Medical Student Poster Competition

NM Scientific Chapter Meeting

November 5, 6, 7, 2015

Residents’ Committee
Alisha Parada, MD
Patrick Rendon, MD
Co-Chairs
Background: Currently, the most accurate diagnosis of osteomyelitis is gadolinium-enhanced MRI. Ferumoxytol is FDA approved for use as an iron replacement in chronic kidney disease patients, but is also highly paramagnetic and thus active on MRI. Ferumoxytol is selectively taken up by macrophages, and thus can be used in macrophage imaging. In osteomyelitis, there is evidence that iron contrast agents are more specific for active infection than gadolinium. Further, many patients with diabetic osteomyelitis have comorbid kidney disease and are not candidates for gadolinium due to risk of nephrogenic systemic fibrosis. Therefore, Ferumoxytol can be a potential diagnostic modality in patients with suspected osteomyelitis. Methods: Inclusion criteria for participation includes diabetic patients with suspected pedal osteomyelitis and 18 years or older. Exclusion criteria included septic, unstable patients, contraindication to MRI, pregnancy, or hemochromatosis. Patients fitting these criteria are identified by UNM radiologists to undergo standard of care MRI for osteomyelitis. Within 48 hours of standard of care MRI, patients are injected with 7 mg/kg iron, with a maximum dose of 510 mg. Within 12-36 hours of iron infusion, repeat MRI is obtained. Surgical resection patients have tissue analyzed by Pathology. Results: Patient 1 was found to have small osteomyelitis in distal phalanx by standard of care MRI. Pathology was negative for evidence of osteomyelitis. Patient was treated with 6 weeks of antibiotics. Osteomyelitis was not apparent on Ferumoxytol MRI. Patient 2 was found to have osteomyelitis in the distal 1st phalanx by MRI imaging. Pathology demonstrated osteomyelitis and abscess formation. Further quantitative analysis of imaging is pending. Patient 3 was found to have osteomyelitis in the distal phalanx by standard of care MRI. Pathology was not performed and the patient was treated with 6 weeks of antibiotics. Osteomyelitis was not apparent on Ferumoxytol MRI. Patient 4 was found to have osteomyelitis in second metatarsal by MRI. Pathology was not performed. Conclusion: Further studies are needed to determine if Ferumoxytol is more specific for active infection relative to Gadolinium. Given the wound healing issues of diabetic ulcers, bone biopsies are not always obtained for osteomyelitis. Thus, the decision to amputate is often made on the basis of imaging and clinical presentation. This makes specific identification of osteomyelitis on imaging paramount. The use of Ferumoxytol did not increase the incidence of SIRS, supporting the hypothesis that it does not increase infection.
Mechanism of lipopolysaccharide modulation of intestinal tight junction permeability in mice

Gut-derived bacterial lipopolysaccharides (LPS) play an essential role in inducing intestinal and systemic inflammatory responses and have been implicated as a pathogenic factor of necrotizing enterocolitis (NEC) and inflammatory bowel disease (IBD). The defective intestinal tight junction (TJ) barrier has been shown to be an important factor contributing to the development of intestinal inflammation. Despite its importance in mediating intestinal inflammation, the physiological effects of LPS on the intestinal epithelial barrier remain unclear. The major aims of this study were to determine the effects of physiologically relevant concentrations of LPS (0 to 1 ng/mL) on intestinal barrier function using an in vivo (mouse intestinal perfusion) intestinal epithelial model system. Intraperitoneal injection of LPS (0.1 mg/kg body weight), leading to clinically relevant plasma concentrations (0.3-0.6 ng/ml) caused a time-dependent increase in intestinal permeability in vivo. The LPS-induced increase in intestinal TJ permeability was mediated by an increase in enterocyte membrane to Toll-like Receptor 4 (TLR-4) expression and a TLR-4-dependent increase in membrane co-localization of membrane-associated protein cluster of differentiation 14 (CD14). LPS-induced increase in mouse intestinal permeability was associated with focal adhesion kinase (FAK) and Myeloid Differentiation Primary Response 88 (MyD88) activation; knockdown of intestinal epithelial FAK prevented the LPS-induced increase in intestinal permeability. LPS did not cause an increase in interleukin-1 receptor-associated kinase 4 (IRAK4) phosphorylation in MyD88 knockout mice. Our data show for the first time that LPS-induced increase in intestinal tight junction permeability was regulated by TLR-4 dependent activation of FAK-MyD88-IRAK4 signaling pathway.
What's Growing in the Belly: An Unexpected Case of Listeria monocytogenes Spontaneous Bacterial Peritonitis

Spontaneous bacterial peritonitis (SBP) is a condition that confers an increased risk of mortality in a cirrhotic patient admitted to the hospital. Escherichia coli and Klebsiella pneumoniae account for the majority of cases with an increasing prevalence also being seen with gram-positive cocci, and candida. Listeria monocytogenes is a gram-positive facultatively anaerobic bacterium that is a rare cause of SBP. The bacteria are intrinsically resistant to first line empiric treatment for spontaneous bacterial peritonitis. We present a case of 42-year-old afebrile male with Child Pugh C cirrhosis presenting with complaints of diffuse worsening abdominal pain and found to have profound hyperkalemia that responded to medical therapy. The patient received a total of 2 weeks of IV ampicillin, and was discharged from the hospital to complete a 2 week course of Bactrim DS BID followed by Bactrim SS daily for prophylaxis. The management of listeriosis differs from current SBP guidelines due to the inherent resistance to cephalosporins. While Listeria is an uncommon cause of SBP, it is associated with a high degree of mortality, but only by way of case reports. There are no current guidelines for the management of Listeria SBP, and the lack of this data make the management of these cases difficult.
The Clot: An Atypical Presentation of a Myeloproliferative Disorder

Introduction: This case describes an atypical presentation of portal and splenic vein thrombosis in the absence of decompensated cirrhosis. The presence of non-cirrhotic portal vein thrombosis should lead to consideration of hypercoagulable states including myeloproliferative disorders. Case Description: A 39 year old obese male with a history of non-alcoholic steatohepatitis (NASH), migraines, a remote peptic ulcer, and hypothyroidism presented to the Emergency Department with worsening nausea for one month, anorexia and 20 pound weight loss, and three days of upper abdominal pain. On admission the patient was normotensive and afebrile and physical exam was significant only for diffuse abdominal tenderness. Hemoglobin and hematocrit (16.9 and 50% respectively), platelets, and LFTs were all within normal limits and hepatic synthetic function was intact. His family history was significant for a clotting disorder in his paternal grandfather but the patient had no personal history of clotting. Abdominal imaging showed intra-hepatic portal vein thrombosis (PVT) with portal hypertension, splenic vein thrombosis (SVT) with splenomegaly, left upper quadrant varices, and a nodular liver concerning for cirrhosis. A liver biopsy was significant for steatohepatitis with only mild fibrosis. Further work-up included erythropoietin at the lower end of normal and a hypercoaguable work-up revealed a Jak2 v617f mutation concerning for an occult myeloproliferative neoplasm. The patient was discharged on anticoagulation and followed in the hematology clinic where he subsequently received therapeutic phlebotomy for occult polycythemia vera. Discussion: This atypical presentation of an occult myeloproliferative disorder (MPD) reveals the importance of a complete work-up for hypercoagulable states when acute PVT or SVT are not explained by other predisposing factors. The thromboses in this case were initially attributed to NASH, but the lack of significant cirrhosis or cirrhotic decompensation prompted further testing. An underlying prothrombotic factor is found in 80% of acute non-cirrhotic PVT cases that are fully evaluated for hypercoagulability. A Jak2 mutation is present in up to 35% of PVT cases and is now a major criterion for diagnosis of a MPD. While polycythemia vera, the most common MPD associated with the Jak2 mutation, classically presents with an elevated hemoglobin and hematocrit, this is not required to cause thrombosis. An occult MPD is defined by presentation with intra-abdominal thrombosis in the setting of normal blood counts and is diagnosed in 16.7% of all PVT cases. Additionally, hypersplenism due to acute thrombosis and subsequent portal hypertension may obscure elevated blood counts and overt disease. Identification of an occult MPD through Jak2 screening identifies patients who should be monitored for the development of overt disease and potentially changes management, which may include therapeutic phlebotomy, in addition to standard anticoagulation.
Tomas Cordova

Category: High Value, Cost-Conscious Care

Additional Authors: Keith Davis, Mary Lacy MD

Reducing Overutilization of Cardiac Telemetry through Targeted Education

A 70 year old male patient with history of Usual Interstitial Pneumonitis with unilateral left sided lung transplant done in 2012. Patient was on chronic immunosuppressive regimen of tacrolimus, mycophenolic acid and prednisone. Patient’s baseline creatinine level was 1-1.1. Patient was in his usual state of health when he was noticed to have gradual creatinine elevation to 1.8 in May 2014 and up to 3 in July, 2014 with estimated glomerular filtration rate 21. Patient’s tacrolimus level were mostly between 5-8, urine total protein/creatinine ratio of 0.3, urine microscopy was essentially bland at first and subsequently did show presence of decoy cells. Ultrasound kidneys was unremarkable. CMV status was negative, however serum BK virus was at 10 million copies/ml initially. Native renal Biopsy was done which showed active polyomavirus nephropathy, with visible viral inclusions, positive staining for SV-40 large T antigen, and associated tubular cell injury/necrosis and mainly mononuclear tubulitis. There was moderately severe interstitial fibrosis and tubular atrophy (about 40-45% that was out of proportion to the degree of global glomerulosclerosis (13%), and this was likely felt to be due to the polyomavirus nephropathy. Immunofluorescence was unremarkable. Patient’s mycophenolic acid was discontinued secondary to both the BK viropathy as well as leucopenia with continuation of his tacrolimus and prednisone. A course of leflunomide has been started at 10 mg once a day as well as Intravenous Immunoglobulin at 1gm/kg to be given monthly for three months. At present time, approximately two months after initiation of treatment, patient’s serum BK virus is down to 3.5 million copies, however creatinine has remained relatively stable at 2.6 with gfr of 24. BK nephropathy is an important cause of allograft dysfunction in renal transplant patients. In non-renal solid organ transplant and bone marrow transplant patients, renal dysfunction can occur and is often attributed to calcineurin inhibitor toxicity. However, a review of the literature suggests that BK nephropathy of the native kidneys is becoming an emerging problem in non-renal transplant patients.
Refractory Hypophosphatemia in Sunny New Mexico

Hypophosphatemia can have multiple etiologies, and in a hospitalized patient it is not an electrolyte abnormality that normally warrants much concern. However, severe, refractory hypophosphatemia is much less common and may be the only sign of a more serious underlying deficiency. A 43-year-old woman with a history of alcohol dependence presented to the emergency department with severe right upper quadrant (RUQ) pain, nausea and fever. She was admitted to the Medical ICU for septic shock secondary to cholangitis complicated by acute alcoholic hepatitis. Once stabilized, the patient was transferred to the floor for management of her acute conditions. At time of transfer the patient was afebrile with persistent nausea and RUQ pain. She was alert, oriented and answering questions appropriately. Physical exam showed scleral icterus, jaundice and scattered spider angiomas. Her abdomen was moderately distended and painful to palpation in the RUQ. Hepatomegaly was appreciated. Labs were notable for leukocytosis, transaminitis, hypoalbuminemia and multiple electrolyte abnormalities including hypokalemia, hypomagnesemia, hypophosphatemia and hypocalcemia. The patient’s corrected serum calcium was within normal limits and her renal function was normal. The patient’s electrolytes were repleted but a chemistry panel the next morning was significant for critically low phosphorus at 0.7 mg/dL. Her phosphorus was again replaced, and her serum phosphorus showed a transient response. Over the next few days, the patient’s phosphorus continued to decline, reaching a nadir of 0.1 mg/dL despite aggressive replacement. Aside from mild irritability, the patient remained asymptomatic, and denied bone pain or muscle weakness. Further work up revealed a parathyroid hormone level of 176 with a low ionized calcium, indicating secondary hyperparathyroidism concerning for vitamin D deficiency given her normal renal function. The patient was started on calcium replacement and a standard over-the-counter dose of vitamin D. Five days later, when her 25,OH vitamin D level returned measuring undetectably low, treatment with Ergocalciferol 50,000 IU twice weekly for six weeks was initiated. Once three treatments with high-dose vitamin D were given, the patient’s serum calcium and phosphorus levels began to stabilize. Severe vitamin D deficiency, leading to refractory hypophosphatemia is an uncommon diagnosis in the inpatient setting of New Mexico, where the sun shines 300 days of the year. In the setting of generalized electrolyte deficiencies, this patient’s low phosphorus levels were initially considered to be secondary to poor oral intake, refeeding syndrome, alcoholism and alcohol withdrawal. However, the severe, refractory nature of her hypophosphatemia prompted a more complete workup. This case illustrates the importance of considering vitamin D deficiency in the patient with refractory hypophosphatemia, as low phosphorus levels may be the first, subtle sign that a deficiency is present.
A rare case of cardiac tamponade secondary to peritoneopericardial fistula

Introduction: Communication between the peritoneal cavity and the pericardium is rare. Cases are described as either peritoneopericardial fistulas or herniations. The communication is categorized as either acquired (more frequent) or congenital. Acquired communications are described as complications of abdominal and thoracic traumas, surgeries or procedures. Congenital communications can be associated with chromosomal abnormalities. In patients with ascites, peritoneopericardial communication can allow the development of pericardial effusions, potentially leading to cardiac tamponade. Case Presentation: We present the case of a 41-year-old man with hepatitis C, active intravenous drug and alcohol abuse, with no known history of cirrhosis, and remote history of upper abdominal stab wound with exploratory laparotomy in the 1990’s, who presented with two months of abdominal and lower extremity swelling. He was noted to be dyspneic, tachycardic (120’s) and hypotensive (90/50). Physical exam revealed distant heart sounds, jugular venous distention, clear lungs and swollen abdomen with fluid wave. Labs revealed hyponatremia, renal dysfunction, thrombocytopenia, hypoalbuminemia and coagulopathy. Chest x-ray showed an enlarged cardiac silhouette and echocardiogram confirmed a large pericardial effusion with tamponade physiology. He underwent emergent pericardiocentesis with drain placement, an initial 1.5 liters of yellow fluid was drained. Paracentesis was done and fluid analysis revealed SAAG >1.1 consistent with portal hypertension. The combination of clinical, laboratory and imaging studies were consistent with a new diagnosis of cirrhosis with portal hypertension. Imaging with chest x-ray, echocardiogram, and PET CT did not reveal the fistula tract. The tract was only elucidated with SPECT with MAA which was ordered due to clinical suspicion of fistula. Discussion: Communication between the peritoneal cavity and thorax is unusual, with communication to the pericardium occurring in <1% of cases. This case presents an interesting etiology of a massive pericardial effusion in a patient with peritoneopericardial fistula from a prior upper abdominal stab wound in the setting of new a diagnosis of cirrhosis. Imaging with chest x-ray, echocardiogram, and PET CT did not reveal the fistula tract. The tract was only elucidated with SPECT with MAA which was likely more easily visualized with less specific imaging modalities.
A 61 year old male with past medical history significant for type 2 diabetes mellitus with diabetic neuropathy and depression presented with acute onset altered mental status (AMS) and weakness with leukocytosis and Acute Kidney Injury (AKI). Home medications, including gabapentin and tramadol, were stopped on admission to rule out medications as contributing to his AMS. He was found to have Methicillin-sensitive Staphylococcus aureus (MSSA) bacteremia. His mentation improved after 1 day of IV antibiotics and hydration, AKI began to resolve and leukocytosis was trending down. Overnight between days 2 and 3, the patient became increasingly agitated and had altered mentation. He was experiencing visual and auditory hallucinations that were paranoid in nature. His agitation was not controlled with low dose haloperidol or lorazepam and required 4-point restraints and increasing doses of sedatives. He remained intermittently agitated and obtunded for the following day. His WBCs continued to trend down with antibiotics and his creatinine was down trending with IV fluids. After consultation from psychiatry and careful review of the patient's symptoms and past medical history, it was determined that the patient was suffering from an atypical withdrawal from tramadol, possibly exacerbated by gabapentin withdrawal. Patient was started back on his home doses of gabapentin and tramadol. He remained obtunded but was arousable for the first 24 hours after restarting the medications. His mentation continued to improve to baseline over the next 48 hours with his home doses of tramadol and gabapentin. Tramadol was approved by the FDA in 1995 for treating acute and chronic pain. Tramadol is primarily a centrally acting opioid analgesic with effects on the µ receptors. But it also increases central serotonin and norepinephrine by mechanisms similar to SSRIs and NSRIs. Tramadol withdrawal has been estimated to occur in 1/100,000 patients using tramadol. Due to the primary opioid mechanism, most instances of tramadol withdrawal consist of classical opioid withdrawal symptoms including but not limited to GI symptoms (diarrhea, nausea, abdominal cramps), lacrimation, rhinorrhea and anxiety. It has been estimated that 1 in 8 tramadol withdrawals are atypical in nature with CNS symptoms, including paranoia, hallucinations and sensory disturbances. This atypical withdrawal is likely secondary to the mixed serotonergic and noradrenergic effects of tramadol. In our patient, his AMS, paranoia, and hallucinations were likely secondary to the abrupt discontinuation of tramadol on admission.
Susan Muraida, M.D.

Category: Quality Improvement/Patient Safety

Additional Authors: Percy Pentecost, MD and J. Rush Pierce, MD

USING PDSA METHODOLOGY TO DEVELOP AN INTER-FACILITY TRANSFER FORM THAT IMPROVES INFORMATION TRANSFER AND PATIENT SAFETY

Introduction: Research indicates that 15-20% of Americans have taken some form of supplement in the last 12 months. One study demonstrated over half of patients taking supplements don’t disclose because providers don’t specifically ask. Joint Commission requires documentation of supplements during medication reconciliation but the current EMR formulary does not contain a complete registry of common over-the-counter (OTC) supplements. Supplements must be tediously entered manually as miscellaneous medications. The lack of auto-populating makes documenting patient’s supplement use prohibitively difficult. This lack of medical documentation could result in adverse drug-supplement interactions. Our objective is to improve the documentation of supplements during electronic medication reconciliation. Process Mapping: The current system for documenting supplements fails in multiple ways, but three main pathways were identified for intervention. The areas for improvement included changing the electronic medication reconciliation process by creating an auto-populating folder of supplements. Educating providers about the importance of supplement documentation and instructing providers to directly ask patients about supplement use. Interventions: A computerized folder entitled “Supplements” was created in the EMR. This folder contains evidence based dosing of 65 supplements, 32 Chinese herbal preparations and 5 OTC proprietary herbal blends. The folder appears whenever the add medication tab is activated. When a supplement is selected from the folder, a dose, route, and frequency are automatically reconciled into the patient’s medication list. Fifty providers were polled regarding their attitudes toward supplements. 20% indicated that they did not perform supplement reconciliation and 68% indicated that supplement reconciliation was somewhat difficult with the current system. 23% responded that they were least concerned about patient safety and supplement use. Providers were educated about JCAHO requirements for supplement reconciliation and the possible dangers of common supplement-medicinal interactions. Providers were trained on how to use and document supplements with the new folder. In order to prompt patients to disclose supplement use providers were instructed to ask patients “In addition to your prescribed medications, do you use any supplements, vitamins, minerals, herbs, nutritional supplements or over the counter medications?” Measuring the Outcome: 27 supplements were documented as patient medications in the EMR the month before the interventions. Thirty one days after the interventions, 41 documented supplements appeared in patient’s medication lists. This represents a 51% increase in supplement documentation. Conclusion: Using a dedicated supplement folder that automatically populates dose, route, frequency and indication improves the documentation of OTC supplements in an outpatient primary care clinic.
AN INTERVENTION IN THE ASSESSMENT OF THROMBOPHILIA DECREASES INAPPROPRIATE TESTING

Introduction Thrombophilias occur as a result of a variety of inherited and acquired abnormalities. However, the risk for developing a venous thromboembolism (VTE) is not fully dependent on having an acquired or genetic abnormality. The question then remains, after a VTE, when is testing indicated for a potential thrombophilic pre-disposition? The main reasons to consider testing are to: a) Look for an underlying cause of an unprovoked VTE and b) assess the probability of a repeat event thereby guiding duration of anticoagulation therapy. In order to improve testing efficiency, the hypercoagulable panel order set (HCPAN), a set of 8 tests indicated for inherited thrombophilia, was instituted at the University of New Mexico at the outset of induction of the electronic medical record. However, we hypothesized that the HCPAN was being utilized outside of guideline-directed diagnostic utility. After our initial analysis of the data, we concluded that the majority of tests were being ordered outside of the recommended guideline. At this time, we implemented an intervention, eliminating the order set from the routine laboratory orders for subspecialties that ordered the most tests. Methods: An intervention was implemented to decrease the number of routinely “over-ordered” HCPAN tests per international guidelines. The HCPAN was removed from the general order sets in place of specified order sets (e.g. APLA panel) for medical disciplines with statistically higher usage (e.g. Rheumatology). We compared the number of panels ordered from 9/13-3/14 to those ordered from 9/14-3/15. This included adult inpatient and outpatient HCPAN tests. We conducted a manual chart review, evaluating risk factors for VTE, the rationale for ordering the HCPAN, and the level and service of the ordering provider. The protocol was approved by the Institutional Review Board for the UNM School of Medicine. Results: While preliminary data indicate minimal correlation with ordering the panel and the status of the thrombophilic event, we did decrease the number of panels ordered by approximately 57%. With a cost analysis, we concluded that the intervention saved the hospital $49,023. The results also indicated that although the overall number of panels ordered decreased, approximately 93% of those still being ordered were classified as ‘inappropriate’ as defined by our criteria. Conclusion: The data indicates that most HCPAN tests are ordered outside of recommended guidelines, including being ordered for a provoked VTE, in the presence of anticoagulant, or during an active VTE. Furthermore, our results indicate that the HCPANs are ordered as a whole when only a small number of tests were indicated. Although removal of the panel from the general routine order sets significantly decreased the number of panels being ordered, many of those being ordered are still not within guidelines, indicating a need for further quality improvement-based educational interventions.
An Alien Sighting in a New Mexico Axilla

A 41-year-old man with a history of Type II diabetes mellitus and hypertension presented with chest pain, fever, and pain to his left arm. The patient was diagnosed with both hypertensive emergency and sepsis, and was later diagnosed with a type 2 myocardial infarction (MI) secondary to hypoperfusion. His symptoms quickly resolved although the patient noted concern of a large mass under his left arm as the cause of the pain. He stated that the mass had been steadily growing for the past two years; he referred to the mass as his “alien baby” and used an elastic bandage to support the weight. There were no associated symptoms of chronic fever (only the aforementioned acute fever), chills, or weight loss but the mass did cause pain in the left arm at the site of attachment. On exam an approximately 8 cm polypoid, warm, pedunculated erythematous mass was found dangling from a 9.5 cm stalk attached to the left arm immediately distal to the axilla. It was non-tender to palpation and did not demonstrate fluctuance or induration. There was no left axillary lymphadenopathy. The mass was thought to be a source of infection and the patient was treated with doxycycline for a suspected cellulitis. An incisional biopsy taken in the Emergency Department suggested a fibrolipomatous variant of acrochordon (i.e., skin tag), but the differential was broadened to include plexiform neurofibroma, malignant nerve sheath tumor, and dermatofibrosarcoma protuberans because of concerns of sampling error in a mass so large. The patient also underwent computerized tomography (CT) scan to rule out any infiltrating lesions, which yielded a 12.6 mm, enlarged left axillary lymph node without other signs of infiltration. The patient later underwent sentinel lymph node biopsy and excisional biopsy of the mass. The mass was found to weigh 322 grams, measuring 17.5 cm in its greatest dimension. The surgical site healed without any complications and final pathology confirmed initial findings of a benign fibro-epithelial polyp (acrochordon). Acrochordons, or skin tags, are a common finding in patients. They occur in approximately 25% of the general population, but occur more frequently in the elderly and patients with metabolic syndrome, acromegaly, or colonic polyps. They are generally pedunculated, no larger than 1 cm, and occur most commonly on the neck, axilla, or groin. This case illustrates an uncommon presentation for a common dermatologic finding with special emphasis on the workup and expanded differential diagnosis. To our knowledge, an acrochordon this large is a rare finding and is uncommon in the dermatology literature.
Jules Ryan Tabilona

Category: Clinical Vignette

Additional Authors: Mary Ramirez, MD, Sarah Safadi, MD, Jennifer Jernigan, MD

An Unusual Case of Persistent Lactic Acidosis

J. Tabilona, M. Ramirez MD, S. Safadi MD, J. Jernigan MD Department of Internal Medicine, UNM Health Sciences Center, Albuquerque, NM  A 22-year old female with past medical history of diabetes mellitus type I, gastroparesis, depression, and cocaine and marijuana use was brought in by ambulance after being found semi-responsive by her mother following a possible suicide attempt. She had ingested “a handful” of acetaminophen, drank more than 10 servings of alcohol, and did not take her last dose of insulin. In the ED the patient reported that she also used cocaine the previous night. She complained of nausea and left-sided jaw pain. On physical exam, she was alert, tachycardic, and had moderate left-jaw tenderness. Her labs showed a normal WBC, glucose of 583, bicarbonate of 17, elevated anion gap of 17, low VBG pH of 7.28, and an elevated lactate of 3.5. Her alcohol level was 186. Interestingly, her urine drug screen, acetaminophen and salicylate levels were all negative. Imaging showed a periapical abscess involving her left lower molars. She was treated for diabetic ketoacidosis secondary to dental abscess and given insulin, fluids, and ampicillin/sulbactam. On day 2 of hospitalization, her lactate continued to be elevated as high as 6.0 despite aggressive fluid administration and normalization of her blood glucose, bicarbonate and anion gap. She did not meet SIRS criteria at any point during her admission, so sepsis was thought to be an unlikely. At this point, with no other explanation for persistent elevated lactate it was hypothesized that she may have ingested a drug such as synthetic cannabinoid, which does not show up on urine drug screens. This was suspected given the patient’s history of frequent marijuana use and her presenting symptoms of altered mental status, nausea and tachycardia. Her home medications were insulin, metoclopramide, and levothyroxine, none of which are reported to cause elevated lactate. Despite continuing IV fluids, her lactate remained elevated, fluctuating from 3 to 6.5 until day 4 of hospitalization, when she was ultimately discharged. There are multiple causal agents of lactic acidosis including sepsis, alcohol intoxication, diabetic ketoacidosis, and medications. Although our patient did not report using synthetic cannabinoids, it was suspected based on history. Synthetic cannabinoids are becoming more of a health concern due to increased recreation use and unpredictable potential toxicity. This case highlights the fact that this type of drug can cause elevated lactate. In addition, they have been reported to cause hyperglycemia, rhabdomyolysis, seizures, cardiotoxicity, acute kidney injury, psychosis, and even death. We recommend that clinicians maintain a high index of suspicion for these drugs in patients with suspected drug use, negative drug screens and unexplained clinical symptoms including elevated lactate.
Atypical, Atypical HUS: Rare Squared

Noopur Goyal, B.A. Biology, University of New Mexico School of Medicine, Albuquerque, NM Adrian Moretti, M.D., Resident Physician, Department of Internal Medicine, University of New Mexico School of Medicine, Albuquerque, NM Deepti Rao, M.D., Associate Professor, Department of Internal Medicine, University of New Mexico School of Medicine, Albuquerque, NM

Atypical hemolytic uremic syndrome (aHUS) is a rare disease, categorized along with thrombotic thrombocytopenic purpura (TTP), as thrombotic microangiopathies consisting of thrombocytopenia, microangiopathic hemolytic anemia (MAHA), and organ ischemia. It affects approximately two per one million adults annually. aHUS is typically associated with a genetic deficiency in complement proteins and is marked by uncontrolled activity of the alternative complement pathway, resulting in 25% mortality. In this case we highlight a rare genetic condition as the cause of aHUS. A 27 year old male with a history of cerebral palsy presented with acute hypoxic respiratory failure secondary to alveolar hemorrhage that was confirmed with emergent bronchoscopy and bronchoalveolar lavage. The patient was found to have thrombocytopenia, acute kidney injury (AKI) requiring dialysis, and a MAHA evidenced by schistocytes on peripheral smear along with markedly elevated LDH and low haptoglobin. He was started on high dose steroids and plasmapharesis for treatment of his thrombotic microangiopathy. With no significant improvement on initial therapy, further testing to differentiate TTP from HUS was performed, including assessing the ADAMS13 enzyme activity level, a plasma derived proteolytic enzyme that cleaves large multimers of von Willebrand factor. Labs indicated low levels of ADAMS13 and negative ADAMS13 inhibitor antibody levels, consistent with aHUS. He then began treatment with a recently FDA approved humanized monoclonal antibody, Eculizumab, that binds complement protein C5 and inhibits complement activation. Further workup showed complement factor H mutation and a rare plasminogen PLG genetic mutation, confirming aHUS diagnosis. This case illustrates a rare cause of thrombotic microangiopathy, aHUS, due to a rare genetic mutation. The patient’s brother passed away one month earlier from aHUS, further highlighting the genetic correlation. Although factor H complement mutation is most associated with aHUS, a PLG mutation adds to the rarity of this presentation. New research indicates that there are three known plasminogen deficiency mutations and a recorded pathogenic variant in several identified cases of aHUS. Plasminogen deficiency prevents thrombi degradation, causing ineffective coagulation, contributing significantly to aHUS pathogenesis. The findings of this case are significant as research of coagulation pathway genes, not only complement factor genes, are under consideration as an emerging cause in aHUS pathogenesis. This case highlights a rare cause of a rare disease.
An Unusual Cause of Severe Hypoglycemia

Introduction: As a class, quinolone antibiotics have been rarely associated with hypoglycemia in non-diabetic patients and the documented reversible episodes of hypoglycemia typically occur within the duration of action of the medication. In this case, the effect of the medication started later and persisted longer. Case Description: A thin 61-year-old man with a past medical history significant for schizoaffective disorder, bipolar disorder and recent community-acquired pneumonia (CAP) presented tremulous and confused after falling. A random blood glucose measurement taken following the fall was 26 mg/dL. The initial hypoglycemic episode was rapidly reversed with oral glucose tablets; however, the patient continued to suffer from recurrent hypoglycemia requiring intervention with intravenous dextrose and oral glucose until hospital day 4 when his blood glucose stabilized to normal. 

The patient’s c-peptide, TSH, fasting insulin, insulin antibody, sulfonylurea level and consyntropin stimulation test were all within normal limits. The patient had recently been treated for CAP with moxifloxacin and received his last dose of the medication 4 days prior to presentation. Discussion: Upon presentation, the origin of the patient’s hypoglycemia was unclear. He had no significant medical comorbidities including diabetes. His psychiatric illness had been stable on the same treatment regimen for many years and he was previously in an in-patient ward with a low likelihood of having been given insulin or any other hypoglycemic agents. Fluoroquinolones as a class have been shown to cause hypoglycemia by inappropriate activation of pancreatic beta cells resulting in increased insulin release. Considering the patient’s recent course of moxifloxacin, this was thought to be the most likely culprit. Among diabetic patients, especially those taking sulfonylureas, dysglycemia is a well-established side effect of fluoroquinolone antibiotics with moxifloxacin being most likely to cause hypoglycemia in these patients. Rarely fluoroquinolones have been shown to cause persistent hypoglycemia in non-diabetic patients. In non-diabetic patients, a handful of case reports have been published showing an association between levofloxacin and hypoglycemia. Moxifloxacin is much less commonly cited as a cause of hypoglycemia among non-diabetic patients. Additionally, a perplexing aspect of this case was the duration of time from discontinuation of moxifloxacin to appearance of severe, symptomatic hypoglycemia and the duration of hypoglycemia after discontinuation of moxifloxacin. The patient reported decreased oral intake the morning preceding presentation, which may have worsened any persistent underlying hypoglycemia and resulted in altered mental status and fall. Advanced age and reduced renal function are thought to be risk factors for the development of hypoglycemia among non-diabetic patients treated with fluoroquinolones. The patient had neither of these risk factors. This case adds to the base of literature regarding severe hypoglycemia resulting from moxifloxacin administration and brings awareness to the rare, but potentially fatal side effect of this commonly used medication.
Pulmonary embolism from venous thromboembolism is a common condition with a robust body of literature to support guidelines and treatment recommendations. Septic pulmonary emboli, although clinically and radiographically similar to venous thromboemboli, lack data to inform treatment decisions. Guidelines for treatment are based mainly on expert opinion and small case series or observational data. One area of controversy in treatment is anticoagulation. This case of a patient with likely septic pulmonary emboli provides an opportunity to review management decisions using the literature regarding septic emboli and septic thrombophlebitis.

A 33-year-old male with Type 2 diabetes and a history of recurrent neck abscesses presented with severe sepsis, thought initially secondary to community-acquired pneumonia. He presented with subjective fever, chills, and cough. On initial evaluation, he had elevated WBC of 18.7, elevated HR to 120s, and elevated lactate to 4.1. He had new-onset hypoxia with 6L oxygen requirement. Initial blood cultures grew MRSA and chest CT Angiogram on hospital day 2 showed numerous pulmonary emboli in the middle and right lower lobes, as well as multifocal pneumonia. Dopplers of lower extremities were without thrombi, and in consultation with Infectious Disease, it was decided that the bilateral emboli were most likely consistent with septic emboli. TTE and TEE were without valvular vegetations. CT of the abdomen to look for other sources of infection showed worsening septic emboli in the imaged thorax, left internal iliac vein thrombophlebitis, and probable abscesses in the left piriformis and right adductor longus muscles. The patient was treated with intravenous antibiotics and anticoagulation and discharged on these medications.

One clinical question that arose during the care of this patient was the safety of treating patients with septic emboli with anticoagulation. The primary concern centered on septic emboli to the brain resulting in stroke, with ensuing hemorrhagic transformation. A literature search revealed that this concern is primarily theoretical and is extrapolated from evidence from infectious endocarditis, and even these data are unclear. A recent case series of patients with septic pulmonary embolism found that only 27% had infective endocarditis as the source of infection, raising the question of the relevance of this extrapolation. Forty-four percent had skin and soft tissue source of infection, as is likely in this case. No literature supports the connection between septic thrombophlebitis and cerebrovascular accidents. Furthermore, a systematic review illustrated that anticoagulation is a useful adjunct to antibiotics in some patients with septic thrombophlebitis, as the mortality rate is low and the benefits may be significant. Lack of comparative trials precludes recommending anticoagulation to all patients with septic thrombophlebitis or septic pulmonary emboli, but based on our review, we feel that in many patients, the relative benefit likely outweighs any theoretical risks.
Richard Collier

Category: Clinical Vignette

Additional Authors:  Sosa, David; Pierce, John Rush Jr., MD; Lacy, Mary, MD

But Where was the Cat?

Introduction. Klippel-Trenaunay Syndrome (KTS) is a rare congenital vascular disorder that classically involves a combination of capillary malformations, venous malformations, and hypertrophy of bone and soft tissue, often of just one extremity. Common complications include lymphedema, anemia, and skin ulceration. It typically appears in infancy, though occasionally appears during childhood and rarely adulthood. Infectious complications have not been commonly reported but include bacteremia, usually secondary to vascular malformations in the gut, which can predispose to gram-negative sepsis following bacterial translocation. Case Description. A 30 year-old Navajo woman presented to the hospital with severe right flank pain, malaise, and nausea. She had a history of KTS with massive edema of the right leg. Though she had no fever, leukocytosis, or focal infection at presentation, she was tachypneic, hypotensive, and had profound acute on chronic anemia; in addition to volume resuscitation and transfusions, blood cultures were drawn and ultimately grew Pasteurella multocida. Though the patient had recently visited her family on the Navajo Nation and had peripheral contact with domesticated animals, she denied close contact including being bitten or licked by these animals. She responded well to 14 days treatment with IV Penicillin, though she remains chronically anemic and requires intermittent blood transfusions. Her past history included massive edema of her right leg that developed and progressed after four pregnancies. KTS was diagnosed in 2011 when imaging showed typical findings of severe, diffuse edema throughout the right lower extremity in the subcutaneous soft tissues and interposed within the muscular septae, infiltrative changes of fat consistent with lymphangiectasia, subcutaneous varicosities, and a heterogeneous, mottled appearance to the bone marrow throughout the right lower extremity without marrow enhancement. Discussion. Pasteurella multocida is a gram negative coccobacilli that is quite commonly linked with animal bites, as it is frequently found in the oropharynx of dogs and cats. It occasionally can occur following animal licks of nonintact skin. In cases with trivial animal exposure, an underlying disorder can be a risk factor. When acquired in a human host, Pasteurella most often causes a cellulitis, but can cause systemic infection as well as respiratory or meningeal involvement. Cellulitis is a known complication of primary lymphedema. Our patient did not have obvious cellulitis, but we postulate that her lymphedema put at her risk for entry of bacteria through skin that had become contaminated with Pasteurella from trivial animal exposure. This case is especially instructive in that it emphasizes (1) the importance of considering Pasteurella infections in patients even with trivial animal exposure, (2) patients with KTS may be at risk for bacteremia with gram negative organisms, and (3) considering KTS as a cause of adult-onset or unilateral lymphedema.
A Rare Tango of Pulmonary Renal Syndrome After Streptococcal Pharyngitis

Pulmonary renal syndrome (PRS) is a class of small vessel vasculitides that are characterized by the dual presentation of diffuse alveolar hemorrhage (DAH) and glomerulonephritis. While it has multiple etiologies, PRS subsequent to group A streptococcal pharyngitis has rarely been documented. A 36-year-old Native American man presented after a hyperacute development of hemoptysis and rapidly worsening respiratory distress. Upon admission from an outside facility, he was intubated and sedated and showed signs of tachycardia and hypertension with SaO2 of 88% on a ventilator. A history of smoking, hypertension, hyperlipidemia and gout was elucidated by chart review. Two weeks prior to admission, he was diagnosed with streptococcal pharyngitis and was prescribed penicillin. He had no history of recent travel, autoimmune, or clotting disorders. Physical exam showed mild generalized edema in his lower extremities. Chest CT showed diffuse centralized alveolar densities in both the lungs. Bronchoscopy revealed a bloody lavage, in addition to a diffuse, erythematous mucosa, findings consistent with DAH. Labs were significant for a creatinine of 1.78 mg/dL, blood urea nitrogen of 52 mg/dL and proteinuria as well as dysmorphic red blood cells on urinalysis. An extensive workup for autoimmune disease and an ongoing infectious process was performed. It was predominantly negative, but did demonstrate a WBC of 20.8 x 103 cell/μl, Hgb 12.8 g/dL, Hct 39%, albumin 2.5 g/dL, elevated anti-streptolysin-O (ASO) antibodies at 1270 IU/ml (normal < 408 IU/ml) and hypocomplementemia (C3=9 mg/dL and C4=8.9 mg/dL). Given the patient’s history of strep pharyngitis and a disease process consistent with autoimmune inflammation in the lungs and the kidneys, he was diagnosed with DAH with post-streptococcal glomerulonephritis. He was started on diuretics, high-dose intravenous Methylprednisolone, prophylactic antibiotics and plasmapheresis. The patient’s renal function worsened initially, but was restored after four sessions of plasmapheresis during the first week. The ASO levels also showed progressive decline with each session of serum exchange. The patient’s respiratory status improved and he was extubated successfully. Upon further questioning the patient admitted to poor compliance to penicillin therapy after strep throat. He recovered fully and was discharged home. This case describes a patient with unusual presentation of DAH after a recent bout of streptococcal infection. He had hemoptysis, decreased hematocrit and diffuse alveolar infiltrates-the defining triad of DAH. He also showed classic manifestations of post-streptococcal glomerulonephritis, which included oliguric acute renal failure, microscopic hematuria, edema and hypertension. The patient’s clinical course and lab findings led to the diagnosis of DAH with post-streptococcal glomerulonephritis. A thorough medical history and timely recognition of the autoimmune inflammatory nature of streptococcal infection despite negative rheumatologic workup is vital in diagnosing and managing these patients. It further illustrates the effectiveness of plasmapheresis and high-dose corticosteroids in treating DAH with post-streptococcal glomerulonephritis.
Methemoglobinemia after topical benzocaine during transesophageal echocardiogram

Introduction Methemoglobinemia is a rare cause of hypoxia that can be life threatening; it requires specific clinical suspicion for diagnosis. Methemoglobin is an oxidized form of hemoglobin that is unable to bind oxygen. Common precipitating agents are topical analgesics (lidocaine, benzocaine) and dapsone. Pre-existing hypoxic conditions such as anemia lower the threshold for developing symptoms. In patients with methemoglobinemia, true hypoxia is out of proportion of hypoxia reported by pulse oximetry and arterial blood gas. Low levels of methemoglobin may be asymptomatic; higher levels may be life threatening by causing seizures, respiratory depression, and shock. The blood turns a dark chocolate color, an important sign to increase clinical suspicion. Diagnosis is made by measuring methemoglobin level in the blood. Treatment is intravenous methylene blue, which reduces methemoglobin back to hemoglobin, which can transport oxygen. Treatment is generally effective within an hour. Patients may also benefit from blood transfusions. We present the case of a patient who developed methemoglobinemia after exposure to topical benzocaine used for analgesia prior to transesophageal echocardiogram. Case presentation A 57-year-old male with end-stage renal disease secondary to diabetes mellitus, status post renal transplant, presented to an outside hospital with abdominal pain. He was transferred to our university hospital for further evaluation. The patient was diagnosed with acute cholecystitis and underwent cholecystectomy. During the hospitalization the patient developed new-onset atrial fibrillation and was started on warfarin. The patient was discharged without complications. Three days later the patient was readmitted with severe abdominal pain, CT scan showed hemoperitoneum. To allow for reversal of anticoagulation, a transesophageal echocardiogram was performed to rule out cardiac thrombus. The throat was anesthetized with topical benzocaine prior to the procedure. Thirty minutes after the procedure the patient became altered with seizure-like activity. Pulse oximeter showed oxygen saturation 86-90%. Chocolate brown blood was noted in the arterial line. Arterial blood gas showed pH 7.22, pCO2 54, pO2 89; methemoglobin level was > 31%. The patient administered 100 mg intravenous methylene blue and improved. One hour later methemoglobin level was 6.1%, and eight hours later was normal at 1.5%. With no findings of cardiac thrombus, the patient’s anticoagulation was reversed and the patient’s hemoperitoneum stabilized. There were no further complications during the hospitalization and the patient was discharged four days later. Discussion Although methemoglobinemia is rare disorder, it is important to have the clinical knowledge of this disorder as it may be life threatening and is reversible. Internists are most likely encounter patients with exposure to topical lidocaine and benzocaine analgesics in the setting of upper endoscopic procedures. Methemoglobinemia should be a consideration of causes of altered mental status, respiratory insufficiency, seizures, in shock in patients with exposure to these medications.
A community-based approach to cardiovascular risk reduction in northern New Mexico

Introduction and background: Hypertension is the most common modifiable risk factor of premature cardiovascular disease and events including heart attacks and stroke. One in three American adults have hypertension and about half of those with a formal diagnosis do not have it controlled. A major challenge in reducing the prevalence of hypertension is that it is a chronic, progressive and asymptomatic condition. While hypertension is a serious medical condition, there are well-defined and effective ways to monitor and control it with medical and lifestyle interventions. Therefore, improving early detection efforts and access to and use of medical care has the potential to considerably reduce the rate of hypertension and its cardiovascular consequences. According to state cause-of-death records, New Mexicans under the age of 75 have increased mortality due to hypertension relative to national rates, most notably in Hispanic and Native American men. We hypothesize that limited access to preventive and primary medical care for non-senior adults is a significant factor leading to this and other cardiovascular health outcome disparities in the state. Current estimates of hypertension and cardiovascular disease burden in northern New Mexico are limited to data collected from national surveys like the Behavioral Risk Factor Surveillance System, hospital records and state death records. These epidemiological estimates have been found to underestimate disease burden in communities, particularly those with significant minority populations. Thus, improved, community-specific estimations of disease burden are necessary to accurately determine risk, and can profoundly impact the allocation of resources and support given to the community. Community blood pressure screening has been utilized as a public health strategy for decades. Recent studies on this method of intervention found that public screening coupled with direct medical referral is most effective in improving blood pressure control. In order to facilitate a reduction in cardiovascular risk, a clear route to medical follow-up is vital. We present our efforts in a community blood pressure screening, education and medical referral program called Impact Heart Health (IHH), a collaborative quality improvement (QI) community health project with the non-profit Impact Health New Mexico in Santa Fe. Methods and Results: To date we have screened 380 individuals for high blood pressure at 28 community events primarily in Santa Fe, providing individuals with heart health education and medical referral. In addition to direct services to community members, we evaluate the rates of hypertension in the community as well as risk factors of smoking history and access to primary care (presentation of results pending IRB approval). Conclusions: We present the structure of our ongoing community program as a successful model of community engagement and community-based participatory research in northern New Mexico that includes disease screening, health education, medical referral and assessment of risk factor burden.
Successful Inpatient Buprenorphine and Benzodiazepine Titration for Co-existing Alcohol and Opiate Dependence in a Patient with Torsades de Pointes and QT Prolongation

Successful Inpatient Buprenorphine and Benzodiazepine Titration for Co-existing Alcohol and Opiate Dependence in a Patient with Torsades de Pointes and QT Prolongation Eric Quintana, MS-III; Elyce Sheehan MD; Christopher Bailey DO; Seth Scott MD While it is known that buprenorphine is the preferred agent for opiate dependence in patients with prolonged QT, it interacts significantly with benzodiazepines by causing potentially significant respiratory depression. This makes treatment of patients with alcohol and opiate use disorders challenging. We report the safe initiation of buprenorphine in a patient who was simultaneously undergoing treatment for alcohol withdrawal. A 29 year-old female consented to voluntary treatment for alcohol and heroin dependence at the advisement of her primary provider and care team. She presented with a history of alcohol abuse, opiate dependence, and depression. She also had a history of QT prolongation with five episodes of torsades de pointes. An AICD was placed after suffering cardiac arrest in the setting of methadone treatment, which made methadone a contraindication. We planned to use titration of buprenorphine due to its safety profile in patients with QT prolongation. The patient had been using 1.5 pints of alcohol and 2 grams of snorted heroin daily prior to admission. Her initial QTc on admission was 469. She was successfully treated for her alcohol withdrawal with benzodiazepines using the standard inpatient CIWA protocol. Once lack of significant sedation from benzodiazepines was confirmed, the presence of opiate withdrawal was determined with a clinical opiate withdrawal scale. On hospital day two, buprenorphine was started at a lower than usual dose of two milligrams. We continued to increase her buprenorphine dosages incrementally to a target of 16 mg daily while the sedation from her benzodiazepines was decreased. During this transition, we monitored her cardiac rhythm with continuous telemetry monitoring and daily electrocardiograms, which showed a QTc range of 392-555. Simultaneously, her sedation and respiratory status were carefully monitored with continuous pulse oximetry and serial clinical exams, and the dose of buprenorphine was titrated accordingly. During her hospitalization, she did not show any cardiac ventricular arrhythmias or episodes of torsades de pointes. She was discharged home on hospital day five without signs of opiate or ongoing alcohol withdrawal. She was prescribed 16 mg of buprenorphine daily and was arranged for close follow up by her primary provider. In patients with coexisting opiate and alcohol dependence and prolonged QT, there is a risk of respiratory depression when treating simultaneous opiate and alcohol withdrawal due to an interaction between buprenorphine and benzodiazepines. In patients with opioid dependence and QT prolongation, buprenorphine is the safer option for treatment. However, in a patient with co-existing alcohol withdrawal, this should be titrated in a controlled monitored environment to optimize patient safety.
Kaitlyn McCranie

Category: Clinical Vignette

Additional Authors: Alisha Parada MD FACP, Barbara Welcer RN, Mark Lee MD FACP

**Pain Together: an Integrative Approach to Chronic Pain Management via Group Medical Visits**

Chronic pain is defined as pain which has persisted for longer than the expected time course and which is non-malignant in nature. Although there is a relative paucity of epidemiological data regarding chronic pain in the United States, some estimates suggest that chronic pain affects up to 55% of people during their lifetime. Consequently, managing chronic pain patients in the outpatient setting has been challenging to healthcare providers often in connection with challenges in the safety and efficacy of prescribing narcotics. Interdisciplinary care has been promoted since the 1950’s, but little research has been done using the group medical visits (GMVs) model to manage chronic pain. We describe an integrative medicine-GMV model as an ideal vehicle for efficient and cost effective healthcare delivery for chronic pain patients. At the UNM Center for Life, we piloted our model with chronic pain patients to encourage pain prevention and promote patient self-care in a cost effective setting. Utilizing a single-provider model, our curriculum offers several techniques for the management of chronic pain, including acupuncture, guided meditation, and lifestyle changes with the goal of minimizing the use of controlled drugs. Inherent to the GMV model, more cost effective and efficient care is provided, while encouraging social support and interaction. During each weekly visit, patients took their own vitals and participated in a group learning activity on a set of curricular topics. In the single provider model, each patient met briefly with a provider for individual assessment, plan and E&M Coding. Group visits were structured into 8 week synchronous cohorts, with formal assessments utilizing a validated SF-12 survey of physical and mental wellness at the first and last visit. Attrition rate was measured and feedback from participants was collected. Our pilot study recruited 27 patients over 3 cohorts in February-April, April-June, June-August 2015. Seven of 27 participants attended both the first and last sessions with variable attendance from week to week. Preliminary survey data suggested that the integrative medicine-GMV model for chronic pain was well received by patients, with specific comments on the benefit of the GMV providing a forum for social support. Providers described positive experiences in chronic pain management while maintaining a cost effective E&M productivity. The quantitative efficacy of our integrative medicine group medical visit model is still under study. Based on our initial experience with our pilot, patients and providers perceive it as a potentially effective model to deliver cost effective integrative medicine healthcare to serve the needs of chronic pain patients.