BREAKOUT ROOM NUMBER ELEVEN CV 101 - 110
A MISSED EPIDURAL ANESTHESIA INDUCED BILATERAL SUBDURAL HEMATOMA

Epidural anesthesia is a pain relief modality used by most pregnant women in the United States. If the dura is punctured during the procedure, post-dural puncture headache could occur due to intracranial hypotension secondary to occult spinal fluid leakage which should be immediately recognized and addressed.

A 31-year-old woman with unremarkable past medical history underwent epidural anesthesia and had an uneventful vaginal delivery. Shortly after delivery, she developed a headache which continued despite treatment with caffeine and acetaminophen until day 4 when it was controlled by adding tramadol and oxycodone. Patient was then discharged home; however, her headache continued to worsen and gradually became associated with neck and bilateral shoulder pain. On day 13 postpartum, the patient's primary care provider attributed the headache to sinusitis and prescribed nasal steroid spray along with the acetaminophen which did not control the headache. On day 14, the patient was seen by her obstetrician-gynecologist who ordered a non-contrast MRI in response to the patient's insistence on the worsening nature of the headache. The MRI returned positive for a large bilateral subdural hematoma (SDH) with no midline shift. After 48 hours, the patient developed excruciating headache, double vision, drowsiness and intractable vomiting and presented to the emergency department where a non-contrast brain CT scan confirmed the diagnosis of bilateral SDH. Urgent epidural blood patch was done and the patient was given bedrest in the flat position and levetiracetam was started for seizure prophylaxis. On admission day 4, the patient was discharged home with dexamethasone and levetiracetam. At 1-week follow-up, the patient's headache had improved, and brain CT showed decreased hemorrhage and slowly improving mass effect. Steroids were tapered down and levetiracetam continued. At 2-months, repeat brain CT confirmed resolution of hematoma and the patient's symptoms had nearly resolved.

This case illustrates the hazardous impact of diagnostic bias on patient safety and alerts clinicians to consider rarer causes of headache such as SDH in cases of worsening headache after epidural anesthesia. Despite being potentially life-threatening, such events can be safely managed with conservative treatment if they are recognized and diagnosed early.

CV 101
BREAKOUT ROOM 11

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AMERICAN COLLEGE OF PHYSICIANS – MD CHAPTER

MULHOLLAND MOHLER RESIDENTS MEETING
TUESDAY, MAY 12, 2020

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VITAMIN C: A DAILY PILL TO KIDNEY ILL

Introduction: Vitamin C as a daily supplement is generally considered safe. It is metabolized to oxalate within the body. Oxalate nephropathy is a rare, but serious, condition that is associated with vitamin C intake.

Case presentation: A 46-year-old male with chronic kidney disease stage III (baseline Cr 1.4 mg/dL), diabetes mellitus type I, untreated hepatitis C, and iron deficiency anemia presented after his routine outpatient labs showed creatinine 14.9 mg/dL and hemoglobin 4.9 g/dL. He had generalized weakness and oliguria on admission but no other complaints. Physical exam was notable for left above-the-knee amputation with clean stump, and colostomy with increased output. Pertinent labs: creatinine 15.2 mg/dL, hemoglobin 5 g/dL, CO2: 14 mEq/L. Urine analysis showed +2 proteinuria, moderate blood, no casts. Renal ultrasound showed non-obstructive bilateral renal calculi. CT abdomen/pelvis showed tiny non-obstructing renal stones. Patient was treated with IV fluids and creatinine improved but plateaued at 10-11 mg/dL after which hemodialysis was started. Further work-up for his acute kidney injury including immune, infectious, and protein electrophoresis was non-diagnostic. Renal biopsy showed calcium oxalate deposits in the tubules, interstitial fibrosis, significant scarring and chronic diabetic nephropathy changes. Gastroenterology was consulted for concern for short bowel syndrome secondary to colostomy; however, that was determined unlikely. On further investigation, he denied ethanol glycol ingestion or any other medications not prescribed to him but reported taking vitamin C 500 mg daily for 4 years. Vitamin C supplements were discontinued. He remained clinically stable but continued to need hemodialysis after discharge.

Discussion: Vitamin C intake greater than 2 g/day can induce oxalate crystal nephropathy; however, the kidney injury may happen at lower doses, especially in patients with baseline renal impairment or association with other acute kidney insults. There are no clear guidelines regarding Vitamin C dosage in chronic kidney disease non-dialysis patients despite being dangerous to this group. The prognosis is guarded with almost half of patients being dialysis-dependent.

Conclusion: A cautious use of high doses of vitamin C should be considered in all patients given the rare but potentially devastating outcomes. Dosage adjustment should be considered in all patients with chronic kidney disease.

CV 102
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PUFFY LIMBS - ITS NOT ALWAYS THE HEART

Introduction: Remitting Seronegative Symmetrical Synovitis with Pitting Edema (RS3PE) is a rare syndrome that is a subset of the seronegative symmetrical polyarthritis of elderly initially described by Dr. Daniel McCarty et al. in 1985. There is no studied incidence rate due to rarity of the disease, but systemic literature review reported 250 cases of RS3PE from 91 articles from 1985 to 2014.

Case Description: 61 year-old male with hypertension presented with progressive swelling and pain of bilateral upper and lower extremities for 2 months. He denied exertional dyspnea or orthopnea. On exam, he had mild JVD and pitting edema of bilateral upper and lower extremities. Labs showed WBC 11.1, normocytic anemia with Hb 8.9, platelets 233, pro-BNP 1210, normal renal/liver panel, ESR 129, CRP 15.4. Chest x-ray showed mild pulmonary venous congestion. He was admitted for possible new-onset heart failure and treated with IV furosemide. However, echocardiography showed normal heart function. Rheumatology was consulted. Further evaluation revealed loss of appetite with unintentional weight loss of 35 lbs in 6 months and acute synovitis in 2nd/3rd MCP and PIP. He also had diffuse stiffness without involvement of shoulder or hip joints. Radiography of hands and feet showed osteopenia, mild soft tissue edema with no evidence of erosions. He was suspected to have acute rheumatoid arthritis flare and was started on prednisone. Other differentials included chronic inflammatory polyarthritis, polyarticular gouty attack, polymyalgia rheumatica, infectious arthritis, connective tissue disease or myopathy. Lab work at outpatient follow up showed negative ANA, Rheumatoid Factor (RF), anti-citrullinated peptide, HIV, Hep C, normal uric acid, CPK and Aldolase. Ferritin was mildly elevated. His pain and swelling significantly improved with steroids. Given his clinical presentation and workup, he was diagnosed with RS3PE and steroids were tapered. Age appropriate cancer screening was recommended.

Discussion: RS3PE is characterized by abrupt onset of symmetrical distal synovitis associated with pitting edema of the hands and/or feet, absence of RF and favorable response to corticosteroids. It is predominantly found in males. It does not have specific diagnostic criteria. Inflammatory markers are elevated with no evidence of erosion in radiography. Vasculo-endothelial growth factor has been found to be elevated contributing to pathological changes. It has been associated with concomitant malignancy (13.6%) and rheumatologic disease (2%). Prognosis is very good with low dose steroids except if associated with underlying malignancy. Age appropriate cancer screening is recommended specifically for steroid non-responders.

CV 103

BREAKOUT ROOM 11

ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
SEIZURE-LIKE-ACTIVITY AS A PRESENTATION OF EHLER DANLOS SYNDROME

Davis IC, MD and Fitch J, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Ehler-Danlos syndrome (EDS) is a group of genetic disorders that result in derangements of connective tissues. EDS commonly presents with joint hypermobility, joint dislocations, poor wound healing, atypical scarring and translucent skin. However, diagnosis is challenging as there are 13 subtypes of EDS with varying phenotypes and genotypes. The Hypermobile EDS subtype is defined by large and small joint hypermobility and can also be associated with autonomic dysfunction and syncope.

A 26-year-old woman with a history of well controlled Juvenile Myoclonic Epilepsy and postural orthostatic tachycardia syndrome (POTS) was electively admitted to the epilepsy monitoring unit for characterization of new, frequent seizure like activity. Her prior epilepsy was characterized by myoclonic jerking that started at age eight but had not occurred for ten years. For months prior to admission, the patient experienced absence-like episodes most mornings when rising from bed. She experienced a blank look and fluttering eyes that lasted seconds and without post-ictal confusion.

During her hospitalization, the patient was on continuous electroencephalography, electrocardiography and audio/video monitoring. Her antiepileptic medicines were withdrawn. After a routine blood draw, she intermittently lost consciousness with a weakly palpable pulse. Telemetry demonstrated sinus bradycardia, sinus pauses and a junctional rhythm. The episode was characterized as a vasovagal syncopal episode and consistent with the absence-like seizure activity experienced at home.

On physical exam, her rhythm was regular tachycardia and the remainder of her cardiopulmonary exam was normal. Exam of the joints revealed hyperextension of the fifth metacarpal joint past 90 degrees, apposition of the thumb to the flexor aspect of the forearm, hyperextension of the elbow past ten degrees, hyperextension of the knee past ten degrees and with active flexion of the trunk the palms rested flat on the floor. She was diagnosed with Hypermobile EDS with a 9/9 Beighton Score. Her presentation was explained by laxity in the venous systems, venous pooling and subsequently decreased preload to the heart resulting in syncope and compensatory tachycardia.

EDS is rare disorder however it should be considered in a patient presenting with syncope and dysautonomia that is not otherwise explained.

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ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
A RARE CASE OF DISSEMINATED CUTANEOUS ZOSTER WITH BACTERIAL SUPERINFECTION AND SEPSIS IN AN IMMUNOCOMPETENT PATIENT. Motwani K, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD

Varicella-zoster virus (VZV) may present with a painful, vesicular, single dermatome eruption, or with rapid progression involving multiple dermatomes and additional organ involvement. Immunocompromised patients are more likely to present with disseminated disease, but it is also seen in immunocompetent hosts, such as the one in this case.

A 63-year-old male with a history of lymphoma and prior stem cell transplant in remission presented to the emergency department (ED) with a painful vesicular rash involving the right V2 dermatome. He endorsed a right frontal headache, photophobia, and right eye erythema and pruritus. Neurological and ophthalmologic exams were benign. He was diagnosed with herpes zoster and discharged with 7 days of acyclovir and scheduled for an ophthalmology appointment the next day. The patient did not fill the prescription nor attend the appointment.

He represented to the ED 6 days later with worsening facial rash, vision loss, inability to open his right eye due to pain and edema, and throat pain. He was febrile to 39.2°C, tachycardic to 130 bpm, and blood pressure was 103/63 mmHg. The vesicular rash involved the right trigeminal dermatome, marked by erythema and edema. Additional lesions were noted on the right lateral flank, thigh, and right oropharynx extending along the soft palate. There was bilateral eyelid edema and crusting of the right eyelid limiting eye opening. Ophthalmologic exam of the right eye revealed conjunctival and scleral injection, corneal haziness, an irregular pupil, minimally reactive pupil, and right lateral gaze deficit. The left eye was normal. Laboratory values were significant for lactate 2.5 mmol, bicarbonate 16 mEq/L, and creatinine 3.7 mg/dL. MRI brain revealed right peripapillary enhancement, particularly of the posterior intraorbital optic nerve.

The patient had herpes zoster with superimposed bacterial infection. He received intravenous (IV) fluid resuscitation, 14-days of IV acyclovir, and 5 days of antibiotic coverage with cefepime and vancomycin, later switched to linezolid for renal protection. Ophthalmic moxifloxacin and methylprednisolone, and topical mupirocin were applied to the eyes and skin. Despite treatment he had decreased sensation of the right hemi-face, visual deficits and right gaze palsy.

This case illustrates that patients with VZV merit prompt attention and close follow up, as delays in treatment can result in diffuse organ involvement, bacterial super-infection, and even sepsis.

CV 105
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ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
ACCELERATED IDIOVENTRICULAR RHYTHM ALWAYS REQUIRES AN ELECTROCARDIOGRAM TO EVALUATE Na J, MD and Desai K, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Accelerated Idioventricular Rhythm (AIVR) is a wide-complex regular rhythm defined as 3 or more ventricular beats with a QRS complex greater than 120 milliseconds at a rate between 50-110 beats per minute. Its pathophysiology remains unclear, but the proposed mechanism involves the enhanced automaticity of a ventricular pacemaker with associated triggered activity that competes with the sinus node. [1] Usually well-tolerated and self-limiting, AIVR has been associated with drugs, such as cocaine, halothane, and digitalis, electrolyte disturbances, and cardiomyopathies. Importantly, this rhythm can also occur with acute myocardial ischemia or reperfusion of a previously occluded artery. [2] Thus, patients with AIVR should always be further evaluated especially if there is suspicion for an electrically silent infarction, such as that involving the left circumflex artery.

A 32-year-old man with diabetes mellitus and schizophrenia presented to the Emergency Room with substernal, non-radiating chest pain. Prior to admission, he had developed nausea with multiple episodes of non-bloody, non-bilious emesis. Although most of his history could not be further clarified in the setting of alcohol inebriation and marijuana use, initial evaluation showed a regular wide-complex rhythm on EKG and telemetry showed an alternating pattern between this and normal sinus rhythm. Initial troponin was 7.71 ng/mL, and he was promptly taken for left heart catheterization when subsequent troponin resulted as 28.2 ng/mL.

His left circumflex artery was found to be 100% occluded due to an extensive clot in an aneurysmal segment. Rheolytic thrombolysis achieved complete revascularization of the entire left circumflex system. Post-procedural transhoracic echocardiogram showed an ejection fraction of 35%, lateral wall akinesis, and posterior wall hypokinesis. After further stabilization, he was discharged on aspirin, clopidogrel, atorvastatin, lisinopril, and metoprolol with instructions to follow up at cardiology clinic.

In this case, the patient likely had vasodilatation and collateral flow causing reperfusion of his infarction, leading to AIVR on EKG. While he could not provide a classic history for acute coronary syndrome, early recognition of this rhythm prevented a delay to catheterization. This case highlights the subtlety of an acute occlusion of the left circumflex artery and that AIVR can be a crucial clue to ensure a prompt diagnosis of a myocardial infarction.

CV 106
BREAKOUT ROOM 11
A Case of Pancreatic Cancer presenting with Constipation in a Patient with Chronic Opioid Dependence

Introduction: Pancreatic cancer can present as diarrhea, with foul smelling stools due to exocrine dysfunction caused by malignancy. However, it can also present with constipation due to mechanical bowel obstruction.

Case Presentation: A 48 year-old male with past medical history of hypertension and chronic back pain, who was recently released from incarceration, presented with lower abdominal pain and constipation for over 3 weeks. He stated that he had been having very small, hard stools every 1-2 days, despite eating a full oral diet every day. The patient also reported a 1-day history of headache and 3-day history of chills. He denies nausea or vomiting, hematocritia. The patient had taken codeine, calcium carbonate, and omeprazole for his abdominal pain, but he did not experience any relief from his abdominal pain symptoms. He went to an outside institution for acute pain exacerbation and an abdominal x-ray was taken, but he did not stay to get the abdominal x-ray report.

Over the past 3 weeks while in incarceration, the patient had also received detoxification treatment for methadone (for which he had been taking for 16 months), consisting of clonidine and promethazine. The patient's abdominal pain temporarily worsened during methadone weaning but returned to his pain baseline after methadone weaning was completed.

On physical examination, the patient's abdomen was distended and tender to palpation in the lower abdomen. A digital rectal examination demonstrated hard stool in the rectum, without any visualized of bright red blood or melena.

The differential for this patient included fecalith impaction and abdominal obstruction, warranting imaging workup to assess for any malignant obstruction. A CT abdomen and pelvis revealed a hypodense lesion in the head of the pancreas and numerous hypodense lesions in the liver, suggestive of metastatic pancreatic cancer. The patient was discharged with follow-up plan his treatment course for pancreatic cancer.

Conclusion: Abdominal CT imaging should be considered in patients with new constipation and abdominal pain to rule out pancreatic cancer.
Steroids for Systemic Lupus Erythematosus, a Double-edged Sword

Introduction: Reversible posterior leukoencephalopathy syndrome (RPLS), also known as Posterior reversible encephalopathy syndrome (PRES) is a rare complication associated with systemic lupus erythematosus (SLE). It can occur due to the pathogenic changes caused by the disease itself or the medications used to treat it. Symptoms progress rapidly and can mimic other SLE complications which makes an accurate diagnosis crucial.

Case: A 25-year-old female with SLE and lupus nephritis diagnosed one year prior, nonadherent with medical therapy and follow-up, presented to the hospital with a 2-week history of generalized weakness and polyarthritis. Laboratory workup revealed a potassium of 8.2 meq/l, bicarbonate of 8.3 meq/l, anion gap 19 meq/l, blood urea nitrogen 130 mg/dl, creatinine 7.1 mg/dl, venous blood gas PH of 7.11, leukocytes 6 K/ul, hemoglobin 6.7 g/dl, C3 34 mg/dl, C4 7 mg/dl, positive antinuclear antibody screen, and anti-double-stranded DNA IgG antibody titers 1:10240. She was admitted for an SLE flare and was treated with pulse steroids for three days followed by a taper, mycophenolic acid and hydroxychloroquine. She responded well with improvement in her symptoms and renal function. On day 4 of her hospitalization she had two seizures four hours apart and was treated with phenytoin. Electroencephalogram (EEG) was consistent with partial focal seizures. Her mental status failed to improve despite adequate treatment and after ruling out status epilepticus. Lumbar puncture was unremarkable. Brain computed tomography and magnetic resonance imaging were suggestive of PRES. Blood pressure at the time was not significantly above her baseline hypertension, but she was managed with a nicardipine drip. Her mental status improved after four days.

Discussion: RPLS/PRES is a rare (prevalence 0.69%) but well known clinical-radiological diagnosis associated with SLE. It presents as encephalopathy, seizures, headache, and visual disturbances. Risk factors include young age at presentation, hypertension, lupus nephritis, heart failure with reduced ejection fraction, and leukopenia. PRES in lupus has been reported to occur during augmentation and discontinuation of steroid therapy.
Refactory Hyponatremia, A Case of Cisplatin Induced Salt Wasting
Sinai Hospital of Baltimore1
Sakshi, J. Burley, N. Adashek, M. Aghera, D

Introduction
Cisplatin is a common chemotherapeutic agent in oncologic practice, and is a component of multiple therapeutic regimens for malignancies ranging from lymphomas to germ cell tumors. However, it has significant toxicities including ototoxicity and nephrotoxicity. Nephrotoxicity is most commonly manifested as acute kidney injury (AKI). However, renal tubular acidosis and electrolyte abnormalities in the absence of AKI can occur as well. We describe a case of a patient who received cisplatin-based chemotherapy with resultant renal salt wasting syndrome (RSWS).

Case report
A 23-year-old male presented to the hospital with cough, malaise and left-sided testicular mass. Ultrasound of testis showed a 4cm left testicular mass. AFP and B-HCG levels were significantly elevated. CT chest, abdomen and pelvis revealed multiple lesions in lungs and liver concerning for metastasis. Left orchectomy showed choriocarcinoma. The patient was started on VIP (cisplatin, ifosfamide and etoposide) chemotherapy. Within 24 hours of initiation, the patient developed polyuria and severe hyponatremia. Despite aggressive volume repletion with IV normal saline, he became hyponatremic and hypotensive requiring pressors. On C1D4, total urine output was 14L, serum Na level was 120mEq/L, urine Na was 200mEq/L, serum osmolality was 282mosm/kg and urine osmolality was 656mosm/kg. Given the constellation of polyuria, hypoosmolar hyponatremia, hypotension, hypovolemia and elevated urine sodium levels, a diagnosis of cisplatin-induced RSWS was made. 3% NS was commenced and rate titrated on a day-to-day basis according to sodium levels. Trial of cetimidine was administered to prevent further cisplatin induced RSWS. The patient was eventually able to wean off of 3% NS and was transitioned to oral salt tablets. For the next chemotherapy cycle, cetimidine was given to prevent RSWS. The patient tolerated the next cycle without any electrolyte abnormality.

Conclusion
Cisplatin induced RSWS can be treated with 3% NS and Cimetidine. Cimetidine competes with Cisplatin for the tubular uptake at PCT and can potentially reduce risk of Cisplatin induced nephrotoxicity. In situations where Cisplatin is a necessity to treat cancer such as germ cell tumor, Cimetidine should be considered as an adjunctive treatment to reduce risk of nephrotoxicity.

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CV 109

BREAKOUT ROOM 11

ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
**ABSENT THORACIC PEDICLE SIGN, A CASE REPORT**

Michael Farrell, MD – ACP Member, David Heltzel, MD – WRNMMC, Bethesda, MD

**Introduction:** The absent pedicle sign is a subtle yet clinically important radiographic finding. Etiologies range from life-threatening metastatic neoplasia to the completely benign congenitally absent cervical pedicle. Within this spectrum, primary neoplasms such as osteoblastomas may result in significant morbidity if undiscovered.

**Case Presentation** A 26-year-old male developed upper back pain after heavy-lifting while on deployment. His pain progressed over two years despite over-the-counter analgesics, chiropractic adjustments, and physical therapy. Initial review of spine radiographs described grossly normal anatomy. Subsequent MRI showed a locally aggressive process of the left T9 posterior elements, including the pedicle and superior articular process, with borders obscured by inflammation. CT showed a mixed lytic and sclerotic lesion arising from a nearly unidentifiable pedicle and articular process. CT-guided core needle biopsy confirmed osteoblastoma. He underwent laminectomy, facetectomy, and pedicle resection followed by T7-T11 fixation and posterolateral fusion with good response. On retrospective review of the initial radiograph, there was absence of the left T9 pedicle cortical ring.

**Discussion:** This case highlights a nearly missed absent pedicle on radiograph and contrasts the clinical and radiologic features of three important etiologies to consider. As the treatments and prognoses are significantly different amongst the three, arriving at the right diagnosis depends on imaging evaluation and clinical correlation. Osteoblastomas, as demonstrated in this case, may arise as delayed diagnoses given their gradual progression in pain. Osteoblastomas may demonstrate the “flare phenomenon” on MRI, which is a perifocal T2 hyperintense signal obscuring the osseous margins due to soft tissue inflammatory changes. The asymptomatic nature of congenital absence of pedicle often limits discovery to incidental findings amongst a variety of ages. The diagnostic triad described by Wiener et al includes enlargement of the neural foramen, dorsal displacement of the articular pillar, and a dysplastic appearance of the transverse process. Metastatic disease predominates over age 50 and may present as pain from compression fracture, nerve root impingement, or cord compression. Metastatic disease typically has a more infiltrative or destructive appearance, often with a large soft tissue component and vertebral body extension.

**Conclusion:** The absent pedicle is a subtle yet important sign to recognize, and key features on imaging facilitate differentiation between benign and highly morbid and potentially lethal etiologies.

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