RA diagnosis were used to evaluate associations between geographic factors and treatment initiation. Results: The study included 1,573 patients (67% female) diagnosed with RA, which includes 802 urban, 426 micropolitan, and 345 rural residents. Age, sex, and seropositivity was similar across groups. Higher area level deprivation was more common in micropolitan (27%) and rural (24%) residents compared with urban residents (6%) ($p<0.001$; Table 1). Micropolitan residents were 14% more likely to initiate GC therapy than urban residents (HR 1.14, 95% CI 1.01-1.29), suggesting greater reliance on glucocorticoids in these communities. In contrast, initiation of DMARD therapy was less frequent among micropolitan patients (HR 0.86, 95% CI 0.75-0.98) and modestly lower among rural patients compared with urban residents. No significant geographic differences were observed in biologic initiation. Lower social vulnerability was associated with a greater likelihood of DMARD initiation, while area-level deprivation and social vulnerability were not significantly associated with GC or biologic initiation. Older age was associated with lower rates of DMARD and biologic initiation, whereas female patients were 13% less likely to initiate GC therapy as compared to males.

	Conclusion: Geographic disparities in RA treatment patterns persist despite advances in therapeutic options. Patients living in micropolitan communities were more likely to receive glucocorticoids and less likely to initiate DMARD therapy than their urban counterparts, suggesting potential differences in access to timely, guideline-concordant rheumatology care. Although biologic initiation did not vary by geographic location, the observed disparities in early treatment approaches highlight the continuing influence of geographic and socioeconomic factors on RA management. Efforts to improve access to specialty care and optimize early treatment strategies may help reduce these inequities and improve outcomes for patients with RA.
Clinical Vignette - Transitional Medical Graduates	
Hiba Ahmed Saman Pathan Dr. Mithun Suresh	*Tire Explosion: The Science, Pathophysiology, and A Case* Case Presentation: 67-year-old male with medical history of history of oxygen dependent COPD, neck neoplasm previously treated with surgery and radiation therapy, chronic hypotension, pharyngeal dysphagia, esophageal stricture, recurrent aspiration pneumonias, hypothyroidism, anxiety, previous stroke, low BMI who presented to the hospital after being struck by an exploding tire. The patient was blowing up a tire when it exploded, and he was struck in the chest by debris from the explosion. Pain was located primarily in the right chest. No head trauma or loss of consciousness. The area was red with minor abrasions around it. X-ray imaging was notable for a fracture through the medial right clavicle with approximately 1 cm inferior and lateral displacement, a displaced fracture anterior right first rib, and diffuse infiltrates in the right lung (slightly decreased in the base and increased in the mid and upper lung), left sided perihilar infiltrates, left basilar atelectasis, and hyperinflation in both lungs. His symptoms were able to be controlled with oral pain medications, so he was placed in a sling and discharged with plans to follow-up with orthopedics as an outpatient. However, his chest pain worsened, and along with the development of some shortness of breath and hypoxia that was detected with a home oxygen saturation probe, he returned to the hospital. His pain was most intense with breathing, talking, and moving. Aside from pain and shortness of breath, he denied any other symptoms. CT imaging was performed and demonstrated moderate consolidation of both lower lobes with air bronchograms, acute fractures of the medial right clavicle and anterior right first/second/third ribs, and multiple noncalcified varying sized pulmonary nodules in the right lung concerning for metastatic disease. He was subsequently admitted for further management. He was seen by the trauma surgery and orthopedic surgery teams, and both recommended non-operative management and supportive cares. Over the next three days, his oxygen needs progressively worsened. His cough was weak secondary to his chest trauma but also, prior neck surgeries and radiation therapy to this area. Aggressive pulmonary toilet was attempted, including the use of incentive spirometry,

	nebulizers, and nasotracheal suctioning. Serial chest x-rays during the time that oxygen needs were worsening demonstrated progressive interstitial opacities. Antibiotics were also initiated as well. Steroids were started in case of concurrent COPD exacerbation. Despite all of this, along with escalating respiratory support to include high flow oxygen therapy and BIPAP, oxygenation needs continued to worsen such that intubation was recommended. During intubation, cardiac arrest occurred. ROSC was obtained, but the post-ROSC period was characterized by worsening shock and multi-system organ failure over the next several hours. Accordingly, family elected to transition the patient to comfort cares, and he passed away in the evening.
Nithish Nanda Palanisamy Dr. Gokulesh Gurumurthy Dr. Pranaya Baskaran Dr. Moira Hilscher	*CST Syndrome with Hereditary Recurrent Pancreatitis* Introduction: Hypohidrotic ectodermal dysplasia (HED)/ Christ–Siemens–Touraine syndrome (CST) is characterized by hypotrichosis, hypohidrosis, and hypodontia. Clinical features include thin, lightly pigmented, slow-growing scalp hair and deficient sweating, leading to episodes of hyperthermia[Wright JT et al, 1993]. Pancreatitis results from the uncontrolled activation of trypsinogen to trypsin. The chymotrypsin C gene (CTRC) codes chymotrypsin C. Trypsinogen degradation prevents conversion of active trypsinogen to active trypsin. Loss-of-function mutations in CTRC reduce the secretion or activity of chymotrypsin C, thereby impairing the protective trypsinogen degradation [Zhou et al, 2011]. Case Report: A 13-year-old male child, a known case of hypohydrotic ectodermal dysplasia (Christ–Siemens–Touraine syndrome), presented with symptoms of acute pancreatitis. Labs revealed anemia and elevated lipase and amylase levels. CT abdomen showed a bulky pancreas and peri-pancreatic fluid suggestive of acute pancreatitis. Past history was notable for acute pancreatitis 2 months back. There's no family history of pancreatitis. Given his pre-existing genetic predisposition, a hereditary cause for the recurrent pancreatitis was suspected. Additional genetic testing was done, which revealed that the patient was a carrier of the pathogenic CTRC gene associated with hereditary pancreatitis. Discussion: Classic HED can be diagnosed after infancy based on physical features in most affected individuals. Mild HED is diagnosed by genetic testing of pathogenic variant of EDA, EDAR, EDARADD or WNT10A. These genes can be transmitted in an X-linked recessive/ autosomal recessive/ autosomal dominant fashion [Wright JT et al 1993, Tridico LA et al 2015]. Our patient had classical clinical features and was confirmed with genetic testing. Since he had a pathogenic EDA variant, HED was transmitted through an X-linked recessive fashion. Our patient presented with multiple episodes of pancreatitis. Since he had a pre-existing genetic disorder, we suspected hereditary pancreatitis. The pathophysiology behind hereditary pancreatitis is the

	uncontrolled activation of trypsinogen to trypsin. The genes involved include mutations in PRSS1, SPINK1, PRSS2, CTRC, and CTRB1-CTRB2 locus inversion [Mayerle, J. et al 2019]. The majority of patients have a mutation in the trypsinogen gene (PRSS1) [Charnley et al 2003]. CTRC Mutation resulted in 5 fold increase risk of chronic pancreatitis [Mayerle.J et al 2019]. The CTRC gene is found to have an autosomal dominant, low-penetrance pattern of inheritance. [9] Our patient had a pathogenic CTRC variant, which substantiates episodes of recurrent acute pancreatitis at an early age. Once a patient carries one confirmed monogenic diagnosis, clinicians should lower their threshold for genetic workup for unrelated new symptoms. Also, the absence of family history does not exclude hereditary pancreatitis, as CTRC-related pancreatitis follows low penetrance. Management of our patient includes coordination between multiple departments and a multidisciplinary approach and follow-up. Conclusion: This case highlights an unusual co-occurrence of Christ–Siemens–Touraine syndrome and hereditary pancreatitis secondary to a pathogenic CTRC variant in the same child. Although HED and hereditary pancreatitis are genetically unrelated, their coexistence highlights the need to consider genetic causes in children with monogenic syndromes who develop atypical or recurrent illnesses.
Bhumika Rimal Dr. Selene Rubino Dr. Shiva Aryal Dr. Courtney Arment	*Title: JAK1 Inhibition with Upadacitinib Achieves Rapid Clinical and Serologic Remission in Refractory Adult-Onset Still's Disease* Background: Adult-onset Still's disease (AOSD) is a rare systemic autoinflammatory disorder characterized by quotidian fevers, evanescent rash, arthritis, hyperferritinemia, and systemic inflammation. While interleukin (IL)-1 and IL-6 inhibitors have transformed management, a subset of patients remain refractory to conventional and biologic therapies. Janus kinase (JAK) inhibitors have emerged as a potential therapeutic option for difficult to treat diseases. Case Presentation: A 28-year-old woman was diagnosed with AOSD four years prior to presentation after developing daily fevers, salmon-colored rash, arthralgias, splenomegaly, elevated inflammatory markers, and marked hyperferritinemia. Despite treatment with anakinra (IL-1 inhibition), methotrexate, canakinumab (IL-1 inhibition), and tocilizumab (IL-6 inhibition), she experienced recurrent flares requiring prolonged glucocorticoid therapy. She was readmitted with recurrent fevers, severe fatigue, elevated liver enzymes, and ferritin of 1,253 ng/mL while on canakinumab 300 mg subcutaneously every 14 days and IV methylprednisolone 1000 mg every 2 weeks. She received intravenous methylprednisolone 500 mg daily for three days and was started on upadacitinib 15 mg daily.

	At one-month follow-up, she reported complete resolution of fevers, fatigue, rash, sore throat, and systemic symptoms, describing her condition as ""the best she has felt in years."" Laboratory evaluation demonstrated normalization of inflammatory markers, including CRP, liver enzymes, and ferritin (148 ng/mL). Physical examination showed no active synovitis, rash, or systemic manifestations of disease. At three months, she was able to taper down to prednisone 20 mg daily with the goal of complete discontinuation. Discussion: This case illustrates successful disease control in refractory AOSD following initiation of a JAK inhibitor after failure of multiple standard therapies, including methotrexate, IL-1 inhibition, and IL-6 inhibition. The rapid improvement in both clinical manifestations and laboratory parameters suggest that JAK-STAT pathway blockade may effectively suppress persistent inflammatory signaling in treatment-resistant AOSD. Furthermore, upadacitinib provided an opportunity for glucocorticoid tapering, an important goal given this patient's significant steroid related complication. Conclusion: Upadacitinib induced rapid clinical and serologic remission in a patient with severe refractory AOSD who had failed first line therapy with IL-1 inhibitors. This case supports the growing role of JAK inhibition as a promising therapeutic and steroid-sparing strategy in difficult-to-treat AOSD.
Residents	
Quality Improvement - Residents	
Sara Busche Dr. Jesse Abelson Dr. Wyatt Hahn Dr. Breanna Zarmbinski	*Clearing the Air: Improving Lung Cancer Screening Through Standardized Tobacco Assessment* Background: Lung cancer remains the leading cause of cancer-related mortality in the U.S., and low-dose computed tomography (LDCT) screening reduces mortality among high-risk individuals. Despite guideline recommendations, LDCT screening remains underutilized, in part because tobacco use documentation within the electronic health record (EHR) is often incomplete or inaccurate. Approximately 12.8 million U.S. adults meet USPSTF eligibility criteria for LDCT screening, yet many eligible patients are not recognized within clinical systems. Consequently, only about 15% of eligible individuals have undergone screening. This quality improvement (QI) project aimed to improve identification of patients eligible for LDCT screening through implementation of a standardized tobacco use worksheet completed during clinic visits. Methods: This initiative was conducted over 6 months in a primary care resident clinic. A standardized tobacco use worksheet was implemented for completion at every in-person clinic visit, regardless of previous completion history. The form was completed by patients or

	medical assistants (MAs) during the rooming process, and patients were informed that participation was voluntary. The worksheet collected detailed smoking history elements required to determine LDCT eligibility, including smoking status, pack-year history, and quit date. The worksheet was available in English, Spanish, and Somali. Completed worksheets were uploaded into the EHR, allowing information to populate structured tobacco-use fields recognized by clinical decision support tools. A post-implementation survey assessed ease of use and perceived impact on clinic workflow. In addition, randomly selected worksheets were reviewed to evaluate completeness, accuracy, and appropriate upload into the EHR. Primary outcomes included changes in the number and proportion of the clinic population recognized by the EHR as eligible for LDCT screening. The secondary outcome was the percent of identified individuals who were overdue for screening. Results: During the 4 weeks preceding implementation, the number of patients deemed eligible for LDCT screening via EHR minimally increased from 31 to 33. Across the 6-month intervention period, this increased from 33 to 87, corresponding to an increase from 2.1% to 5.6% of the clinic population identified as eligible. Within the eligible population, the number of patients overdue increased from 25 to 46, while the proportion of eligible patients who were overdue decreased from 71% to 53%. This reflects improved recognition of unmet screening needs and increased completion of screening. A random review of 213 completed worksheets demonstrated high fidelity to the intervention. Of these, 79.8% were fully completed and appropriately uploaded into the EHR, 18.8% were partially completed, and 0.9% were not completed. MA surveys indicated the worksheet was user-friendly and did not significantly burden clinic workflows. Discussion: Implementation of a standardized tobacco use worksheet improved smoking history documentation and enhanced the EHR's ability to identify patients eligible for LDCT screening. The intervention exposed previously unrecognized care gaps while improving physicians' ability to address them, ultimately increasing the number of individuals undergoing LDCT screening. Integration into the rooming process was feasible, with high completion and upload rates. Limitations include the single-site design, short duration, and reliance on in-person visits, potentially excluding patients utilizing telehealth or those who infrequently present for care.
Yahye Farajuun Dr. Fiona He Dr. Emily Stickney	*Optimizing Treatment of Iron Overload in Myelodysplastic Syndrome (MDS)* Background: Iron overload is a recognized complication of transfusion-dependent myelodysplastic syndrome (MDS), yet

Gabe Steinwand, PharmD, BCOP Erin Kiernan-Johnson, RN, MS, CPHQ Michelle Mastous, MS, MLS	identification of eligible patients and implementation of chelation strategies remain challenging. A retrospective review evaluated low- and intermediate-risk MDS patients receiving ≥20 red blood cell transfusions at Allina over a period of 10 years. Among 221 patients screened, 116 met criteria after exclusions. Median age was 75 years and median transfusion burden was 27 units (range 20-182). Thirty patients (25%) received iron chelation therapy, 46 (40%) had documented chelation discussions. Methods: A multidisciplinary quality improvement initiative at Allina Health Cancer Institute (AHCI) aimed to standardize iron overload identification and management. Interventions included clinician education, an Epic SmartPhrase that provided transfusion count history directly into the clinical note, a standardized therapy plan for chelation integrated with specialty pharmacy management, and a data dashboard that flagged patients at risk for iron overload based on transfusion history and serum ferritin levels. Results: We identified 10 living low-risk MDS patients with >20 lifetime transfusions at AHCI. Five had current or prior chelation, one declined therapy, one had ferritin <200, and two were potential candidates without documented discussions. These findings suggest chelation was considered for many eligible patients and shifted the improvement focus from increasing treatment uptake to improving patient identification, documentation, and workflow reliability. Conclusion: A systematic approach using EHR-enabled interventions can help clinicians identify and treat patients with lower risk MDS who may be eligible for chelation therapy. Future efforts will monitor adoption of EHR tools and expand scalable interventions across AHCI practice sites.
Ashley Grunwald	*Rethinking Sulfonylureas in the Hospital: A QI Success Story* Background: Sulfonylureas (SU) carry a significant risk of inducing hypoglycemia, and inpatient hypoglycemia in non-ICU settings is associated with prolonged length of stay, higher hospitalization costs, and increased mortality both during admission and after discharge (Korytkowski et al., Journal of Clinical Endocrinology & Metabolism, 2022; Mendez and Umpierrez, Diabetes Spectrum, 2014). Initial data from hospitalized patients administered an SU between 8/1/2022 and 7/30/2023 demonstrated a 10% rate of hypoglycemia, prompting a system-wide safety initiative. Objectives: As Part 2 of a multiphase quality improvement (QI) project on inpatient SU use, this study evaluated the effectiveness of a multistep intervention designed to improve patient safety. The intervention comprised: (1) an alternative electronic medical record (EMR) alert warning providers of hypoglycemia risk and recommending a sliding scale insulin (SSI) order set; (2) an SU order

	set panel bundling the medication with QID blood glucose checks and a hypoglycemia order set; and (3) targeted provider education. Methods: A system-wide retrospective chart review was performed across all Allina Health hospitals. Patients treated from 8/1/2022–7/30/2023 (control group) were compared with those treated from 4/12/2025–4/12/2026 (intervention group). Inclusion criteria were type 2 diabetes mellitus and administration of a sulfonylurea (glyburide, glimepiride, or glipizide) during the inpatient stay. Patients on the inpatient rehabilitation unit (Courage Kenny) and the inpatient mental health unit were excluded. Outcomes included the rate of hypoglycemic events, SU prescribing volume, and event rates within predefined high-risk subgroups (chronic kidney disease [CKD] and NPO status). Results: A total of 426 unique patients received a sulfonylurea (mean age 68 years). Following implementation, SU prescribing decreased by 68%, accompanied by a statistically significant reduction in hypoglycemic events, corresponding to a 55% relative risk reduction. Among patients in redefined high-risk groups who experienced a hypoglycemic event, the intervention was associated with an 89% reduction in patients with CKD and a 100% reduction in patients who were NPO. Conclusions: A multistep, EMR-based safety intervention—combining an alternative alert, a standardized SU order set with structured glucose monitoring and hypoglycemia protocols, and provider education—was associated with a substantial decline in inpatient sulfonylurea use and a significant reduction in hypoglycemic events, with the greatest benefit in high-risk CKD and NPO populations. Although sulfonylureas remain commonly used for their glucose-lowering efficacy, familiarity, and low cost, their inpatient use warrants careful monitoring and consideration of alternatives. System-level EMR alerts, ongoing education, and standardized safety practices can meaningfully reduce adverse events in hospitalized patients. Limitations: Retrospective design; clear liquid diet not analyzed as a distinct category within NPO status; other hypoglycemia-inducing agents (e.g., fluoroquinolones, beta-blockers) not accounted for; inclusion of mixed capillary and venous glucose measurements; and no adjustment for baseline differences between groups.
Research - Residents	
Siddhant Solanki Dr. Simranjeet Singh Nagoke Dr. Sreekant Avula	*Once-Weekly Versus Once-Daily Basal Insulin in Type 1 and Type 2 Diabetes: A Systematic Review and Meta-Analysis of Twelve Phase 3 Randomized Trials* Background: Two once-weekly basal insulins, insulin icodec and insulin efsitora alfa, reduce basal injections from 365 to 52 per year. Existing meta-analyses have examined one molecule at a time, report

	pooled HbA1c differences ranging from favoring once-daily to favoring once-weekly insulin, and predate the most recently published trial. We pooled the complete phase 3 evidence base and compared the two molecules directly. Methods: The review was registered with PROSPERO (CRD420261511906) and conducted following PRISMA 2020. We searched PubMed/MEDLINE, Europe PMC, Cochrane CENTRAL, Crossref, Google Scholar, OpenAlex and Semantic Scholar from inception to 13 September 2026, without language or date restriction, supplemented by ClinicalTrials.gov, WHO ICTRP, and ADA and EASD abstract archives. Eligible studies were phase 3 randomized trials comparing insulin icodec or insulin efsitora alfa with a once-daily basal insulin analogue in adults with type 1 or type 2 diabetes. Two reviewers screened and extracted independently. Outcomes were change in HbA1c (estimated treatment difference) and combined level 2 or 3 hypoglycemia (rate ratio), pooled with DerSimonian-Laird random-effects models; heterogeneity was quantified by I-squared. Diabetes type and molecule were prespecified subgroups. Results: Of 3,451 records identified, 2,323 remained after deduplication; 12 trials randomizing 8,890 adults met eligibility (1,274 type 1, 7,616 type 2). The two molecules diverged on efficacy. Across the efsitora program, HbA1c reduction was equivalent to once-daily analogues (pooled estimated treatment difference -0.03 percentage points, 95% CI -0.09 to 0.03; I-squared 1%). Icodec trials favored once-weekly dosing, with four demonstrating statistical superiority for HbA1c reduction. For combined level 2 or 3 hypoglycemia, both type 1 diabetes trials showed significantly higher rates with once-weekly insulin (rate ratio 1.90, 95% CI 1.50 to 2.30; and 1.21, 95% CI 1.04 to 1.41). None of the ten type 2 diabetes trials showed an increase, and one insulin-initiation trial using fixed-dose escalation showed a reduction (0.57, 95% CI 0.39 to 0.84). Heterogeneity was substantial and the formal interaction test by diabetes type was not significant ($p = 0.14$). Conclusions: Across all twelve completed phase 3 trials, once-weekly basal insulin achieved glycemic control at least equivalent to once-daily analogues, but the two agents are not interchangeable and the safety signal is not uniform. Every type 1 diabetes trial showed a significant excess of clinically significant or severe hypoglycemia with once-weekly insulin; no type 2 diabetes trial did. For the general internist, weekly basal insulin is a reasonable option in type 2 diabetes, most favorably at insulin initiation, while its use in type 1 diabetes should await dose-optimization data. Prescribers should treat icodec and efsitora as distinct agents rather than a single class.
Justin Woolridge Dr. Jeffrey Moorhead	*Iterative Physician-Guided Prompt Design for Detecting Acute Coronary Syndrome from Clinical Encounter Notes*

Vinod Kaggal, MS Dr. Adelaid Arruda-Olson	Background: Atherosclerotic cardiovascular disease (ASCVD)—including acute coronary syndrome (ACS)—is a leading cause of global morbidity and among the most frequently documented conditions in clinical notes. Chart review is time-intensive, consuming an estimated 33% of physician electronic health record (EHR) time. Large language models (LLMs) can extract diagnoses from clinical text, but few studies describe structured multi-physician workflows that iteratively refine LLM performance before clinical use. We evaluated whether an iterative physician-in-the-loop workflow improved LLM accuracy for identifying ACS attributes from clinical notes, hypothesizing that targeted, error-driven model revision would yield higher per-attribute precision and recall. Methods: In this validation study, Gemini 2.5 Flash extracted ACS and five related myocardial infarction (MI) attributes from 240 cardiology notes obtained from the Mayo Clinic Explorer database. The model extracted six concepts—ACS, shock, acute MI, history of MI, MI type, and any mention of MI—per the Fourth Universal Definition of MI. Two mutually blinded physicians scored each prediction. Round one errors (120 notes) refined the prompt; round two evaluated 120 additional notes, with a senior cardiologist adjudicating discrepancies. Outcomes were per-attribute precision, recall, F1, and inter-rater reliability (Cohen κ). Results: After second-round adjudication, precision/recall/F1 were: history of MI 1.00/1.00/1.00; shock 1.00/1.00/1.00; acute MI 0.99/1.00/0.99; MI type 0.99/0.99/0.99; mention of MI 0.96/1.00/0.98; ACS 0.93/1.00/0.96. Recall was ≥0.99 for every attribute, with one false negative (MI type). Residual errors were mostly false positives, chiefly ACS (7) and mention of MI (3). Cohen κ ranged from 0.76 (mention of MI) to 0.98 (history of MI). Conclusions: An iterative, physician-in-the-loop workflow refined an LLM identifying ACS and related MI attributes from unstructured cardiology notes with high precision and near-perfect recall and substantial-to-almost-perfect inter-rater agreement. Structured multi-physician validation with targeted refinement can yield clinically credible LLM performance for automated diagnosis extraction.
Clinical Vignette - Residents	
Ryeesa Amin Dr. Molly Beier Dr. Malak Helal Dr. Zelalem Temesgen	*When Two Pathogens Collide: Interpreting Concurrent STEC and Clostridioides difficile Detection in Severe Hemorrhagic Colitis* Introduction: Shiga toxin-producing Escherichia coli (STEC) colitis and Clostridioides difficile infection (CDI) share clinical and radiographic features, yet management conflicts: antibiotics are recommended for CDI but avoided in STEC due to hemolytic uremic syndrome (HUS) risk. C. difficile testing complicates this further: a nucleic acid amplification test (NAAT) detects toxigenic strains but cannot distinguish active infection from asymptomatic colonization.

	Multiplex gastrointestinal (GI) panels identify pathogens quickly but don't establish causality. Co-detection, particularly when one result is equivocal, creates diagnostic and treatment uncertainty. We report a case of severe hemorrhagic colitis in an immunosuppressed woman; her GI pathogen panel detected STEC and toxigenic C. difficile while toxin enzyme immunoassay (EIA) testing was negative, posing uncertainty about the primary pathogen and need for CDI-directed therapy. Case Presentation: A 64-year-old woman with psoriatic arthritis on methotrexate and certolizumab presented with five days of worsening abdominal pain, nausea, vomiting, diarrhea, hematochezia, and subjective fever. She was afebrile, hypotensive (74/57 mmHg), and tachycardic (129 beats/min), with neutrophil-predominant leukocytosis (16.3×10^9/L), hemoconcentration (hemoglobin 17.1 g/dL), and CRP 174 mg/L. CT showed severe pancolitis. A multiplex GI panel was positive for STEC and C. difficile by PCR, with confirmatory EIA pending. STEC was considered the primary driver, but CDI could not be excluded; given severe colitis, immunosuppression, and hemodynamic instability, empiric CDI treatment was started with fidaxomicin before the toxin EIA returned negative. Discussion: This case describes an immunosuppressed woman with severe pancolitis whose panel simultaneously detected STEC and C. difficile (PCR-positive, toxin-negative), forcing a decision between two pathogens whose treatments directly conflict. Antibiotics are generally avoided in STEC because they may increase Shiga toxin release and precipitate HUS, yet possible CDI in a hypotensive, immunosuppressed patient with severe colitis could not be safely ignored. Because empiric management was required before confirmatory toxin EIA results were available, fidaxomicin was selected: its minimal systemic absorption and negligible activity against Enterobacterales supported treating possible CDI while limiting selective pressure on STEC. Multiplex GI panels detect more than one organism in 10–20% of positive samples, and up to nearly half in some cohorts. For C. difficile, NAAT is highly sensitive (~96%) with excellent negative predictive value, whereas toxin EIA is highly specific (~99%) yet insensitive (as low as ~48% by latent class analysis). NAAT-positive/toxin-negative patients have lower recurrence and severe-CDI rates, yet a meta-analysis showed comparable mortality to toxin-positive patients and an association between treatment and reduced mortality. Conclusion: When a multiplex panel co-detects pathogens whose treatments conflict, management must be guided by clinical judgment over any single test result—here, treating possible CDI with a non-absorbed agent while maintaining HUS surveillance addressed one infection without heightening the risk of the other.
Jacob Arel	*A Hole in the Diagnosis: Ventricular Septal Defect Complicating MINOCA*

Dr. Leeore Levinstein	Introduction: Chest pain accounts for the second-largest volume of emergency department visits annually, often leading to a highly regimented diagnostic workup. However, standard protocols can obscure atypical etiologies. This clinical case demonstrates the importance of a thorough, broad-differential evaluation for a common chief complaint, particularly when the initial coronary and diagnostic evaluations fail to explain the patient's presentation. Case Description: A 71-year-old female with no prior cardiac history presented to the emergency department with chest pain and nausea. She described her chest pain as located at her sternum and squeezing in nature; it was not relieved with nitroglycerin. While in route patient was found to have EKG with ST elevations in leads V2 and V3, with reciprocal depressions in V5 and V6. The physical exam was unremarkable. Patient was subsequently taken to the catheterization lab and found to have normal coronary arteries. Follow up transthoracic echocardiogram revealed hypokinesis of basal and mid anteroseptal and anterior wall, prompting evaluation with cardiac MRI with gadolinium. Imaging revealed a large acute VSD 19 x 7 mm with significant interventricular shunting. The patient underwent Impella bridging to allow for maturation of infarcted tissue with subsequent surgical closure of her VSD. Patient recovered adequately in the post-operative period and follow up transthoracic echocardiogram at 1 month showed normal ejection fraction with no residual shunting. Discussion: This case highlights the rare but catastrophic complication of VSD in myocardial infarction, even in patients presenting with normal coronary arteries. While a normal angiogram can be reassuring, it may mask critical underlying pathologies such as coronary vasospasm, spontaneous coronary artery dissection, or transient thromboembolism that can cause profound myocardial injury. MINOCA should be considered in patients who meet criteria for acute myocardial infarction but have no obstructive coronary artery disease on angiography. It occurs more commonly in women, younger patients, patients with substance use, and those presenting with NSTEMI. When standard diagnostic modalities, including EKG, transthoracic echocardiography, and coronary angiography, yield conflicting results, advanced imaging is paramount to further characterize myocardial structure and tissue injury, in this case via cardiac MRI. Importantly, MINOCA is a working diagnosis rather than a definitive diagnosis and should prompt further evaluation for the underlying etiology. Cardiac MRI can help distinguish ischemic from nonischemic causes of myocardial injury, including myocarditis, Takotsubo cardiomyopathy, and true myocardial infarction, and may provide critical diagnostic information when initial testing is inconclusive. Maintaining a high index of suspicion for MINOCA despite a normal coronary angiogram is essential, as timely identification of the underlying mechanism and associated myocardial injury may allow for recognition and management of potentially life-threatening complications such as VSD.

	Key Takeaways: 1. Do not dismiss MI with a normal coronary angiogram: MINOCA should remain on the differential when clinical and laboratory findings support myocardial infarction despite the absence of obstructive coronary disease. 2. MINOCA is a working diagnosis, not an endpoint: Further evaluation is necessary to identify the underlying mechanism.
Juliana Bacigalupi Hester Dr. Nadir Bhuiyan	*Optimal MRI Timing in a Case of Transient Global Amnesia* Background: Transient global amnesia (TGA) is a disorder characterized by sudden inability to form new memories, often combined with the inability to recall events preceding the episode by hours or days. This is considered a benign phenomenon, since anterograde memory is restored within 24 hours, non-cognitive functions are always preserved, and recurrence is rare. Often, initial brain imaging is negative and etiology of the episode unknown. Case Presentation: We report a case of a 57-year-old woman who presented to her local hospital abroad for chest pain that woke her from sleep. From the time she woke at 4:00AM to being in the hospital at 10:30AM, she had no recollection of events. Family observed her asking the same questions repeatedly, and she was unable to retain information. She had no recollection of the day prior to presentation and believes she may have taken hydroxyzine for a rash related to her recent allergies. Other associated symptoms included dizziness and headaches. Her medical history is significant for mild hyperlipidemia. She had no history of venous thromboembolism or known prior stroke. In the emergency room she had new, markedly elevated blood pressure (192/111). Work up was negative for myocardial infarction, head CT normal, and MRI performed 28 hours post event was interpreted to have no correlated findings. MRI visualized a small, chronic appearing subcortical infarct adjacent to the right caudate head. She was treated with IV diphenhydramine and labetalol. Anxiety was considered, and she was given antidepressants. She presented to our internal medicine clinic for a second opinion ten days later. Her history was highly suspicious for TGA. Second read of original MRI revealed multiple punctate foci of diffusion restriction within both hippocampal formations on DWI sequence, no evidence of chronic regional infarct. This finding was consistent with our clinical diagnosis. Her neurological exam was normal, Kokmen mental status scale was 33/38, with points lost on attention, calculation, and recall. Additional workup revealed diastolic hypertension on 24-hour BP monitoring. Carotid US was negative for stenosis with minimal plaque at the bifurcation. Evaluation of her chest pain with exercise EKG and Holter monitor did not reveal abnormalities. On exam, chest pain was reproducible with palpation of the costochondral junction at the 3-4th

	left intercostal border, suggesting costochondritis. She was placed on losartan 25 mg daily, atorvastatin 20 mg to reduce her risk of ASCVD. Conclusion: TGA is a disorder that can be encountered in the inpatient or outpatient practice of internal medicine physicians. While symptoms can be distressing to patients and family members, reassurance can be provided about prognosis. Atypical symptoms include any neurological deficits beyond an anterograde and retrograde memory impairment, or memory impairment without resolution. Diffusion restriction at the hippocampus can be seen in up to 70% of cases, but it is easily missed if MRI is performed too early, or after 48 hours. Findings are transient and not associated with infarct. A detailed history is essential to consider alternative diagnosis, but in the absence of atypical symptoms, extensive work up is not necessary.
Jasson Barrios Abid Anwar, BS Dr. Misha Gautam Dr. Motaz Ashkar	*Gallstone Ileus at an End Ileostomy: A Rare Site of Impaction* Introduction: Gallstone ileus is a rare mechanical bowel obstruction that occurs when a gallstone passes through a cholecystoenteric fistula and impacts within the small bowel, usually at the ileocecal valve. It accounts for an estimated 0.1–4% of mechanical bowel obstructions and is more commonly seen in the elderly population. The site of impaction largely depends on the size of the stone and the diameter of the bowel lumen. In rare situations, a stone may impact at a pre-existing point of narrowing, such as an anastomotic stricture, where the functional lumen diameter is already reduced. We present a unique case in which a gallstone impacted at an end ileostomy in a patient with altered surgical anatomy, ultimately removed through surgical retrieval. Case Presentation: An 83-year-old man presented for evaluation of recurrent small bowel obstruction. His past medical history was notable for ulcerative colitis, status post total abdominal colectomy with end ileostomy, and cirrhosis with portal hypertension. Notably, four years earlier he had experienced painless jaundice with pericholecystic inflammation that resolved spontaneously. This event was attributed to the passage of a gallstone. Two weeks prior to presenting to our team, he had experienced abdominal distension, nausea, and vomiting and was found to have a small bowel obstruction at his ileostomy, initially managed conservatively with decompression. His symptoms resolved and he was discharged. He then re-presented to our team with ongoing abdominal pain, poor oral intake, and increased stomal output. CT demonstrated small bowel dilation with inflammation at the stoma. Ileoscopy revealed a strictured stoma with two calculi impacted in the distal neo-terminal ileum with stercoral ulceration. Initially, both were deemed to be stercoliths. The patient underwent ileostomy revision with segmental resection, and both stones were retrieved and found to

	be gallstones. Patient had clinical improvement after the removal of the gallstones. Discussion: We present a rare case of gallstone ileus found at an atypical anatomical location, where the stoma effectively became the functional point of narrowing. Normally, there is no connection between the gallbladder and the bowel, making gallstone ileus very rare. It typically requires a preceding fistula to form between the two structures, which is created when a large stone erodes through the gallbladder wall. The patient's earlier episode of painless jaundice that resolved spontaneously likely marked the formation of the fistula, though whether the same or subsequent stones went on to cause the obstruction cannot be fully determined. To our knowledge, gallstone impaction at an end ileostomy has rarely been reported. It was managed successfully with a segmental bowel resection that removed the stones. This case provides an excellent example of a gallstone ileus visualized directly on endoscopy with resolution through surgery. Conclusion: Gallstone ileus with impaction at an end ileostomy is exceptionally rare. This case reinforces the importance of keeping gallstone ileus on the differential for bowel obstruction, particularly in the elderly population. Furthermore, it demonstrates successful management by surgical management of the stones.
Jacob Beery	*A Swell of Trouble: Severe Lower Extremity Edema from an Over-the-Counter Supplement–Amlodipine Interaction* Background: An estimated 60% of Americans take non-prescription supplements. While the efficacy of these over-the-counter pills can range significantly anywhere from generic replacement of prescription medications to placebo; there exist clear dangers of certain supplements when taken at the same time of prescription medications. This danger can come from a variety of mechanisms including drug absorption interference, drug breakdown acceleration or delay, an/or metabolism acceleration or delay. In this case, we present a case of severe lower extremity edema due to health supplement side effects. Case Presentation: Ms. K is a 75 y/o F with a past medical history of HTN, NSTEMI s/p PCI,, MDD, and HLD. Her medications included amlodipine, aspirin, carvedilol, and sertraline. She presented to clinic with 5 months of pitting lower extremity edema that began in late spring of that year without any clear inciting events at that time. BP was 117/75 with other vitals also normal. She denied any clinical signs or symptoms concerning for acute or chronic kidney, liver, or heart failure besides noted edema. She had also recently had labs showing normal, unchanged renal, hepatic function and a normal NT-proBNP. She did endorse taking a hair supplement called "Nutrafol," however stated her symptoms began months after starting this. Given a lack of

	clear cause and known side-effects of amlodipine, she was instructed to discontinue and start losartan for HTN management. Prior to making this switch, she called back to clinic to state that she was also taking a weight loss supplement called "Mitolyn." In this communication she noted she stopped taking this Mitolyn and her symptoms resolved within 3 days. Upon literature review, it was noted that Mitolyn contains Gomisin C and Gomisin N, lignan molecules contained in Schisandra Fruit Extract which is one component of the Mitolyn supplement. These are both potent, irreversible inhibitors of the CYP3A4 pathway which inactivates amlodipine. Ingestion of Mitolyn therefore is thought to lead to supra therapeutic amlodipine levels. Discussion: There are several key learning points from this case including pearls and pitfalls of edema complaints in the outpatient setting, medication reconciliation, and anti-hypertensive therapy side effects. For working up new edema in the outpatient setting, it is important to rule out acute renal, hepatic, or cardiac issues before considering non-life-threatening issues. Also important is complete medication reconciliation when dealing with new issues, knowing that patients may forget certain non-prescription medications that they are taking if not prompted with drug names. The teaching point in this case is regarding amlodipine and other anti-HTN medication use. Typically going from moderate to high dose or even supratherapeutic doses of amlodipine results in little BP change but significant increase in side effects. In this case, BP remained stable but significant edema began with CY3A4 inhibition. Importantly, this supplement was stopped and her symptoms resolved.
Caroline Behling-Hess Dr. Leeore Levinstein	*Tale of a Trojan Horse: Cauda Equina as a Presenting Symptom of Primary Bone Lymphoma* Introduction: Low back pain is one of the most common presenting symptoms in general internal medicine. The vast majority of cases are self-limiting, and red flag symptoms such as progressive pain or weakness warranting further workup often overlap with the natural progression of benign disease. Case Presentation: A 78-year-old man presented to his PCP with gradually progressive low back pain with occasional radiation of discomfort and tightness into both of his legs. Symptoms were generally worse with walking and improved with rest. Initial XR imaging noted mild degenerative changes in the bilateral hips and L4-5 vertebrae. He was diagnosed with neurogenic claudication and referred to physical therapy. Symptoms persisted despite some gains in strength with PT. An MRI was ordered due to development of numbness in the right foot, which showed a lytic-appearing lesion in the dorsal S1 body with concern for extraosseous lesion extension. Repeat MRI three months later showed tumor extending into the adjacent ventral epidural space. Biopsy of the lesion was delayed by patient limitations from pain; results showed no evidence of malignancy. Two weeks later, the

	patient presented with new inability to walk, constipation, and urinary incontinence. He was admitted to the hospital with concern for cauda equina syndrome. Repeat MRI and subsequent PET scan 6 weeks after his previous imaging showed new leptomeningeal enhancement concerning for metastases. Primary cancer at this point was still unknown; however, workup for likely causes was thus far negative. Repeat sacral biopsy again showed no malignancy and lumbar punctures were without diagnostic findings. He was treated with dexamethasone for cord edema and surgical biopsy was pursued. Tissue showed a small focus of neoplastic cells consistent with primary bone lymphoma, likely diffuse large B-cell lymphoma (DLBCL). The patient ultimately elected to pursue hospice and passed away 10 months after his initial presentation with low back pain. Discussion: This case demonstrates the progression of an initially benign presentation of low back pain to cauda equina syndrome from malignant compression, and highlights the importance of prompt investigation of red-flag findings such as lytic bone lesions. While an uncommon presentation of DLBCL, primary bone lymphomas should remain on the differential as highly treatable but easily missed etiology for lytic bone lesions with an unknown primary. Additionally, primary bone lymphomas have a high non-diagnostic biopsy rate; when clinical suspicion is high, additional biopsies or escalation to surgical intervention should be pursued early in the clinical course.
Vishwesh Bharadiya Dr. Emma Johns Dr. Parul Berry Dr. Ellen McPhail Dr. Mary Finta	*Anaplasmosis-Associated Hemophagocytic Lymphohistiocytosis Masquerading as Active Sarcoidosis: A Diagnostic Challenge* Background: Human granulocytic anaplasmosis (HGA) is an increasingly recognized tick-borne illness that can rarely trigger secondary hemophagocytic lymphohistiocytosis (HLH). The diagnosis may be obscured by coexisting inflammatory disorders and nonspecific clinical manifestations. Case Presentation: A 79-year-old man with pulmonary sarcoidosis not receiving immunosuppression, heart failure with mildly reduced ejection fraction, and recently diagnosed dysautonomia presented with acute hypoxemic respiratory failure and recurrent high-grade fevers. Initial evaluation demonstrated elevated inflammatory markers, while CT pulmonary angiography showed stable pulmonary sarcoidosis without pulmonary embolism or consolidation. Despite empiric treatment for community-acquired pneumonia, fevers persisted. Over the subsequent four days, he developed progressive cytopenias, transaminitis, and splenomegaly. Tick-borne testing, including Anaplasma phagocytophilum polymerase chain reaction (PCR), was sent early in the hospitalization but remained pending. An initial peripheral blood smear was unrevealing. FDG-PET/CT demonstrated avid pulmonary and thoracic nodal lesions and abnormal splenic uptake, raising concern for active sarcoidosis, malignancy, or primary

	inflammatory disease. Ferritin increased to 18,122 mcg/L, triglycerides to 366 mg/dL, and soluble interleukin-2 receptor levels exceeded 4,000 pg/mL. The patient fulfilled five HLH-2004 criteria and had an HScore corresponding to a 98%–99% probability of HLH. An extensive infectious evaluation was otherwise unrevealing, including negative blood and urine cultures, respiratory viral testing, HIV, viral hepatitis, Epstein-Barr virus, QuantiFERON-TB, endemic fungal testing, Lyme serology, and Bartonella testing. Repeat peripheral blood smear obtained 48 hours later for worsening thrombocytopenia revealed intracytoplasmic morulae within neutrophils, establishing the diagnosis of HGA prior to PCR confirmation. Doxycycline therapy resulted in rapid defervescence and hematologic recovery without HLH-directed immunosuppression. Retrospective history subsequently revealed that his wife had sustained multiple recent tick bites near their home. Conclusion: Anaplasmosis should remain in the differential diagnosis of unexplained fever, cytopenias, and HLH in endemic regions. Repeat peripheral smear examination may be diagnostic even when initial studies are unrevealing and can establish the diagnosis before molecular testing becomes available.
Susanna Goggans Dr. Rade Jibawi Rivera Dr. Alan Hu Dr. Emily Olson	*I Got an Itch that this is Sepsis: MRSA Bacteremia Presenting as Diffuse Pruritus* Introduction: Methicillin-resistant staphylococcus aureus (MRSA) bacteremia typically presents with fever, chills, and hypotension. In this case, the patient's chief complaint was "itching all over." Case Presentation: A 68-year-old woman with end-stage renal disease on hemodialysis through a tunneled catheter, type 2 diabetes mellitus, and chronic obstructive pulmonary disease presented with 1 day of generalized pruritus, one episode of emesis, and brown, subjectively purulent output from a surgical drain. She was recently discharged following a prolonged hospitalization for colonic ischemia requiring multiple bowel resections with ileocolonic anastomosis. That admission was complicated by postoperative intra-abdominal abscesses requiring multiple drains, with cultures growing Candida albicans and Staphylococcus epidermidis, treated with ceftriaxone, metronidazole, and fluconazole placement which were discontinued approximately 23 days prior to presentation. On presentation, her heart rate was 103 beats/min, blood pressure 111/66 mmHg, temperature 36.8°C, and oxygen saturation 93% on 2 L/min nasal cannula. Laboratory studies demonstrated leukocytosis (white blood cell count 24.1 × 10^3/μL), C-reactive protein 203.1 mg/L, lactate 2.9 mmol/L, blood urea nitrogen 36 mg/dL, and potassium 6.6 mmol/L. Computed tomography of the abdomen and pelvis showed persistent inflammation surrounding the ileocolonic anastomosis concerning for ongoing infection. Physical exam was notable for no

	rash present on skin, mild tenderness to palpation of right lower quadrant, patient alert and answering questions appropriately, and serosanguinous drain output. Empiric ceftriaxone, metronidazole, and vancomycin were initiated for suspected progression of intra-abdominal infection. She was admitted to the medical intensive care unit for urgent hemodialysis to treat severe hyperkalemia. Within 8 hours, peripheral and hemodialysis catheter blood cultures grew MRSA. Shortly thereafter, she developed septic shock requiring norepinephrine. Hemodialysis catheter was removed and patient's pruritus improved with loratadine and adequate treatment of bacteremia with vancomycin. Additionally, she continued on ceftriaxone and metronidazole for ongoing intra-abdominal infection with quick improvement. Discussion: This case illustrates an atypical presentation of MRSA bacteremia, in which diffuse pruritus preceded classic signs of sepsis. S. aureus has been proposed to induce itch through V8 protease-mediated cleavage of protease-activated receptor-1 (PAR1) on pruriceptive sensory neurons. Additionally, patients receiving hemodialysis who are colonized with S. aureus have higher rates and greater severity of uremic pruritus, potentially related to α-toxin-mediated skin barrier dysfunction. We hypothesize that underlying uremia lowered this patient's pruritic threshold, allowing early bacteremia to manifest primarily as generalized itching. Initially, the explanation for patient's SIRS was anchored on progression of her intra-abdominal infection. However, due to the initial collection of blood cultures, the diagnosis of MRSA bacteremia was made promptly with subsequent appropriate anti-microbials and source control. This case reinforces Hickman's dictum, "A patient can have as many diseases as they want," and emphasizes the importance of a broad infectious differential, even when the source seems clear.
Jamie Burke Dr. Jeremy Taylor	*Spontaneous Isolated Superior Mesenteric Artery Dissection Presenting as Chronic Abdominal Pain* Introduction: Chronic abdominal pain is a common presentation with broad differential diagnosis. Although less common, vascular etiologies should be considered in patients with risk factors when more common causes have been excluded. Spontaneous isolated superior mesenteric artery dissection (SISMAD) is characterized by dissection of the superior mesenteric artery (SMA) without trauma, iatrogenic injury, or associated aortic or celiac artery dissection. SISMAD classically presents with acute abdominal pain, often in middle-aged men, but presentations may vary. This case highlights the importance of considering SISMAD in patients with persistent unexplained abdominal pain. Case Presentation: A 44-year-old male veteran with hypertension presented with several months of acute-on-chronic left lower quadrant

	abdominal pain. Symptoms had previously prompted an emergency department visit and hospitalization. Prior evaluation included upper endoscopy, colonoscopy, broad lab work, and CT of the abdomen and pelvis, which was largely unrevealing aside from findings suggestive of colitis two months prior. On presentation, he was hemodynamically stable with focal left lower quadrant tenderness. Laboratory studies were largely unremarkable. Given persistent unexplained symptoms and concern for a vascular etiology, contrast-enhanced CTA was obtained and demonstrated a shelf-like filling defect along the posterior aspect of the main SMA trunk without flow limitation, consistent with SMA dissection. Vascular surgery and gastroenterology recommended conservative management with anticoagulation and aspirin. Repeat CTA two days later showed the dissection was "slightly less apparent or resolved." His pain improved but did not completely resolve. He was discharged on apixaban and aspirin with planned vascular surgery follow-up and repeat CTA in three months. Dicyclomine was prescribed for a possible component of irritable bowel syndrome. He subsequently followed with gastroenterology and primary care but did not attend vascular surgery follow-up. Repeat CT four months after diagnosis demonstrated complete resolution of the dissection. Discussion: SISMAD is an uncommon cause of abdominal pain that may be overlooked when symptoms are chronic or atypical. Patients are predominantly middle-aged men, with reported risk factors including hypertension, smoking, and hyperlipidemia (1,2). An increased aortomesenteric angle has also been associated with symptomatic SISMAD (3). CTA is the preferred diagnostic modality and may demonstrate an intimal flap, double-lumen appearance, or eccentric mural thrombus (1). Early recognition is important because progression can result in mesenteric ischemia, aneurysmal degeneration, or rupture. In patients without bowel ischemia or hemorrhage, conservative management with bowel rest, analgesia, and blood pressure control is appropriate in most cases. Endovascular stenting may be considered for refractory pain, dissection progression, aneurysmal degeneration, or threatened bowel, while surgery is generally reserved for complications such as bowel infarction or rupture (4). This case emphasizes maintaining a broad differential for persistent abdominal pain. When common gastrointestinal etiologies fail to explain ongoing symptoms, particularly in patients with vascular risk factors, vascular imaging should be considered. Recognition of SISMAD can prevent diagnostic delay and identify patients at risk for serious vascular complications.

Kaelan Byrd Dr. Rebecca Kummer	*Septic Shock from Recurrent Pyogenic Hepatic Abscess: When Bacteria Move into a Second-Hand Home in the Liver* Introduction: Concurrent intrahepatic abscess, choledocholithiasis, and MSSA bacteremia are a known but uncommon cause for septic shock. Shock of this etiology begins with acute obstructive pathology of the biliary tree. This leads to diffuse biliary congestion, inflammation, and translocation of extrabiliary bacteria which later develop pyogenic liver abscesses and sepsis. We present here a patient with recurrent hepatic abscess in a similar distribution within the liver from a prior abscess, which suggests scarring from the initial abscess as a risk factor for recurrent pathology. This case also highlights an occurrence of pyogenic hepatic abscess with an atypical pathogen years after a more typical infection. Case Presentation: A 75-year-old male with a history of prior liver abscess and sepsis with Streptococcus anginosus and Prevotella histicola presented with three days of confusion, weakness, and nausea with vomiting; he was admitted for septic shock. On exam, he was jaundiced and encephalopathic with a diffusely tender abdomen. Initial labs showed elevated white blood cells (25.3k/L), elevated LFTs with a cholestatic pattern (ALK 263, ALT 109, AST 151), and elevated bilirubin (total 13.8, direct 10.4). CT identified multiple hepatic abscesses throughout the liver measuring up to 9 cm and common bile duct dilation to 1.4 cm with intrahepatic ductal dilation concerning for ascending cholangitis secondary to choledocholithiasis, which was confirmed on MRCP. Review of prior CT studies show his right lobe abscess was similar in shape and quality to the current study, suggesting that a cavitary lesion or scar was present from prior infection. A percutaneous CT-guided drain was placed for the largest hepatic abscess, while ERCP was performed to treat choledocholithiasis. Blood and abscess cultures were significant for methicillin-sensitive Staphylococcus aureus (MSSA), for which he was treated antibiotics 17-days while his failure-to-thrive and multifactorial encephalopathy were managed. He improved in his clinical course and successfully transitioned to a short-term rehabilitation facility. Discussion: This case highlights an atypical case of hepatic parenchymal scarring causing susceptibility to subsequent infection. The similar lesion pattern on CT imaging suggests that a remnant cavitary lesion or other scar likely formed from prior infection, though follow-up CT study from prior infection demonstrated mostly healed hepatic parenchyma one month after the first percutaneous drain was placed. Beyond this, the etiology of recurrent infection is unclear. Most intrahepatic abscesses develop from obstructive cholestatic disease typically involving Klebsiella pneumoniae, Escherichia coli, and mostly gram-negative enteric pathogens due to proximity of the GI lumen. This patient's prior abscess-causing agents are enteric, yet now the causative agent is MSSA which is typically extraluminal and spreads hematogenously. Though we cannot determine exactly the primary site of infection on exam, CT, or patient history - we can state

	that MSSA is less able to prevail within a cavitary hepatic lesion as a facultative anaerobe. This is supported by retrospective studies showing Staphylococcus species accounting for 8% of sampled pyogenic liver abscesses. This case study may better inform the approach and treatment of recurrent hepatic abscesses from preexisting parenchymal liver scarring.
Abhishek Chandra Dr. Jesse Abelson	*Persistent Fever after CABG: Occult Streptococcus Anginosus Cervical Epidural Abscess Mimicking Post-Pericardiotomy Syndrome* Introduction: Persistent fever after coronary artery bypass grafting (CABG) is common and often attributed to inflammatory processes such as post-pericardiotomy syndrome (PPS). However, persistent fever without a clear source warrants continued reassessment for occult invasive infection. Case Presentation: We present the case of 69-year-old male patient who developed recurrent fevers shortly after CABG initially suspected to represent PPS. Despite broad-spectrum antibiotics and anti-inflammatory therapy, his fevers persisted and serial thoracoabdominal imaging failed to find an infectious source. His blood cultures eventually grew Streptococcus angionsus. He shortly after developed progressive weakness, respiratory decompensation, and sustained a pulseless electrical activity arrest followed by quadriplegia. MRI ultimately identified extensive cervical discitis and osteomyelitis with a large ventral epidural abscess extending from C2-T1 with resultant spinal cord compression and diffuse cord edema. Neurosurgical intervention was not considered viable given the extent of neurologic injury. The patient transitioned to comfort-focused care and died shortly after. Discussion: This case highlights the diagnostic complexity of persistent postoperative fever after cardiac surgery and emphasizes the importance of aggressive investigation for occult abscess in patients with bacteremia and evolving neurologic deficits.
Jenny Chen Dr. Madeline Franke Dr. Madeleine O'Keefe Dr. Aleander Niven	*Diagnostic and Therapeutic Challenges of Adult Secondary HLH in a Critically Ill Patient with Distributive Shock* Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a life-threatening hyperinflammatory syndrome associated with multiorgan failure and high mortality. Secondary HLH is often triggered by infection, malignancy, or autoimmune disorders, and a systematic approach to the diagnosis of secondary HLH is essential to guide appropriate and increasingly targeted therapy for this complex condition. Case Presentation: A 23-year-old woman with trisomy 21 and B-ALL s/p induction chemotherapy, with a history of obesity, hypothyroidism, and type 2 diabetes was transferred to the MICU with fever and distributive shock. Initial evaluation revealed a significantly elevated

	ferritin, hypertriglyceridemia, pancytopenia, hypofibrinogenemia, and elevated hepatic enzymes. Admission Hscore was 256. The patient was treated with targeted crystalloid resuscitation, broad spectrum antibiotics, and L-carnitine due to concerns for pegaspargase associated liver toxicity. An extensive evaluation for infection identified patchy bilateral lung infiltrates with focal right upper lobe consolidation, an atypical epidural fluid collection following intrathecal chemotherapy, positive Karius, and hidradenitis suppurativa, but BAL, aspirate, and other cultures were all negative. Bone marrow biopsy showed low-grade hemophagocytosis, and soluble IL-2 receptor alpha, IL-18, IL-6, MCP-1, and CXCL9 levels were elevated concerning for HLH. The patient received escalating doses of corticosteroids, tocilizumab, anakinra, and IVIG. Despite initial improvement in her inflammatory parameters with treatment, she developed abrupt vasopressor refractory shock concerning for massive pulmonary embolism and died. Discussion/Conclusion: HLH is driven by dysregulated T cell and NK cell activity leading to excessive cytokine production and subsequent multiorgan failure. Diagnosis relies on clinical and laboratory markers. The HLH 2004 criteria is commonly used, which requires meeting at least 5 of 8 criteria for diagnosis: fever, splenomegaly, cytopenia affecting >2 cell lines, hypertriglyceridemia or hypofibrinogenemia, hemophagocytosis, hyperferritinemia, low or absent NK cell activity, and elevated sCD25 levels. The HScore is another validated tool used to estimate the probability of HLH with high sensitivity and specificity. Cytokine panels help differentiate the etiology of HLH and may guide treatment options. This case highlights the diagnostic challenges differentiating HLH from other inflammatory states like sepsis and chemotherapy related toxicity, as many of the criteria she met could also be explained by her chemotherapy exposures. HLH should be considered in unexplained or uncontrolled inflammation (fever, cytopenia, hyperferritinemia, hepatosplenomegaly, or coagulopathy) in critically ill patients, especially in immunocompromised patients or underlying malignancy unresponsive to targeted resuscitation and antimicrobial therapy. HLH testing, in addition to systematically evaluating possible triggers and excluding untreated infection, is essential to guide HLH treatment. Novel biomarkers such as CXCL9 and T cell activation profiles offer promising diagnostic utility. Treatment options for HLH should include treating the underlying trigger; additional treatments depend on disease severity, CXCL9 levels, and response to initial therapy. Traditionally corticosteroids and etoposide have been first-line treatment, but emerging cytokine-directed therapy with anakinra, emapalumab, and ruxolitinib offers an individualized approach that targets affected inflammatory pathways. Increased awareness of HLH and its mimics, use of a systematic approach to diagnosis, and new therapeutic approaches offer opportunities to improve the prognosis of HLH in adults.

Marie-Lise Chrysostome Dr. Samantha Vuong Dr. Samuel Garcia	*Methadone and Ozempic-Induced Emesis - A Potentially Arrhythmogenic Combination* Introduction: As GLP-1 receptor agonists become more ubiquitous, complex side effect profiles arise alongside them. These medications do not generally increase the risk of cardiac arrhythmias, however prolonged emesis combined with poor oral intake may promote arrhythmogenic conditions, especially in those who are already at increased risk. Case Presentation: We present the case of a 49-year-old female with a history of type 2 diabetes mellitus and chronic pain who was brought into the emergency room after experiencing out-of-hospital cardiac arrest. Bystander CPR was performed before EMS arrived and continued resuscitation attempts. The AED detected polymorphic ventricular tachycardia and ROSC was achieved after 1 shock. On arrival to the hospital she was noted to be hypokalemic with a potassium of 2.9 mmol/L, and lactate of 8.05 mmol/L. ECG demonstrated a QTc of 569. History obtained from the patient and family revealed that she had been on methadone for approximately 20 years for chronic pain after a motor vehicle accident. Over time her prescription was uptitrated to 320 mg of methadone daily. She was also started on semaglutide four months prior to presentation and had been uptitrated to 2mg weekly. Since starting the medication she experienced worsening emesis with episodes up to three times a day prior to admission. TTE, cardiac MRI, and coronary angiography did not reveal a clear ischemic or structural etiology of cardiac arrest. As workup pointed away from an intrinsic cardiac cause, it was deemed that the cardiac arrest likely resulted from a combination of QTc prolongation in the setting of high-dose methadone, and electrolyte derangements secondary to semaglutide side effects. Given the possibility of recurrence of life-threatening arrhythmia, an implantable cardioverter-defibrillator was placed, methadone was immediately discontinued, and an oxycodone pain regimen was initiated. She was discharged home with close follow-up with pain medicine and cardiology. Discussion: Several commonly prescribed medications are associated with QT-prolongation. GLP-1 receptor agonists are becoming increasingly ubiquitous and emesis remains one of the most common side effects. In light of these facts, we must remember that while GLP-1 receptor agonists themselves are not arrhythmogenic, a known side effect of these medications can promote electrolyte derangements thus increasing the risk of life-threatening arrhythmias. This is especially relevant to patient education upon initiation of treatment. The case we present was further complicated by the complex symptomology involved in sudden discontinuation of methadone. As there is no established protocol for rapid discontinuation, addressing pain, hyperalgesia, and physical dependence posed a challenge for the patient in both the inpatient and outpatient settings. In order for patients to make informed decisions, it is important that they are aware

	of the possible downstream complications of GLP-1 receptor agonists, and how their unique comorbidity profile may be impacted.
Alexis Clark	*When the Effusion Follows the Calendar: A Case of Thoracic Endometriosis* Introduction: Thoracic endometriosis (TES) is the most common extrapelvic manifestation of endometriosis, typically presenting with catamenial right-sided chest pain, hemothorax, or hemoptysis. Nonspecific imaging findings and effusion resolution between menstrual cycles often results in TES symptoms being attributed to infectious or malignant etiologies, delaying appropriate treatment. This case highlights a woman from a tuberculosis-endemic region whose recurrent effusions were treated empirically as pleural tuberculosis before her monthly pattern of recurrence revealed the true diagnosis. Case Presentation: A 41-year-old woman from Ivory Coast with a history of anemia, uterine fibroids, and menorrhagia presented to clinic in 2021 with right-sided chest pain since 2018. Recurrent right pleural effusions were documented on serial imaging from 2020-2021, often fluctuating in size. Given her origin in a TB-endemic area, pleural tuberculosis was favored despite three negative AFB cultures and a negative interferon-gamma release assay; thoracentesis additionally confirmed an exudative effusion with negative cultures. After discussing treatment options, she elected to proceed with empiric anti-tuberculous therapy. Despite this intervention, symptoms persisted. Repeat thoracenteses yielded grossly bloody fluid. She reported right upper quadrant and shoulder pain occurring immediately before menses consisting of heavy periods and significant abdominal pain. In her history, the only recent period of time where she was free of symptoms occurred while on hormonal menstrual suppression. Ultimately, her catamenial bloody effusions, in association with her menses, led to a clinical diagnosis of TES. A GnRH agonist (leuprolide) was started but adherence was limited by frequent international travel. She subsequently developed a small apical pneumothorax and a persistent effusion with concern for trapped lung requiring further intervention. In 2024, she underwent a VATS with talc pleurodesis, experiencing good initial response; however, follow up imaging demonstrated new pleural nodular densities. Her clinical team decided sole operative intervention was unlikely to give her complete remission and leuprolide was resumed. Imaging following resumption showed great improvement, as expected in TES. On continued therapy, she has remained minimally symptomatic with a improved chest radiography to current date. Discussion: This case illustrates the diagnostic hazard of anchoring bias. Although this patient was from an endemic region, in continuing to pursue treatment of pleural tuberculosis, the patient's complete picture including her monthly pattern of recurrence was overlooked. TES is diagnosed clinically based on the timing of thoracic symptoms during menstruation. MRI is the most sensitive radiographic modality

	for diagnosis, revealing hemorrhagic pleural and diaphragmatic implants, as most other imaging will demonstrate nonspecific findings. Histopathology from thoracoscopic biopsy, however, remains the gold standard of diagnosis. The correct diagnosis for this patient was ultimately based on her recurrent effusions, timing of symptoms and MRI-confirmed coexisting pelvic endometriosis. Although this patient lacked the critical finding of endometrial glands on biopsy, hemosiderin-laden macrophages with pleural fibrosis - which was confirmed in this patient - are typical supportive findings. Optimal management is multidisciplinary; surgical treatment, with pleural resection vs pleurodesis, combined with postoperative hormonal therapy offers the lowest rate of recurrence. Fertility goals and treatment adherence can critically shape therapeutic plans and likelihood of recurrence, as seen in this patient.
Karen Dale Dr. John Park	*A Severe Cutaneous Reaction Following Trimethoprim-Sulfamethoxazole Exposure: A Case Report* Case Presentation: A 68-year-old male with past medical history of benign prostatic hyperplasia complicated by urinary retention requiring frequent intermittent catheterizations presented to an outside emergency department for symptoms of a urinary tract infection. He was prescribed trimethoprim-sulfamethoxazole (Bactrim). Within an hour of receiving this medication, he developed a headache, new oxygen requirements, and hypotension. His physical exam was notable for new bilateral conjunctivitis and a diffuse, confluent erythematous rash. Labs were notable for new onset lymphopenia. His drug reaction was attributable to a rare hypersensitivity reaction known as SCoRCH (sudden conjunctivitis, lymphopenia. and rash combined with hemodynamic changes). While this reaction has been documented in rare case series, this case is novel in that there were documented progressive clinical manifestations across multiple exposures to TMP-SMX, rapid clinical improvement following drug discontinuation despite his concurrent infectious process, and concurrent bacterial Klebsiella aerogenes urinary tract infections during his recurrent episodes. Finally, while this patient was hypotensive, he remained largely fluid responsive and did not demonstrate the profound hemodynamic instability described in previous reported cases.
Connie Dale	*Small Lungs, Big Clue: Shrinking Lung Syndrome as the First Presentation of SLE* Introduction: Shrinking lung syndrome (SLS) is a rare pulmonary manifestation of systemic lupus erythematosus (SLE), characterized by dyspnea, pleuritic pain, and restrictive physiology with small lung volumes and elevated hemidiaphragms, without parenchymal or pulmonary vascular disease. It usually arises in established SLE and is rarely the presenting feature, which can delay diagnosis. We describe SLS as the initial manifestation of undiagnosed SLE.

	Case Presentation: A 40-year-old woman with a recent admission for hypoxic respiratory failure, and no known rheumatologic disease, presented with acute respiratory distress: Her previous admission was thought to be due to CAP, given improvement with antibiotics and steroids. She presented with tachypnea, hypoxia, and severe exercise intolerance. She had a newly developing malar rash, pleuritic pain, and a steady respiratory rate of 34 bpm. Her VBG was remarkable for a pH of 7.4, with a pCO2 of 35. Infectious workup was negative. Chest radiograph showed small lung volumes, bilateral elevated hemidiaphragms, and small bilateral effusions. CT chest showed prominent anterior mediastinal/axillary lymph nodes without ILD or PE. Pulmonary function testing could not be completed due to the patient's tolerance. Bedside ultrasound showed markedly reduced bilateral diaphragmatic excursion, about 1 cm at maximum inspiration. Serologies showed markedly elevated ANA >1:2560 and anti-dsDNA 1:1280. Additionally, she had an increased SSA 52 and SSA 60, with a low C4. Diagnosis: She was diagnosed with SLS in the setting of newly diagnosed SLE, meeting 2019 EULAR/ACR classification criteria (ANA entry criterion plus 20 points: anti-dsDNA/anti-Sm 6, acute cutaneous lupus 6, pleural effusion 5, low C4 3). Because PFTs were not obtainable, SLS was diagnosed on clinical, radiographic, and sonographic findings (small lung volumes, elevated hemidiaphragms, and markedly reduced diaphragmatic excursion) with a negative infectious workup and exclusion of ILD, pulmonary embolism, and cardiac causes. Management and Outcome: She responded well to inpatient intravenous methylprednisolone and was discharged on a steroid taper, hydroxychloroquine, and long-term azathioprine. At pulmonary clinic follow-up 4 months later, diaphragmatic excursion had improved to 3.3 cm, and she could walk up to six blocks, compared with less than one block at presentation. Discussion: SLS is uncommon, reported in about 1% or fewer of patients with SLE, and its pathogenesis remains uncertain. Proposed mechanisms include diaphragmatic myopathy or dysfunction, phrenic neuropathy, pleural inflammation and reduced surfactant or chest wall compliance. This case is unusual because SLS was the first manifestation of SLE, following a presumed CAP respiratory failure episode, and was diagnosed without PFTs, using bedside diaphragm ultrasound, which also allowed objective tracking of recovery. Treatment evidence is limited to case series and reports, but response to immunosuppression is often favorable, as it was here. Take-Home Points: Consider SLS in unexplained dyspnea with small lung volumes and elevated diaphragms. When PFTs are unfeasible, diaphragm ultrasound is a useful alternative. Screen for connective

	tissue disease even without a prior rheumatologic diagnosis. Early immunosuppression may improve diaphragm function and symptoms.
Martin Davis Dr. Agata Sularz Dr. Malcolm Bell	*DOACs without Borders – A Challenging Anticoagulation Decision Impacted by International Travel* Background: Coronary artery embolism is an under-recognized cause of acute myocardial infarction, accounting for 3% of cases. [1] In a young patient without atherosclerotic risk factors, an embolic mechanism should prompt a search for a cardioembolic source and, if possible, a specific driver of hypercoagulability which can direct treatment. Acute treatment is usually initiated while the cause is still being explored, making close follow-up crucial. This case exemplifies a test pending at discharge (TPAD) that might have altered a patient's care. Case presentation: A 40-year-old woman visiting from Europe presented after an episode of syncope with chest pain, palpitations, and emesis while in Minnesota visiting family. Her ECG was ischemic and high sensitivity troponins were elevated and dynamic. CT cardiac angiography demonstrated a perfusion defect and inferolateral hypokinesis. A diagnosis of acute myocardial infarction (MI) was made. Invasive coronary angiography revealed occlusion of the distal posterior descending and posterolateral branches, consistent with an embolic etiology. Transesophageal echocardiography showed a tiny patent foramen ovale with no intracardiac embolic source. CT angiography of the abdomen/pelvis revealed a prior splenic infarct, consistent with remote systemic embolization. A broad workup initially found elevated ESR and CRP, ANA 1:160, and anti–SS-A/Ro positivity. Lupus anticoagulant and anti-cardiolipin antibodies were negative. She reported one prior miscarriage. Facing imminent travel back to Europe and concern about heparin bridging with warfarin and transition of care, she was discharged on a direct oral anticoagulant (DOAC) for presumed hypercoagulability. Following discharge her anti–β2-glycoprotein I antibody returned positive, raising the possibility of antiphospholipid syndrome (APLS) and, with it, a reason to prefer warfarin. Updated results and recommendations for follow-up testing were provided to the patient via electronic portal. Discussion: Research shows 30% to 40% of TPADs reveal actionable results, physicians are often unaware, and immunologic serologies are among the slowest. [2] This case is an illustration of a potentially actionable TPAD. However, single anti–β2-glycoprotein I positivity drawn during an acute thrombosis does not establish APLS, as antiphospholipid antibodies are frequently transient during acute illness and inflammation. Classification requires persistence on repeat

	testing ≥12 weeks apart. Regardless, suspicion for APLS favors warfarin over a DOAC for arterial thrombosis. Conclusions: When an embolic mechanism for acute MI is suspected, particularly in young patients, a search for an intracardiac source and signs of other systemic embolization and complete thrombophilia testing in these patients is required. Treat thrombophilia/autoimmune serologies as high-consequence TPADs requiring closed-loop follow-up, especially across health systems and borders. 1. Raphael CE, Heit JA, Reeder GS, Bois MC, Maleszewski JJ, Tilbury RT, Holmes DR Jr. Coronary Embolus: An Underappreciated Cause of Acute Coronary Syndromes. JACC Cardiovasc Interv. 2018 Jan 22;11(2):172-180. doi: 10.1016/j.jcin.2017.08.057. PMID: 29348012. 2. Darragh PJ, Bodley T, Orchanian-Cheff A, Shojania KG, Kwan JL, Cram P. A Systematic Review of Interventions to Follow-Up Test Results Pending at Discharge. J Gen Intern Med. 2018 May;33(5):750-758. doi: 10.1007/s11606-017-4290-9. Epub 2018 Jan 19. PMID: 29352419; PMCID: PMC5910344.
Alexandra Dobos Dr. Khushboo Gala	*Refractory Reflux with Recalcitrant Esophageal Ulceration: The Importance of Reassessment* Introduction: Gastroesophageal reflux disease (GERD) affects approximately 1/3rd of adults in the United States. Management varies from lifestyle changes, antisecretory therapy including proton pump inhibitor therapy (PPI), histamine H2 receptor antagonists (H2RAs) and now with emerging potassium-competitive acid blockers (P-CABs), as well as endoscopic and surgical therapy. Refractory GERD is generally defined as evidence of pathologic reflux while on optimal antisecretory therapy; and can be both a diagnostic and therapeutic challenge. Recalcitrant gastrointestinal (GI) ulceration may be in the setting of severe or refractory GERD; but rarely, may be due to underlying factors like foreign bodies, ischemia or chronic tobacco use. We present a case of recurrent esophageal ulceration and gastrointestinal bleeding refractory to maximal medical therapy, ultimately attributed to an embedded metal suture fastener. Case Description: A 63-year-old male with a longstanding history of refractory GERD, 5 cm hiatal hernia, Barrett's esophagus with low-grade dysplasia presented for multiple admissions with melena. During his initial presentation, esophagogastroduodenoscopy (EGD) demonstrated esophagitis with ulceration, at which point he was started on high dose PPI, and repeat EGD revealed healing of this ulcer. Given persistent GERD symptoms and hiatal hernia, he underwent a Dor fundoplication. Despite this, his GERD symptoms did not resolve, and he was restarted on high dose PPI, later augmented with an H2RA and then switched to a P-CAB. Despite multiple measures at escalating

	therapy, he had persistent GERD symptoms and multiple admissions for recurrent melena and anemia. Repeat EGDs now showed a cratered, large esophageal ulcer with esophagitis. Biopsy of the ulcer was unrevealing. CT showed a titanium TI-KNOT suture fastener at the presumed location of the ulcer, placed during the fundoplication. He underwent an endoscopic ultrasound (EUS), which revealed a metal TI-KNOT suture embedded adjacent to the site of his nonhealing esophageal ulcer. After multidisciplinary discussion with gastroenterology and general surgery, it was determined that the non-healing esophageal ulcer may be related to suture material from his fundoplication. He has now been referred for revision of surgery. Discussion: Recalcitrant and non-healing ulceration in the GI tract can often present a diagnostic dilemma. Along with evaluating and medically escalating therapy for GERD, evaluating for secondary causes and risk factors of ulceration is also important. This case demonstrates the importance of reassessing the diagnosis when GI ulceration persists despite maximal medical therapy, and the role of multidisciplinary reassessment.
Jacob Fuchs Dr. Amy Holbrook	*A Complement Conundrum: From Creatinine Crisis to Renal Recovery* Introduction: Atypical Hemolytic Uremic Syndrome (HUS) is a rare but serious condition identified by the hallmarks of acute kidney injury (AKI), thrombocytopenia, and microangiopathic hemolytic anemia. It has significant overlap with Thrombotic Thrombocytopenic Purpura (TTP) and typical HUS - typically induced by Shiga toxin-producing E. coli. However, atypical HUS is caused by a rare genetic disorder in the alternative compliment pathway, which poses a unique diagnostic challenge. Case Presentation: A 28-year-old female presented to urgent care after several days of nausea and vomiting. She was found to have severe AKI (creatinine 7.25), anemia (hemoglobin 6.7) and thrombocytopenia (platelets 16) which prompted admission to the hospital. During admission, she had laboratory findings consistent with microangiopathic hemolytic anemia and the top differential considerations were HUS and TTP. Patient received a blood transfusion and started on plasma exchange later that same day for presumed TTP. With the history of nausea and vomiting, some concern was raised for typical HUS induced by Shiga toxin-producing E. coli (STEC), though this testing was negative. On further discussion and history taking, it was discovered that the patient had a family history of atypical HUS in a sister. ADAMTS-13 testing returned negative 4 days after admission ruling out TTP, and atypical HUS was the primary consideration. The patient was empirically started on eculizumab infusions, and plasma exchange was discontinued. Despite this treatment, azotemia worsened with creatinine 9.3 and BUN 128 and patient began to have symptoms of uremia including altered mental status and was started on hemodialysis. Patient stabilized on hemodialysis and was discharged from the hospital on weekly

	eculizumab infusions and planned hemodialysis as an outpatient. The initial atypical HUS panel was unrevealing. Patient underwent specialized genetic testing which identified a mutation in the complement factor H gene, attributed to the cause of her atypical HUS. Her renal function recovered and remains off dialysis on eculizumab infusions. Discussion: This case illustrates the diagnostic challenges of diagnosing atypical HUS and the value of a complete history. While the potential for TTP requires prompt treatment before a diagnosis can be made, alternate etiologies must be considered when diagnostic testing returns ruling out TTP and typical HUS. Treatment for atypical HUS requires the compliment-inhibiting monoclonal antibody eculizumab, which differs significantly from supportive care only for typical HUS. A high level of suspicion for atypical HUS must be maintained in such cases to prevent long-term kidney injury that could otherwise be reversed with appropriate treatment.
Ahmed Gad Dr. Christopher Behrend Dr. Abdelrahman Abrahim Dr. Ahmad Malli	*A Dangerous Masquerade: Colonic Histoplasmosis in the Setting of Anti-TNF Therapy* Introduction: Gastrointestinal histoplasmosis is an uncommon manifestation of Histoplasma capsulatum infection that can closely mimic inflammatory bowel disease. Distinguishing these entities is particularly important in patients receiving immunosuppressive therapy, in whom escalating treatment for a presumed inflammatory flare may worsen an unrecognized fungal infection. Case Presentation: A 31-year-old man with ankylosing spondylitis and psoriasis treated with infliximab, recent corticosteroid exposure, unexplained granulomatous hepatitis, and suspected Crohn's disease presented with fever, worsening right upper-quadrant abdominal pain, and an acute increase in chronic diarrhea. His C-reactive protein was 162 mg/L. Abdominal computed tomography demonstrated multifocal discontinuous ileal thickening and hyperenhancement with mild upstream bowel dilation and reactive mesenteric lymphadenopathy, raising concern for stricturing Crohn’s disease. Stool polymerase chain reaction detected norovirus and enteropathogenic Escherichia coli, further complicating the clinical picture. Colonoscopy revealed a normal terminal ileum and otherwise normal colon except for two large, cratered, broad-based ulcers measuring 4–6 cm at the hepatic flexure and distal transverse colon—an appearance considered highly atypical for Crohn’s disease. Biopsies demonstrated ulceration, non-necrotizing granulomas, and numerous uniform, small, intracellular yeasts with narrow-based budding on silver staining, morphologically consistent with Histoplasma. Cytomegalovirus and herpes simplex virus immunostains were negative. Although urine Histoplasma antigen was undetectable, the serum Histoplasma complement-fixation titer was elevated at 1:32, and colonic tissue culture subsequently grew H. capsulatum, confirming the diagnosis. Infliximab and corticosteroids were withheld, and itraconazole was initiated. Within 13 days, fever and chills had resolved, diarrhea had decreased from

	daily to sporadic episodes, and the abdominal pain had markedly improved. Discussion: This case illustrates the substantial clinical, radiographic, and histologic overlap between gastrointestinal histoplasmosis and Crohn's disease. The isolated distribution and unusual morphology of the colonic ulcers prompted broader tissue sampling and prevented further escalation of immunosuppression. The negative urine antigen initially argued against systemic histoplasmosis but did not exclude tissue-limited gastrointestinal disease. Concurrent norovirus and enteropathogenic E. coli detection may have contributed to the acute worsening but could not explain the chronic symptoms, granulomatous ulcers, intracellular yeast, and positive tissue culture. Learning Points: Gastrointestinal histoplasmosis should be considered in immunosuppressed patients with presumed inflammatory bowel disease when the endoscopic appearance is atypical or the response to therapy is incomplete. A negative urine Histoplasma antigen does not exclude localized gastrointestinal infection. Histopathologic examination and dedicated fungal culture remain essential before intensifying immunosuppression in diagnostically uncertain cases.
Issac Georgy	*Invasive Hypervirulent Klebsiella pneumoniae Infection with Multifocal Metastatic Abscesses, Venous Thrombosis, and Spinal Subdural Empyema* Background: Hypervirulent Klebsiella pneumoniae (hvKp) is an emerging pathogen distinguished from classical Klebsiella pneumoniae by its propensity for invasive and metastatic infection in younger, often immunocompetent hosts. The characteristic syndrome involves pyogenic abscess formation with hematogenous seeding to distant sites, including the central nervous system. Spinal subdural empyema is a rare manifestation of hvKp infection and represents a potentially devastating complication of disseminated disease. Case Presentation: We present a 26-year-old man with a history of traumatic brain injury, status post left-sided craniectomy in May 2026 with subsequent bone flap replacement in June 2026, who presented in August 2026 after being found altered with concern for seizure activity. On admission, he was tachycardic to the 130s and tachypneic to the 30s, with marked leukocytosis (WBC 53.08 K/μL) and elevated lactate (4.4 mmol/L). He was started on broad spectrum antibiotics, levetiracetam, and pressors. EEG showed no seizure activity and initial neuroimaging was unrevealing. However, CT of the abdomen and pelvis demonstrated hepatic and prostatic abscesses with portal and mesenteric venous thrombosis. Lumbar puncture yielded cloudy cerebrospinal fluid with gram-negative bacilli, with cultures growing Klebsiella pneumoniae. Culture of the prostatic abscess also grew Klebsiella pneumoniae. His course was complicated by development of fixed, dilated pupils, with repeat neuroimaging demonstrating cerebral edema requiring external ventricular drain placement. On hospital day 6, he developed recurrent tachycardia and worsening

	fevers. MRI of the spine revealed a thoracic spinal subdural empyema, prompting laminectomy and surgical drainage with placement of a thoracic spinal catheter and hemovac drain. Microbiologic findings and the pattern of multifocal infection were consistent with hypervirulent Klebsiella pneumoniae causing disseminated invasive infection with metastatic seeding. Following surgical source control and targeted antimicrobial therapy with Ceftriaxone 2 gram bid, his mental status significantly improved, and the external ventricular drain was removed. Discussion: This case illustrates the aggressive metastatic potential of hypervirulent Klebsiella pneumoniae (hvKp), with concurrent hepatic and prostatic abscesses, portal and mesenteric venous thrombosis, and a rare thoracic spinal subdural empyema requiring surgical decompression. Unlike classical K. pneumoniae, hvKp is associated with community-acquired invasive disease and hematogenous dissemination, often involving multiple distant sites. Central nervous system involvement is uncommon but can result in severe neurologic morbidity. In this patient, the recent craniectomy and bone-flap replacement may have represented a potential risk factor for CNS involvement, although a direct causal relationship cannot be established. The case underscores the importance of early recognition of disseminated infection, prompt cross-sectional imaging when the clinical course is atypical, and aggressive source control. Successful management required coordinated multidisciplinary care, including external ventricular drainage, surgical drainage of the spinal empyema, and targeted antimicrobial therapy. Conclusion: Hypervirulent Klebsiella pneumoniae can present with widespread metastatic infection and unexpected CNS complications. The presence of multiple abscesses should prompt a search for other sites of dissemination, particularly when new neurologic findings develop. Early recognition and aggressive source control, combined with targeted antimicrobial therapy, are essential in these rapidly progressive infections.
Hiam Ghunaim Dr. Hesham Haj Ebrahimi	*A Difficult Diagnosis and an Unexpected Response: Metastatic Lung Adenocarcinoma* Case Presentation: A 58-year-old woman with tobacco use, COPD/asthma, and hypertension presented to the ED three times in June for progressive chest, arm, and back pain. An April ED visit for back pain after a motor vehicle collision showed no acute spinal abnormality on CT. On 6/2, she presented with severe pain, cough, rhinorrhea, and dyspnea. CXR showed right lung interstitial opacities, and she was treated for presumed CAP. She returned 6/8 with worsening pain; repeat CXR was unrevealing, and reproducible chest-wall tenderness favored musculoskeletal pain. On 6/22, she presented again with chest pain, dyspnea, back and arm pain, weight loss, and night sweats, with symptoms markedly worse

	over two weeks, and was admitted. Echocardiography revealed a large pericardial effusion with right atrial and ventricular diastolic collapse. Despite initially preserved hemodynamics, worsening hypoxia prompted pericardiocentesis, yielding 500 mL of bloody fluid. Cytology confirmed lung adenocarcinoma, establishing stage IV disease. Molecular testing identified EGFR G719A. CT demonstrated thoracic and abdominal adenopathy with pulmonary infiltrates, while a bloody exudative pleural effusion required thoracentesis. Her respiratory status deteriorated with extensive mediastinal and peribronchial tumor, severe bronchial stenoses, lymphangitic carcinomatosis, and complete RML/RLL collapse. She developed acute-on-chronic hypoxemic respiratory failure requiring continuous BiPAP. Bone scan showed no osseous metastases. She developed a hypersensitivity reaction upon initiation of paclitaxel. Afatinib was planned but never given because of insurance/formulary barriers. After discharge, she developed a pathologic C7 fracture from new C6-C7 metastatic disease and was readmitted on 8/20 for dyspnea. She received two inpatient cycles of carboplatin and nab-paclitaxel (last on 9/2), as well as palliative radiation. An enlarging malignant pleural effusion required right chest tube placement. CT on 9/10 showed decreased pleural effusions and improving bilateral GGOs, yet she remained continuously BiPAP-dependent without meaningful respiratory improvement. With ECOG 3-4 and functional decline, further chemotherapy was not recommended, and goals of care became comfort-focused, with a goal of returning home on BiPAP or CPAP. Discussion: This case illustrates how metastatic lung cancer can initially present through dyspnea and nonspecific cardiopulmonary symptoms. From an April ED visit for back pain to three June presentations for progressive pain and dyspnea, the clinical picture evolved through several plausible alternative diagnoses before hemorrhagic pericardial tamponade revealed disseminated malignancy. This highlights the importance of reassessing recurrent/progressive symptoms, particularly when accompanied by weight loss and other systemic features. The case also posed a difficult therapeutic dilemma: whether severe respiratory failure remained sufficiently reversible to justify continued cancer-directed therapy. Multiple contributors to dyspnea were addressed, including pleural effusions, pulmonary opacities, airway obstruction, and tumor burden. Despite drainage, radiation, and two cycles of chemotherapy, she remained continuously BiPAP-dependent. Follow-up imaging showed improvement in pleural effusions and GGOs without meaningful physiologic recovery, while extensive malignant airway and parenchymal disease persisted. Conclusion: This case highlights a distinction in advanced cancer: radiographic response does not necessarily translate into clinical benefit. When severe dyspnea persists despite improvement in some imaging findings, treatment decisions must integrate the patient's

	clinical trajectory, residual structural disease, physiologic reserve, and performance status rather than imaging response alone.
Samarth Goyal Dr. Cassandra Verghese Dr. Rishabh Tandon Dr. Laith Alhuneafat Dr. Pratik Velangi Dr. Ranjit John Dr. Andrea Elliott	*Elusive Origins: A Rare Case of Polymicrobial Purulent Pericarditis Post-Nissen Fundoplication* Introduction: Purulent pericarditis is rare but life-threatening and requires rapid source identification, drainage, and antimicrobial therapy. We describe polymicrobial purulent pericarditis with intrapericardial air after prior Nissen fundoplication, suggesting an occult esophagopericardial source despite negative fistula testing. Case Presentation: A 66-year-old woman with hypertension, gastroesophageal reflux disease, gastric ulcer disease, and hiatal hernia status post Nissen fundoplication one year earlier presented with episodic chest pain, dyspnea, and sinus tachycardia. She was afebrile and hemodynamically stable. Clinical Findings & Diagnostic Assessment: Initial testing showed leukocytosis to 24 × 10^9/L, C-reactive protein 621 mg/L, procalcitonin 2.83 ng/mL, mild hyponatremia, nonspecific ST-segment changes, and negative blood cultures. Computed tomography demonstrated a large loculated pericardial effusion with intrapericardial air. Transthoracic echocardiography showed a large circumferential loculated effusion without tamponade, suggesting an indolent process. Pericardiocentesis yielded 600 mL of purulent fluid with white blood cell count 14,000/µL, neutrophilic predominance, protein 5.1 g/dL, lactate dehydrogenase 1,474 U/L, and glucose <2 mg/dL. Cultures grew Streptococcus salivarius, Streptococcus parasanguinis, Rothia mucilaginosa, Clostridium butyricum, Candida glabrata, and yeast, consistent with oral and upper gastrointestinal flora. Barium swallow, esophagogastroduodenoscopy, and methylene blue challenge did not identify a fistula. Therapeutic Interventions: Empiric clindamycin, piperacillin-tazobactam, and vancomycin were narrowed to ceftriaxone, micafungin, and metronidazole for 4 to 6 weeks. Persistent symptoms prompted median sternotomy, pericardial washout, and anterior pericardiectomy with debridement of a thickened pericardial ring. Follow-up & Outcomes: Postoperatively the patient had marked improvement, particularly resolution of dyspnea. She was discharged on intravenous ceftriaxone and micafungin with oral metronidazole to complete her antibiotic course. Discussion: Polymicrobial purulent pericarditis with oral/esophageal flora after Nissen fundoplication is highly suggestive of esophagopericardial contamination, even when imaging and endoscopic evaluation are unrevealing. A spontaneously sealed fistula may explain the negative workup, while the large effusion without tamponade supports chronic accumulation. This case highlights the need to integrate microbiology with surgical history and imaging, and

	supports aggressive management with drainage, culture-directed antimicrobial therapy, and pericardiectomy when organized infection persists.
Catherine Gray Dr. Amy Holbrook	*Face the Pressure: When SVC Syndrome Unmasks Malignancy* Introduction: Superior vena cava (SVC) syndrome is most often caused by malignancy, and in 60% of cases it is the presenting symptom of a not yet diagnosed cancer [1]. While lung cancer is the most common malignant cause, primary mediastinal large B-cell lymphoma (PMBCL) is a fast-growing, highly aggressive non-Hodgkin's lymphoma subtype that can also present with SVC syndrome. Early recognition of SVC syndrome, pursuing appropriate workup, and coordinating timely treatment can significantly impact morbidity and mortality. Case Presentation: A 43-year-old woman presented to the emergency department with several days of facial swelling, shortness of breath, and fatigue. Initial CBC and BMP were unremarkable, and she was discharged home with a course of oral prednisone. She presented again to the ED one month later with progressive symptoms. Additional workup at that time included elevated inflammatory markers with an ESR of 53 and CRP of 26. CT of the chest revealed a large superior mediastinal mass occupying the majority of the upper mediastinum with near complete obstruction of the SVC and left innominate vein, scattered abutment and partial encasement of the aortic arch, and mild narrowing of the right main pulmonary artery. Given these imaging findings and her worsening symptoms, she was admitted for further management. CT-guided biopsy of the mediastinal mass confirmed the new diagnosis of primary mediastinal large B-cell lymphoma (PMBCL). Oncology was consulted and recommended treatment for both the lymphoma and SVC syndrome with chemotherapy and steroids. Despite PMBCL being a rapidly proliferative cancer, they anticipated a good prognosis with an intense treatment regimen of R-EPOCH. Given the aggressiveness of the regimen, the patient remained inpatient for 5 days of the first cycle. The patient tolerated her first cycle of chemotherapy well and was discharged home with resolution of her facial swelling and plan for ongoing treatment with oncology. Discussion: This case illustrates the importance of considering mediastinal malignancy as a potential cause of facial swelling. Recognition of SVC syndrome should prompt imaging of the chest if not yet already obtained. Although lung cancer is the more common cause of malignancy-related SVC syndrome, PMBCL is another potential cause, especially in young adults [2]. [1] Friedman, T., Quericer, K.B., Kishore, S.A., Winokur, R.S., & Madoff, D.C. Malignant venous obstruction: Superior vena cava syndrome and beyond. Seminars in Interventional Radiology. 2017; 34(4): 398-408.

	[2] Besien, K.V., Kelta, M., & Bahaguna, P. Primary mediastinal B-cell lymphoma: A review of pathology and management. Journal of Clinical Oncology. 2001; 19(6): 1855-1864.
William Guns	*When Thrombosis Precedes the Overt Phenotype: JAK2-Positive Myeloproliferative Neoplasm with Noncirrhotic Portal Hypertension* Introduction: Myeloproliferative neoplasms (MPNs) are the most common underlying systemic prothrombotic disorder associated with noncirrhotic, nonmalignant splanchnic vein thrombosis (SVT). Thrombotic complications may precede an overt hematologic phenotype, creating a diagnostic challenge, particularly in younger patients without erythrocytosis, thrombocytosis, leukocytosis, or definitive bone marrow findings. Chronic SVT may also cause noncirrhotic portal hypertension and gastroesophageal varices, making gastrointestinal complications an important component of management. Case Presentation: A 35-year-old man with no significant PMH presented with lower abdominal/epigastric pain and nausea. Hemoglobin was 16.6 g/dL, hematocrit 50.5%, and leukocyte and platelet counts were normal. CT A/P showed extensive thrombosis of the main and intrahepatic portal veins, splenic vein, and distal superior mesenteric vein, with splenomegaly. Thrombophilia evaluation while inpatient for initial presentation was notable for low protein S, and apixaban was started. Despite extensive SVT and erythrocytosis, hematologic evaluation was not initially pursued. Follow-up imaging after 3 months of treatment showed cavernous transformation of the portal vein, consistent with chronic thrombosis. Sixteen months after presentation, he was evaluated by hematology-oncology. Work-up revealed low-normal erythropoietin and positive JAK2 V617F testing (VAF 23%). Bone marrow biopsy showed trilineage hematopoiesis with mildly increased pleomorphic megakaryocytes. GI was consulted. EGD demonstrated grade III esophageal varices requiring banding, grade I gastric varices, and portal hypertensive gastropathy. FibroScan showed no cirrhosis. He was started on phlebotomies to keep hematocrit <45% and Ropeginterferon alfa-2b for further cytoreduction given high-risk disease. Apixaban was maintained at prophylactic dose (2.5mg BID) without aspirin given high bleeding risk. Discussion: This case highlights an important diagnostic lesson: SVT may be the first manifestation of an underlying MPN even when the hematologic phenotype is subtle. Consensus recommendations support JAK2 V617F testing in adults with primary SVT regardless of peripheral blood counts. If negative, CALR, MPL, and JAK2-exon 12 mutations with broader next generation sequencing should be considered when suspicion for a MPN remains high. In our patient, extensive SVT, erythrocytosis, and splenomegaly were present at initial presentation, yet hematologic evaluation was delayed more than

	a year. The presentation along with the presence of a JAK2 V617F mutation make polycythemia vera the most likely diagnosis. Earlier JAK2 testing may have accelerated diagnosis and disease-directed therapy. Gastrointestinal consequences are also significant in MPN-associated SVT. In a cohort of 519 MPN-SVT cases, 67% had or developed gastroesophageal varices and 44.5% experienced bleeding. These complications can occur in MPN-associated SVT even without cirrhosis, as demonstrated by our patient. This highlights gastroenterology's role in longitudinal management of MPN-associated SVT, particularly when thrombosis becomes chronic. Guidelines support endoscopic screening when SVT fails to recanalize, ongoing surveillance, and long-term anticoagulation in patients with persistent prothrombotic conditions such as MPN. Conclusion: MPN should be on the differential diagnosis in patients presenting with unexplained SVT, even when peripheral blood findings are not suggestive of this diagnosis. Molecular testing, particularly JAK2 V617F, should be performed in patients presenting with SVT. Close collaboration between hematology and gastroenterology is important, as recognition and treatment of the underlying MPN must be coordinated with management of gastrointestinal complications.
Lian Hagage	*Rebound-Associated Multiple Vertebral Fractures Following Denosumab (Xgeva) Discontinuation in a Patient with Bone-Metastatic Breast Cancer* Introduction: Denosumab is a fully reversible RANKL inhibitor; unlike bisphosphonates, its discontinuation triggers a rebound in bone turnover. This is well described after osteoporosis-dose (Prolia) discontinuation, but evidence at oncology dosing (Xgeva) is scarce, and guidelines for oncology-dose bone metastases do not include transition strategies Case Presentation: A 71-year-old woman with a history of ER/PR-positive, HER2-negative metastatic breast cancer (bone metastases since 2019) received denosumab 120 mg from November 2020, initially every 3 months and later every 6 months, until November 2024, when it was discontinued after 2 years of stable disease without a plan for antiresorptive bridging therapy. Approximately 13 months later, she developed acute back pain after shoveling snow in December 2025. Over the next 10 weeks, serial imaging revealed four new vertebral compression fractures (L1, T11, T9, L4) in addition to chronic T5–T7 deformities. DEXA scan in January 2026 showed a 14.5% decline in lumbar spine bone mineral density from her last on-treatment scan. Labs showed elevated CTX (400 pg/mL) and PTH (143 pg/mL) with normal calcium and renal function, arguing against primary hyperparathyroidism and myeloma; PET-CT confirmed stable oncologic disease without progression. She was diagnosed with

	rebound-associated osteoporosis with multiple vertebral fractures (MVF) and treated with kyphoplasty at all four levels and IV zoledronic acid with CTX-guided monitoring. CTX remained elevated after one dose, and a second is planned. Discussion: This rebound phenomenon is well established and recognized in the osteoporosis and endocrinology guidelines, but it remains overlooked in oncology. In the FREEDOM Extension post hoc analysis, 60.7% of denosumab-treated patients with an off-treatment vertebral fracture had multiple fractures, compared with 38.7% on placebo. Risk increased further with longer exposure: patients treated with denosumab for more than 3 years had a multiple vertebral fracture rate of 7.5 per 100 patient-years, versus 2.9 for those treated 3 years or less. Our patient had both risk factors, a prior vertebral fracture and over 3 years of continuous denosumab therapy, and received no bridging antiresorptive at discontinuation. Oncology-specific outcome data remain limited to one retrospective cohort study and a small number of case reports. This scarcity may partly reflect survival in this patient population: rebound-associated fractures typically occur 7 to 20 months after the last dose, and patients with bone-metastatic disease may not survive long enough, or be followed closely enough, for this complication to be captured. In the absence of oncology-specific data, clinicians often extrapolate transition-off strategies from osteoporosis guidelines. Both NCCN and ASCO guidelines clearly address denosumab initiation for skeletal-related event prevention in solid-tumor bone metastases, but neither offers guidance on transitioning off therapy. As emerging cancer therapies extend survival, more patients with metastatic bone disease are living long enough for this rebound phenomenon to manifest as clinically significant complications such as multiple vertebral fractures.
Wyatt Hahn Dr. Leeore Levinstein	*One Tooth, One Thumb, One Limb: Purpura Fulminans from Capnocytophaga Canimorsus* Introduction: Capnocytophaga canimorsus (C. canimorsus) is a slow-growing, Gram-negative rod and commensal organism of the canine and feline mouth that can have catastrophic consequences in humans. Although immunocompromised and asplenic hosts are at highest risk, fulminant infection also occurs in healthy individuals. This case describes a patient with alcohol use disorder who presented with fever, fatigue, and cough and was found to have severe sepsis and purpura fulminans secondary to C. canimorsus. Case Presentation: A 55-year-old male presented to a rural emergency department for 1.5 days of fatigue, fever, rigors, and non-productive cough. Medical history included hypertension, COPD, alcohol use disorder, and tobacco use disorder. He was tachycardic and tachypneic with severe lactic acidosis and acute kidney injury; white blood count was normal and procalcitonin was 19.60 ng/mL. Chest x-ray showed a subtle left base nodularity. He was admitted for severe sepsis

	secondary to left lower lobe pneumonia and started on broad-spectrum antibiotics. Shortly after admission, he developed a purpuric rash of the extremities and face, a cold left foot with Doppler-only pulses, and ongoing lactic acidosis. Given his worsening clinical status, he was transferred to a quaternary care center. Upon arrival to the ICU, his mottling and purpuric rash were diffuse with additional concern for left lower extremity ischemia. White blood count increased to 23,000, platelets decreased to 42,000, and creatinine increased to 2.36. He was started on piperacillin-tazobactam monotherapy. Concern for vasculitis as a primary driver prompted plasmapheresis and methylprednisolone. Left lower extremity CTA showed occlusion of the left superficial femoral artery, so he was subsequently started on heparin. After several days, blood cultures were growing Gram-negative rods. Upon additional questioning, the patient noted he sustained a minor cut to his left thumb from his dog's tooth the day prior to symptom onset. There was then growing suspicion for infection secondary to C. canimorsus. Autoimmune workup was largely negative. Blood cultures subsequently confirmed C. canimorsus. Rheumatology felt his bacteremia was the primary cause of his purpura fulminans, so methylprednisolone was stopped. The patient then developed worsening anemia and recurrent thrombocytopenia, so antibiotics were changed to ampicillin-sulbactam. Laboratory workup was consistent with autoimmune hemolytic anemia requiring high-dose steroids. Heparin was discontinued due to epistaxis; however, perfusion of the left foot decreased, so heparin was restarted. Angiogram, suction thrombectomy, and angioplasty of the left distal superficial femoral and popliteal arteries were performed with transition to oral anticoagulation. Despite aggressive multidisciplinary care, he ultimately required a below-knee amputation after discharge. Conclusion: This case highlights a rare cause of sepsis and purpura fulminans complicated by limb thrombosis. The slow-growing and fastidious nature of C. canimorsus emphasizes the importance of an early thorough history and physical exam for expeditious treatment. Bacterial sepsis with purpura fulminans can closely mimic systemic vasculitis, underscoring the caution needed to avoid premature immunosuppression before excluding fulminant infection.
Hashem Haj Ebrahimi Dr. Hiam Ghunaim	*Two Stroke Codes, Two Antibodies, One Culprit* Introduction: Two Stroke Codes, Two Antibodies, One Culprit Paraneoplastic encephalitis is an immune-mediated neurologic disorder associated with cancer. Its presentation with focal seizures, weakness, and neglect can make it difficult to distinguish from stroke. Case Presentation: A 66-year-old man with a history of over 25 pack-years of smoking, severe alcohol use disorder, and prior withdrawal seizures was found down at his assisted living facility on May 13,

	2026. Confusion, left-sided weakness, left gaze preference, and increased left arm tone prompted a stroke alert upon arrival in the emergency department. Stroke series showed no acute lesion or large-vessel occlusion, and initial brain MRI showed no acute infarct. A seizure was witnessed in the emergency department. Alcohol withdrawal and postictal weakness were considered as his confusion and weakness improved, but gait instability prevented discharge, and he was admitted. Following admission, he went on to develop recurrent left arm jerking, fluctuating weakness, and left visuospatial neglect. CSF testing on May 16 was positive for antineuronal nuclear antibody type 1 (ANNA-1, also known as anti-Hu) and collapsin response mediator protein 5 (CRMP-5) antibodies. Infectious testing was negative. Electroencephalography captured a right temporoparietal seizure. A May 19 brain MRI showed new, nonspecific signal changes in the right paracentral and possible occipital cortex, with inflammation, seizure-related changes, and ischemia considered. Later MRIs remained stable and showed chronic infarcts without convincing brain metastases. Chest CT identified a 3.5 cm right upper lobe mass, enlarged from 1.6 cm five months earlier, with two additional new right lung nodules. A May 28 core biopsy confirmed small-cell lung carcinoma. Seizure treatment required levetiracetam, lacosamide, and clobazam. He received high-dose methylprednisolone and intravenous immunoglobulin for paraneoplastic encephalitis. Alertness improved, but left-sided deficits and severe cognitive and mobility impairment persisted. Previously ambulatory, he could no longer walk. Psychiatry confirmed his decision-making capacity on June 1, when he declined chemotherapy despite repeated counseling. On July 9, a new headache, worsening left arm and leg weakness with neglect prompted a second stroke alert. Acute stroke CT imaging was negative, and MRI showed stable findings. Clinicians judged his deficits close to baseline. Continuous electroencephalography showed frequent right-sided periodic discharges, prompting intensified antiseizure treatment and another steroid course. Chest CT on July 9th showed an enlarging lung mass up to 4.7 cm and additional pulmonary nodules. He agreed to chemotherapy on July 10 and began carboplatin and etoposide on July 11. He completed four cycles by September. An August scan showed the dominant lung lesion had decreased to 1.7 cm. On hospital day 133, he remains hospitalized awaiting guardianship and long-term care placement. This case illustrates how paraneoplastic encephalitis can present with recurrent focal deficits suggestive of stroke. Electroencephalography, CSF antibodies, and lung biopsy supported the diagnosis despite plausible vascular and alcohol-related explanations. Conclusion: Early recognition of paraneoplastic encephalitis can expedite the search for an underlying malignancy and the initiation of treatment, a critical advantage when the associated cancer is rapidly progressive. The substantial tumor response did not restore independent mobility, showing that radiographic and neurologic outcomes must be assessed separately.

Austin Hoeg Dr. Hassan Karoam Dr. Kianoush Kashani	*Black Stool, Red Herring: Bismuth Ingestion as an Acute Gastrointestinal Bleed Mimic* Case Presentation: A 57-year-old woman was brought to the emergency department by EMS after an intentional ingestion with suicidal intent. She reported ingesting approximately 30 tablets of trazodone 100 mg, 30 tablets of baclofen 10 mg, and 30 tablets of quetiapine 300 mg. She denied other ingestions. Past medical history included major depressive disorder, generalized anxiety disorder, borderline personality disorder with multiple prior suicide attempts, Crohn's disease, Roux-en-Y gastric bypass, and recurrent foreign body ingestion (e.g., coins, metal charms) requiring endoscopic removal. On arrival to the ED, she was somnolent and hypotensive, with a blood pressure of 75/30 mmHg, heart rate of 88 beats/min, and respiratory rate of 15 breaths/min. EKG showed sinus rhythm with a QTc of 512. Hemoglobin was 11.3 g/dL, and serum lactate, transaminases, ethanol, acetaminophen, and salicylates were all within normal limits. After receiving a 2 L fluid bolus, she was transferred to the medical intensive care unit per the recommendation of Minnesota Poison Control. Upon arrival to the unit, she had a very large, black, tarry bowel movement with continued hypotension. Hemoglobin initially dropped to 10.6 g/dL, but lactate remained stable. On re-examination, the patient's oropharynx was coated with a pink-tinged substance. A portable abdominal X-ray was ordered and demonstrated multifocal radiolucency throughout her colon, consistent with bismuth ingestion. Her blood pressure improved with continued fluid resuscitation, and follow-up hemoglobin and lactate stayed within normal limits. Gastroenterology was consulted to rule out an upper intestinal bleed, and endoscopic evaluation was unremarkable. Given her continued hemodynamic stability, her black, tarry stool was attributed to large-volume bismuth ingestion. Upon further questioning when the patient was alert and oriented the following day, she endorsed also taking liquid bismuth amidst the other ingested substances the day prior as well. In this particular patient, the presence of hypotension and altered mental status further complicated her presentation of black, tarry stools. Though similar to findings of an acute gastrointestinal bleed, they are also common toxidromes of sedative and centrally acting medications that may be seen in the setting of intentional overdose. Baclofen toxicity can cause prolonged somnolence, hypotension, and respiratory depression. Quetiapine and trazodone are associated with QT interval prolongation, increasing the risk of ventricular arrhythmias. Bismuth subsalicylate (e.g., Pepto-Bismol) travels through the gastrointestinal tract and reacts with hydrogen sulfide produced by commensal colonic microbes, forming bismuth sulfide, the black pigment responsible for stool darkening. Discussion: Taken together, this case underscores the importance of avoiding premature anchoring and diagnostic closure based on a single clinical feature. In patients with known or suspected toxic ingestion,

	gastrointestinal mimics should remain on the differential, even when stool appears to be melanotic in nature. Serial hemodynamic and hemoglobin measurements, careful medication reconciliation and ingestion histories, and judicious use of endoscopy may help avoid unnecessary procedures and improve the quality of care given and received.
Albert Huang Dr. Frank Brozovich Dr. Hana Rosenow Dr. Kate Haley	*5-Fluorouracil Coronary Vasospasm Demonstrated on Coronary CT Angiography: Diagnosis and Successful Rechallenge* Introduction: 5-Fluorouracil (5-FU) is the second most common cause of chemotherapy-associated cardiotoxicity, usually presenting as angina from coronary vasospasm. Studies estimate vasospasm occurs in 2.2% of patients receiving 5-FU, with a paradoxically higher risk in patients with fewer cardiovascular risk factors. Diffuse ST-segment elevations during these episodes can mimic pericarditis if clinical context is not considered. We describe infusional 5-FU vasospasm confirmed by dynamic electrocardiographic, echocardiographic, and coronary CT angiographic findings, followed by successful rechallenge with a bolus-based plan. Case Presentation: A 42-year-old woman with hypertension and newly diagnosed stage IV colon adenocarcinoma with hepatic metastases who after normal DPYD testing began mFOLFOX6 with bevacizumab. On day 2 of her first 46-hour 5-FU infusion, she developed recurrent 15–30-minute episodes of severe substernal chest pain radiating to her jaw and arms, with nausea and dyspnea; the pain was non-pleuritic and non-positional. Her initial ECG was unremarkable. During subsequent episodes, serial ECGs showed diffuse ST-segment elevation in I, II, aVL, and V3–V6 that resolved and recurred in step with her pain over 12 hours, normalizing within 30 minutes of pain relief. High-sensitivity troponin T was mildly elevated without dynamic change (peak 29 ng/L). Coronary CT angiography during symptoms showed no coronary artery disease (CAD-RADS 0) but, on further review, focal LAD narrowing suggestive of vasospasm was observed.Transthoracic echocardiography (TTE) performed during an episode showed LVEF 47% with global hypokinesis and global longitudinal strain (GLS) of −10% with reduced right ventricular function.. The 5-FU infusion was stopped, amlodipine was increased, and isosorbide mononitrate was added. Acute chest pain episodes were controllable with nitroglycerin and morphine. After chest pain episodes subsided, repeat TTE on hospital day 3 showed LVEF 56%, GLS −21%, and normalized right ventricular function. Oncology transitioned her to FLOX, giving 5-FU as a weekly bolus. Prophylaxis was de-escalated to amlodipine 5 mg and isosorbide mononitrate 30 mg for adverse effects. She has since received 2 weekly boluses without chest pain or nitrostat use.

	Discussion: This case offers two lessons. First, while transient diffuse ST elevation and angina most often prompts internists to think of pericarditis, ST elevations that resolve between angina episodes are more indicative of coronary vasospasm. Here, vasospasm visualized on CT corresponded temporally with diffuse ST elevations and hypokinesis on TTE that improved between episodes. To our knowledge, focal vasospasm captured on coronary CT angiography during symptoms, concurrent with dynamic ST elevation, has not previously been reported with 5-FU cardiotoxicity. New angina with ST changes on a fluoropyrimidine should prompt stopping the infusion and considering vasospasm treatment first. Second, symptomatic vasospasm does not contraindicate further fluoropyrimidine therapy if prophylaxis is tolerated. Prospective studies show 5-FU cardiotoxicity is nearly three times as likely with continuous-infusion than with bolus-only regimens. Switching from infusional FOLFOX to bolus FLOX with calcium channel blocker and nitrate prophylaxis preserved first-line therapy in a patient whose UGT1A1 poor-metabolizer status made irinotecan alternatives less attractive. Prompt recognition and cardio-oncology collaboration can preserve therapy that might otherwise be abandoned.
Rachel Koller Dr. Leeore Levinstein	*Spleen on the Scene: A Case of Thoracic Splenosis* Introduction: Thoracic splenosis is a rare condition resulting from autotransplantation of splenic tissue following trauma to the spleen or diaphragm. It commonly presents as a left-sided thoracic mass and is typically found incidentally. CT characteristics are often non-diagnostic, and tissue sampling is typically required to confirm the diagnosis. Case Presentation: A 70-year-old male with past medical history of alcohol use disorder complicated by cirrhosis, heart failure with reduced ejection fraction, tobacco use disorder with 47 pack-year history, vocal cord paralysis complicated by chronic aspiration, history of splenectomy in 2003 following a snowmobile accident, and unhoused status presented to the emergency department after being found down by bystanders. On presentation, he was tachycardic but otherwise vitally stable. Labs were notable for Na 132, Cr 1.45, WBC 12, lactate 3.9, elevated procalcitonin. Chest-xray showed a chronic left upper lobe cavitary lesion, as well as a possible left midlung opacity. He was admitted for further workup. Infectious disease and pulmonology were consulted for the chronic cavitary lesion. Upon review of imaging, left apical lung pathology had been present dating back to at least 2015, initially noted as pleural thickening with some parenchymal fibrosis. CT chest was notable for a 5.6 x 4.8 cm cavitary lesion in the L apical lung. His prior workup had included multiple sputum cultures with pseudomonas aeruginosa which was thought to represent colonization. He received numerous antibiotic courses during prior hospitalizations including meropenem,

	ciprofloxacin, and doxycycline without resolution in his imaging. He had negative histoplasmosis and blastomycoses urine studies and negative TB QuantiFERON testing one month prior to hospitalization. Workup also included a PET CT scan in which the cavitary lesion had mild peripheral uptake, "favored to represent an infectious or inflammatory process." He also had a prior biopsy of the lesion which was non-diagnostic. Given the broad differential, the patient was started on piperacillin-tazobactam for possible aspiration pneumonia in the setting of vocal cord paralysis and alcohol use disorder. Workup for TB, fungal infections, and malignancy was pursued. He underwent IR-guided lung biopsy. Pathology ultimately resulted with "a core biopsy of splenic tissue, including red and white pulp, surrounded by fibrosis, chronic inflammation, and focal alveolar lung parenchyma. The histologic features, supported by immunohistochemistry, are consistent with thoracic splenosis." No further management was indicated for his thoracic splenosis. Conclusion: As illustrated in this case, the differential for pulmonary masses is quite broad and often requires tissue pathology to confirm the diagnosis. Though thoracic splenosis is rare, it should be considered on the differential for patients with known splenic trauma or splenectomy, and a biopsy should be pursued. Given the asymptomatic nature of the disorder, diagnosis can often go unnoticed, or patients, such as in this case, often undergo extensive workup prior to diagnosis. The diagnosis of thoracic splenosis via biopsy and tissue confirmation can limit further unnecessary imaging, treatment, and procedures for this benign process.
Anna Krauss Dr. Molly Lindstrom Dr. Suk Yin Chan-Colenbrander	*The Great Imitator: A Case of Primary Cerebral Nocardiosis* Introduction: Nocardia is a ubiquitous environmental bacteria. While considered opportunistic, it can still cause infections -- typically pulmonary or cutaneous -- in greater than one third of immunocompetent individuals. Nocardiosis is considered a "great imitator" as it typically presents with non-specific or insidious symptoms. This case highlights a relatively rare presentation of solitary cerebral nocardiosis in a patient without obvious risk factors except for advanced age. Case Presentation: An 87-year-old male patient with a medical history of atrial fibrillation on anticoagulation presented with two weeks of word finding difficulty progressing to slurred speech. He had no associated headache, vision changes, focal motor or sensory deficit, fevers, chills, or dyspnea. He was afebrile without leukocytosis, and his other labs were largely unremarkable. Imaging of the head with CT was negative for infarct, but did show pronounced edema with mass effect and mild sinus mucosal thickening. Brain MRI was subsequently obtained and was concerning for a 3.5cm rim-enhancing lesion in the left parietal lobe with significant vasogenic edema. Unfortunately, his aphasia worsened and he began to have facial spasms concerning for

	refractory focal seizures. He was intubated for airway protection. Due to acute decompensation, he was taken to the OR for expedited biopsy of the lesion. Purulent material was aspirated and sent for Gram stain and aerobic, anaerobic, and fungal cultures. Gram stain revealed 2+ modified acid-fast bacilli shortly after specimen submission. Meanwhile, CT scan of the chest and abdomen was obtained to assess for possible primary site of infection or malignancy and was without acute pathology. HIV and toxoplasma were negative, T cell subsets, including the CD4 count, were normal. Antibiotics were quickly transitioned from broad spectrum to trimethoprim-sulfamethoxazole, meropenem, and linezolid for empiric therapy targeting the most likely possible organisms. On culture day three, aerobic culture grew 4+ Nocardia cyriacigeorgica. Conclusion: This case presents a solitary Nocardia cerebral abscess initially thought to be malignancy in the setting of normal labs and lack of infectious symptoms in an elderly, but otherwise immunocompetent, individual. Because cerebral nocardiosis is rare and insidious, it is not often identified until after biopsy and culture. In this case, biopsy was ultimately expedited as the patient acutely decompensated. Obtaining a Gram stain was crucial as it allowed for immediate empiric treatment of modified acid fast bacteria, with a differential of Nocardia, Rhodococcus, Gordonia, or Tsukamurella. Nocardia is most often contracted via direct inoculation either cutaneously or in the lungs. When cerebral Nocardia is identified, it is most often after dissemination from a pulmonary source. However, primary CNS infections are not uncommon, even in patients without any predisposing factors. This case promotes a broad differential once ring-enhancing lesions are identified with imaging. These findings underscore the importance of considering nocardiosis and obtaining prompt specimens for Gram stain and modified acid-fast staining, with appropriate antimicrobial coverage when positive, even in the absence of infectious or pulmonary symptoms. Reference: Beaman BL, Beaman L. Nocardia species: host-parasite relationships. Clin Microbiol Rev. 1994 Apr;7(2):213-64. doi: 10.1128/CMR.7.2.213. PMID: 8055469; PMCID: PMC358319.
Walker Lahr Dr. Brian Berglund Mahima Devarajan Nived Kethireddy	*Hidden in Plain Sight: How a Lab Technician's Curiosity Unraveled the Diagnostic Mystery* Case Presentation: An 83-year-old Caucasian gentleman with Type 2 Diabetes, Polymyalgia Rheumatica, Obstructive Sleep Apnea, and Chronic Immune Thrombocytopenia presented with fever and altered mental status. Earlier that day, while biking outdoors his friends noted him confused and driving erratically. Later, his wife found him febrile and altered from baseline and brought him to the emergency department. On presentation, he was febrile to 103°F but otherwise hemodynamically stable. No evidence of meningismus on examination. Laboratory studies revealed mild leukopenia (WBC at 4200/ µL), anemia (Hemoglobin of 10 g/dL, around 11 g/dL at

	baseline), thrombocytopenia (23,000/μL, around 30,000/μL at his baseline), mild hyponatremia, and an elevated procalcitonin of 0.53 ng/mL. Hepatic Panel, lactate, urinalysis, and CTA of the head and neck were unremarkable. CT chest, abdomen, and pelvis demonstrated scattered reticulonodular pulmonary opacities suggesting infectious or inflammatory processes. He was hospitalized and treated with ceftriaxone and azithromycin for presumed community-acquired pneumonia and sepsis-associated encephalopathy. Despite antibiotics, he continued to spike fevers and remained delirious during the first two hospital days. Respiratory pathogen PCR was positive for rhinovirus/enterovirus. Given his recent outdoor exposure, Lyme serology was obtained and was nonreactive. On day 2, his platelet count abruptly dropped to 5,000/μL, prompting hematology consultation. He received intravenous corticosteroids and two courses of intravenous immunoglobulin (IVIG), with only modest improvement in his platelet count to above 16,000/μL. Initial peripheral smear review demonstrated his preexisting macrocytic anemia and thrombocytopenia without additional diagnostic findings. A Laboratory technician noted an abnormality on manual review of CBC with differential. His requested buffy coat exam demonstrated intracellular organisms within neutrophils suspicious of Human Granulocytic Ehrlichiosis (HGA). Antibody testing for Ehrlichiosis/Anaplasmosis came back negative. He was started on Doxycycline resulting in rapid improvement in fever and encephalopathy. PCR testing resulted late but confirmed Anaplasma phagocytophilum. He continued to improve and ultimately was discharged home. His platelet count improved above baseline at follow-up with his primary care physician. HGA is a vector-borne bacterial infection caused by Anaplasma Phagocytophilum, transmitted to humans through the bite of nymphal or adult Ixodes Ricinus complex (black-legged or deer) ticks. First cases in the USA were described by Dr. Bakken in the upper Midwest (1994). Symptoms of anaplasmosis are often nonspecific, overlap with various viral syndromes, and include fever, headache, malaise, loss of appetite, and aches. Illness is often mild and self-limited but can progress to severe illness and death in older individuals, in immunocompromised and those with comorbid conditions. Laboratory findings include leukopenia, thrombocytopenia, and elevated hepatic transaminases. Morulae may be identified within neutrophils on peripheral blood smear and provide an important diagnostic clue. PCR is the most specific and preferred confirmation test, especially during acute illness, whereas IFA antibody testing may be negative early in the course. This case highlights the importance of considering HGA in patients with unexplained fever, cytopenias, and potential tick exposure, even when another infection is identified or initial serology is negative. When clinical suspicion is high, doxycycline should be initiated

	empirically and continued for 7-10 days without waiting for confirmatory testing.
Shan Lakhani	*The Elusive Occlusion: A Case of Vasospastic MINOCA* Introduction: Myocardial infarction without obstructive coronary artery disease (MINOCA) is defined as myocardial infarction with mild or no obstructive coronary artery disease (CAD) on angiogram. Causes of MINOCA Include coronary disruption, coronary vasospasm, coronary embolism, coronary artery dissection, and coronary microvascular dysfunction. About 10% of patients undergoing cardiac catheterization for myocardial infarction (MI) are found to have MINOCA. Case Presentation: We present a 44-year-old woman with sudden-onset, non-exertional substernal chest pain and dyspnea. Initial troponin was 3 ng/L, but electrocardiogram (ECG) demonstrated ST-segment depressions in the inferior and anterior leads. Emergent coronary angiography revealed severe ostial left anterior descending (LAD) artery stenosis. After administration of heparin, ticagrelor, and intracoronary nitroglycerin, repeat angiography demonstrated complete resolution of the lesion without distal embolization, and percutaneous coronary intervention was deferred. Intravascular ultrasound (IVUS) demonstrated lipid-rich plaque with layered thrombus in the proximal LAD, preserved luminal area, and no evidence of subintimal hematoma. Serial troponins subsequently rose to 4,000, 9,000, and 11,000 ng/L, confirming acute myocardial infarction. Her symptoms and ECG changes resolved following nitroglycerin. transthoracic echocardiogram (TTE) demonstrated regional wall-motion abnormalities with a left ventricular ejection fraction (LVEF) of 55%. The presentation was consistent with MINOCA, with transient coronary vasospasm and plaque rupture with thrombus formation considered the leading etiologies. She remained asymptomatic during 48 hours of telemetry and was discharged on dual antiplatelet therapy, atorvastatin, nifedipine, nitroglycerin, and isosorbide mononitrate. Outcome: Following discharge, the patient remained compliant with her medications and cardiac rehabilitation but reported intermittent exertional chest discomfort. Isosorbide mononitrate was discontinued and her nifedipine dose was adjusted. Three days later, she was re-hospitalized with chest pain, nausea, and vomiting. Initial troponin was 90 ng/L, rising to 1,188 ng/L on repeat testing. While the initial ECG was normal, repeat ECG demonstrated ST elevation in V1 with significant ST depressions in V2–V4. She had partial symptomatic improvement with nitroglycerin. TTE demonstrated new regional wall-motion abnormalities, prompting

	repeat coronary angiography, which revealed severe multivessel coronary vasospasm that resolved with intracoronary nitroglycerin. The presentation was consistent with recurrent MINOCA secondary to coronary vasospasm. On discharge, nifedipine was transitioned to amlodipine and isosorbide mononitrate was restarted. The patient has remained clinically stable following discharge. Conclusion: This case highlights coronary vasospasm as an important cause of MINOCA and demonstrates that transient coronary occlusion may result in significant myocardial injury despite rapid angiographic resolution. Serial ECGs and troponins, along with intravascular imaging and response to intracoronary nitroglycerin, can help establish the diagnosis.
Olivia Langton Dr. Taylor Bowler Dr. Idil Oksuz Dr. Jeremy Taylor	*Development of a Cecal Bascule After Ingestion of Alendronate* Introduction: A cecal volvulus, defined as a twisting of the cecum around itself, is a rare clinical presentation that accounts for 1-2% of all large bowel obstructions [1]. There are multiple subsets of cecal volvuli defined by the pattern of rotation or folding. Cecal bascules account for only ~5-20% of cases of cecal volvuli and present as anterior migration of the enlarged cecum in a folding pattern without a rotational component [1, 2]. This folding can compromise blood flow, leading to areas of ischemia and necrosis that often require surgical intervention [1]. Case Presentation: A 73-year-old man with seronegative rheumatoid arthritis and osteoporosis presented to the emergency room with sharp, progressive, right-sided abdominal pain after taking oral alendronate. His pain was worse with oral intake, but he denied nausea or vomiting. He had two bowel movements on the morning of presentation, both formed and without frank blood. He had never undergone a colonoscopy or abdominal surgery. Physical exam was notable for guarding and tenderness to palpation in the epigastric region. A complete blood count demonstrated a hemoglobin of 13.6 g/dL without leukocytosis. A complete metabolic panel, lipase, and lactic acid were within normal limits. Computed tomography of the abdomen demonstrated a distended cecum in the right upper quadrant with a swirl-like appearance of the adjacent mesentery consistent with a cecal bascule. General surgery was consulted and the patient underwent an exploratory laparotomy and right-sided hemicolectomy with end-to-end anastomosis of the terminal ileum and the transverse colon. Pathology of the resected tissue demonstrated focal mucosal flattening and vascular congestion consistent with cecal bascule without evidence of malignancy. The patient's post-operative course was uncomplicated, and alendronate was discontinued on hospital discharge as a precaution. Discussion: A cecal bascule is a rare cause of large bowel obstructions that typically arise in a patient with a hypermobile cecum and a known history of abdominal surgery, adhesions, or constipation [1]. Our case

	highlights a rare presentation of cecal bascule in a patient who lacked these typical risk factors and developed symptoms after ingestion of bisphosphonates. While there is currently no literature outlining the correlation of bisphosphonates and cecal bascules, this case highlights the importance of maintaining a broad differential for abdominal pain even in the absence of typical anatomic and surgical risk factors. References: 1. Lung BE, Yelika SB, Murthy AS, Gachabayov M, Denoya P. Cecal bascule: a systematic review of the literature. Tech Coloproctol. 2018 Feb;22(2):75-80. doi: 10.1007/s10151-017-1725-6. Epub 2017 Nov 20. PMID: 29159782. 2. Park JS, Ng KS, Young CJ. Caecal bascule: a case series and literature review. ANZ J Surg. 2018 May;88(5):E386-E389. doi: 10.1111/ans.13898. Epub 2017 Mar 20. PMID: 28318090.
Christine Loo Dr. Rawad Nasr Dr. James Leatherman	*A Breathless Diagnosis: Recognizing Shrinking Lung Syndrome in Systemic Lupus Erythematosus* Introduction: Shrinking lung syndrome (SLS) is a rare pulmonary manifestation of systemic lupus erythematosus (SLE) characterized by diaphragmatic dysfunction, reduced lung volumes, and restrictive physiology. Case Presentation: We describe the case of a 43-year-old man who presented with fevers, diffuse myalgias, tachycardia, fatigue, generalized weakness, sore throat, and diffuse joint pain one month after a COVID-19 infection. Physical examination revealed exquisite tenderness of the hands, wrists, forearms, elbows, and knees. Laboratory evaluation demonstrated markedly elevated inflammatory markers (CRP 146, ESR >120), progressive microcytic anemia, positive TB-Quantiferon and CMV IgM/IgG with negative CMV NAAT, and a highly positive ANA (1:2560, homogeneous) with normal complement levels. CT imaging demonstrated marked right axillary lymphadenopathy concerning for infection or malignancy; however, lymph node biopsy revealed necrosis with acute and chronic inflammation consistent with a reactive process. Review of chest imaging demonstrated right-sided atelectasis and significantly decreased lung volumes. Diaphragm ultrasound showed decreased right diaphragmatic excursion without pain on inspiration. Pulmonary function testing revealed severely reduced vital capacity of 1.45 L supine and 1.65 L sitting. In the setting of a highly positive ANA, diaphragmatic dysfunction, restrictive physiology, and exclusion of infectious and malignant etiologies, these findings supported a diagnosis of SLS associated with SLE. The patient received methylprednisolone 125 mg IV daily for 5 days with marked clinical improvement, followed by a prednisone taper and hydroxychloroquine 300 mg daily. At one-month follow-up, he reported significant improvement in myalgias, dyspnea, and overall

	well-being. Repeat spirometry demonstrated improved vital capacity to 2.57 L, while bedside ultrasound showed improved diaphragmatic excursion from 1.1 cm to 2.95 cm. Discussion: This case highlights the importance of recognizing SLS as a rare but potentially severe manifestation of SLE, particularly when respiratory symptoms and restrictive physiology occur in the setting of otherwise unexplained systemic inflammation. In patients with unexplained dyspnea and systemic inflammatory symptoms, recognition of diaphragmatic dysfunction and restrictive physiology may facilitate diagnosis of SLS and prompt treatment.
Wendy Lopez Moreno	*Unmasking Distal Renal Tubular Acidosis in a Patient with Primary Biliary Cholangitis and Cirrhosis* Introduction: Hypokalemia is frequently encountered in patients with cirrhosis and is usually multifactorial, reflecting diuretic therapy, gastrointestinal losses, and malnutrition. However, persistent hypokalemia accompanied by a normal anion gap metabolic acidosis should prompt evaluation for an underlying renal tubular acidosis. Case Presentation: A 42-year-old woman with cirrhosis secondary to alcohol use and biopsy confirmed primary biliary cholangitis (PBC) was hospitalized for skin burns, with a course complicated by decompensated liver disease. She had longstanding low-normal serum potassium. In the hospital, K was as low as 2.6 mEq/L and was initially attributed to cirrhosis and diuretic use. She was also noted to have serum bicarbonate of 19 mEq/L which subsequently downtrended to 14-15 mEq/L during hospitalization, with a normal anion gap (5-9). While on diuretics, a spot urine anion gap (UAG) was negative (Na 57, K 81.5, Cl 141 mEq/L; UAG -2.5), falsely suggesting preserved renal ammonium excretion, an expected confounder given loop diuretic driven distal sodium delivery and kaliuresis. After diuretics were held, reassessment demonstrated a positive UAG (Na 21, K 32, Cl 37 mEq/L; UAG +6) indicating impaired renal ammonium excretion. A 24-hour urinary potassium of 34 mmol despite a serum potassium of 3.2 mEq/L (threshold being urine K > 20 mmol/day for renal wasting) confirmed inappropriate renal potassium wasting (of note, renal function was preserved). Urine pH remained approximately 6 despite systemic acidosis, the hallmark of impaired distal urinary acidification. Discussion: Both PBC and chronic alcohol use are recognized causes of dRTA. The normal anion gap metabolic acidosis, inappropriately alkaline urine pH, positive off-diuretics UAG and renal potassium wasting with preserved GFR, established a clinical diagnosis of distal RTA. The patient was treated with oral alkali therapy and potassium supplementation, with marked improvement of serum potassium to 4.3 mEq/L. Conclusion: Persistent hypokalemia accompanied by a normal anion gap metabolic acidosis should prompt evaluation for renal tubular disorders, particularly in patients with underlying autoimmune disease.

	This case highlights the importance of assessing acid-base status and urinary potassium excretion when evaluating hypokalemia.
Matthew Lukasik Dr. Colin Turner	*Adult Onset Still's Disease with a Miller Fisher Like Presentation* Introduction: This case report describes a unique presentation of Adult-Onset Still's Disease (AOSD) complicated by neurologic involvement either as a manifestation of Still's disease itself or as a separate, concomitant Guillain-Barré syndrome (GBS)/acute inflammatory demyelinating polyneuropathy (AIDP) with an incomplete Miller-Fisher (MF) like presentation. Case Presentation: A 74-year-old male was admitted for quotidian fevers, diffuse myalgias, and concern for sepsis. An extensive infectious workup was negative and unrevealing for a source of infection. He was transferred to our hospital for further workup and specialty care. On arrival, he presented with diffuse myalgias, weakness, and voice changes. He reported that the presentation was nearly identical to a similar unexplained hospitalization 15 years earlier, at which time ID workup was similarly unremarkable. PET scan and MRI of the brain and spine were both unrevealing for malignancy or an acute neurologic condition to explain the symptoms. Rheumatology was consulted for concern for AOSD. Although no rash was seen on examination or reported by the patient, he met both major and minor Yamaguchi criteria, including recurrent fevers, arthralgias, and leukocytosis, as well as minor criteria of transaminitis and negative rheumatoid factor and ANA. Inflammatory markers remained persistently elevated during the hospital stay, including a ferritin peak of 1,393 ng/mL (remaining above 700 ng/mL for the duration of admission), a peak CRP of 18.8 mg/dL, and leukocytosis of 25.8 × 10^9/L. The persistent elevations raised concern for immune overactivation with evolving macrophage activation syndrome (MAS) or hemophagocytic lymphohistiocytosis (HLH). High-dose corticosteroids and anakinra were started. Myalgias began to improve after steroid initiation, but the hospitalization was complicated by worsening neuropathic pain, sensory ataxia, loss of lower extremity deep tendon reflexes, and an acute kidney injury secondary to urinary retention that resolved with Foley catheter placement. Neurology was consulted for concern for CNS involvement or a separate neurologic condition such as GBS/AIDP. An incomplete MF presentation was also considered with no evidence of ophthalmoplegia and negative GQ1b antibodies. Lumbar puncture showed cytoalbuminologic dissociation with no evidence of meningitis, negative ganglioside antibodies, and a negative chronic inflammatory demyelinating polyneuropathy panel. Further disease progression was halted with corticosteroids, anakinra, and IVIG. Anakinra was transitioned to canakinumab (subcutaneous injection once monthly), and the patient was transferred to a swing bed for rehabilitation following a prolonged 3-week hospital stay. The patient continues to

	make very slow improvements in strength, mobility, and speech with the assistance of physical, occupational, and speech therapy. Discussion: This case represents a complex presentation of AOSD complicated by CNS involvement or a separate polyneuropathy such as an incomplete MF-like variant of GBS. On literature review, few cases of AOSD complicated by MF syndrome exist, with no reports found on an incomplete MF presentation with negative ganglioside antibodies. This case highlights potential neurological complications of AOSD and adds to the limited case discussions surrounding peripheral neuropathies and ataxias as sequela of the condition. In our treatment, the goal of halting disease progression was achieved with long term patient outcomes still being monitored.
Brianna Lupo Dr. Rebecca Windschitl	*Small Vessels, Big Consequences: A Challenging Case of ANCA Vasculitis* Introduction: Antineutrophil cytoplasmic antibody (ANCA) vasculitis is a subset of vasculitides that affects small vessels including capillaries, arterioles, and venules. ANCA vasculitis primarily affects adults ages 40 to 60 and presents with general symptoms of fatigue, weight loss, or rash. Given the generalized nature of presenting symptoms, the differential diagnosis is broad and requires a high degree of suspicion to avoid critical delays in diagnosis. This case describes a patient with a delayed diagnosis of ANCA vasculitis with crescentic glomerulonephritis that presented with generalized symptoms and worsening acute kidney injury (AKI). Case Presentation;: A 31-year-old female presented to her primary care provider (PCP) for ongoing fatigue, weight loss, and epistaxis. Medical history was otherwise significant for celiac disease and asthma. She was referred to otolaryngology who noted significant nasal inflammation with workup showing a weakly positive (1:20) c-ANCA titer. She subsequently had numerous outpatient visits with her PCP and the emergency department (ED) for weight loss and fatigue. Workup showed an elevated C-reactive protein (CRP) of 16.1mg/L and erythrocyte sedimentation rate of 29 mm/hr. Computed tomography (CT) of the abdomen and pelvis noted splenomegaly with several areas suspicious for infarcts. Presentation was felt consistent with mononucleosis with recommendation for supportive management. She re-presented to the ED due to worsening facial rash, arthralgias, and epistaxis. Vital signs were normal. Exam was notable for scleral injection and malar rash. Labs revealed leukocytosis of 13.6 x10(9)/L, new anemia with hemoglobin of 9.4 g/dL, elevated creatinine of 0.94 mg/dL from her baseline of 0.46 mg/dL, worsening proteinuria with urine protein to creatinine ratio of 1.3, and hematuria with red blood cell casts. CRP and ESR remained elevated at 11.4 mg/L and 49 mm/h respectively. She was admitted to the hospital for further diagnostic evaluation.

	With worsening renal function and positive ANCA titer three months prior, a broad workup was pursued with suspicion for glomerulonephritis. CT scan of the chest, abdomen, and pelvis revealed left renal and worsened multifocal splenic infarcts. Renal function worsened with creatinine doubling to 1.17 mg/dL. A renal biopsy was obtained. She was empirically started on high-dose methylprednisolone while awaiting biopsy results due to high suspicion for glomerulonephritis. c-ANCA titer returned positive at 1:320 with proteinase-3 antibody of 888, myeloperoxidase antibody negative. Renal function began to improve and biopsy results ultimately revealed crescentic injury of glomeruli (48%) due to pauci-immune glomerulonephritis. She began a steroid taper and was started on rituximab for ongoing management of ANCA vasculitis with marked improvement in ANCA titers and renal function. Discussion: AKI is a leading cause of hospitalization and a common complication during hospital stays that can be incited by several mechanisms internists are familiar with elucidating. More infrequently, patients with AKI have insidious pathology contributing to organ function decline with time-sensitive diagnostic needs. It is particularly crucial to consider atypical AKI pathology when concurrent symptoms are present to avoid irreversible renal damage. Here, failure to recognize and comprehensively assess symptoms across the renal, musculoskeletal, hematologic, and integumentary systems led to delay in diagnosis.
Lindsey Martens Dr. Scott Reule Dr. Khushboo Gala	*Non-Surgical Management of Cholecystitis in Patients on Peritoneal Dialysis: Is Endoscopic Gallbladder Drainage the Answer?* Introduction: Management of cholecystitis in peritoneal dialysis (PD) patients is a complex medical problem carrying higher than usual risk. Cholecystectomy remains the preferred treatment, however, patients on PD may have higher risk of infection, perioperative morbidity, and bile peritonitis. In patients who are suboptimal surgical candidates, gallbladder drainage (percutaneous vs endoscopic) is the therapy of choice. Percutaneous therapy may pose challenges to resumption of PD afterwards, given risk of peritonitis. We describe a case of cholecystitis in a patient on PD, treated successfully with endoscopic ultrasound (EUS)-guided gallbladder drainage. Case Description: A 75-year-old male with a past medical history significant for end stage renal disease on peritoneal dialysis, type 2 diabetes, and locally advanced pancreatic cancer with a biliary stent in place presented to the emergency department for evaluation of abdominal pain. On admission, CT abdomen and pelvis showed soft tissue density in the distal lumen of the biliary stent and distended gallbladder with thickened walls. Laboratory evaluation was notable for an elevated alkaline phosphatase. An endoscopic retrograde cholangiopancreatography (ERCP) revealed significant tumor ingrowth into the previously placed common bile duct stent; it was also noted that the cystic duct and gallbladder could not be opacified

	despite pressured occlusion cholangiography. A fully covered metal stent was placed through the pre-existing stent with significant drainage of purulent material from the bile duct consistent with ascending cholangitis. The patient did not have relief of pain despite treatment of biliary stent ingrowth; hence, a HIDA scan was performed, which confirmed cholecystitis. After a multidisciplinary discussion, decision was made to pursue endoscopic gallbladder drainage via EUS. Peritoneal dialysis was stopped 3 days prior to procedure and the patient was dried of dialysate. A cholecystogastrostomy was created with placement of 15 x 10 mm lumen apposing metal stent (LAMS) and coaxial double pigtail plastic stent with drainage of a large amount of sludge from the gallbladder. He was then transitioned from PD to hemodialysis (HD) for a planned 8-week course, to allow for maturation of the cholecystogastrostomy fistula and minimize the risk of leak and infection. Two months later, the patient underwent repeat ERCP, with the LAMS exchanged for two double-pigtail plastic stents, intended to serve as destination therapy for cholecystitis. Two weeks later, the patient was successfully transitioned to home PD. Discussion: This case highlights the viable option of EUS gallbladder drainage with a LAMS in PD patients. Patients on PD prefer the greater lifestyle flexibility, fewer dietary restrictions, and lesser physical strain compared to HD, and the ability to transition back to PD is highly desired. Endoscopic gallbladder drainage offers a potentially safer alternative to percutaneous drainage in patients who are suboptimal surgical candidates. There are no clear guidelines on management of renal replacement therapy in patients undergoing gallbladder drainage; a multidisciplinary approach with a safe window of hemodialysis before and after procedure is advised. This case emphasizes a team- and patient-based approach to care between internal medicine, gastroenterology, and nephrology for the care of a PD patient with cholecystitis.
Jeffrey Moorhead	*Eosinophilic Aortitis: A Case of Precipitous Debility* A man in his 70s with a cardiomyopathy, recurrent sinusitis and psoriasis treated with secukinumab developed three years of progressive exertional dyspnea, fatigue and weakness. Investigation revealed late gadolinium enhancement on cardiac MRI, moderate-to-severe mitral regurgitation, diffuse concentric thickening of the thoracoabdominal aorta and supra-aortic branches, peripheral eosinophilia, and elevated inflammatory markers. An intraoperative biopsy of the ascending aorta taken during mitral valve repair showed active aortitis with a dense eosinophil-predominant infiltrate and rare giant cells, without fibrinoid necrosis, granulomas or IgG4 features, favoring eosinophilic granulomatosis with polyangiitis-spectrum disease. High-dose glucocorticoids and discontinuation of secukinumab produced rapid improvement, with relapse on tapering. Unexplained cardiovascular decline with persistent eosinophilia

	warrants evaluation for large-vessel vasculitis, and surgical exposure can yield diagnostic tissue when peripheral workup is inconclusive
Andre Morse Dr. Idil Oksuz Dr. Jeremy Taylor	*Delayed Ventricular Embolization of a Transcatheter Aortic Valve Following Cardiopulmonary Resuscitation* Introduction: Transcatheter valve embolization complicates approximately 0.92% of transcatheter aortic valve replacements (TAVR), most often into the aorta (1). Left ventricular embolization is rarer, deadlier, and usually requires surgical retrieval (2). We herein report delayed ventricular embolization of a TAVR valve after peri-deployment cardiac arrest. Case Presentation: A 78-year-old man with prior CABG, right bundle branch block, and heart failure with improved ejection fraction was admitted with decompensated heart failure in the setting of severe aortic stenosis. Six months earlier, his aortic stenosis was moderate (valve area 1.2 cm^2). Cardiac CT at that time showed a calcified aortic valve (1293 Agatston units) of probable bicuspid morphology, an annular area of 442 mm^2, and left ventricular ejection fraction (LVEF) of 40–45%. Echocardiography now showed LVEF of 25–30%; thickened, calcified cusps with markedly restricted excursion; Vmax 2.3 m/s; mean gradient 14 mmHg; and dimensionless index 0.39. Left ventricular outflow tract (LVOT) spectral broadening precluded reliable valve area calculation. Severe low-flow, low-gradient aortic stenosis was diagnosed morphologically. Due to high surgical risk, TAVR was recommended. In discussion, the patient stated he would not accept surgical bailout in the event of TAVR complications. A 23-mm SAPIEN 3 Ultra valve, overfilled by 2.5 mL, was deployed transfemorally at an 80% aortic and 20% ventricular position. Immediately after deployment, he developed pulseless electrical activity (PEA) arrest requiring approximately 20 minutes of cardiopulmonary resuscitation. Post-arrest echocardiography showed a seated valve without paravalvular regurgitation, no pericardial effusion, and preserved coronary flow. LVEF was 10–15%, and an intra-aortic balloon pump (IABP) was placed. The patient developed new ventricular ectopy 95 minutes after IABP implantation, prompting fluoroscopy, which demonstrated ventricular embolization of the valve. Based on the patient's previously expressed wishes to avoid open-chest surgery, immediate surgical retrieval was not pursued. He was transferred to the ICU on vasopressors and IABP support with ongoing goals of care discussions. Four hours after IABP deployment, he died of PEA arrest, presumably caused by LVOT obstruction and concurrent cardiogenic shock. Autopsy was declined. Discussion: The prosthesis was well-seated immediately post-arrest and was found displaced only during hemodynamic recovery, making gross malposition unlikely. Its conventional-to-high implant depth also argues against migration from low deployment. We hypothesize that chest compressions compromised radial fixation, after which IABP diastolic augmentation, rising aortic pressure against the closed

	prosthesis, and still-recovering left ventricular contractility contributed to ventricular migration (3,4). Borderline annular sizing, lower calcium burden, and probable bicuspid anatomy may also have limited anchoring. Our case demonstrates that displacement can follow apparently successful deployment; therefore, any peri-procedural arrest should prompt serial imaging to confirm stable valve position. Declining open surgery forfeits the main rescue for this rare complication, a risk worth making explicit during consent. References: 1. Kim WK, et al. Eur Heart J. 2019. 2. Ibebuogu UN, et al. Am J Cardiol. 2015. 3. Chakravarty T, et al. Catheter Cardiovasc Interv. 2018. 4. Kim EK, et al. Catheter Cardiovasc Interv. 2014.
Emilee Ohman Dr. Leeore Levinstein	*Beyond the Esophagus: A Rare Case of Bleeding Jejunal Varices* Introduction: A common non-cirrhotic cause of portal hypertension (pHTN) is thromboses of the portal venous system. Development of varices, both esophageal and ectopic, is a common sequela of pHTN that can lead to life threatening gastrointestinal hemorrhage. Here we present a case of recurrent GI bleeding from ectopic jejunal varices caused by chronic splenic and superior mesenteric vein (SMV) thromboses. Case Presentation: A 74-year-old male with history significant for splenic vein and SMV thromboses on rivaroxaban and T cell large granular lymphocytic leukemia was admitted with five-month history of melena and acute blood loss anemia with hemoglobin drop from 12.0 to 9.0 on admission. The patient had been following with gastroenterology outpatient for iron deficiency due to chronic blood loss, and EGD/colonoscopy/video capsule endoscopy done one month prior to presentation were all normal. At presentation he had been off a proton-pump inhibitor for 2 weeks in anticipation of Helicobacter pylori testing. Given low hemoglobin and concern for new bleed, anticoagulation was held. EGD was completed and notable for few moderate jejunal erosions without bleeding. Hemostatic clips were placed on these erosions with subsequent torrential bleeding, hemostasis was achieved with epinephrine and a diagnosis of jejunal varices was confirmed. Unfortunately, the patient experienced a PEA arrest due to hypovolemic shock from blood loss requiring CPR, intubation, pressors, massive transfusion protocol, and ICU admission. He immediately underwent CTA with recanalization of his SMV occlusion and embolization of duodenal and jejunal varices by interventional radiology with subsequent improvement in clinical status. H pylori testing returned positive and treatment was initiated prior to hospital discharge. Discussion: This patient was at increased risk of pHTN and ectopic variceal formation due to his chronic hypercoagulable state in the setting of active malignancy resulting in both splenic and SMV

	thromboses. Ectopic varices can form anywhere in the small or large bowel and can be notoriously difficult to identify and treat given the limited access by gastroenterologists beyond the duodenum. Bleeding from these ectopic varices represents 1-5% of variceal bleeding but carries a mortality rate of 40%. Generally, if a patient presents with chronic anemia and melanic stools with no clear source of upper or lower GI bleeding, a video capsule endoscopy (VCE) should be performed to evaluate the small bowel. In the case presented, a repeat EGD was warranted after a negative VCE given the patient's ongoing symptoms and anemia, eventually leading to discovery of jejunal varices. While there are no current guidelines established on the best way to treat varices of the small intestine, interventional radiology is often involved for recanalization of the portal venous system and embolization of bleeding varices. Management of stable esophageal, gastric, or ectopic varices includes band ligation, vasoconstrictors and/or non-selective beta blockers, and consideration of a TIPS procedure. This case highlights the importance of considering ectopic varices as a cause for recurrent melena despite a previously normal workup.
Lauren Pomerantz Dr. Natalie Griffin	*Beyond Recurrent Infections: Common Variable Immunodeficiency Presenting with Autoimmune Hemolytic Anemia and Pulmonary MALT Lymphoma* Introduction: Common variable immunodeficiency (CVID) is characterized by hypogammaglobulinemia and impaired specific antibody production but may manifest with substantial immune dysregulation, including autoimmune cytopenias, lymphoproliferation, interstitial lung disease (ILD), and malignancy. Noninfectious manifestations may precede recurrent infections, contributing to delayed diagnosis. We present a case of probable CVID complicated by autoimmune hemolytic anemia (AIHA) and pulmonary MALT lymphoma, highlighting the diagnostic challenges of multisystem immune dysregulation. Case Presentation: A 49-year-old woman initially presented with E. coli pyelonephritis and bacteremia in the summer of 2023, with incidental marked splenomegaly, pulmonary nodules, and interstitial abnormalities on computed tomography (CT) imaging. Following treatment of her infection, she did not experience recurrent infections but instead developed progressive autoimmune and pulmonary manifestations. Over subsequent months, she developed severe Coombs-positive AIHA requiring corticosteroids, intravenous immunoglobulin, and rituximab. Multiple lymph node and bone marrow biopsies were negative for malignancy. Her pulmonary abnormalities initially improved following immunosuppressive therapy. In the spring of 2025, she developed a recurrent dry cough with numerous bilateral pulmonary nodules. Extensive infectious evaluation and multiple tissue samples, including bronchoscopy, endobronchial

	ultrasound, transbronchial biopsy, CT-guided lung biopsy, and mediastinal lymph node biopsies, were negative for malignancy. Lung pathology demonstrated organizing pneumonia with dense lymphoplasmacytic infiltrates. She was treated with prolonged corticosteroids, with transient clinical improvement and waxing and waning pulmonary nodules. Following steroid discontinuation in early 2026, her respiratory symptoms recurred. CT demonstrated persistent bilateral pulmonary nodules, several with a reverse halo sign, mediastinal lymphadenopathy, septal thickening, and splenomegaly. PET-CT showed FDG-avid lymphadenopathy above and below the diaphragm and multiple FDG-avid pulmonary nodules. Profound hypogammaglobulinemia (IgG 151 mg/dL, IgA <5 mg/dL, IgM 44 mg/dL), together with her history of AIHA, splenomegaly, lymphadenopathy, and pulmonary disease, raised concern for CVID. Repeat lung biopsy ultimately demonstrated extranodal marginal zone (MALT) lymphoma. Multidisciplinary evaluation concluded that her pulmonary disease was most consistent with lymphoma arising in the setting of probable CVID. Rituximab and immunoglobulin replacement were recommended, with gradual corticosteroid taper. Genetic and specific antibody response testing are planned. Discussion: This case demonstrates that CVID may initially present with autoimmune and lymphoproliferative complications rather than recurrent infection. CVID-associated immune dysregulation can promote autoimmune cytopenias, including AIHA, through loss of immune tolerance and dysregulated B- and T-cell function. CVID is also associated with lymphoproliferative disorders and an increased risk of lymphoma, including extranodal marginal zone (MALT) lymphoma, reflecting chronic immune stimulation and abnormal B-cell proliferation. In the lungs, these manifestations may overlap with infection, granulomatous-lymphocytic interstitial lung disease, organizing pneumonia, and malignancy, making diagnosis particularly challenging. In this patient, multiple nondiagnostic biopsies did not ultimately exclude malignancy. Persistent or waxing-and-waning pulmonary nodules, particularly in the setting of AIHA, hypogammaglobulinemia, lymphadenopathy, and splenomegaly, warrant continued multidisciplinary evaluation and, when clinically indicated, repeat pulmonary tissue sampling to establish a definitive diagnosis. In patients presenting with AIHA and pulmonary findings concerning for MALT lymphoma, evaluation for an underlying immunodeficiency, including quantitative immunoglobulins, should also be considered, as recognition of CVID may guide immunoglobulin replacement, surveillance, and management of associated lymphoproliferative disease.
Willis Pullman	*Not Every Recurrent "Pneumonia" Is Infection: Aureobasidium Pullulans Hypersensitivity Pneumonitis from an Indoor Marijuana Farm in a Person Living with HIV*

	Case Presentation: A 41-year-old male with well-controlled HIV presented to clinic with three weeks of flu-like symptoms, scant hemoptysis, and progressive exertional dyspnea with hypoxia. He was initially treated with cefuroxime/azithromycin, then levofloxacin without resolution. Initial chest x-ray showed extensive bilateral interstitial opacities. Laboratory studies at the time were significant for leukocytosis and reactive thrombocytosis. He was later hospitalized twice for hypoxemic respiratory failure requiring supplemental oxygen. Chest CT demonstrated diffuse centrilobular micronodules with scattered ground-glass opacities. Infectious workup was unrevealing: respiratory viral panel, TB, legionella, BAL bacterial/fungal/AFB cultures and PJP PCR were all negative. BAL showed predominantly neutrophils with a CD4:CD8 ratio of 1.02, and normal IgE. A hypersensitivity pneumonitis panel returned positive IgG to Aureobasidium pullulans. A key feature was rapid symptom recurrence within 12 hours of returning home. Exposure history revealed an indoor marijuana farm in a damp basement and manipulation of a dirty dehumidifier. Symptoms resolved with antigen avoidance and corticosteroids; a follow-up chest radiograph and CD4:CD8 findings supported acute HP. Pullulans is an environmental mold and a known cause of HP. The reduced BAL CD4:CD8 ratio reflects the CD8-weighted alveolitis characteristic of extrinsic allergic alveolitis. Although BAL lymphocytosis is classic for HP, an early neutrophil-predominant pattern can occur shortly after acute antigen exposure. The reproducible clinical worsening on home re-exposure was diagnostically decisive alongside laboratory findings. Discussion: This case serves as a reminder that HP should be considered in the differential for a recurrent pneumonia that fails to respond to antibiotics, with a detailed environmental and hobby exposure history, in order to identify a culprit antigen.
Matthew Record Dr. Kambiz Kalantari	*More Than GI Intolerance: Anchoring on Common Causes of AKI Delays a Myeloma Diagnosis* Introduction: Nausea with acute kidney injury (AKI) in older adults with nephrotoxic drug exposures is more often attributed to hemodynamic causes or direct drug-induced injury. However, AKI warrants a thorough evaluation and broader diagnostic considerations. In the setting of monoclonal gammopathy, AKI may be the first sign of multiple myeloma. Case Presentation: A 67-year-old woman with type 2 diabetes, uncontrolled hypertension, hyperlipidemia, gout, and obesity presented with a two-month history of nausea, malaise, and headache. She had been started on tirzepatide two months prior for management of diabetes and weight loss, but this was discontinued one month later

	due to intolerance with significant nausea. She had been taking daily indomethacin for chronic bilateral foot pain and in the past week she noted elevated home blood pressures for which she was taking extra losartan and hydrochlorothiazide doses. On admission, blood pressure was 180/102 mmHg. Physical examination was notable for generalized abdominal tenderness, and the neurologic exam was unremarkable. Creatinine was 7.18 mg/dL (baseline 0.6), bicarbonate was 13 mEq/L, hemoglobin was 7.0mg/L, MCV 92 fL. Urinalysis demonstrated clear urine with 3+ protein, 3-10/HPF RBCs, 21-30/HPF WBCs and large leukocyte esterase with negative nitrites and no casts. FENa was 5.8%, consistent with intrinsic injury. Workup for hemolysis and thrombotic microangiopathy was negative (normal platelets and peripheral smear without schistocytes). Renal ultrasound was unremarkable without hydronephrosis. Serum free light chains demonstrated a markedly elevated lambda of 1059 mg/dL and a kappa:lambda ratio of 0.0018 with suppressed quantitative immunoglobulins. Nephrology was consulted, and renal biopsy demonstrated lambda light chains in the glomeruli and tubular basement membranes with tubular casts. Hematology was consulted, and she underwent bone marrow biopsy that revealed plasma cell myeloma. Management for newly diagnosed multiple myeloma with light chain nephropathy was initiated, including therapeutic plasma exchange for cast nephropathy. Discussion: This case illustrates the dangers of anchoring on common explanations for AKI without assessing the entire clinical picture. Increased ARB, thiazide, and NSAID use, along with GLP-1 related nausea offered a convenient explanation for volume depletion and NSAID-induced nephrotoxicity. Upon further review, an unexplained, years-long mild anemia along with acute tubular injury with proteinuria, and newly disproportionate anemia should place paraprotein related diagnoses on the differential. Prompt lab studies testing serum free light chains helped identify a monoclonal associated kidney injury, which helped immediately start disease-directed therapy. Clinicians need to consider paraprotein evaluation in patients with unexplained AKI, particularly with co-existing anemia.
Shameel Shafqat	*When Hemolysis Starts in the Marrow: Pernicious Anemia Presenting with Pancytopenia* Introduction: Severe vitamin B12 deficiency may cause ineffective hematopoiesis with intramedullary destruction of erythroid precursors, producing pancytopenia and evidence of hemolysis with an inadequate reticulocyte response. This uncommon presentation may mimic peripheral hemolysis or thrombotic microangiopathy, and prompt unnecessary testing or plasma exchange, making recognition of the underlying marrow-production defect clinically important. Case Presentation: An 86-year-old woman with dementia, hypertension, chronic liver disease, prior cholecystectomy and remote gynecologic malignancy, presented after one week of progressive

	weakness, lightheadedness, poor oral intake, and near-syncope. She denied overt gastrointestinal or current vaginal bleeding. At presentation, hemoglobin was 5.2 g/dL, hematocrit 15.0%, with an elevated mean corpuscular volume 126.1 fL. Furthermore, she had a low white blood cell count 3.28 ×10^9/L, and platelets 104 ×10^9/L. CT chest/abdomen/pelvis showed no bleeding and she received two units of packed red blood cells. Further evaluation demonstrated vitamin B12 below the assay limit (<150 pg/ml), with elevated methylmalonic acid of 9.45 umol/L, and homocysteine of 73 umol/L, suggestive of severe vitamin B12 deficiency. Reticulocytes were 2.0%, inappropriately low for the severity of anemia. Lactate dehydrogenase was 1,638 U/L, haptoglobin <3 mg/dL, and total bilirubin peaked at 1.5 mg/dL. Direct antiglobulin testing was negative. Peripheral smear showed marked macrocytic normochromic anemia, mild neutropenia and monocytopenia, and thrombocytopenia without red-cell fragmentation. Renal function was preserved. Intrinsic-factor blocking antibody, which had been pending throughout admission, subsequently resulted positive, supporting autoimmune gastritis/pernicious anemia as the cause of severe vitamin B12 deficiency. Upon further chart review, the patient was found to have a singular vitamin B12 level of 66 pg/ml at a prior admission 12 years ago, suggesting longstanding or recurrent deficiency. The combination of profound macrocytosis, pancytopenia, hemolysis with inadequate reticulocyte response and without schistocytes favored intramedullary hemolysis from ineffective megaloblastic erythropoiesis rather than immune hemolysis or a true microangiopathic process. The patient was treated with intramuscular cyanocobalamin 1,000 mcg daily for seven doses, then weekly for four doses, followed by monthly therapy, plus folate 1 mg daily. One month after discharge and follow-up in clinic, her hemoglobin increased to 9.2 g/dL, platelets to 221 ×10^9/L, and white blood cells to 6.55 ×10^9/L. Her vitamin B12 had also normalized to 731 pg/ml and haptoglobin to 86 mg/dL. Discussion/Conclusion: Profound vitamin B12 deficiency can produce a convincing hemolytic profile while marrow output remains inadequate. Marked macrocytosis, pancytopenia, a disproportionately low reticulocyte response, negative antiglobulin testing, and confirmatory B12 metabolites should prompt consideration of intramedullary hemolysis. Recognizing this reversible presentation can distinguish vitamin B12 deficiency-related ineffective erythropoiesis from peripheral hemolytic processes, avoid unnecessary interventions, and direct definitive lifelong replacement when pernicious anemia is confirmed.
Harsh Sharma Dr. Abigail Rice Dr. Adriana Dhawan	*Sepsis in the Setting of Pancreatic Tail Mass Masquerading as a Neuroendocrine Tumor*

	Introduction: Neuroendocrine tumors (NET) are a group of malignant neoplasms that often secrete bioactive hormones. Functional neuroendocrine tumors include carcinoid tumors, gastrinomas, glucagonomas, insulinomas, somatostatinomas and VIPomas. Their combined incidence of about 3.56 per 100,000 makes them quite rare. We describe the case of a middle-aged woman who presented with subacute symptoms of fatigue, diarrhea and abdominal pain, and was found to have a well-defined mass on initial imaging concerning a NET. She underwent endoscopic and serologic evaluation and was ultimately found to have an accessory splenule. She was treated for suspected sepsis which improved with antibiotics and was discharged. Case Description: A 57-year-old female with a past medical history of chronic PE, lymphedema, tardive dyskinesia, obesity (status post gastric bypass) presented to the hospital with several weeks of abdominal pain, generalized fatigue, diarrhea, vomiting, and intermittent subjective fevers that had worsened the past 3 days. She reported her abdominal pain was worse on an empty stomach, along with dark colored stools and a new cough with chest discomfort on laying supine. Notably she mentioned that she has a family history of stomach ulcers in her father. She denied rashes, myalgias, unintentional weight loss, or bruising. She did not take routine post-bypass vitamin supplementation. Her vitals at presentation were: Temperature 100.5F, Pulse 122/min, BP 99/75 mm Hg, SpO2 95% on room air, BMI - 48.9Kg/m2. Her physical exam was notable for involuntary movements of her head and face and right upper quadrant abdomen tenderness. Her labs demonstrated leukocytosis (WBC 13.64K), mild anemia (Hb 10.6, MCV 79.2), and suspected mild kidney injury (creatinine 1.17, previously at baseline ~0.7). Her CRP (165) and LDH (251) were elevated. A CT abdomen and pelvis noted ‘Mildly enhancing lesion in the pancreatic tail’ along with ‘Multiple enlarged para-aortic and iliac lymph nodes’. She was started on empiric broad spectrum antibiotics in the setting of suspected sepsis Additional workup revealed severe deficiencies of iron, B12, and vitamin D. We obtained Serum Gastrin and VIP levels for suspected peptic ulcers - which were normal. Gastroenterology was consulted and she underwent EUS with a biopsy of the pancreatic tail mass. Her symptoms improved with antibiotics though workup revealed no specific infectious etiology, and her micronutrient deficiencies were addressed with supplementation. Biopsy later revealed the pancreatic tail mass was consistent with an accessory spleen / splenule. Discussion: Functional NETs present with a group of symptoms depending on the hormone they secrete including diarrhea, flushing, electrolyte abnormalities, abdominal pain and glucose fluctuations. Our patient's presentation and imaging raised suspicion for a true NET. She was treated for sepsis while undergoing workup for this rare

	diagnosis, and ultimately the mass was confirmed to be an accessory spleen. An accessory spleen is the presence of splenic tissue outside of its typical anatomic location. These benign masses are usually found incidentally on imaging via CT or MRI (often misidentified as reactive lymph nodes or malignant GI tumors) and can share features common to NETs.
William Sieling Dr. Nikolai Mendonca Dr. Craig Mescher Dr. Balkrishna Jahagirdar Dr. Gaurav Guliani Dr. Courtney Burnett Dr. Mary Fredrickson Dr. Oana Dickinson	*A Boxed Trifecta with No Winners: Triple M Overlap Syndrome Due to Immune Checkpoint Inhibitor Toxicity* Immune checkpoint inhibitors (ICIs) such as pembrolizumab have revolutionized cancer care and are increasingly used in the treatment of many cancer types, but they can trigger rare, life-threatening immune-mediated toxicities affecting nearly every organ system. We describe a patient with metastatic breast cancer recently started on pembrolizumab who presented with myalgias, diplopia, shortness of breath and elevated troponin levels. Symptoms and diagnostics supported myositis, myocarditis and myasthenia gravis, consistent with Triple-M Overlap Syndrome - a rare but incredibly severe ICI-related toxicity.
Jacqueline Turner Heather Montane Dr. Svetomir Markovic	*Liver-Directed Therapy in Metastatic Uveal Melanoma: A Case Report Evaluation of Overcoming Therapeutic Resistance and Metastatic Organotropism* Background: Uveal melanoma is a rare subtype of melanoma that occurs within the orbit and has a unique propensity to metastasize to the liver. Hepatic involvement occurs in up to 95% of patients with stage IV disease, and these metastases are strongly associated with poor prognosis and mortality. Despite advances in systemic immunotherapies, uveal melanoma remains largely refractory to traditional and novel interventions, highlighting the need for effective liver-directed therapies. Case Presentation: We report the case of a 56-year-old man with uveal melanoma involving the choroid and ciliary body who developed diffuse hepatic metastases. He was ineligible for tebentafusp due to HLA-A*02:01 negativity and was treated sequentially with combination ipilimumab/nivolumab, single-agent nivolumab, and nivolumab-relatlimab, but progressed with persistent hepatic tumor burden. The patient subsequently underwent three cycles of percutaneous isolated hepatic perfusion with melphalan (HEPZATO), achieving significant radiographic improvement with >30% reduction in hepatic tumor volume on MRI. Suspected extrahepatic disease, including lung and peritoneal nodules, remained stable during treatment. While he developed immune related-adverse events from the prior immune checkpoint blockade, the HEPZATO treatment was well tolerated without significant toxicity.

	Conclusion: This case highlights HEPZATO as a promising liver-directed therapy for refractory uveal melanoma, capable of achieving meaningful hepatic disease control and contributing to prolonged survival. HEPZATO is a promising therapeutic option for patients with refractory uveal melanoma and illustrates the importance of liver-directed interventions.
Abby Sigurdson Dr. Breanna Zarmbinski	*Seizing Under Pressure: When Delivery Is Not the End of Critical Illness* Introduction: Delivery is the definitive treatment for pregnancy-related hypertensive disease but does not necessarily mark the end of maternal critical illness. Severe hypertension can initiate progressive vascular and end-organ injury, and the postpartum period is characterized by ongoing hemodynamic and cardiovascular changes. Consequently, maternal deterioration may persist, or new complications can emerge after delivery. Recognition of severe hypertension is a time-sensitive emergency and continued evaluation of postpartum neurologic and multisystem dysfunction is essential. Case Description: A 28-year-old previously normotensive woman at 40w4d gestation presented with severe-range hypertension (257/121 mmHg) and headache following spontaneous rupture of membranes. Blood pressure remained severely elevated despite analgesia, and recurred following transient improvement with nifedipine. Initial evaluation demonstrated marked proteinuria, ALT 709 U/L, and platelets of $174 \times 10^3/\mu L$. Over several hours, severe hypertension was followed by rapidly progressive multisystem injury, including thrombocytopenia, hemolysis, hyperbilirubinemia, transaminitis, and acute kidney injury. She underwent vaginal delivery; however, delivery did not halt her clinical deterioration. Postpartum thrombocytopenia progressed to a nadir of $27 \times 10^3/\mu L$ with active bleeding requiring platelet transfusion. Her postpartum course was subsequently complicated by a generalized tonic-clonic seizure with transient unresponsiveness, prompting ICU transfer. Blood pressure was again in the 190s/100s mmHg. She experienced a second generalized tonic-clonic seizure requiring lorazepam and levetiracetam. Persistent severe hypertension required intravenous nicardipine and labetalol. Continued seizures and encephalopathy prompted neurologic evaluation. Head CT demonstrated multifocal hypodensities, while MRI demonstrated bilateral frontal cortical abnormalities. Continuous EEG was notable for posterior and generalized cortical dysfunction. In the setting of severe hypertension, recurrent seizures, encephalopathy, and neuroimaging abnormalities, findings were most consistent with posterior reversible encephalopathy syndrome (PRES). With continued blood pressure control and supportive critical care, seizures resolved

	and neurologic, renal, hepatic, and hematologic dysfunction progressively improved. Follow-up MRI demonstrated complete resolution of cerebral abnormalities. Discussion: Severe hypertension was the earliest manifestation of an evolving hypertensive emergency in this patient, proceeding progressive hematologic, hepatic, renal, and neurologic injury. Acute severe hypertension in pregnancy requires prompt treatment to reduce the risk of maternal end-organ damage. Importantly, delivery should not create diagnostic closure. Removal of the inciting pregnancy does not immediately reverse established endothelial or end-organ injury, and the postpartum period remains a time of ongoing cardiovascular, hemodynamic, and multisystem risk. Continued deterioration therefore warrants ongoing critical care surveillance and reassessment. Recurrent seizures and encephalopathy in the setting of persistent severe hypertension should broaden the differential beyond eclampsia alone and prompt evaluation for neurological complications such as PRES. Early recognition and effective blood pressure control may prevent further neurologic injury and permit complete clinical and radiographic recovery.
Dakota Snustad Dr. Alisa Duran Dr. Shahidali Jaffer	*Wearing Your Heart on Your Skin: Afebrile Implantable Cardioverter Defibrillator Lead Endocarditis and Infection-Associated Glomerulonephritis Initially Presenting as Leukocytoclastic Vasculitis* Introduction: Leukocytoclastic vasculitis can be a benign, transient, skin-limited condition, but it can also be a sign of underlying infection, adverse drug reaction, or neoplasm, making it imperative to evaluate for a broad differential diagnosis to ensure accurate diagnosis and treatment. Case Presentation: In this case, we present a 65-year-old male with a past medical history of ischemic cardiomyopathy and ventricular tachycardia status post implantable cardioverter-defibrillator (ICD) placement 8 years prior to presentation, type 2 diabetes mellitus, and tobacco use, who presented to his primary care clinic for evaluation of a new purpuric rash over his bilateral lower extremities, an unintentional 30-pound weight loss, and fatigue. He had a skin biopsy performed which revealed leukocytoclastic vasculitis. During a subsequent hospitalization, a transthoracic echocardiogram (TTE) revealed a 1.2cm mobile vegetation attached to the atrial portion of the right-sided ICD lead, and subsequent blood cultures were positive for Enterococcus faecalis. The patient was started on ceftriaxone and ampicillin for a diagnosis of Enterococcus faecalis ICD lead endocarditis requiring ICD removal. The patient's clinical course was complicated by infection-associated glomerulonephritis, confirmed with biopsy showing endocapillary and mesangial proliferative glomerulonephritis which required temporary hemodialysis, and severe tricuspid regurgitation. He required surgical tricuspid valve replacement along with ICD replacement following a 6-week course of IV antibiotic therapy for his endocarditis followed by oral amoxicillin

	for suppressive therapy prior to his tricuspid valve replacement procedure. Following these interventions and treatment of his endocarditis with antibiotics, the patient is currently attending cardiac rehabilitation and working with his cardiology team to further titrate his guideline-directed medical therapy for heart failure. Discussion: This case serves to highlight unique presentations of endocarditis with leukocytoclastic vasculitis and infection-associated glomerulonephritis, and it emphasizes the importance of maintaining a wide differential diagnosis and further evaluating for underlying systemic etiologies in patients with newly diagnosed leukocytoclastic vasculitis.
Anna Staudacher Dr. Sakshi Mishra Dr. Charles Bruen Dr. Elie Gertner	*VV-ECMO as Bridge Therapy for Life-Threatening Diffuse Alveolar Hemorrhage in Granulomatosis with Polyangiitis* Case Presentation: A 50-year-old man with hypertension and obesity presented with five weeks of cough, migratory arthralgias, and progressive dyspnea with scant hemoptysis, and tea-colored urine. He developed acute hypoxemic respiratory failure, and CT angiography demonstrated extensive symmetric bilateral ground-glass and consolidative opacities. He was intubated and bronchoscopy confirmed diffuse alveolar hemorrhage (DAH). Due to refractory hypoxemia (P/F 66.6, AC/VC, TV 390, PEEP 15) he was cannulated for VV-ECMO on hospital day (HD) 1. Infectious workup was negative. Rheumatology was consulted, and serologic workup revealed PR3-ANCA >160, supporting a diagnosis of granulomatosis with polyangiitis (GPA) in the setting of DAH and renal involvement. For remission induction, he received IV glucocorticoids on HD1–HD4 (followed by daily oral prednisone starting HD5) and rituximab (HD3, HD10, and HD17), with adjunctive plasma exchange (HD3–HD8). Creatinine rose from 1.66 on admission (baseline ~1) to 2.99, prompting CRRT initiation on HD4 for worsening AKI and hypervolemia. Given his high bleeding risk while on ECMO, a lower-intensity anticoagulation strategy was used, with a PTT goal of 40–60 seconds while on heparin. Following ECMO decannulation on HD8, he was found to have multiple deep vein thromboses (DVTs) and was transitioned to oral anticoagulation. He continued to improve and was discharged on HD19 with a steroid taper per the PEXIVAS protocol. At outpatient follow-up approximately four weeks later after completing five doses of rituximab, he had no recurrent dyspnea or hemoptysis. Renal function had improved, and he did not require iHD after discontinuation of CRRT (HD8). Discussion: Management of granulomatosis with polyangiitis (GPA) with diffuse alveolar hemorrhage (DAH) is particularly challenging when severe hypoxemia necessitates ECMO, as the anticoagulation necessary for ECMO may exacerbate ongoing bleeding. A recent systematic review of ECMO in rheumatologic cardiopulmonary disease reported 73.2% survival in ANCA-associated vasculitis and 100% survival (16/16) among patients with GPA supported with VV-

	ECMO for severe disease, including DAH (Falaas et al., ACR Open Rheumatology, 2025). This case aligns closely with the findings in the review. In this case, VV-ECMO provided critical respiratory support while immunosuppressive therapy controlled fulminant GPA-associated alveolar hemorrhage. The development of multiple DVTs further highlights the challenge of balancing pulmonary bleeding with thrombotic risk during extracorporeal support. Conclusion: Early recognition of GPA and multidisciplinary coordination among rheumatology, critical care, pulmonology, nephrology, and ECMO teams were essential to successful stabilization.
Emily Stickney Dr. Amy Holbrook	*Having a Blast(o) Kayaking* Introduction: Blastomycosis is a fungal infection typically caught from inhaling Blastomyces from soil or decaying matter. Blastomycosis can cause focal pneumonia that appears similar to community acquired pneumonia and is an important diagnosis to consider on the differential in CAP not responding to antibiotics. Case Presentation: A 27-year-old female with history of exercise-induced asthma presented to the Emergency Room for productive cough, fevers, chills, and post-tussive emesis. Chest X-ray showed possible consolidation, and she was treated with doxycycline for community acquired pneumonia. She presented to urgent care 6 days later with ongoing symptoms. X-ray showed worsened infiltrate in left lower lobe, her WBC was newly elevated to 13.6, and procalcitonin was 0.31; she was prescribed azithromycin and cefuroxime. Three days later she was directed to the Emergency Room for evaluation, given a lack of response to antibiotics. Chest CT showed patchy left lower lobe consolidation with numerous pulmonary nodules and ground-glass opacities and recommended consideration of fungal etiologies. Additional history notable for an event where a large amount of soil and debris had fallen onto her kayak while she was fishing in the Mississippi River, as well as clearing of decayed trees and frequent gardening. She was admitted for workup and management, and IV ceftazidime and vancomycin were initiated. Serologies and bronchoscopy testing for histoplasmosis, blastomycosis, coccidioides, and cryptococcus were obtained. She continued to fever intermittently to the 103s and was started on empiric itraconazole. Workup subsequently returned with positive blastomycoses and histoplasmosis urine antigens. Fungitell remained negative throughout illness. Bronchoscopy pathology was consistent with blastomycosis, and cultures eventually grew blastomycosis as well. Antibiotics were discontinued. She continued on itraconazole for 6 months with gradual improvement in symptoms and CT scans. The patient had sick contacts with mild viral symptoms preceding her illness, and post-viral bacterial pneumonia had appeared most likely initially. Chest X-ray and symptoms were very consistent with CAP.

	Important differentiating findings included nodular appearance on imaging suggesting possible fungal pathology as well as ongoing high fevers despite appropriate antibiotics, and of course positive fungal testing once this resulted. Discussion: Key points of this case include considering fungal etiology when CAP is unresponsive to antibiotics. ID's recommendation to start empiric antifungals was uncommon but warranted. This case illustrates the concept that high inoculum (large amount of debris falling on the patient) is associated with more severe disease and the importance of taking a detailed social history. "
Anna Strauss Dr. Joshua Trujeque	*Fungal Foes: Unmasking Tracheobronchial Aspergillosis in an Immunocompetent Host* Introduction: Aspergillus fumigatus is the most common fungal pathogen found in the lungs, and is often present in immunocompetent hosts without leading to invasive infection or symptoms. In these cases, Aspergillus fumigatus in a sputum culture usually represents host colonization and does not require treatment. When clinically significant invasive infections arise, called aspergillosis, they are typically opportunistic and occur almost exclusively in moderately to severely immunocompromised hosts. However, invasive tracheobronchial aspergillosis can present in patients who have only mild immunosuppression or critical illness, and may be treated as an invasive infection in these cases. Case Presentation: An 80-year-old male with a history of non-insulin dependent, well-controlled type II diabetes, as well as COPD on an inhaled corticosteroid, presented to the emergency department with generalized fatigue and poor appetite. He was found to be in diabetic ketoacidosis (DKA) and was admitted to the intensive care unit. Approximately 12 hours later, the patient developed distributive shock requiring vasopressor support. A broad infectious workup was obtained, including blood and sputum cultures, as well as whole-body imaging. The patient was found to have acute mesenteric ischemia and underwent multiple small bowel resections over the course of six days, for which he remained intubated between surgeries. After several days, his initial sputum culture began growing Aspergillus fumigatus. This was felt to represent colonization given the patient was not significantly immunocompromised, he had no infiltrates on chest imaging, and he had a stable respiratory status as he only remained intubated for surgical purposes. However, later in his course, the patient had a persistent leukocytosis and then developed hemoptysis, for which bronchoscopy was performed. This revealed multiple shallow tracheobronchial ulcers without active bleeding. With these new ulcers, the patient's presentation was now felt to represent invasive ulcerative tracheobronchial aspergillosis rather than colonization, and treatment for invasive fungal infection with voriconazole was started. The next

	day, he was successfully extubated but four days later required re-intubation at which time he was noted to have significant airway edema and mucosal inflammation, possibly related to his tracheal ulcers. A repeat bronchoscopy at that time showed changes to the tracheal mucosa suggestive of pseudomembranous tracheobronchial aspergillosis. At this time, amphotericin B nebulizers were added for further treatment. Despite this patient's classically opportunistic pulmonary infection, no underlying cause for significant immunosuppression was ever discovered. As such, and based on existing literature, it was believed that the patient's type II diabetes with associated DKA, inhaled corticosteroid use, and critical illness led to mild immunosuppression that allowed for the development of this invasive pulmonary fungal infection. Discussion: This case demonstrates a rare example of critical illness and an only mildly immunocompromised state allowing for the development of invasive tracheobronchial aspergillosis. Although uncommon, Aspergillus fumigatus in a host with only mild immunosuppression may represent active infection rather than colonization when persistent leukocytosis exists or tracheal ulcers are visualized on bronchoscopy. In these cases, treatment for invasive fungal infection may be warranted.
Cassandra Sundaram and Mary McConville	*Vegetation Virulence: Mysterious Mobile Intracardiac Masses in a Patient with Bacteremia and Malignancy* Introduction: For many clinicians, the presence of valvular vegetations in the heart is considered a fast-track to evaluation for an infectious process. In a patient without obvious infectious signs and lab findings, clinical suspicion should also be raised for non-bacterial thrombotic endocarditis (NBTE). NBTE is most often associated with hypercoagulable states including malignancy and autoimmune disease, with pathophysiology involving prothrombotic activation of the coagulation cascade amid endothelial injury. Determining the origin of intracardiac vegetations in the setting of malignancy and concurrent bacterial infection can be challenging. We present an interesting case of suspected malignancy associated NBTE in an 80-year-old patient found to have primary metastatic pancreatic cancer amidst confounding infectious findings. Case Presentation: An 80-year-old male with complete heart block with a pacemaker, persistent atrial fibrillation on anticoagulation with apixaban, HFpEF and history of pulmonary histoplasmosis was admitted for months of generalized fatigue, dyspnea and weight loss. His dyspnea and troponin elevation were concerning for acute coronary syndrome. After ruling out ACS, echocardiogram was obtained which showed multiple mobile intracardiac masses on both sides of the heart, including a mass seemingly adherent to the RV

	pacemaker lead, and small masses attached to the aortic valve. Of four blood culture sets (8 bottles) obtained, two bottles were positive: one for streptococcus mitis and one for streptococcus parasanguinis. The patient was started on ceftriaxone, but with only a mild leukocytosis and no physical exam findings concerning for infectious endocarditis sequelae (no vascular phenomena, no fever), his history of weight loss prompted ordering CT Chest/Abdomen/Pelvis. This showed widespread scattered lesions throughout the liver, lungs and pancreas concerning for malignancy. His prior history of confirmed pulmonary histoplasmosis also raised concern for disseminated histoplasmosis, as it can present with lesions in the liver mimicking malignancy, and, although rare, has been documented in a case of causal mitral valve vegetation. He underwent a nuclear tagged WBC study, which showed septic emboli in the lungs. The patient had a liver biopsy, which showed moderately differentiated adenocarcinoma, pancreatic in origin. Oncology was consulted and the patient began evaluation for palliative chemotherapy. Unfortunately, during his hospitalization, he developed acute limb ischemia of his right lower extremity while on apixaban, and two days later suffered a cardiac arrest and passed away. Discussion: This case highlights the diagnostic challenges of intracardiac masses with concurrent malignancy and infection. Although blood cultures and tagged WBC scan raised concern for infectious endocarditis, his subacute infectious presentation did not meet Duke criteria, and diagnosis of metastatic pancreatic adenocarcinoma and subsequent acute limb ischemia are most consistent with NBTE. However, the presence of septic pulmonary emboli in the setting of positive blood cultures prevents exclusion of bacterial contribution. Recognition of NBTE in patients is particularly important given the high risk of systemic thromboembolic sequelae and need for appropriate anticoagulation. Heparin products are most effective for anticoagulation in this population, as some studies have suggested treatment failure with warfarin or direct oral anticoagulants.
Brianne Vanderheyden	*A Breath of Suspicion: Pulmonic Valve Sarcoma Presenting as Pulmonary Embolism* Case Presentation: A 40-year-old woman with hypertension, moderate persistent asthma, gastroesophageal reflux disease, depression, anxiety, and tobacco use disorder presented with two weeks of right-sided pleuritic chest pain and dyspnea. On presentation, BP was 162/75 mmHg without hypoxia, tachypnea, or tachycardia. Examination revealed clear bilateral lung fields and reproducible right chest wall tenderness. CBC and BMP were unremarkable. CT chest for pulmonary embolism revealed a 1.4-cm lesion along the anterior pulmonic valve without pulmonary embolism. She was started on heparin for suspected thrombus. Transthoracic echocardiography showed no significant valvular pathology, although the pulmonic valve was poorly visualized. Subsequent cardiovascular magnetic resonance imaging with and without gadolinium demonstrated a mobile, smooth

	mass on the ventricular aspect of the pulmonic valve. She underwent pulmonic valve replacement and excision of the native valve. Intraoperatively, the anterior leaflet was "essentially replaced with a smooth sessile tumor extending to but not involving the pulmonary valve annulus." Her postoperative course was complicated by a right upper-lobe pneumothorax requiring chest tube placement. Pathology demonstrated low-grade myxoid sarcoma. She was discharged on dual antiplatelet therapy with oncology follow-up. Given the tumor's low-grade histology, oncology recommended surveillance CT imaging without chemotherapy or radiation. Eight years later, progressive prosthetic valve stenosis and right-sided heart failure prompted redo sternotomy and valve replacement. Cost and transportation barriers complicated achievement of a therapeutic INR, resulting in prosthetic valve thrombosis. PET-CT eight years and four months after initial presentation demonstrated intermediate uptake involving the prosthetic valve and outflow tract, favored to represent reactive or inflammatory changes following redo sternotomy. Discussion: This case highlights the rare presentation of myxoid sarcoma involving the right heart outflow tract, an exceedingly uncommon location with an estimated incidence of 0.001–0.03%, mean age at diagnosis of 45–54 years, and slight female predominance [1]. Myxoid sarcomas more commonly arise in the deep soft tissues of the extremities, particularly the medial thigh and popliteal fossa, where they typically present as painless, slowly enlarging masses [2]. Cardiac presentations are rare and may originate from the arterial intima or extend from the pulmonic valve into the pulmonary trunk [3]. Symptoms are often nonspecific and may mimic pulmonary disease, particularly pulmonary embolism, contributing to diagnostic delay [4]. This case emphasizes the importance of multimodal cardiac imaging when initial echocardiography is nondiagnostic, particularly the role of cardiac MRI for tissue characterization and anatomic definition of poorly visualized structures [5]. PET-CT may provide additional information for assessing tumor activity and distinguishing recurrence from postoperative or inflammatory changes, although some sarcomas demonstrate low metabolic uptake [6]. Management of myxoid sarcoma requires individualized multidisciplinary decision-making. Complete surgical resection remains the primary potentially curative approach, while the roles of chemotherapy and radiation depend on tumor grade, location, and extent of disease [7]. This case underscores the importance of coordinated care among cardiovascular surgery, cardiology, cardio-oncology, and sarcoma oncology, as well as consideration of longitudinal barriers to optimal postoperative management.
Samantha Vuong	*From Enteritis to Encephalopathy: A Case of Suspected Colchicine Toxicity*

Dr. Marie-Lisa Chrysostome Dr. Madeline Jenkin Dr. Remy Johnson Dr. Elie Salloum Dr. Yang Zheng Dr. Ben Weidemann Dr. Ellenor Chi Dr. Sorawit Ongsupankul Dr. Sumera Ahmad	Introduction: Colchicine toxicity is rare but potentially life-threatening and can initially present with nonspecific gastrointestinal symptoms before progressing to cytopenias and multiorgan failure. Case Presentation: A 35-year-old woman presented to the emergency department with several days of abdominal pain, vomiting, and diarrhea. She had a history of bipolar disorder, post-traumatic stress disorder, prior suicide attempts, pseudotumor cerebri status post right transverse sinus stenting, class I obesity, obstructive sleep apnea, polyendocrine metabolic ovarian syndrome, and nicotine dependence. She was undergoing evaluation for a presumed auto-immune inflammatory condition. On presentation, she was afebrile and normotensive but tachycardic to the 120s and tachypneic to the 20s on room air. Physical exam notable for diffuse abdominal tenderness. Initial laboratory studies demonstrated a white blood cell count 14.1 × 10^9/L, lactate 5.9 mmol/L, metabolic acidosis with an anion gap of 25, blood urea nitrogen 33 mg/dL, creatinine 2.70 mg/dL, aspartate aminotransferase 707 U/L, alanine aminotransferase 189 U/L, alkaline phosphatase 741 U/L, and creatine kinase 966 U/L. Computed tomography demonstrated infectious or inflammatory small-bowel enteritis. She received intravenous fluids and empiric ceftriaxone and metronidazole for presumed sepsis but subsequently deteriorated. She progressed to anuric acute kidney injury requiring intermittent hemodialysis, rhabdomyolysis, hematochezia, and abrupt thrombocytopenia, with platelet counts declining from 300 to 50 ×10^9/L within 24 hours. Markedly elevated lactate dehydrogenase of 2,429 U/L and D-dimer of 16,898 ng/mL prompted evaluation for disseminated intravascular coagulation (DIC) and thrombotic microangiopathy. However, a normal international normalized ratio, elevated fibrinogen of 695 mg/dL, absence of schistocytes, and only mildly reduced ADAMTS13 activity argued against these diagnoses. Computed tomographic angiography demonstrated worsening enteritis without mesenteric ischemia. She subsequently developed severe electrolyte abnormalities, hypertriglyceridemia, encephalopathy with hallucinations, and acute hypoxemic respiratory failure requiring intubation. Bone marrow biopsy demonstrated hemophagocytosis, raising concern for secondary hemophagocytic lymphohistiocytosis (HLH). She received pulse-dose methylprednisolone with improvement in inflammatory markers. Extensive hematologic and rheumatologic evaluation identified no convincing alternative etiology, and whole-genome sequencing for primary HLH revealed no pathogenic variant. A colchicine level of 5.1 ng/mL, drawn on hospital day five, raised concern for colchicine toxicity as a potential unifying trigger. She subsequently developed generalized tonic-clonic seizures, with magnetic resonance imaging of the brain demonstrating posterior cerebral edema consistent with posterior reversible encephalopathy

	syndrome. She was treated with lorazepam, levetiracetam, and lacosamide. Renal, respiratory, and neurologic function eventually improved. Discussion: This case highlights the broad and rapidly evolving manifestations of suspected colchicine toxicity. The initial gastrointestinal presentation and inflammatory profile suggested sepsis, while subsequent thrombocytopenia, hemophagocytosis, hypertriglyceridemia, and multiorgan dysfunction raised concern for HLH, thrombotic microangiopathy, DIC, and autoimmune disease. Multidisciplinary evaluation did not identify an alternative diagnosis. The temporal progression of symptoms, together with a detectable colchicine concentration, supported colchicine toxicity as a potential inciting event. Nevertheless, the absence of a clearly documented exposure history and delayed colchicine level measurement limited definitive attribution. Clinicians should maintain a high index of suspicion for drug toxicity when severe systemic illness evolves rapidly without an established cause, particularly when gastrointestinal symptoms precede cytopenias and multiorgan dysfunction.
Kyle Wansing	*Clot in Every Vessel: Systemic Thrombolysis for Catastrophic Biventricular and Multiorgan Thrombosis in Suspected Antiphospholipid Syndrome* Introduction: Catastrophic antiphospholipid syndrome (CAPS) affects fewer than 1% of patients with antiphospholipid syndrome and is associated with approximately 40% mortality during the acute event. Infection, particularly staphylococcal infection, is the most common trigger, likely due to molecular mimicry with β2-glycoprotein I (1,2). When CAPS leads to an intracardiac thrombus involving both ventricles, published literature is limited to isolated case reports. The underlying hypercoagulable state restricts the use of extracorporeal membrane oxygenation (ECMO), percutaneous ventricular assist devices, and surgical intervention, often leaving systemic thrombolysis as the only remaining therapeutic option (3). Case Presentation: We present the case of a 73-year-old man with rheumatoid arthritis treated with methotrexate, who was admitted for weakness and hyponatremia and was found to have transient pancytopenia. Bone marrow biopsy demonstrated regenerating marrow without evidence of malignancy. His hospital course was complicated by methicillin-sensitive Staphylococcus aureus (MSSA) bacteremia originating from a groin wound and progressive respiratory failure necessitating intubation. Within 24 hours of intubation, he developed cardiogenic shock: ejection fraction fell from 57% to 20%, with new thrombi filling both ventricles (right ventricular mass 2.6 × 2.0 cm), along with CT showing emboli extending from the thoracic aorta through the abdominal aorta into the iliac arteries, with bilateral renal infarcts. An

	intra-aortic balloon pump was ruled out by the aortic emboli, an Impella by the LV thrombus, and surgery by the overall clot burden. As the patient's shock worsened, with escalating pressor requirements and rising lactate, a multidisciplinary cardiogenic shock team, including cardiology, cardiothoracic surgery, rheumatology, and critical care, was rapidly convened. The team elected to administer systemic tPA, 50 mg over 6 hours, with heparin. This off-label use was considered a rescue option when other means of support became largely unavailable; thrombolysis has been used successfully off-label in APS in acute stroke with some success, but its use in this situation is largely unstudied (4). Repeat echocardiography the following day showed the ejection fraction had returned to 50–55% with markedly reduced clot burden. However, brain MRI performed the same day revealed new embolic strokes, and transesophageal echocardiography was deferred due to the bleeding risk shortly after thrombolysis. Rheumatology initiated empirical treatment for CAPS given the multiorgan involvement and history of rheumatoid arthritis. Although antiphospholipid antibodies were ultimately negative, the development of multiorgan, multi-vascular-bed thrombosis over several days (biventricular intracardiac thrombi, aortic thrombus, renal infarcts) with rapid progression to cardiogenic shock fulfilled the criteria for a ""thrombotic storm"" of CAPS, particularly when precipitated by infection such as MSSA bacteremia (5,6). The patient subsequently developed acute limb ischemia and recurrent respiratory failure. After discussion with his family, care was transitioned to comfort measures. He died on hospital day 20. Conclusion: This case suggests that systemic thrombolysis may be a reasonable option for biventricular thrombus at imminent risk of embolization when the clot burden precludes all device-based and surgical alternatives. Additionally, multidisciplinary teams can facilitate prompt decision-making in such difficult situations.
Jade Wexler Dr. Clayton Brady Dr. Upasana Agrawal Dr. Clement Michel	*Atypical Sweet Syndrome in a Patient with PR-3 ANCA Vasculitis and Psoriatic Arthritis on Immunosuppressive Therapy* Introduction: Sweet syndrome, or acute febrile neutrophilic dermatosis, is a rare inflammatory syndrome characterized by abrupt eruption of erythematous, tender nodules with neutrophilic infiltration of upper dermis on histopathology. While classically associated with neutrophilia and pyrexia, Sweet syndrome can exhibit extracutaneous manifestations. Given its association with many infections, malignancies, and inflammatory conditions, recognition can pose a diagnostic challenge. We report a case of Sweet syndrome occurring after purulent otitis media in a patient with PR-3 positive ANCA vasculitis and psoriatic arthritis treated with ustekinumab.

	Case Presentation: 52-year-old man was admitted for hyperkalemia secondary to missed dialysis sessions. On arrival, he was febrile, tachycardic, and noted to have a cough and a day of vesiculopapular rash, headache, and eye pain with neutrophilic-predominant leukocytosis. Medical history was notable for psoriatic arthritis treated with ustekinumab and PR-3 positive ANCA vasculitis diagnosed 7 years ago after initial presentation with rapidly progressive glomerulonephritis which was treated with steroids and rituximab. However, he progressed to end-stage renal failure requiring hemodialysis. He was recently treated with amoxicillin-clavulanate and underwent myringotomy for right purulent otitis media, which cultured Corynebacterium striatum. While the vesiculopapular nodules originated around the right ear, they quickly progressed to the trunk, face, and upper extremities. Ophthalmological exam demonstrated anterior scleritis with superimposed hemorrhagic keratitis, which was treated with prednisolone ophthalmic drops. Given rapid progression and his relative immunosuppression on ustekinumab, he was initiated on broad spectrum antimicrobial therapy with concern for disseminated herpes zoster. Skin biopsy of the nodules revealed a neutrophil-rich superficial dermal infiltrate, epidermal separation and partial thickness necrosis without vasculitis, consistent with Sweet syndrome. An extensive work-up for bacterial, viral, and fungal infections ensued. CT-Chest revealed a necrotizing, spiculated left upper lobe mass invading the mediastinum, suspicious for malignancy. However, CT-guided pulmonary aspiration revealed necrotizing granulomatous inflammation without dysplasia. DNA sequencing for myeloid neoplasms, UBA1 mutation (VEXAS Syndrome), and peripheral smear were unremarkable. Cultures and PCR of skin and pulmonary samples were negative for microbiologic growth. On admission, C-reactive protein was elevated at 265 mg/dL and he re-demonstrated high titers (>8.0) for PR-3 ANCA antibodies. With negative workup for infection and malignancy, his overall presentation was consistent with an atypical ANCA vasculitis flare and co-occurring Sweet syndrome. He was treated with high-dose IV steroids and rituximab with rapid clinical improvement and discharged home after a 3-week hospitalization. Discussion: This case highlights multiple challenges to diagnosing Sweet syndrome. Patients may exhibit extra-cutaneous symptoms, including eye involvement that may mimic other conditions. While Sweet syndrome is often associated with hematologic malignancies, it can also be seen in autoimmunity. Our patient's overlapping symptoms of ANCA vasculitis flare, history of psoriatic arthritis and recent otitis media demonstrates how Sweet syndrome not only occurs in the setting of autoimmunity but is likely predisposed by shared mechanisms of neutrophilic dysregulation. Clinicians should sustain a high degree of suspicion for Sweet syndrome given potential for overlapping presentations while also avoiding diagnostic anchoring

	and excluding infectious, malignant, and autoimmune triggers. Ultimately, diagnosis of Sweet syndrome remains challenging.
Abbey Whiting Dr. Leeore Levinstein	*Eosinophils Gone Wild: A Case of ANCA-Negative EGPA* Introduction: Eosinophilic granulomatosis with polyangiitis (EGPA), formerly known as Churg-Strauss syndrome, is a rare ANCA-associated vasculitis of small to medium sized vessels. Timely diagnosis is challenging not only because of the rare frequency of disease but also due to the variety of symptom presentations given its multi-organ system involvement. Here we present a case of EGPA affecting cardiac, pulmonary, and upper respiratory systems. Case Presentation: A 32-year-old male with a past medical history of asthma, recurrent sinusitis, and nasal polyps presented with persistent shortness of breath and worsening cough for three weeks despite treatment for community acquired pneumonia. On presentation to the ED, he was found to be hypoxic and tachycardic. Labs were notable for elevated WBC to 31 with an absolute eosinophil count of 17, high-sensitivity troponin elevation to 1012, and pro-BNP of 2,441. He was found to have extensive multifocal consolidations on CT of the chest. He was admitted to the hospital and initially started on broad spectrum antibiotics. On further workup with cardiac MRI, he was found to have myopericarditis with biopsy confirming eosinophilic infiltrates. Bone marrow biopsy without evidence of malignancy or hypereosinophilic syndrome. Rheumatology consulted, ultimately diagnosed with ANCA-negative EGPA with cardiac and respiratory manifestations. He started on high dose steroids with rapid improvement of symptoms. He was discharged home with a long steroid taper and was eventually started on rituximab. Subsequent follow-up with cardiac MRI and CT chest showed radiographic improvement along with persistent resolution of symptoms. Discussion: Patients with EGPA often fail to receive a timely diagnosis. Internists should be aware of potential manifestations of the disease along with early red flag signs of EGPA to trigger further investigation and prompt coordination among specialists. EGPA often mimics pneumonia. This specific case highlights how EGPA should be considered if there is lack of improvement with standard antibiotics, especially in the setting of additional extra-pulmonary symptoms such as cardiac, peripheral nerve, or skin involvement and less commonly involvement of GI and renal systems. Adult-onset asthma is a key manifestation in majority of EGPA patients and may precede other systemic manifestations by several years. Other prodromal presentations to be aware include chronic recurrent rhinosinusitis and nasal polyposis. As in this case, past medical history, multi-organ system involvement, and eosinophilia were major clues in prompting workup, timely diagnosis, and treatment of EGPA.
David Wong Dr. Armond Isaak	*Twinkling Artifact in Tumefactive Gallbladder Sludge Mimicking a Large Gallbladder Neoplasm*

Dr. William Miller Dr. Ahmad Malli	Introduction: Gallbladder masses identified on ultrasound often prompt concern for malignancy. Tumefactive sludge is an uncommon benign entity that can closely mimic gallbladder cancer, particularly when Doppler twinkling artifact is mistaken for internal vascularity. We present a case illustrating this important diagnostic pitfall. Case Presentation: A 41-year-old man with no significant medical history presented with his second episode of intermittent postprandial epigastric and right upper quadrant pain that progressed to constant pain with nausea and vomiting. He denied fever, alcohol or illicit drug use, recent illness, and prior abdominal surgery. He reported a 20-pound weight loss over the preceding year while taking a GLP-1 receptor agonist. On presentation, he was afebrile, hemodynamically stable, and had mild epigastric tenderness. Laboratory studies demonstrated mild leukocytosis with mixed hepatocellular and cholestatic liver injury (AST 298 U/L, ALT 200 U/L, alkaline phosphatase 144 U/L, total bilirubin 2.6 mg/dL, direct bilirubin 1.5 mg/dL). Lipase was normal, and HIV, RPR, and hepatitis A, B, and C testing were negative. Right upper quadrant ultrasound demonstrated a heterogeneous 7-cm intraluminal gallbladder mass with apparent internal color Doppler flow, adjacent gallstones and sludge, and a common bile duct measuring 5 mm, raising concern for gallbladder malignancy. However, contrast-enhanced CT and MRI obtained only four hours later showed no gallbladder mass or abnormal enhancement, instead demonstrating a distended gallbladder with biliary sludge and no evidence of acute cholecystitis. After multidisciplinary review with abdominal radiology, the apparent Doppler flow was determined to represent twinkling artifact rather than true vascularity, suggesting that the initial mass represented tumefactive sludge that had subsequently redistributed or disaggregated. The patient's symptoms improved with conservative management. Liver chemistries rapidly improved and were nearly normalized five days after discharge (AST 15 U/L, ALT 73 U/L, alkaline phosphatase 143 U/L, total bilirubin 0.7 mg/dL). He is scheduled to undergo elective outpatient cholecystectomy. Discussion: Tumefactive sludge is a recognized mimic of gallbladder malignancy, but apparent internal color Doppler flow from twinkling artifact may further increase suspicion for cancer. Typically, tumefactive sludge remains visible on subsequent CT or MRI, where lack of enhancement and diffusion restriction can help distinguish it from neoplasm. In our patient, however, the 7-cm lesion was no longer present on cross-sectional imaging obtained only four hours later. The rapid disappearance of the mass-like lesion likely reflects redistribution or disaggregation of tumefactive sludge, while his abdominal pain, transient liver enzyme elevation, and subsequent biochemical improvement suggest concomitant passage of sludge or

	microlithiasis causing transient biliary obstruction. Recognition of Doppler twinkling artifact and correlation with the clinical course and contrast-enhanced cross-sectional imaging can prevent misdiagnosis of gallbladder malignancy and avoid unnecessary invasive procedures.