Purpose: Background: In addition to the positive therapeutic impact of anti-tumor necrosis factor (anti-TNF) therapy in the treatment of inflammatory bowel disease (IBD), there are adverse effects of which the clinician must be aware. One of these adverse events, recurrent shingles caused by the varicella zoster virus (VZV), can be not only frustrating but also life-threatening. Although the current literature is sparse, there is literature stating a two-fold increased incidence of shingles in patients on anti-TNF therapy, although it is not statistically significant (Failla et al). Literature exists supporting vaccination against VZV in IBD patients who are initiating anti-TNF therapy (Rahier, et al). However, to our knowledge, this is the first case report addressing the role of antiviral suppressive therapy in an IBD patient with recurrent VZV infection on anti-TNF therapy. Case: A 52-year-old male with a 15-year history chronic ulcerative colitis (CUC) was initiated on anti-TNF co-therapy due to persistent symptoms of CUC, despite treatment with standard therapies. Prior to the onset of anti-TNF therapy, his physical examination, routine chemistries, CBC and stool studies were normal. His most recent colonoscopy showed diffuse mucosal granularity, edema and loss of vascularity throughout the colon. Multiple biopsies were consistent with active chronic colitis. Adalimumab was initiated in addition to mesalamine for CUC. Adalimumab, loaded as per protocol, was well-tolerated and was subsequently scheduled for maintenance dosing every other week. The patient’s symptoms improved. However, prior to receiving his second maintenance dose of anti-TNF therapy, the patient developed a pruritic vesicular dermatomal rash on his back consistent with shingles. The patient endorsed a remote history of chickenpox, but denied a prior history of shingles or vaccination for VZV. At this point, anti-TNF therapy was held and the patient was treated with famciclovir 500 mg three times a day for 7 days. The patient recovered from his VZV outbreak and anti-TNF therapy was reinitiated. However, his dermatomal rash returned 8 weeks after reinitiation of anti-TNF therapy, necessitating retreatment with famciclovir and cessation of his anti-TNF therapy. The patient elected to resume anti-TNF therapy in conjunction with antiviral suppressive therapy. Acyclovir 400 mg, twice daily, was initiated in conjunction with adalimumab. To date, the patient has had no further recurrence of VZV outbreaks. Conclusion: Although further case series are needed, this case supports the consideration of use of antiviral suppressive therapy for recurrent VZV infection in patients with IBD on anti-TNF therapy.

Methods: N/A
Results: N/A
Conclusion: N/A

CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease
PRESENTATION TYPE: Oral or Poster

AUTH DESIGN: ACG Membership Status <font color="red">*</font>
Cari Sorrell : ACG Non-Member
Victor Torres : ACG Member

ACG Research Grant Support: No
Supported by Industry Grant: No
Commercial Products or Services: No
Initiated Research: Investigator
Financial Relationships: Not Applicable
FDA Approval: No
Designed Study: Investigator
Abstract Author: Investigator
Yasmin Alishahi : ACG Member
Tisha Lunsford : ACG Member
(No Image Selected)
(no table selected)

**AVERAGE SCORE:** 4.75

**REVIEWER FLAGS:** (none)

**REVIEWER RECOMMENDATION CODE DESCRIPTION:** None

**REVIEWER COMMENTS:**
Somashekar Krishna: [No Comments]
Julia LeBlanc: [No Comments]
Girish Mishra: [No Comments]
Rayburn Rego: [No Comments]
Purpose: Introduction: Interstitial pneumonitis as an adverse effect of mesalamine therapy is a rare complication with no documented cases of respiratory failure. Patients typically have a mild disease course and may present with non-specific clinical signs and symptoms. Case Presentation: A 65-year-old man presented with intermittent low-grade fever, non-productive cough and exertional shortness of breath for a period of one week. The patient had been diagnosed with ulcerative colitis (UC) two weeks prior during an outpatient evaluation for chronic bloody diarrhea. He was started on therapy with mesalamine 1.2 gm four times daily. On physical examination, the patient was febrile, tachycardic and tachypneic. Arterial blood gas values revealed a pH of 7.50, pCO2 of 20 mm Hg, PO2 of 70.7 mm Hg and bicarbonate level of 19 mmol/L on 2L oxygen. Computed tomography of the chest showed bilateral lower lobe infiltrates with trace pleural effusions [Figure1]. Mesalamine was discontinued and intravenous antibiotics were initiated. However, the patient developed worsening respiratory distress and was intubated later the same day. A transbronchial biopsy was performed, which revealed interstitial lymphocytic infiltrates, few histiocytes and mild fibrosis. A diagnosis of drug-induced interstitial pneumonitis was made and intravenous corticosteroids were started. The patient showed gradual clinical improvement, was extubated four days later and discharged on oral prednisone. Discussion: The exact pathophysiology of mesalamine-induced lung injury is not known. It is important to distinguish pulmonary manifestations in patients with IBD secondary to drug-related toxicity, as opposed to the disease process itself. The most important aspect of treatment involves the safe discontinuation of therapy with mesalamine. The role of steroids is unclear. Given the complexities in diagnosis and management, it is essential that a joint evaluation be carried out by a multi-disciplinary team.

Methods: N/A
Results: N/A
Conclusion: N/A

CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease
PRESENTATION TYPE: Oral or Poster
ACG Research Grant Support: No
Supported by Industry Grant: No
Commercial Products or Services: No
Initiated Research: Investigator
Financial Relationships: Not Applicable
FDA Approval: No
Designed Study: Investigator
Abstract Author: Investigator

AUTH DESIG: ACG Membership Status: Albin Abraham : ACG Non-Member
Ali Karakurum : ACG Member
Computed Tomography of the chest with intravenous contrast showed the presence of bilateral lower lobe infiltrates and trace pleural effusion.

**IMAGE CAPTION:** Computed Tomography of the chest with intravenous contrast showed the presence of bilateral lower lobe infiltrates and trace pleural effusion.

(no table selected)

**AVERAGE SCORE:** 4.75

**REVIEWER FLAGS:** (none)

**REVIEWER RECOMMENDATION CODE DESCRIPTION:** None

**REVIEWER COMMENTS:**
Somashekar Krishna: [No Comments]
Julia LeBlanc: [No Comments]
Girish Mishra: [No Comments]
Rayburn Rego: [No Comments]
Purpose: Case: A 26-year-old man with recurrent perianal fistula presented in August, 2012 with a one-month history of bloody diarrhea, abdominal pain and 10 Lb weight loss. He had been previously healthy, and did not take any medication. Further investigation at that time revealed Hb 11.4, MCV 91, WBC 14.8, PLT 309, CRP 24.8, normal electrolytes, creatinine and LFT, negative stool C&S, O&P and Clostridium difficile. Colonoscopy demonstrated moderately severe and continuous mucosal inflammation from rectum to cecum, with normal terminal ileum. Colonic biopsies demonstrated chronic active inflammation with architectural distortion, consistent with IBD. He was started on Asacol, prednisone and imuran. He responded well, but started to flare (Harvey Bradshaw Index=5) when prednisone was tapered to 20 mg per day. He quickly responded with an increased prednisone dose, but bloody diarrhea and abdominal pain recurred (HBI=12) when prednisone dose was tapered over the following 4 weeks. Stool was again negative for C&S and C. difficile. He needed to go on a biological agent, but his insurer refused to cover the cost. The discussion of fecal microbiota transplantation (FMT) as an experimental therapy was brought up with the patient as an option, and he agreed to proceed. He underwent FMT (universal donor) by colonoscopy, which demonstrated diffuse moderate mucosal inflammation, more severe in the proximal than the distal colon, and normal terminal ileum (Photo 1). Colonic biopsy subsequently ruled out CMV. Approximately 400 cc of fresh fecal suspension was infused in the cecum. Two days after FMT, he started to have 2-3 soft BMs per day, and his abdominal pain started to improve. Two weeks after FMT, his HBI was 0. The patient decided to pursue further FMT therapy as his maintenance therapy, and had a second FMT by colonoscopy four weeks after initial FMT. It showed complete mucosal healing (Photo 2). He continued to do well six weeks after the second FMT, and had the third FMT eight weeks after the second FMT.

Conclusion: To our knowledge, this is the first report of complete mucosal healing following a single FMT infusion in an IBD patient. These results suggest that FMT may be a promising therapy in a subgroup of IBD patients. Further research is needed to further characterize the IBD population most likely to benefit from FMT.
REVIEWER COMMENTS:
Purpose: Mesalamine and other 5-ASA compounds remain the first-line treatment modality for induction and maintenance of remission in patients with mild to moderate ulcerative colitis (UC), however up to 8% of patients are unable to tolerate these agents due to allergy, intolerance or idiosyncratic reactions. Rarely, neurologic sequelae result from the use of these compounds. We report the case of a 19-year-old woman with UC who developed sensorimotor neuropathy on mesalamine therapy. The patient initially presented with bloody diarrhea and abdominal pain. She was not taking any medication at presentation and reported a childhood allergy to Pepto Bismol ® (bismuth subsalicylate) causing dizziness and weakness. On exam she appeared in mild distress with voluntary guarding and tenderness to palpation in the left lower quadrant. Colonoscopy revealed features of colitis extending from the rectum to descending colon and biopsies were consistent with features of UC. The patient was started on oral mesalamine 800mg TID. She was reevaluated six weeks later when she reported new symptoms of progressive dizziness and bilateral lower extremity weakness since initiating therapy. Her gastrointestinal symptoms were mildly improved but still active. The symptoms of dizziness were worse in the mornings associated with near-syncopal episodes. Bilateral lower extremity weakness was described as a sensation of "leg heaviness" necessitating use of a cane for walking. Physical exam was notable for bilateral lower extremity weakness with evidence of slowed gait and episodic unsteadiness. There was no evidence of overt cranial nerve dysfunction or changes in upper extremity sensation or strength. No further infectious source or micronutrient deficiency was detected. In light of these findings, mesalamine was discontinued and 40mg of oral prednisone was started to treat her gastrointestinal symptoms. Within two weeks, the dizziness and muscle weakness had completely resolved. At that time, Azathioprine 50mg daily was initiated and the prednisone was tapered. On subsequent visits to the clinic, the patient was asymptomatic and tolerating the new regimen. The literature regarding 5-ASA induced neuropathy is limited to few case reports with patients manifesting predominantly sensory symptoms. Our patient is unique in that she developed evidence of sensorimotor neuropathy with brisk resolution off mesalamine. Given the patient’s history of significant allergy to another salicylate containing compound and no signs of infection or micronutrient deficiency, mesalamine was strongly suspected as the culprit of her new symptoms.
AVERAGE SCORE: 4.25
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Somashekar Krishna: [No Comments]
Julia LeBlanc: [No Comments]
Girish Mishra: [No Comments]
Rayburn Rego: [No Comments]
Purpose: Flexible sigmoidoscopy is recommended over colonoscopy in severe or fulminant ulcerative colitis (UC) due to concerns for colonic perforation or toxic megacolon, although the magnitude of this risk over that of screening colonoscopy is unknown. Here we report the use of water exchange colonoscopy to safely evaluate three patients with severe ulcerative colitis. Cases (Table 1): The water exchange method was performed as follows. Air pump was turned off. Upon entry into the rectum air was aspirated. The tip of the scope was placed in close proximity to the slit-like opening of the next bowel segment. Water was infused to confirm the orientation of the scope; as the lumen opened, the scope was advanced. Infused water was suctioned during insertion to remove fecal material that impaired visualization and to minimize distension. Air pockets were suctioned to minimize angulations of the flexures. The scope was withdrawn to regain view of the lumen whenever the scope tip was too close to the mucosa. Discussion: Using the water exchange method, colonoscopy was performed in each case by supervised trainee endoscopists without complications despite severe colitis. Two of the 3 patients were examined to the cecum without sedation. Conclusion: Water exchange colonoscopy was safely performed in severe UC patients, and provided a more thorough examination compared to flexible sigmoidoscopy.
Case 2: Inflammation, ulceration of transverse colon (water-filled lumen).

Case 3: Colitis, pseudopolyps near ileocecal valve.

**IMAGE CAPTION:** Case 1: Severe inflammation at 120cm (water-filled lumen). Case 2: Inflammation, ulceration of transverse colon (water-filled lumen). Case 3: Colitis, pseudopolyps near ileocecal valve.
<table>
<thead>
<tr>
<th>Case</th>
<th>Age / Sex</th>
<th>New UC Diagnosis</th>
<th>Symptoms</th>
<th>WBC</th>
<th>ESR</th>
<th>CRP</th>
<th>Sedation</th>
<th>Extent of Exam</th>
<th>Endoscopic Findings</th>
<th>Biopsy Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>47 / Male</td>
<td>Yes</td>
<td>Severe abdominal pain and up to 30 watery bowel movements (BMs) with occasional blood daily for 1 month</td>
<td>13.5</td>
<td>49</td>
<td>29.30</td>
<td>Versed 3mg / Demerol 75mg</td>
<td>Probable mid-ascending colon</td>
<td>Severe pancolitis without any normal mucosa throughout, no identifiable landmarks</td>
<td>Severe active chronic colitis through out with ulceration and exudate, minimal sparing of crypt architecture</td>
</tr>
<tr>
<td>2</td>
<td>46 / Male</td>
<td>Yes</td>
<td>Shortness of breath due to anemia, 8-10 mostly bloody BMs daily for 5 months</td>
<td>9.6</td>
<td>59</td>
<td>9.45</td>
<td>None</td>
<td>Cecum</td>
<td>Severe pancolitis with greatest severity in transverse and descending colon, pseudo polyps</td>
<td>Normal cecum, severe active chronic colitis from rectum to ascending colon</td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>Gender</td>
<td>Recent UC flare but relapsed during steroid taper, had flexible sigmoidoscopy on admission, now on prednisone 60mg/day but still with 10 bloody BMs daily</td>
<td>3.9</td>
<td>41</td>
<td>4.10</td>
<td>None</td>
<td>Terminal ileum (TI)</td>
<td>Severe pancolitis from rectum to cecum, diffuse pseudo polyps, normal TI</td>
<td>Normal TI, hyperplastic changes in rectum, chronic colitis from rectum to cecum, ulceration from rectum to transverse colon</td>
</tr>
</tbody>
</table>

**TABLE TITLE:** Table 1: Case Summary  
**AVERAGE SCORE:** 4  
**REVIEWER FLAGS:** (none)  
**REVIEWER RECOMMENDATION CODE DESCRIPTION:** None  
**REVIEWER COMMENTS:**  
Somashekar Krishna: [No Comments]  
Julia LeBlanc: [No Comments]  
Girish Mishra: [No Comments]  
Rayburn Rego: [No Comments]  
[No Comments]
ABSTRACT BODY:

Purpose: Urachal anomalies are found early in life and include urachal cyst or sinus, patent urachus or vesicourachal diverticulum. Persistence into adulthood is very rare, typically presenting as septic cysts, which can rupture, causing peritonitis or dissect to the umbilicus, causing drainage of pus. This can easily be mistaken for other pathologies, requiring heightened awareness. We describe a case of infected ruptured urachal cyst mimicking a cutaneous fistula in an adult with a flare of Crohn’s colitis. Case: A 26-year-old male with Crohn’s colitis presented with fever, severe abdominal pain and yellowish fluid draining from his umbilicus, in addition to bloody diarrhea and weight loss. Physical exam revealed acute abdomen and palpable umbilical nodule. Abdominal abscess and enterocutaneous fistula were suspected. CT scan showed inflammation in the cecum and possible fistualization versus small bowel herniation at the umbilicus (Figure 1). Diagnostic laparoscopy showed no communication between the bowel and abdominal wall, but a midline anterior mass was seen (Figure 2). Given diffuse inflammation around the right colon, the procedure was converted to an open laparotomy with excision of the mass and right hemicolectomy. Pathology revealed an infected urachal cyst and severely diseased cecum due to Crohn’s disease, with no evidence of Crohn’s disease extension outside the gut. Discussion: Urachal cysts are exceptional in adults. When infected, they may be mistaken for other pathology, namely complicated Crohn’s disease, as illustrated. There is no reported direct correlation between these two distinct entities. The differential diagnosis of a midline anterior abdominal lesion with umbilical drainage should include urachal pathology. Diagnosis is made by laparoscopy, and complete excision is warranted to prevent re-infection and malignant transformation.

Methods: N/A

Results: N/A

Conclusion: N/A
Purpose: A 68-year-old woman was referred for evaluation of chronic abdominal symptoms. She had a long list of non-specific complaints. Most notably, her constipation alternating with diarrhea had become more diarrheal-predominant, associated with right lower abdominal pain and a 12-pound weight loss over the last year. Screening colonoscopy three years ago was normal to the cecum. Other medical history includes anxiety. She denied use of non-steroidal anti-inflammatory medications. She has a son with ulcerative colitis. Colonoscopy to the terminal ileum showed an 8-mm polyp 10 cm from the ileocecal valve, and multiple 1-3 mm ulcers in the terminal ileum up to 20 cm from the ileocecal valve. Biopsy showed active ileitis with ulceration, no granulomas or dysplasia. Biopsy of the 8-mm polyp showed well-differentiated neuroendocrine neoplasm suggestive of carcinoid tumor. CT enterography revealed no masses or liver lesions. Capsule endoscopy showed few 1-mm jejunal ulcers, and many 2-mm ulcers throughout the ileum, consistent with small bowel Crohn's disease (CD). Subsequent ileo-cecectomy confirmed pathologic diagnoses of Crohn's ileitis and carcinoid tumor (stage IIIB, Ki67<1%). The coexistence of carcinoid tumor and CD is rare. To date, there have been about 50 reported cases. A recent study noted that carcinoid tumors are 15 times more common in patients with CD. Carcinoid is a slow-growing tumor. When arising in the small bowel, it can mimic CD with symptoms of abdominal pain, weight loss, diarrhea and small bowel obstruction. They have some similar gross pathologic appearance of fibrosis and thickening of the bowel wall. Histology provides the definitive diagnosis. However, a small focus of carcinoid can be easily missed in a resected specimen of small bowel with CD. Therefore, the true incidence of carcinoid in CD may be underestimated. The pathogenesis of the two diseases' association is unclear, but inflammation, pro-inflammatory cytokines or hyperplasia may play a role in the development of carcinoid tumors, which can even be found in areas with no Crohn's involvement. Given the similarity in clinical presentation, it is important to be aware of their association so that carcinoid tumor can be detected and resected prior to metastasis. Optimal management of CD after a diagnosis of malignancy, such as carcinoid tumor, can also be a challenge. Given the rare coexistence of the two conditions, there are currently no reports, to our knowledge, on the risk of recurrence with immunomodulators or anti-TNF use in CD after carcinoid tumor resection. Future research on the use of these agents in Crohn’s management after a recent diagnosis of malignancy would be of interest.

Methods: N/A
Results: N/A
Conclusion: N/A
Purpose: A 22-year-old woman with familial Mediterranean fever (FMF) treated with colchicine and a strong family history of Crohn's disease presented with severe lower abdominal pain, nausea, vomiting and fevers up to 104°F. The differential diagnosis for her symptoms included Crohn's, ulcerative colitis, appendicitis, intussusception, cholecystitis, pelvic inflammatory disease, peptic ulcer and colchicine-refractory FMF flare. Physical exam was unremarkable, except for abdominal tenderness predominantly in the RLQ with modest guarding. Laboratory evaluation showed normal electrolytes, CBC, amylase/lipase, TFTs, PTH and vitamin levels. Stool and urine cultures were negative. Gynecologic evaluation, including transvaginal ultrasound, was normal. MRE showed no evidence of bowel wall inflammation or obstruction. Upper endoscopy biopsies showed a chronic chemical gastropathy with no Barrett's, no H. pylori, and normal jejunal villous architecture. Colonoscopy was normal, with normal terminal ileum and colonic mucosal biopsies. Given the recurrence of symptoms and greater than one febrile attack per month, the patient was deemed to have colchicine-resistant FMF and was referred to rheumatology for initiation of anti-IL1β biologic therapy.

Discussion: FMF is a hereditary auto-inflammatory disease characterized by periodic fevers and serositis triggered by deregulated inflammasome production of IL-1β. With peritonitis, abdominal pain and fever being the most common manifestations, gastroenterologists need to consider FMF in the DDX of IBD-associated inflammatory symptoms. FMF is caused by mutations in the MEFV gene encoding pyrin. Mutant pyrin fails to regulate caspase-1 activity and additionally activates NFκB. Careful follow-up of FMF patients is critical as they are at increased risk of developing late-onset IBD. This increased risk for IBD may be related to the close association of the CD-susceptibility gene NOD2 with the MEFV gene on chromosome 16. Pyrin also interacts with the innate immune intracellular NOD-like receptor Nlpr3, which has been shown to contribute to Crohn’s susceptibility. Nlrp3 is a crucial regulator of intestinal homeostasis, mediating assembly of the inflammasome complex in the presence of microbial ligands, triggering caspase-1 activation and secretion of IL-1β and IL-18. Although colchicine is the first line of treatment for FMF, case reports have shown some efficacy with anti-TNF biologics. However, IL-1β biologics are emerging as the best second-line therapy for colchicine-resistant FMF patients. Despite the FMF and IBD overlap in inflammasome deregulation of the IL-1β pathway, studies are lacking on the effectiveness of colchicine or anti-IL-1β therapy for IBD.
AVERAGE SCORE: 4
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Purpose: Introduction: Adalimumab is an anti-TNFα agent approved to treat autoimmune disease and Inflammatory Bowel Disease (IBD). Very rarely, central nervous system complications related have been reported.

Case Report: A 50-year-old male with a history of Crohn’s ileitis presented with two days of left leg weakness and numbness. He was diagnosed with Crohn’s ileitis in 1998 and started on adalimumab in 2008 with good control of all symptoms until June, 2012. On presentation, neurologic exam was notable for left leg weakness. MRI showed contrast-enhancing lesions in the right cerebral hemisphere and a lesion of the spine at C5. Visual evoked potentials were abnormal. Lumbar puncture revealed intrathecal synthesis of IgG and oligoclonal bands. He was treated with steroids. His adalimumab was discontinued, and he showed improvement in his strength prior to discharge. Outpatient follow-up at showed significant improvement in symptoms and no new neurological deficits.

Discussion: Extensive review of the literature has revealed only four reported cases of demyelinating disease in patients treated with adalimumab. Only seven similar cases were reported in the global safety trials. There is a suspected link between the development of IBD and MS. Some studies have shown a higher incidence of MS in patients with Crohn’s disease. In patients who develop demyelinating disease while on anti-TNFα therapy, the agent should be discontinued. Additionally, the use of all other TNF-α inhibitors should be avoided. Patients have been shown to generally improve within a few months.

This case highlights an important and rare adverse event associated with the use of anti-TNFα agents. Consideration should be given to avoiding these agents in patients with strong personal or family histories of demyelinating disease and therefore, an inherently increased risk of developing the disease. Furthermore, patients undergoing treatment with this class of medication should be screened with neurologic evaluations on routine follow-up for the development of these disorders.

Methods: N/A
Results: N/A
Conclusion: N/A

AUTH DESIG: ACG Membership Status <font color="red">^</font>:
Caren Bartosz : ACG Member
Donald Tsynman : ACG Member
Asad Ullah : ACG Member
Ashok Shah : ACG Member
Purpose: The patient is a 61-year-old male who presented with several weeks of malaise, weight loss, and several days of vomiting. He has a 25-year history of fibrostenotic Crohn’s disease, for which he underwent an ileo-cecectomy in the past. Six months prior to presentation, the patient began having weight loss, abdominal pain and was found to have strictures and ulcers in his small bowel. He had a negative Quantiferon Gold, and was started on infliximab therapy, 5 mg/kg with a standard initiation protocol. He received a total of six infusions prior to the current presentation, most recently three weeks prior. He was not taking any other medications for IBD, including a 5-ASA, immunologic or steroids. On presentation, the patient reported no fever, chills, nightsweats or cough. He had his baseline abdominal pain, but continued to have regular flatus and bowel movements, as well as a normal appetite. His physical exam demonstrated a thin man with notable pallor. His oropharynx was clear, and there was no palpable cervical lymphadenopathy. He had ronchi in his right lower lobe with deep inspiration. Abdominal exam was notable for a scaphoid abdomen with mild tympanic distention, and hyperactive bowel sounds. On labs, his WBC was 12.2 K/uL with a slight left shift, and his hemoglobin was 7.5 g/dL. He had a CT of the abdomen performed, where a 3.-cm mass-like opacity was seen at the right lung base abutting the pleura. He also has some thickened small bowel loops in the left upper quadrant. Microbiology data was negative for legionella or strep pneumonia, as well as negative for histoplasmosis. The patient then underwent a video-assisted thoracic surgery with removal of the mass lesion. Pathology revealed areas of fibrosis, inflammation and foci with multinucleated giant cells suggestive of granulomas. Stains for AFB and fungus were negative. On sputum cultures, 1/6 grew mycobacterium avium-intracellulare (MAI). The patient was subsequently begun on multidrug treatment of his MAI with resolution of his symptoms and return to his normal weight. Anti-TNF therapy is a well-known risk factor for tuberculosis and other granulomatous diseases. Nontuberculosis mycobacterium has been reported in rheumatologic patients, usually in those taking corticosteroids or methotrexate concomitantly, but a specific warning or testing for MAI is not standard of care for IBD patients receiving anti-TNF therapy.

References:

Methods: N/A
Results: N/A
Conclusion: N/A
Wallace Wang : ACG Member
Moshe Rubin : ACG Member
(No Image Selected)
(no table selected)

**AVERAGE SCORE:** 3.5

**REVIEWER FLAGS:** (none)

**REVIEWER RECOMMENDATION CODE DESCRIPTION:** None

**REVIEWER COMMENTS:**
Devi Rampertab: very well written
Selvi Thirumurthi: [No Comments]
Renu Umashanker: [No Comments]
James Vecchio: [No Comments]
Purpose: Background: Glatiramer Acetate (COPAXONE) is an approved treatment for Multiple Sclerosis with high safety profile. We are reporting a case of Crohn’s disease diagnosed in a patient with multiple sclerosis after Copaxone was stopped. Case: A 57-year-old man with history of multiple sclerosis diagnosed many years ago, in remission achieved by Copaxone for more than two years, presented with bloody diarrhea and anemia. He never had any similar symptoms in the past. The patient admitted that he had not been following with his neurologist and he stopped Copaxone 3-4 months before presentation for financial reasons. CT scan of the abdomen showed Pancolitis. Subsequent colonoscopy showed moderate scattered colitis with submucosal hemorrhage, friability and edema (Figure 1). Biopsies showed acute on chronic inflammatory colitis with granulomas suggestive of Crohn’s disease. The patient received tapering doses of steroids and was started on Mesalamine, his symptoms resolved in 6 weeks.

Discussion: Copaxone is approved drug for multiple sclerosis with relatively safe profile. There is good evidence of therapeutic benefits of Copaxone in treatment of inflammatory bowel disease (IBD) in animal models, however clinical trials are lacking. Case reports in literature, although very few, have some debate, as couple of them reporting potential benefits in Crohn’s disease, with Copaxone helped inducing remission in those cases, while another case report posed the question of potential development of Crohn’s disease as a side effect for Copaxone. Our case reports development of Crohn’s disease after Copaxone was stopped. Experimental studies have shown that Copaxone has immunomodulating property to deviate the immune response from Th1-dominated to a Th2-mediated inflammation. This could favor the potential benefit of Copaxone in maintenance of remission for Crohn’s disease.

Conclusion: Copaxone, supported by animal models, could have a role in treatment of Crohn’s disease. However randomized clinical trials are needed.

Methods: N/A

Results: N/A

Conclusion: N/A

CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease
PRESENTATION TYPE: Oral or Poster
ACG Research Grant Support: No
Supported by Industry Grant: No
Commercial Products or Services: No
Initiated Research: Investigator
Financial Relationships: Not Applicable
FDA Approval: No
Designed Study: Investigator
Abstract Author: Investigator

AUTH DESIG: ACG Membership Status <font color="red">*</font>
Mohammad Telfah : ACG Non-Member
Kaartik Soota : ACG Non-Member
Krishna Tangirala : ACG Non-Member
Deerajnath Lingutla : ACG Non-Member
Manuel Matos : ACG Non-Member

IMAGE CAPTION:
(no table selected)
AVERAGE SCORE: 5
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Devi Rampertab: many errors in grammar and syntax
Selvi Thirumurthi: [No Comments]
Renu Umashanker: [No Comments]
James Vecchio: [No Comments]
CONTROL ID: 1741624
TITLE: Lymphangioma Circumscriptum, A Cutaneous Manifestation of Crohn's Disease
PRESENTER: Adam Schiro
PRESENTER (INSTITUTION ONLY): Medical College of Wisconsin, Department of Medicine
PRESENTER (COUNTRY ONLY): United States

ABSTRACT BODY:
Purpose: A 39-year-old female referred to GI clinic for complaint of bright red blood per rectum that was initially attributed to her hemorrhoids. Blood was noted with every bowel movement, coughing, and intercourse. Associated symptoms were abdominal cramps in the morning that was relieved with 4-5 clusters of loose stool and 20 lbs of unintentional weight loss. Furthermore, she reported a rash on her buttocks that appeared to be enlarging despite using mupirocin and clotrimazole cream. Pertinent physical examination showed 2 large inflamed 2 cm skin tags protruding from the anal canal and multiple verrucous pink papules coalescing into confluent 2-4 cm plaques on both buttocks. Colonoscopy showed a strictured and inflamed IC valve and scattered aphthous ulcerations in the descending and sigmoid colon. Pathology showed mild colitis only in the rectum. CT enterography demonstrated acute inflammatory changes involving the terminal ileum and blind ending sinus tracts extending from TI into the mesentery and cecum suggesting possible entero-cesal fistula. Patient was referred to dermatology and a shave biopsy was performed of the perianal plaques. Pathology revealed many vascular spaces resembling lymphatics with substantial epidermal hyperplasia with elongated rete suggestive of lymphangioma circumscriptum (LC). Patient was diagnosed with Crohn’s disease with LC. Patient was started on Azathioprine 50 mg daily and prednisone 40 mg taper with gradual resolution of the skin lesions and a decrease in abdominal pain and bleeding. A variety of dermatologic extraintestinal manifestations are associated with inflammatory bowel disease. Most common are erythema nodosum, pyoderma gangrenosum, and aphthous stomatitis. LC is a rare proliferation of the lymphatic system that can be caused by infection, fistulizing Crohn’s disease, and radiation therapy. The proposed mechanism for formation is direct mechanical disruption caused by fibrosis associated with fistula tract formation. Composed of dilated native lymphatic channels in the cutaneous and subcutaneous tissues, its manifestation can vary from ill-defined sessile masses resembling cobblestones and pedunculated polypoid lesions to discrete collections of vesicles resembling frog spawn. While treatment options include surgical resection, which is frequently unsuccessful with rapid relapse, sclerotherapy, vaporization with CO2 laser, or observation, there is currently little literature to guide treatment in patients with Crohn’s disease. This case demonstrates the importance of recognizing acquired LC as a dermatologic extraintestinal manifestation of Crohn’s disease and that further research into appropriate long-term clinical management is necessary.

Methods: N/A
Results: N/A
Conclusion: N/A

CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease
PRESENTATION TYPE: Poster Only
ACG Research Grant Support: No
Supported by Industry Grant: No
Commercial Products or Services: No
Initiated Research: Investigator
Financial Relationships: Not Applicable
FDA Approval: No
Designed Study: Investigator
Abstract Author: Investigator

AUTH DESIG: ACG Membership Status <font color="red">*</font>:
Tom Fang : ACG Member
Adam Schiro : ACG Non-Member
Ayan Rag : ACG Member
Barbara Wilson : ACG Non-Member
Daniel Stein: ACG Member
(No Image Selected)
(no table selected)

**AVERAGE SCORE**: 2.75

**REVIEWER FLAGS**: (none)

**REVIEWER RECOMMENDATION CODE DESCRIPTION**: None

**REVIEWER COMMENTS**:

Devi Rampertab: [No Comments]
Selvi Thirumurthi: [No Comments]
Renu Umashanker: [No Comments]
James Vecchio: [No Comments]
Purpose: A 53-year-old African American male with a diagnosis of Crohn’s disease (CD) based on recurrent perianal fistulas since age of 17 was admitted due to increased purulent drainage and pain from existing fistula. Patient was recently started on infliximab after minimum improvement from 6-MP and 2-years of split dosing certolizumab 200mg. Patient underwent seton placement approximately 1 month prior and on subsequent evaluation developed new collections in the leg. Review of previous colonoscopy performed in 2000 and 2008 showed endoscopically and histologically normal findings. Physical exam showed numerous tracks/scars and old inflammation with induration across the buttock and perineum as well as fistulous openings with purulent drainage and setons that was previously placed. A colonoscopy performed to the TI with biopsies taken throughout again demonstrated normal findings. A CT enterography revealed normal appearing small and large bowel without indication of inflammatory bowel disease. Patient underwent radical excision of fistulas from the pus fill cavities down to the subcutaneous tissues. Pathology confirmed the diagnosis of pyoderma fistulans sinifica. Patient underwent successful skin graft with healing of the wound. On follow-up off all IBD medications, the wounds did not show any evidence of disease, and patient reports feeling better than he has felt in years. Approximately 20-30% of patients with CD present at time of diagnosis with perianal lesions with 15-20% having fistulas. Cumulative fistula incidence increases from 33% at 10 years to 50% at 20 years after diagnosis. Severe fistulizing disease is usually associated with rectal mucosal abnormalities. However, not all perianal fistulizing disease is related to CD as demonstrated in our case. Pyoderma fistulans sinifica, first described by Krauspe and Stelzner in 1962, is a rare chronic subcutaneous infection that is both physically and psychologically debilitating. Also termed Fox Den disease due to the multiple deep epifascial epithelialized fistulous tract system that runs in parallel, it predominantly affects the lower body with no relation to skin appendices. The anal canal and rectum are always spared which may serve as a clue as risk of fistula development in CD is higher in patients with rectal involvement. Exact incidence is unknown as diagnosis is often delayed or misdiagnosed. Cure requires wide en-bloc excision and may benefit from skin grafting. This case demonstrates that practitioners should recognize that not all fistulizing perianal disease is CD and the diagnosis should be reconsidered if there is no improvement with escalation of therapy or perianal disease without colonic abnormalities.
AVERAGE SCORE: 2.25
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None

REVIEWER COMMENTS:
Devi Rampertab: Phenomenal
Selvi Thirumurthi: [No Comments]
Renu Umashanker: [No Comments]
James Vecchio: [No Comments]
ABSTRACT BODY:

Purpose: Introduction: Sacro-coccygeal osteomyelitis complicating Crohn’s disease (CD) is a rare phenomenon with less than 20 cases reported in the literature. We report 3 cases of sacro-coccygeal osteomyelitis presenting as low back pain in CD patients. Case A: A 35-year-old male with history of internal penetrating ileo-colonic CD had been on infliximab for 6 years following an ileo-colonic resection, when he developed right-sided low back pain, that would improve transiently following a dose of infliximab. A CT enterography revealed sacro-iliitis; however, a subsequent MRI revealed a fistulous tract from the sigmoid colon to a pre-sacral fluid collection along with radiologic evidence of sacral osteomyelitis. Intravenous antibiotics were initiated; however, the patient eventually required a low-anterior resection with diverting loop ileostomy in addition to antibiotic therapy due to persistent radiologic evidence of osteomyelitis. Case B: A 33-year-old male with history of internal penetrating ileo-colonic CD with perianal involvement presented with perianal as well as low back pain. The patient was naive to biologic as well as immunomodulator therapy. MR enterography showed a perianal fistula with a pre-sacral fluid collection as well as coccygeal osteomyelitis. The patient underwent a diverting ileostomy followed by 7 weeks of intravenous and 8 weeks of oral antibiotics with complete resolution of the coccygeal osteomyelitis. Case C: A 42-year-old male with history of internal penetrating and stricturing ileo-colonic CD presented with low back pain. He did not have any gastrointestinal symptoms and was on infliximab infusions (5 mg/kg every 8 weeks) for his CD. A pelvic MRI showed osteomyelitis of the coccyx. Despite a short course of ciprofloxacin (500 mg BID for 14 days), the back pain persisted and repeat MRI showed persistent enhancement at the distal tip of the coccyx along with an ischio-rectal abscess. The patient underwent a diverting loop ileostomy with drainage of the ischio-rectal abscess prior to initiation of intravenous antibiotics. A coccygectomy was performed 2 months later due to persistent osteomyelitis despite medical therapy.

Discussion: Sacro-coccygeal osteomyelitis is a rare complication of perianal CD. Contrary to the general population, patients with perianal CD develop sacro-coccygeal osteomyelitis by direct extension from a pelvic abscess or fistula rather than hematogenous seeding from a distant source. Pelvic MRI is the most sensitive imaging modality for an accurate diagnosis of sacro-coccygeal osteomyelitis. Surgical intervention is often required in conjunction with antibiotics for complete resolution.

Methods: n/a
Results: n/a
Conclusion: n/a
AVERAGE SCORE: 2.75
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Devi Rampertab: Phenomenal!!|
Selvi Thirumurthi: [No Comments]|
Renu Umashanker: [No Comments]|
James Vecchio: [No Comments]
Purpose: Adalimumab (ADA) is a tumor necrosis factor (TNF) inhibitor, used for treatment of inflammatory bowel disease (IBD). Many studies reported the increased risk of cancer following the exposure to TNF inhibitors, but little has been reported on patients with cancer receiving anti-TNF treatment. We present a female patient with metastatic breast cancer and ulcerative colitis (UC) who was treated with ADA. Case: A 54-year-old African American female developed breast cancer (BC) 21 years prior to development of UC and was initially treated with left breast lumpectomy followed by four cycles of adjuvant chemotherapy. The chemotherapy was discontinued due to side effects. She then received radiotherapy to the left breast. Three years before her UC diagnosis and 18 years after the initial diagnosis of BC, she developed recurrent, bilateral stage IV BC with distal metastases to the bone and lymph nodes. She underwent bilateral mastectomy. Pathology showed invasive ductal carcinoma grade III and ductal carcinoma in situ in the left breast and metastatic cancer in the right breast. She then received trastuzumab, zoledronic acid, and gemcitabine. Three years ago, she was diagnosed with pancolitis by colonoscopy, after an acute episode of diffuse abdominal pain and bloody diarrhea. She was started on 5-aminosalicylates and prednisone, but her UC was not controlled for 5 months. Subcutaneous ADA (40 mg every two weeks) was started and resulted in dramatic improvement. Four months after starting ADA along with ongoing chemotherapy with capecitabine and lapatinib, restaging with CT scan showed the resolution of previously seen internal mammary lymph nodes, and no evidence of distant metastasis. Bone scan and follow-up PET/CT scans performed every 6 months indicated the stability of metastatic bone lesions for the past 2.5 years. Conclusion: While TNF inhibitors could theoretically promote further metastasis in patients with cancer, this case has remained stable for 2.5 years after ADA initiation for UC. The majority of studies have been undertaken to understand whether anti-TNF therapy increases the rate of malignancy. Information regarding the safety of TNF inhibitors prescribed to patients with prior malignancies is available in few studies, one of them indicating a possibly increased recurrence risk for malignancy. On the other hand, there are also few reports of TNF inhibitor treatment of patients with advanced cancer. Some reports suggest progression whereas most reports suggest stability of the cancers. This is the first report of a patient treated with ADA long term, who also has metastatic BC.

Methods: N/A
Results: N/A
Conclusion: N/A
Severe Ulcerative Colitis Associated with Concurrent Cytomegalovirus and Clostridium difficile Infections: Successful Medical Treatment without Corticosteroids

Warren Finkelstein

The Gastroenterology Group of New Jersey

United States

Purpose: Background: Patients with severe ulcerative colitis requiring hospitalization have an increased risk of Clostridium difficile (C. difficile) and Cytomegalovirus (CMV) infections – particularly if immunosuppressed. These infections may be associated with increased morbidity and mortality. Treatment with corticosteroids in such patients without treating these infections may precipitate clinical deterioration. We present a case of an immunosuppressed patient with severe ulcerative colitis who was treated for concurrent C. difficile and CMV infections with a successful clinical outcome without the need for corticosteroids. Case Report: A 27-year-old woman with a 3 year history of ulcerative pancolitis presented with 2 weeks of non-bloody diarrhea, mild abdominal pain, and fevers up to 103°F. She had been taking 50 mg of 6-mercaptopurine per day and 4.8 gm of oral and 4 gm topical mesalamine. On hospital admission, she was febrile to 104°F, tachycardic, but on exam had no abdominal tenderness. Admission laboratory showed a white blood count (WCB) of 2600 with 41% polys and 51% lymphocytes. Hemoglobin was 11 gm. Sedimentation rate was 59, C-reactive protein 88. A CT scan of the abdomen showed colitis extending from transverse colon to the rectum. Flexible sigmoidoscopy showed moderate to severe colitis with diffuse shallow ulcerations from 18cm to 35cm from the anal verge. Stool C. difficile toxin was positive. She was treated with oral vancomycin and intravenous metronidazole and ciprofloxacin. Aminosalicylates were continued both orally and topically. Despite this therapy, she continued to have high fevers and diarrhea. The WBC dropped to 1,300 with 28% neutrophils, 28% bands, and 40% lymphocytes. Meropenem and several doses of neupogen were given. Biopsies from the sigmoidoscopy were consistent with active colitis with immunohistochemical stains consistent with CMV infection. Serum PCR to CMV was 518,000 iu/ml. Intravenous foscarnet was prescribed. Over the next week, her diarrhea and fevers resolved completely, and her WBC rose to 7,000. The patient was discharged 12 days after hospitalization on valganciclovir, vancomycin, and aminosalicylates. Conclusions: Immunosuppressed patients hospitalized with severe ulcerative colitis should undergo a thorough evaluation for multiple infections including both C. difficile and CMV. Non-bloody diarrhea, high fever, and lack of significant abdominal pain should increase clinical awareness for an infectious cause of an ulcerative colitis flare. Prompt recognition and treatment of these precipitating infections and the need to hold off using corticosteroids in such patients may help prevent adverse clinical outcomes including colectomy.

Methods: N/A

Results: N/A

Conclusion: N/A
Yong Kwon : ACG Non-Member
(No Image Selected)
(no table selected)
**AVERAGE SCORE:** 2.75
**REVIEWER FLAGS:** (none)
**REVIEWER RECOMMENDATION CODE DESCRIPTION:** None
**REVIEWER COMMENTS:**
ABSTRACT BODY:

Purpose: We report a case of a 45-year-old male with a history of ileal, fistulizing Crohn’s disease. The patient was originally diagnosed with ulcerative colitis status post ileal-pouch anal anastomosis that was reversed. He later developed severe perianal disease and his disease process appeared consistent with Crohn’s disease (CD). He had multiple ileostomies with multiple complications most significant of which was severe difficult to control peristomal pyoderma gangrenosum (PG). The patient initially was treated with infliximab (Remicade), but experienced an infusion reaction. He was then switched to adalimumab (Humira) with mild benefit, but continued to have severe PG. He was later treated with Anakinra without significant effects. His PG remained severe and required long term steroids. He was later treated with cyclosporine with mild improvement. This had to be stopped in the setting of renal failure. He was also treated with methotrexate, which significantly worsened his symptoms. He was switched to mycophenolate mofetil (CellCept) which was also stopped in the setting of renal failure. Eventually, the patient was started on intravenous immunoglobulin (IVIG) which resulted in dramatic improvement to his PG as well as his CD. One year later, the patient has been off of adalimumab, continuing treatment with only IVIG, on minimal dose of steroids and has been feeling very well with complete clearance of his PG. Pyoderma gangrenosum (PG) is a rare, inflammatory neutrophilic dermatosis of unknown etiology. It is the second more frequent and most debilitating cutaneous extra-intestinal manifestation in inflammatory bowel disease (IBD) patients. It is commonly treated with immunosuppressants and systemic corticosteroids; however, patients have reported that long-term usage of these medications cause serious side effects. Although there are only a few studies available, a very effective and safe therapeutic alternative for PG has been high dose IVIG with minimal side effects. IVIG has also been used in patients with Crohn’s disease with great response. With limited data about the use of IVIG in Crohn’s patients, the exact dose and duration of treatment remain to be defined.

Methods: N/A
Results: N/A
Conclusion: N/A

CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease
PRESENTATION TYPE: Poster Only
ACG Research Grant Support: No
Supported by Industry Grant: No
Commercial Products or Services: No
Initiated Research: Investigator
Financial Relationships: Not Applicable
FDA Approval: No
Designed Study: Investigator
Abstract Author: Investigator

AUTH DESIG: ACG Membership Status <font color="red">*</font>:
Lisa Yoo : ACG Non-Member
Saleh Elwir : ACG Non-Member
Andrew Tinsley : ACG Member
Emmanuelle Williams : ACG Member
(No Image Selected)
(no table selected)
AVERAGE SCORE: 3
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None

REVIEWER COMMENTS:
Devi Rampertab: [No Comments]
Selvi Thirumurthi: [No Comments]
Renu Umashanker: [No Comments]
James Vecchio: [No Comments]
Purpose: We report a case of a 33-year-old woman with a history of ulcerative colitis diagnosed 10 years prior to presentation status post total abdominal colectomy with ileal pouch anal anastomosis (IPAA) 6 years prior to presentation. The patient suffered recurrent severe antibiotic resistant pouchitis shortly after her surgery. Two years ago, a pouchoscopy demonstrated continued pouchitis with more extensive inflammation above the pouch. The patient was started on Azathioprine and responded to a course of steroids and antibiotics. After her endoscopic evaluation, the developed an idiopathic left upper extremity deep vein thrombosis at her IV site and completed 6 months of warfarin therapy. One month prior to presentation, the patient had another episode of severe pouchitis and was treated with antibiotics and a steroid taper. During her taper, the patient presented with dyspnea on exertion. Chest CT scan revealed large bilateral pulmonary emboli (PE) with severe right heart strain, enlargement of the right atrium and main pulmonary artery consistent with pulmonary hypertension. She was treated with enoxaparin and warfarin and discharged home. Our patient appeared to have developed thromboemboli in the setting of two episodes of pouchitis. Hospitalized inflammatory bowel disease (IBD) patients are known to be at increased risk for thromboembolism. The most frequent complication in ulcerative colitis patients after ileal pouch anal anastomosis (IPAA) is pouchitis, a nonspecific inflammation of the ileal pouch reservoir, having a cumulative prevalence of 50%. It is unknown if this inflammatory state increases hypercoagulability. While portal vein thrombi (PVT) have recently been linked to IPAA and patients found with PVT had a higher incidence of postoperative pouchitis, an association with PE has not been described. Hospitalized patients with IBD and pouchitis, such as our patient, must be considered at high risk for thromboembolism and receive appropriate prophylaxis or be considered for long-term anticoagulation regardless of history of thromboembolism.

Methods: N/A
Results: N/A
Conclusion: N/A

CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease
PRESENTATION TYPE: Poster Only
ACG Research Grant Support: No
Supported by Industry Grant: No
Commercial Products or Services: No
Initiated Research: Investigator
Financial Relationships: Not Applicable
FDA Approval: No
Designed Study: Investigator
Abstract Author: Investigator

AUTH DESIG: ACG Membership Status <font color="red">*</font>:
Lisa Yoo : ACG Non-Member
Saleh Elwir : ACG Non-Member
Andrew Tinsley : ACG Member
Emmanuelle Williams : ACG Member
(No Image Selected)
(no table selected)

AVERAGE SCORE: 4.25

REVIEWER FLAGS: (none)

REVIEWER RECOMMENDATION CODE DESCRIPTION: None

REVIEWER COMMENTS:
Ulcerative Colitis Exacerbation in a Young Patient Causing Acute Myocardial Infarction: A Case Report

目的：引言：溃疡性结肠炎（UC）是一种慢性炎症性疾病，主要影响肠道，但也有许多肠外表现。UC最常见的心脏表现是心包炎和心肌炎。患者显示对静脉血栓栓塞并发症和肠系膜缺血的风险增加，但与心肌梗死和心肌炎的关系罕见。案例：33岁女性突然出现胸痛。患者没有心脏病风险因素，包括高血压、高血脂、家族史、吸烟或代谢综合症。她有病史UC，直到入院前两天，腹痛和血便典型地表明UC复发。患者未接受UC治疗。体检仅发现腹部轻度弥漫性压痛。心电图显示II、III和aVF导联ST段抬高。实验室结果显示：心肌肌钙蛋白I升高至25 ng/ml，白细胞11.2×10^3/mm^3，血红蛋白9.5 g/dl，凝血酶原片段0.344 mg/dl，D-二聚体408 ng/ml，PT 11.8秒，总胆固醇185 mg/dl与LDL 121 mg/dl和HDL 38 mg/dl。她接受了急诊冠状动脉造影，显示右冠状动脉完全闭塞，无法通过放置支架来恢复。患者被用β-阻滞剂、ACE抑制剂和他汀类药物管理急性MI。静脉肝素疗法被选择，避免使用阿司匹林和氯吡格雷，因患者有血便。患者对治疗反应良好，没有进一步的直肠出血，没有急性MI的并发症。讨论：UC的高凝状态被归因于血小板、凝血酶原片段、纤维蛋白原、同型半胱氨酸水平的增加和抗凝血水平的降低。凝血酶、抗凝血酶III和凝血酶原与D-二聚体和纤维蛋白降解产物（FDP）的增加表明UC的凝血异常。这些改变在凝血级联中可以导致深静脉血栓形成、肺栓塞、心房血栓和冠状动脉血栓形成。静脉肝素也被用于急性UC加重的治疗，没有增加出血的风险。结论：UC应被视为CAD的潜在风险因素，即使没有其他典型风险因素。如我案例所示，早期诊断和干预对这一亚组患者至关重要，因为有高风险发展血管事件。
Ira Mayer : ACG Member
Rabin Rahmani : ACG Member
(No Image Selected)
(no table selected)

**AVERAGE SCORE:** 3.25

**REVIEWER FLAGS:** (none)

**REVIEWER RECOMMENDATION CODE DESCRIPTION:** None

**REVIEWER COMMENTS:**
Devi Rampertab: [No Comments]
Selvi Thirumurthi: [No Comments]
Renu Umashanker: [No Comments]
James Vecchio: [No Comments]
Purpose: Background: Coronary thrombosis is a rare manifestation of thrombophilia in IBD. We present a case of acute myocardial infarction (MI) in a patient with severe ulcerative colitis (UC) that highlights the therapeutic challenges posed by concomitant MI and active IBD. Case Presentation: A 61-year-old man with UC and coronary artery disease (CAD) presented to his gastroenterologist reporting bloody diarrhea and chest pain prompting an EKG showing T-wave inversions. Cardiac catheterization revealed occlusion of a branch of the left anterior descending artery that was treated with a drug-eluting stent. He was discharged on prednisone for his UC flare but returned five days later with progressive bloody diarrhea. Endoscopy confirmed severe UC. He was treated with intravenous hydrocortisone but had an inadequate response. After extensive discussion regarding risks of surgery, cyclosporine, and infliximab (IFX) he was successfully treated with IFX. He was transitioned to oral steroids and discharged in stable condition. At 18-month follow-up his colitis was in remission on IFX. Discussion: Coronary artery thrombosis is an uncommon manifestation of thrombophilia in IBD. To date there have been six published reports of MI in patients with a UC flare. All cases were postulated to be acute thrombotic events in the setting of active UC. While our patient had a history of CAD, it is likely that the acute inflammatory burden of severe UC contributed to his thrombotic complication. This case highlights the therapeutic dilemma of managing a patient with a severe IBD flare in the setting of a recent MI. All current therapies for severe UC are problematic in this setting. Glucocorticoids are associated with an increased risk of ischemic heart disease, heart failure, and free wall rupture in the setting of acute MI. IFX has been implicated in precipitating acute MI and various arrhythmias. It has been postulated that tumor necrosis factor-α blockade may alter cardiac remodeling and cause progression of CAD. IFX is relatively contraindicated in patients with heart failure. The major adverse cardiovascular effect of cyclosporine is hypertension. However, the utility of cyclosporine may be limited by the need to monitor drug levels. Finally, colectomy is problematic in this setting given the significant increase in major cardiovascular events in patients undergoing non-cardiac surgery after a recent MI. Conclusions: Patients with IBD are at increased risk of thromboembolic events including acute MI. The management of active IBD in the setting of MI poses clinical challenges. The complexity of such cases warrants an interdisciplinary approach involving the patient, gastroenterologist, cardiologist and surgeon.

Methods: N/A
Results: N/A
Conclusion: N/A
AVERAGE SCORE: 4.33
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
David Hass: [No Comments]|Charlene Le Pane: [No Comments]|Renee Young: [No Comments]|Rowen Zetterman: [No Comments]
Complete Portal Vein Thrombosis in a Patient with Active Crohn’s Disease

Chad Cornish

University of Rochester Medical Center, Division of Gastroenterology & Hepatology

United States

Purpose: Introduction: Portal vein thrombosis is an uncommon occurrence in patients with inflammatory bowel disease, but when it does occur it is usually soon after intra-abdominal surgery. We describe a case of complete portal vein thrombosis in a patient with active Crohn’s disease. Case Summary: A 41-year-old gentleman with a 10-year history of Crohn’s colitis, treated with sulfasalazine 1,500 mg BID and mercaptopurine 75 mg daily, presented to the office with a 3-week history of generalized, constant abdominal pain, with associated anorexia and 10-12 loose, bloody bowel movements per day, which began localizing to the right upper quadrant (RUQ) 4 days prior to being seen. His physical exam was notable for RUQ tenderness to even light palpation with guarding and mild hepatomegaly. His labs were significant for AST 209 U/L, ALT 693 U/L, and CRP 62 mg/L. His initial abdominal ultrasound, obtained 1 week after his clinic visit, demonstrated a non-occlusive left portal vein thrombus extending into the bifurcation of the main portal vein. The right and main portal vein were both patent. His abdominal pain gradually resolved and his liver profile returned to normal. 5 weeks later, he presented to the emergency department with intense RUQ abdominal pain. His AST, ALT, and total bilirubin were found to be 1406 U/L, 2527 U/L, and 1.7 mg/dL (direct 0.9 mg/dL), respectively. An abdominal ultrasound was obtained, which demonstrated partial thrombosis of the right portal vein and complete thrombosis of the main portal vein. He was subsequently started on a heparin drip after labs to check for a hypercoagulable state, which were unremarkable, had been drawn. 2 days later, the patient was started on warfarin, as well as an enoxaparin bridge, and discharged to home. The patient’s liver profile quickly improved. A repeat abdominal ultrasound after 6 months of anticoagulation therapy has not yet been performed.

Discussion: Portal vein thrombosis is a potentially life-threatening hepatobiliary manifestation associated with inflammatory bowel disease (IBD) and occurs more often in patients with Crohn’s disease than ulcerative colitis. The formation of a portal vein thrombus in patients with IBD is rare, especially in those who have not recently undergone intra-abdominal surgery. Given that the patient presented with portal vein thrombosis in the setting of a Crohn’s disease flare, we hypothesize that ulceration of the bowel mucosa allowed for translocation of bacteria into the portal venous system leading to portal pyelophlebitis followed by portal vein thrombosis.

Methods: N/A

Results: N/A

Conclusion: N/A

G. Clinical Vignettes/Case Reports

I. Inflammatory Bowel Disease

Poster Only

ACG Research Grant Support: No

Supported by Industry Grant: No

Commercial Products or Services: No

Initiated Research: Investigator

Financial Relationships: Not Applicable

FDA Approval: No

Designed Study: Investigator

Abstract Author: Investigator

ACG Membership Status: Chad Cornish: ACG Member

William Amundson: ACG Non-Member

Dorothy Mason: ACG Non-Member

Vikram Dogra: ACG Non-Member

Vivek Kaul: ACG Member
Ashok Shah: ACG Member
(No Image Selected)
(no table selected)

**AVERAGE SCORE:** 3.67

**REVIEWER FLAGS:** (none)

**REVIEWER RECOMMENDATION CODE DESCRIPTION:** None

**REVIEWER COMMENTS:**
David Hass: [No Comments]
Charlene Le Pane: [No Comments]
Renee Young: [No Comments]
Rowen Zetterman: [No Comments]
Purpose: Opportunistic infections (OI) arise from microorganisms with limited pathogenicity in the immunocompetent, but can cause significant morbidity and mortality in susceptible hosts. Patients with inflammatory bowel disease (IBD) who receive corticosteroids, immunomodulators and anti-TNF agents are at increased risk for developing OIs. Nocardiosis is an OI that can occur locally or systemically, particularly in immunocompromised patients. Here we report a case of nocardia pneumonia in a patient with Crohn’s disease on infliximab.

Methods: An 81-year-old man with colonic and perianal Crohn’s disease presented to the emergency department at our hospital with a three-month history of fatigue, progressive cough and exertional dyspnea. He was evaluated on three occasions elsewhere and was treated with antibiotics without improvement. Physical exam was notable for tachycardia and scattered coarse inspiratory crackles in the right lung field. Chest x-ray revealed new, ill-defined patchy opacities in both lungs. CT Thorax showed multi-focal consolidations and bilateral tree-in-bud opacities with mediastinal and hilar adenopathy. The patient underwent fiberoptic bronchoscopy with bronchoalveolar lavage (BAL). Results: Aerobic cultures of the BAL grew normal flora. Direct fluorescent-antibody (DFA) was negative for Pneumocystis jiroveci (carinii) and acid-fast bacilli. One week later, Nocardia species was isolated from the induced sputum and the BAL. The patient was started on trimethoprim-sulfamethoxazole with marked improvement in pulmonary symptoms. He was continued on trimethoprim-sulfamethoxazole for maintenance and infliximab was restarted several weeks later. Conclusion: We report a case of nocardia pneumonia in a patient with Crohn’s disease on infliximab. The possibility of an OI was not entertained despite the fact that the treating physicians were aware the patient was on infliximab. Seven cases of nocardiosis in the setting of infliximab therapy have been reported to date. In six of these seven cases, the patients were receiving at least one other immunosuppressive agent in addition to infliximab, such as prednisone, 6-MP or azathioprine. Of the seven cases, two patients developed cutaneous nocardiosis, two had disseminated nocardiosis and three patients had pulmonary nocardiosis. There was one death. Although there are no reported cases of nocardia with other anti-TNFs, this drug class is associated with other OIs and all could likely predispose patients to nocardiosis. Thus a high index of suspicion is needed to prevent morbidity and mortality associated with opportunistic infections in immunosuppressed IBD patients.

Methods: N/A
Results: N/A
Conclusion: N/A
(no table selected)

**AVERAGE SCORE:** 2.67

**REVIEWER FLAGS:** (none)

**REVIEWER RECOMMENDATION CODE DESCRIPTION:** None

**REVIEWER COMMENTS:**
David Hass: [No Comments]
Charlene Le Pane: [No Comments]
Renee Young: [No Comments]
Rowen Zetterman: [No Comments]
Aseptic Systemic Abscesses as the Initial Manifestation of Inflammatory Bowel Disease: A Case Report

Sekina Ghuman

Wrexham Maelor Hospital

United Kingdom

Purpose: There have been reports of aseptic abscesses preceding a diagnosis of Crohn's disease, especially from France. We present a case of aseptic liver and splenic abscesses as the presenting feature of ulcerative colitis (UC).

Our aim is to increase awareness of this emerging condition in order to avoid diagnostic delay. Case: A 34-year-old female was admitted with 3 day history of swelling of her right ankle with a rash. She had no medical history. On examination she was pyrexial (39.2°C) and tachycardic. Cardiovascular, respiratory and abdominal examinations were unremarkable. Her right ankle was swollen with an overlying, non-blanching rash. Bloods revealed a microcytic anaemia, thrombocytosis, hypoalbuminaemia and elevated inflammatory markers (Hb 6.8, MCV 68, WCC 12, Platelets 724, CRP 339, Albumin 24). CXR & X-ray of the ankle were normal. Blood and urine cultures were negative. She was commenced on broad spectrum antibiotics and transfused 2 units of blood. She continued to be febrile with raised inflammatory markers. Autoimmune screen, immunoglobulins and rheumatoid factor were negative. A CT scan revealed multiple low attenuation lesions throughout the liver and spleen, retroperitoneal lymphadenopathy and rectosigmoid inflammation, despite absence of any GI symptoms. HIV screen was negative and echocardiogram was normal. Stool cultures were negative. Flexible sigmoidoscopy confirmed colitis with severe mucosal congestion and multiple rectal ulcers. Pus was aspirated from one of the splenic abscesses, which was sterile. She was subsequently commenced on IV steroids, with immediate resolution of her fever. Inflammatory markers returned to normal within four days. Histology of rectal biopsies revealed moderate active chronic inflammation in the lamina propria, cryptitis and crypt abscesses consistent with UC. She was discharged on oral steroids and commenced on azathioprine. She remained well at follow up with normal inflammatory markers and resolution of the liver and splenic abscesses on repeat ultrasound after 4 months. Aseptic Systemic Abscesses Syndrome is characterised by multiple, deep, sterile collections of neutrophils that do not respond to antibiotics but are highly sensitive to corticosteroids. It affects young adults and usually present with fever, abdominal pain and weight loss. It is associated with inflammatory bowel disease and neutrophilic dermatoses. Infectious aetiology should be excluded. Steroids achieve rapid clinical & radiological resolution but steroid sparing drugs such as azathioprine may be required.

Methods: N/A

Results: N/A

Conclusion: N/A

G. Clinical Vignettes/Case Reports

I. Inflammatory Bowel Disease

Poster Only

ACG Research Grant Support: No

Supported by Industry Grant: No

Commercial Products or Services: No

Initiated Research: Investigator

Financial Relationships: Not Applicable

FDA Approval: No

Designed Study: Investigator

Abstract Author: Investigator

AUTH DESIG: ACG Membership Status <font color="red">*</font>

Sekina Ghuman : ACG Non-Member

Hamid Khan : ACG Member

(No Image Selected)

(No table selected)

AVERAGE SCORE: 3.33
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
David Hass: [No Comments] Charlene Le Pane: [No Comments] Renee Young: [No Comments] Rowen Zetterman: [No Comments]
Purpose: Hepatocellular carcinoma (HCC) in Crohn's disease (CD) patients without chronic liver disease is extremely rare, with only 10 cases reported in the English-language literature. We report a case of highly aggressive HCC in a young patient with medically refractory CD. Case report: This is a 24-year-old man with a 10-year history of severe CD status post ileal resection due to strictures, who now primarily has colonic and perianal CD. He has required prolonged immunosuppression (IS) including steroids, azathioprine, methotrexate, infliximab, adalimumab, certolizumab, and natalizumab; none of which has provided durable therapeutic benefit. On a recent MRI performed for staging of his CD, he was incidentally found to have two liver lesions that were biopsy-proven as HCC, with AFP of 1,865 ug/L. Given the absence of underlying liver disease, the presumed etiology was attributed to chronic immune suppression. Natalizumab was discontinued and IVIG was initiated. He subsequently underwent left hepatic lobectomy, which identified 3 lesions (8 to 49 mm), and a 15 mm right lobe lesion which was ablated. Pathology showed "moderately to poorly differentiated HCC." The non-neoplastic hepatic parenchyma showed "minimal macrovesicular steatosis" but no fibrosis. His AFP declined to 274 ug/L post-operatively, but rose to 3162 ug/L two months later. A repeat MRI revealed "innumerable hypovascular lesions in the liver" consistent with rapid, multifocal recurrence. Currently, this patient's CD remains active despite treatment on prednisone, budesonide, and IVIG. Due to theoretical potential of IS contributing to the aggressive phenotype of his HCC, bowel diversion with diverting loop ileostomy has been recommended in hopes of achieving remission in CD while allowing reduction in IS and potential restoring innate anti-cancer immune effects. Control of CD is also hoped to mitigate the risks of systemic therapy with sorafenib for HCC, which include fistula, bleeding, and perforation. His post-op recurrence of multifocal HCC has been treated with serial transarterial chemoembolization with plans to initiate systemic therapy once healed post ileostomy. Conclusion: The use of immunosuppression in Crohn's disease has been associated with an increased risk of lymphoma, skin cancer, and solid malignancies. However, HCC in Crohn's disease patients without underlying liver disease has only been reported in 10 patients to date. This case highlights the complex management of severe Crohn's disease and illustrates an extremely rare complication of chronic immunosuppression in the form of a highly aggressive HCC. Clinicians should be aware of this rare but serious complication of long-term immunosuppression.

Methods: N/A
Results: N/A
Conclusion: N/A

CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease
PRESENTATION TYPE: Oral or Poster

ACG Research Grant Support: No
Supported by Industry Grant: No
Commercial Products or Services: No
Initiated Research: Investigator
Financial Relationships: Not Applicable
FDA Approval: No
Designed Study: Investigator
Abstract Author: Investigator

AUTH DESIGN: ACG Membership Status <font color="red">*</font>:
Edith Ho : ACG Member
Danielle Brandman : ACG Non-Member
R. Kate Kelley : ACG Non-Member
Madhulika Varma : ACG Non-Member
Ryutaro Hirose : ACG Non-Member
Michael Wickham : ACG Non-Member
Andrew Taylor : ACG Non-Member
Jonathan Terdiman : ACG Non-Member
(No Image Selected)
(no table selected)

**AVERAGE SCORE:** 3.67

**REVIEWER FLAGS:** (none)

**REVIEWER RECOMMENDATION CODE DESCRIPTION:** None

**REVIEWER COMMENTS:**
David Hass: [No Comments]
Charlene Le Pane: [No Comments]
Renee Young: [No Comments]
Rowen Zetterman: [No Comments]
Purpose: Both infliximab (IFX) and cyclosporine (CSA) are effective for the treatment of severe ulcerative colitis (UC) that is refractory to intravenous (IV) steroids. A recent randomized trial of these agents found no difference in efficacy, but the selection of which agent to use in a patient and the prediction of response to these different agents has not been clarified. In addition, case series have reported significant infectious events when both agents are tried for salvage therapy in close succession. We report a case of severe UC with salvage therapy guided by serum IFX level.

Case: A 17-year-old boy with a 6 month history of pan-UC presented to our Center. He had primary non-response to mesalamine and oral corticosteroids. He was started on combination therapy with azathioprine (AZA) 50 mg/d and IFX 5 mg/kg IV at 0 and 2 weeks, but due to lack of response, received 7.5 mg/kg IV at 6 weeks. Despite this approach, he had persistent disease activity so he received his next IFX at 7.5 mg/kg at a 6 weeks interval. When this failed to control his colitis, he transferred care here and admitted. At that time (2 w after last IFX dose) we sent serum for IFX level and abs to IFX (ATI). He also was found to have *C. difficile* infection by stool PCR and treated with vancomycin 125 mg QID, metronidazole 500mg IV q8h, and IV methylprednisolone 20 mg q12h. Despite clearance of *C. diff*, he continued to have frequent bloody stool and urgency. Colonoscopy confirmed the presence of diffuse active colitis of moderate to severe endoscopic severity. At this time, the IFX level returned from outside lab and was undetectable, and ATI were high (265 ng/mL, ref< 22 ng/mL). Given this finding, CSA 2 mg/kg IV continuous infusion was started with goal serum level of 300 ng/mL. Standard antibiotic prophylaxis for pneumonia with TMP/SMZ was given and IV steroids continued. By day 4 of CSA, hematochezia had stopped and stool began to form. He was discharged to home on oral CSA, prednisone, TMP/SMZ and AZA. At the time of this report (6 months later), he remains in steroid-free remission only on weight-based AZA dosing. There were no adverse events. Discussion: Primary non-response to IFX in severe colitis may be due to failure of the underlying mechanism of TNF blockade, but may also be due to rapid metabolism or rapid clearance of the drug, with emerging reports of stool loss of the drug as a potential explanation. We believe that this patient's lack of response to IFX was due to rapid clearance and anti-drug antibodies. This provided reassurance for safe use of salvage CSA. This is the first report of the use of therapeutic drug monitoring to minimize toxicity and guide IFX/CSA salvage therapy.

Methods: N/A
Results: N/A
Conclusion: N/A

**CURRENT CATEGORY:** G. Clinical Vignettes/Case Reports

**CURRENT SUB-CATEGORY:** I. Inflammatory Bowel Disease

**PRESENTATION TYPE:** Oral or Poster

**ACG Research Grant Support:** No
**Supported by Industry Grant:** No
**Commercial Products or Services:** Yes
**Initiated Research:** Industry
**Financial Relationships:** Yes

**Extra Info:** Dr. Rubin- Consultant for Prometheus Laboratories- but the assay used in this case report was NOT a Prometheus product, and Dr. Rubin has no conflict of interest to disclose for that one.

**FDA Approval:** No
**Designed Study:** Investigator
**Abstract Author:** Investigator

**AUTH DESIG: ACG Membership Status:** ACG Member

David Rubin : ACG Member
Christopher Chapman : ACG Member
Ashley Bochenek : ACG Non-Member
Adam Stein: ACG Member
(No Image Selected)
(no table selected)
AVERAGE SCORE: 3.67
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
David Hass: [No Comments] Charlene Le Pane: [No Comments] Renee Young: [No Comments] Rowen Zetterman: [No Comments]
Purpose: The number of tuberculosis (TB) cases in patients on anti-TNF therapy reported to the U.S. Food and Drug Administration has recently risen. It is difficult to detect latent tuberculosis infection (LTBI) and de novo TB infection in patients on anti-TNF therapy. Furthermore, there is a higher incidence of life-threatening disseminated disease compared to immunocompetent patients. Over 50% of reported TB cases associated with anti-TNF therapy are extrapulmonary, but ocular TB remains extremely rare. Case: The patient is a 32-year-old Iraq war veteran, diagnosed with Crohn’s colitis in 2008, who was initiated on infliximab in 2010 for gastrointestinal symptoms refractory to prednisone and azathioprine. A purified protein derivative (PPD) test and a chest x-ray were negative prior to initiation of therapy. In 2013, he complained of weight loss and vision loss in his right eye. A dilated eye exam revealed a circular area of diffuse macular whitening in the right eye, suggestive of ocular tuberculosis granuloma. He had no pulmonary symptoms. Sputum cultures confirmed mycobacterium tuberculosis. Treatment was initiated with isoniazid, pyrazinamide, rifampin and ethambutol. Infliximab was held. His vision improved. An HIV test was negative. Onset of active TB after starting infliximab is usually rapid (median of 12 weeks) with 98% of cases occurring within 6 months of initiation of TNF blockade, suggesting this was de novo tuberculosis while on anti-TNF therapy. Discussion: Guidelines in the United States recommend excluding active TB infection with chest x-ray and current PPD status. However, the sensitivity of the PPD test is compromised in patients on immunosuppressive therapy. Data regarding the use of T-cell interferon-gamma release assays (IGRA) such as the QuantiFERON-TB Gold test for the detection of TB in these patients are promising, but remain scarce. There is a need for prospective, longitudinal studies to investigate the prognostic value of IGRA tests in this population, and for guidelines for the management of anti-TNF therapy during active TB infection.
AVERAGE SCORE: 2.67
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None

REVIEWER COMMENTS:
David Hass: [No Comments]
Charlene Le Pane: [No Comments]
Renee Young: [No Comments]
Rowen Zetterman: [No Comments]
Title: A Case of Small Cell Neuroendocrine Carcinoma in a Patient with Long-Standing Ulcerative Colitis

Presenter: Renée Marchioni

Presenter (Institution Only): University of Connecticut Health Center

Presenter (Country Only): United States

Abstract Body:

Purpose: Small cell neuroendocrine carcinomas, or small cell carcinomas (SCCs), are infrequently associated with ulcerative colitis (UC) and comprise <1% of all colorectal cancers. We report a case of extensive-stage SCC in a patient with long-standing inflammatory bowel disease. A 56-year-old male with UC for 26 years presented with decreased frequency of bowel movements and sudden onset of vomiting. His UC had been in prolonged remission until 5 months prior, when he experienced abdominal cramping associated with 15 bloody bowel movements daily, rectal urgency and tenesmus. This improved with tapered prednisone in combination with his established maintenance regimen of sulfasalazine 2 grams daily. Over the subsequent 8 weeks, he reported fatigue, anorexia, 15-pound weight loss and worsening low back pain. Previous surveillance colonoscopies had been stable; his most recent evaluation 1 year prior was biopsy negative for dysplasia or active inflammation. He had never received immunosuppressant therapy. Abdominal radiograph on presentation showed multiple air-fluid levels in the small bowel consistent with obstruction. Computed tomography revealed a proximal sigmoid colon mass with short segment mural thickening resulting in partial distal obstruction with extensive lymphadenopathy. Diffuse metastatic disease was seen throughout the abdomen and pelvis, with serosal, omental, peritoneal, hepatic and osseous foci. Colonoscopy revealed severe ulceration of the rectosigmoid with an impassable mid-sigmoid stricture. Biopsy of the stricture was negative for dysplasia but confirmed exacerbated UC. Laboratory studies showed a serum CEA>1,900 (0.0-0.5 ng/mL) and CA 19-9 of 2,262 (0-37 U/mL). Diagnostic laparoscopy with lymph node and liver biopsies revealed high grade metastatic small cell neuroendocrine carcinoma. The patient was emergently initiated on chemotherapy with cisplatin and etoposide, and a palliative stent was placed in the sigmoid colon. Primary colorectal SCC is a rare but highly aggressive malignancy that requires prompt recognition and treatment; practitioners should maintain a high index of suspicion for this virulent entity and its manifestation in the setting of long-standing UC. Despite adherence to endoscopic surveillance with biopsy protocols, this lesion appeared to arise and metastasize quickly from an area of active colonic inflammation. Further investigation is warranted to elucidate how small cell neuroendocrine tumor development is influenced by extent and duration of disease, genetics, environment, diet and pharmacotherapy. Identifying predisposing factors may modify treatment and endoscopic surveillance guidelines for patients with UC at risk for such neoplasia.

Methods: N/A

Results: N/A

Conclusion: N/A

Current Category: G. Clinical Vignettes/Case Reports

Current Sub-Category: I. Inflammatory Bowel Disease

Presentation Type: Oral or Poster

ACG Research Grant Support: No

Supported by Industry Grant: No

Commercial Products or Services: No

Initiated Research: Investigator

Financial Relationships: Not Applicable

FDA Approval: No

Designed Study: Investigator

Abstract Author: Investigator

Auth Design: ACG Membership Status <font color="red">*</font>:

Renée Marchioni : ACG Member

Thomas Devers : ACG Non-Member

Jessica Clement : ACG Non-Member

(No Image Selected)
AVERAGE SCORE: 3.33
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
David Hass: [No Comments]|Charlene Le Pane: [No Comments]|Renee Young: [No Comments]|Rowen Zetterman: [No Comments]
Massive Eosinophilic Infiltration of the Colon in an Early Case of Inflammatory Bowel Disease

Mustapha El-Halabi
University of Kansas School of Medicine - Wichita
United States

Purpose: Eosinophilic infiltration of the colon is very rare and is usually secondary to eosinophilic colitis (EC), few autoimmune diseases, parasitic infections or intake of certain drugs. We herein report massive eosinophilic infiltration of the whole colon in a case of early Inflammatory Bowel Disease (IBD).

34-year-old African American male, previously healthy, presented with 1 year history of crampy diffuse abdominal pain associated with diarrhea and 3 bowel movements per day that were mostly mucoid but not bloody. He reported only several episodes of fresh blood per rectum over the last 6 months, but denied melena, fever, chills, oral ulcers, rashes, arthralgias, weight loss, or any other complaints. He did not have a family history of IBD. White blood cell count, sedimentation rate and C-reactive protein were within normal limits. Stool studies showed some leukocytes but C. difficile PCR and stool cultures were negative. Colonoscopy showed pan-colitis starting from the anal verge without involvement of the terminal ileum. A linear ulcer was present in the rectosigmoid region extending 4 cm in length. Four quadrant biopsies were obtained every 10cm. Pathology showed mild active chronic colitis with massive eosinophilic infiltration throughout the entire colon. No inflammation was noted on the biopsy from the terminal ileum. No parasites were identified. This colitis was considered, by two gastrointestinal pathologists, not to be eosinophilic colitis but instead to be an early form of IBD because of convincing abnormal architecture. The patient was started on mesalamine and oral steroids. One month later, he reported complete resolution of his symptoms.

Massive eosinophilic infiltration of the colon can be seen very rarely in an early form of inflammatory bowel disease without elevated serum inflammatory markers.

Methods: NA
Results: NA
Conclusion: NA

G. Clinical Vignettes/Case Reports
I. Inflammatory Bowel Disease
Poster Only
No
No
Not Applicable
No
Investigator
Mustapha El-Halabi : ACG Non-Member
Nabil Mansour : ACG Non-Member
Daniel Lalich : ACG Non-Member
William Salyers : ACG Non-Member
NA
4.33
None
David Hass: [No Comments]|Charlene Le Pane: [No Comments]|Renee Young: [No Comments]|Rowen Zetterman: [No Comments]
ABSTRACT BODY:

Purpose: Case report: A 58-year old male with history of Crohn’s iliocolitis diagnosed in 1997, status post right hemicolectomy secondary to obstruction in 2007, was being evaluated for abdominal pain, nausea, and vomiting of 2 days duration. His medications included mesalamine 2.4 gm per day, losartan, and omeprazole. Vital signs were unremarkable and physical examination revealed slightly distended abdomen with hypoactive bowel sounds. Flat and upright x-ray of abdomen was unremarkable without evidence of obstruction. Computerized Tomography scan of abdomen showed early or partial small bowel obstruction most likely secondary to adhesions. Patient was given trial of prednisone with improvement in his symptoms. After all the appropriate workup including negative PPD skin test and hepatitis panel, patient was started on adalimumab. Patient did well with initial injections but developed injection site erythema along with facial swelling and tightness in the throat with second dose. He was treated with methylprednisolone and adalimumab was discontinued. TSH came back elevated at 28.60 mIU/ml (Ref Range: 0.465 to 4.68 mIU/ml). As the patient did not have any history of thyroid disease, repeat TSH was ordered for confirmation of initial result and it showed worsening of TSH at 45.3 mIU/ml in just 3 days. Free T4 was low at 0.59 ng/dl (Ref Range 0.78 to 2.19ng/dl). Thyroid peroxidase antibody was elevated at 150 IU/ml (Ref Range: 0 to 34 IU/ml) and anti-thyroglobulin antibody was negative. Patient was started on levothyroxine 50 MCG per day. Repeat lab works in 2 month showed normal free T4 along with improvement in TSH to 19.6 mIU/ml. Patient is currently being treated with azathioprine for his Crohn’s disease. Discussion: We report a rare case of thyroid dysfunction associated with adalimumab use. Adalimumab is a recombinant anti-tumor necrosis factor alpha (TNF-alpha) IgG1 monoclonal antibody which blocks inflammatory activity of TNF-alpha. Major indications for use of adalimumab are moderate to severe Crohn’s disease, ulcerative colitis, rheumatoid arthritis, psoriatic arthritis, and other autoimmune diseases. The most common adverse reactions are infections (e.g., upper respiratory, sinusitis), injection site reactions, headache, and rash. A study by eHealthMe has reported 15 patients out of 130,505 developing autoimmune thyroiditis while taking Adalimumab which accounts for incidence rate of 0.01 %. Majority of patients were female (84.21%). A case of subacute thyroiditis after being treated with Adalimumab for refractory palmo-plantar pustular psoriasis has also been described in the literature.

Methods: N/A
Results: N/A
Conclusion: N/A
AVERAGE SCORE: 4.33
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
David Hass: [No Comments] Charlene Le Pane: [No Comments] Renee Young: [No Comments] Rowen Zetterman: [No Comments]
Purpose: Introduction: Polyarteritis nodosa (PAN) is a vasculitis of medium and small-sized arteries, primarily occurring in the kidneys, and nervous system. Case: We present a 70-year-old man with two episodes of hematochezia in the last 24 hours. Patient was resuscitated with intravenous (IV) fluids, and 17 units of packed red blood cells (RBC) over the hospital course. Tagged RBC scan demonstrated evidence of active bleeding in the small bowel and the ascending colon. Angiography showed multiple pseudoaneurysms in branches of the left gastric, splenic and superior mesenteric arteries (SMA). Gastroenterology performed an upper gastrointestinal (GI) endoscopy, single balloon enteroscopy and colonoscopy; which did not yield a source of bleed, yet the hematocrit continue to plummet. An alternate diagnosis of PAN was considered. Rheumatology suggested IV steroids, after which the patient’s hematocrit stabilized with complete resolution of symptoms. Further workup demonstrated low complement supporting the diagnosis of PAN. Discussion: PAN typically presents with an array of systemic symptoms, however, a subset of patients have mesenteric arterial involvement with no clinical evidence of extraintestinal symptoms. One study demonstrated that angiographic diagnosis of PAN has a sensitivity of 89% and a specificity of 90%. In patients with severe PAN, characterized by GI bleed, IV steroids should be initiated for three days followed by other immunosuppressant therapy. In this case the patient presented with an acute lower GI bleed and no other clinical symptoms of PAN. Upon failed attempts to visualize a source of GI bleed, a mesenteric angiography demonstrated multiple pseudoaneurysms. Persistent lower GI bleed only subsided with IV steroids, resulting in an interdisciplinary consensus that his GI bleed was likely secondary to PAN. In conclusion this case demonstrates that systemic vasculitis can present with serious GI hemorrhage, even in the absence of other signs and symptoms of PAN. Mesenteric angiography clearly demonstrated that vasculitic lesions can develop and resolve rapidly with prompt, aggressive management.

Methods: N/A
Results: N/A
Conclusion: N/A

CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease
PRESENTATION TYPE: Poster Only
ACG Research Grant Support: No
Supported by Industry Grant: No
Commercial Products or Services: No
Initiated Research: Investigator
Financial Relationships: Not Applicable
FDA Approval: No
Designed Study: Investigator
Abstract Author: Investigator

AUTH DESIG: ACG Membership Status <font color="red">^</font>:
Arman Khorasani-zadeh : ACG Member
Meghan Rane : ACG Member
Nasser Hajar : ACG Member
Renee Williams : ACG Member

IMAGE CAPTION:
(no table selected)
AVERAGE SCORE: 3.33
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
David Hass: [No Comments]
Charlene Le Pane: [No Comments]
Renee Young: [No Comments]
Rowen Zetterman: [No Comments]
Drug Induced Liver Injury (DILI) from Initial Dose of Infliximab

Fong-Kuei Cheng

Walter Reed National Military Medical Center
United States

Purpose: Introduction: Infliximab, a chimeric monoclonal antibody to tumor necrosis factor (TNF)-α, has proven efficacy in treating ulcerative colitis (UC) and Crohn’s disease (CD). Infliximab related hepatotoxicity was first reported in 2001 with 26 cases known to date. The majority of these cases have involved autoimmune features and presented at a median of 12-18 weeks of therapy. Other reported cases without autoimmune features presented at a median of 6.5 weeks of therapy. We present a unique case of infliximab DILI without autoimmune features, occurring after the initial therapy.

Case Report: A previously healthy 2-year-old man was admitted for severe UC flare with 10-15 loose bloody bowel movements (BM) per day, and 18 lbs weight loss. Pulse of 100 and moderate diffuse abdominal tenderness were present on exam. C reactive protein (CRP) was 90 mg/L, abdominal x-ray ruled out megacolon, and infectious workup including Clostridium difficile was negative. Flexible sigmoidoscopy with biopsies demonstrated severe procto-sigmoiditis. Marginal clinical response was achieved after 72 hours of IV methylprednisolone. Salvage therapy with infliximab 10 mg/kg was started on day 5. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) then rose on day 7, peaking to 213 and 124 U/L respectively on day 11, and normalizing on day 13. Potential hepatotoxic medications including rifaximin and omeprazole were previously discontinued. ALT and AST rose again on day 13 following repeat infliximab infusion, peaking to 151 and 454 respectively on day 18. Serology for hepatitis A, B, and C, Epstein-Bar virus, herpes simplex virus I & II, cytomegalovirus (CMV), antinuclear antibody, smooth muscle antibody, mitochondrial antibody, ceruloplasmin, and right upper quadrant with dopplers ultrasound were negative. Due to lack of response, the patient underwent subtotal colectomy with loop ileostomy. Intra-op core liver biopsy showed patchy portal inflammation without cholestasis. CMV stains on liver and colon tissue were negative. Postoperatively, the patient had an uneventful recovery with normalization of aminotransferases.

Discussion: Acute hepatotoxicity secondary to infliximab has been shown to occur with or without autoimmunity. However, a growing body of infliximab DILI cases without autoimmune features is emerging. These cases present earlier, after fewer doses, and suggest a direct pattern of injury without autoantibody formation. This is a unique case that presents DILI on initial infliximab dose, while nearly all other reported cases occurred after at least three doses. Further study is needed to understand the underlying mechanism of infliximab’s direct injury on the liver.

Methods: N/A
Results: N/A
Conclusion: N/A

G. Clinical Vignettes/Case Reports
I. Inflammatory Bowel Disease

Oral or Poster
AVERAGE SCORE: 3
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
David Hass: [No Comments]
Charlene Le Pane: [No Comments]
Renee Young: [No Comments]
Rowen Zetterman: [No Comments]
Purpose: This is a case of a 41-year-old Caucasian female who presented for evaluation of worsening abdominal pain and recurrent bloody diarrhea. Other symptoms included seven years of recurrent multiple painful oral ulcers, genital ulcers, tender nodules around her ankles, and arthralgias. She also had history of recurrent eye redness that was ameliorated by steroid drops. Three months prior, she had developed sudden right-sided hemiparesis thought to be secondary to a left middle cerebral artery stroke although her brain imaging was normal; her symptoms improved with steroids given for presumed vasculitis. Her laboratory data showed normal ANA and rheumatoid factor with elevated ESR, CRP, and immunoglobulins. Colonoscopy showed marked punched out round ulcers in the cecum, ileocecal valve, and terminal ileum. Biopsies revealed chronic active ileitis/colitis. No definite vasculitis was seen; however, submucosal tissue was not present on biopsies. She was diagnosed with Behcet’s disease and was started on infliximab with improvement in her symptoms. Behcet’s disease is classified as a vasculitide with geographic distribution in countries along the Silk Road (eastern Asia to the Mediterranean basin) which affects young adults in their second and fourth decades of life. Diagnosis is based on the following criteria: presence of recurrent oral ulcerations (usually painful, punched-out ulcers with rolled edges located on the inner oral mucosa) plus two of the following; genital ulcers, ocular lesions, skin lesions, or positive pathergy test. GI involvement occurs in about 10% to 15% of Behcet’s disease patients; symptoms usually manifest 5-6 years after oral ulcers and are associated with papulo-pustular lesions and pyoderma gangrenosum. Colonoscopy usually shows ileocecal and right colon ulcers which are described as volcano-type (deeply penetrating ulcers with nodular margins), geographic-type (shallow ulcers with sharp edges), and aphthous-type (small punched out shallow ulcers). Colonic ulcers tend to be round and focal in intestinal Behcet’s disease compared to longitudinal and segmentally diffuse in Crohn’s disease. Biopsies often show vasculitis. Diagnosis is made by presence of typical intestinal ulcers and systemic manifestations of Behcet’s disease. Treatment of intestinal Behcet’s disease usually involves infliximab as it has been shown to cause sustained resolution of GI symptoms; however, this data is based on small studies without standardization of treatment response.

Methods: N/A

Results: N/A

Conclusion: N/A
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
David Hass: [No Comments] Charlene Le Pane: [No Comments] Renee Young: [No Comments] Rowen Zetterman: [No Comments]
Purpose: Acute pericarditis can be a life threatening condition and should be on the differential diagnosis in any patient who presents with chest pain. Diagnosis can be readily made with a careful history, physical examination and appropriate investigational studies. Determining the etiology of the condition can be more difficult as the causes are varied. While drug-induced pericarditis is essentially a diagnosis of exclusion, the clinician must maintain a high index of suspicion in patients receiving mesalamine or other 5-aminosalicylic acid compounds. These medications have been associated with acute pericarditis in rare case reports. A 22-year-old man with a past medical history of ulcerative colitis presented with diffuse abdominal pain and bloody diarrhea. He had been off all treatment for the previous two years. After a thorough evaluation, his symptoms were attributed to an ulcerative colitis flare and was subsequently started on mesalamine 1,600 mg three times a day, in addition to an oral steroid taper. Three weeks after discharge, the patient returned to the emergency room with sudden-onset substernal chest pain. Deep inspiration and sitting up exacerbated the pain. He also endorsed shortness of breath, but no nausea or vomiting, fevers, or cough. His abdominal pain and bloody diarrhea had resolved. On physical exam, the patient was noted to be in mild distress. His jugular venous pressure appeared normal. The chest pain was not reproducible with palpation of the sternum. His cardiovascular exam was unremarkable with no murmurs or rubs. His abdominal exam revealed normal bowel sounds and was soft, non-tender and non-distended. No hepatosplenomegaly was appreciated. Initial laboratory workup revealed an elevated white blood cell count of 15.4 thousand cells/microliter (reference range 4.2-10.8). He was also found to have an elevated troponin of 0.258 ng/ml (normal <0.006, intermediate 0.07-0.49). EKG was remarkable for diffuse ST segment elevations. Echocardiography did not show wall motion abnormalities or a pericardial effusion. Pertinent findings included a negative antinuclear antibody, HIV screen and blood cultures. Based upon the symptoms and initial work-up, the chest pain was diagnosed as pericarditis and was treated with non-steroidal anti-inflammatory medications. Given the temporal relationship between the initiation of mesalamine therapy and the clinical presentation of chest pain, drug-induced pericarditis was suspected, and mesalamine was discontinued.

Methods: N/A
Results: N/A
Conclusion: N/A

CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease
PRESENTATION TYPE: Oral or Poster

AVERAGE SCORE: 3.75
REVIEWER FLAGS: James Buxbaum - Newsworthy?: 1
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
ABSTRACT BODY:

Purpose: Introduction: Patients with inflammatory bowel disease (IBD) are susceptible to thromboembolic complications. This occurs secondary to dysregulation of coagulation activity including disturbances of fibrinolysis, inflammatory reactions and thrombocytosis. The thromboembolic effect may involve both the arterial and venous systems. Thromboembolism (TE) often occurs in the deep veins of the leg and pulmonary circulation, but may also occur less frequently in other sites, including the cerebrovascular system, portal vein, mesenteric veins, and retinal vein. Here we report a case of a man who suffered a cerebellar stroke secondary to ulcerative colitis (UC).

Case: A 38-year-old man with a history of UC presented to an outside hospital for dizziness and severe headache. Non-contrast computed tomography (CT) of the head was negative. He was treated for headache and vertigo and discharged on pain medications and meclizine. Five days later his symptoms persisted and he presented with worsening frontal headache accompanied by blurry vision, loss of balance, and vomiting. Exam was remarkable for right eye ptosis but there was no facial sensory loss and the remainder of the neurological exam was unremarkable. At age 20 he was diagnosed with UC and currently treated with sulfasalazine. He was compliant with his medication until three weeks prior to presentation because failed to meet his appointment with his physician. A non-contrast head CT revealed an infarct involving the right cerebellar hemisphere in the distribution of the superior cerebellar artery. Cerebral angiogram revealed an embolic CVA in the right posterior inferior cerebellar artery (PICA). A hypercoagulable work-up was performed including prothrombin G20210A mutation, Factor V Liden mutation, IgM and IgG for anti-B2 glycoprotein, anticardiolipin antibody, and ANA which were negative; and antithrombin III, homocysteine, protein C and S function which were normal. Therefore, we concluded that UC was the most likely cause of the TE.

Discussion: The consequences of embolic CVA can be devastating and may be more complicated in the setting of active IBD. Studies suggest that TE is more frequent in the active phase of IBD. However, 20-30% of TE complications can occur during disease quiescence, suggesting that the procoagulant tendency in IBD is independent of disease activity. Treatment of TE in this setting involves the management of the underlying IBD. 5-aminosalicylic acid, a mainstay of IBD therapy, induces a wide array of modulatory activities, including the inhibition of platelet activation. Therefore, physicians should be aware of the risk of TE in IBD patients and treatment should be directed at minimizing its risks.

Methods: N/A

Results: N/A

Conclusion: N/A
AVERAGE SCORE: 4
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Purpose: A 28-year-old male with a past medical history of Crohn's colitis presents with worsening abdominal pain and diarrhea. His index colonoscopy revealed pancolitis but later showed patchy colitis with sparing of the rectum and ileum. His symptoms were refractory to adalimumab and infliximab with low-dose 6-MP. The patient was positive for JC virus antibody, so natalizumab was deferred. He was started on ustekinumab and low-dose methotrexate with minimal response and developed weekly muscle swelling with associated pain and redness. Symptoms did not improve with prednisone taper. The patient was admitted for expedited management. Exam revealed abdominal tenderness without rebound as well as painful left forearm and right leg swelling. Initial labs yielded a normal WBC, mild microcytic anemia, and elevated inflammatory markers (CRP 72.5, ESR 40). Metabolic and liver panels and albumin were normal. Infectious stool studies were negative. Flexible sigmoidoscopy biopsies showed inflammation but no CMV infection. The patient was started on methylprednisolone 40mg IV daily and natalizumab 300mg IV q4 weeks and placed on parenteral nutrition. Dermatology performed punch biopsy of acellular plaque on the left forearm which revealed subcutaneous septal leukocytoclastic vasculitis (LCV). A subsequent painless rash developed, which demonstrated LCV on biopsy. Lyme, parvovirus, and hepatitis serologies; anti-Streptolysin O titers; ANCA and complements C3 and C4 were all unremarkable. Urine protein-to-creatinine ratio was normal. The patient was referred for subtotal colectomy with end ileostomy, but he wished to defer surgery. His symptoms gradually improved, and he was transitioned to an oral diet and prednisone and discharged home on natalizumab. Conclusion: LCV is believed to be an immune complex disorder that can be triggered by various drugs, infections, malignancies, and systemic and autoimmune disorders. In this case, the patient’s LCV was felt likely due to his inflammatory bowel disease or possibly medication (eg, methotrexate). The atypical appearance of his initial rash was felt due to the subcutaneous location of his LCV. LCV is less frequently seen in IBD compared to other skin manifestations such as erythema nodosum or pyoderma gangrenosum. Skin lesions of LCV usually spontaneous self-resolve or can be treated with corticosteroids, dapsone, colchicine, or immunosuppressive agents.

Methods: N/A
Results: N/A
Conclusion: N/A
AVERAGE SCORE: 4
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Purpose: Background: The risk of venous thromboembolism (VTE) amongst patients with inflammatory bowel disease (IBD) is increased by a 2-4 time fold compared to non-IBD patients. This case describes a patient who incidentally was diagnosed with Crohn's disease while investigated for a pulmonary embolism. History: A 66-year old lady was admitted to our hospital after a witnessed episode of loss of consciousness. She had recently been on a 2-hour aeroplane flight. She had lost weight recently but her medical history was otherwise non-contributory and was not on any regular medications. She was an ex-smoker. On physical examination a systolic heart murmur was diagnosed. She was pyrexial. The rest of the examination was unremarkable. Laboratory assessment revealed elevated inflammatory markers and a raised d-dimer. Her head CT was normal and echocardiogram showed severe aortic stenosis. A chest radiograph was normal but CT pulmonary angiogram confirmed a pulmonary embolism (PE). Gastroscopy was normal and cross sectional imaging of the thorax, abdomen and pelvis were showed terminal ileal thickening consistent with Crohn's disease. Ileo-colonoscopy with biopsies confirmed mild to moderately active ileitis but no colonic disease. With the possible exception of weight loss she had no other symptoms attributable to IBD. Her son was diagnosed some years ago with ulcerative colitis from which he was asymptomatic after investigations for pulmonary embolism. Management: She has been anticoagulated with warfarin and awaits surgery for aortic valve replacement. She is currently being managed with aminosalicylates despite limited evidence for 5-ASA's in Crohn's disease given the relatively short segment of otherwise asymptomatic disease with a plan to reassess disease after aortic valve replacement. Discussion: Venous thromboembolism (VTE) is a well-recognised complication of IBD with an overall mortality as high as 25% per episode. Despite emerging evidence that Inflammation and coagulation are interdependent processes that perpetuate and intensify each other, this important complication may not be appreciated as well as it should and a high index of suspicion and prompt treatment are necessary for optimal outcomes. Up to 75% of IBD patients may have no identifiable provoking factors but as many as a third of these will have recurrent VTE within 5 years. Between 55-80% have active IBD at diagnosis of VTE. There remains paucity of clinical data driving evidence-based decisions and although anticoagulation is the cornerstone of therapy, randomised trials testing catheter directed thrombolysis or anticoagulation are much needed.

Methods: N/A
Results: N/A
Conclusion: N/A
CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease
PRESENTATION TYPE: Oral or Poster
ACG Research Grant Support: No
Supported by Industry Grant: No
Commercial Products or Services: No
Initiated Research: Investigator
Financial Relationships: Not Applicable
FDA Approval: No
Designed Study: Investigator
Abstract Author: Investigator
AUTH DESIGN: ACG Membership Status <font color="red">*</font>:
Stephanie Soteriadou : ACG Member
Jimmy Limdi : ACG Member
(No Image Selected)
(no table selected)
AVERAGE SCORE: 4.25
ABSTRACT BODY:

Purpose: A 48-year-old man with ulcerative pancolitis diagnosed 6 years prior to admission was transferred to our institution with sharp non-radiating right lower quadrant pain. There was no history of fever, chills, nausea, vomiting, or diarrhea. The patient had been in remission on azathioprine 100 mg and mesalamine 2,000 mg per day after a single course of prednisone at the time of diagnosis. Physical exam was significant for tenderness to palpation in the right lower quadrant. The white blood cell count and hemoglobin were normal. A computed tomography scan of the abdomen with oral and intravenous contrast demonstrated an inflammatory process at the cecal apex with associated 5 cm phlegmon. The appendix was not visualized. Mild lymphadenopathy was present. The small bowel and colon appeared normal with the exception of possible narrowing and hypertrophy of the terminal ileum at the ileocecal valve. The patient was diagnosed with appendicitis and managed conservatively with intravenous antibiotics and bowel rest. His diet was slowly advanced and he was discharged home on a two week course of ciprofloxacin 500 mg twice daily and metronidazole 500 mg three times daily. Subsequent colonoscopy revealed a 4 cm non-obstructing mass in the cecum. The colon distal to the cecum was normal without mucosal breaks or friability. Biopsies of the cecal mass revealed normal colonic mucosa with mild inflammation and mild architectural change. Diagnostic laparoscopy demonstrated an abnormal appearing inflamed appendix. A laparoscopic appendectomy was performed. The surgical specimen measured 5.6 x 4.5 x 3.0 cm and contained no masses. Architectural distortion, transmural acute and chronic inflammation, and epithelioid cell aggregates with giant cell reaction were present. The patient’s diagnosis was changed to Crohn’s colitis with appendiceal involvement. Crohn’s disease (CD) involves the appendix in up to 50% of patients with colonic CD. CD isolated to the appendix is rare and has been described in fewer than 160 cases. We believe that this is the first report of a patient diagnosed with UC to have his diagnosis changed due to symptomatic appendiceal CD. Symptoms of granulomatous disease of the appendix are similar to acute appendicitis and include fever, right lower quadrant pain, nausea, and anorexia. Other causes of appendiceal granulomatous disease include infection (e.g., Yersinia, Mycobacterium tuberculosis, Campylobacter), foreign body reaction, appendiceal or cecal diverticulitis, chronic beryllium poisoning, and sarcoidosis. Our patient did well post-operatively. Azathioprine and mesalamine were continued.

Methods: N/A
Results: N/A
Conclusion: N/A
AVERAGE SCORE: 4.25
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Haritha Avula: [No Comments]
James Buxbaum: [No Comments]
Raquel Davila: [No Comments]
Brian Weston: [No Comments]
Purpose: Three patients, two with ulcerative colitis and the other with Crohn’s disease, tested positive on serologic QFT-G TB testing. In all three the PPD test was negative prior to biologic therapy. Case 1: 54-year-old white male with diagnosis of pancolitis since 1999. He was on maintenance mesalamine and adalimumab. As a child, he lived in Tanzania for 2 years. He reported a cough productive of yellow phlegm. Chest CT showed a partially calcified R hilar node. He denied any fever, night sweats or weight loss but had a positive QFT-G TB. All sputums were negative for AFB. He was started on treatment for latent TB infection (LTBI). Case 2: 35-year-old white male with history of left sided colitis since 2004. While on adalimumab maintenance he developed a fever and shortness of breath with radiographic findings of a left lower lobe pneumonia. He had a positive QFT-G TB test. He lived 3 years as child in Australia and a former roommate was a PPD converter. His pneumonia resolved after antibiotic treatment, all sputums were negative for AFB. He was treated for LTBI. Case 3: 31-year-old asymptomatic male with a 5 year history of colonic Crohn’s disease. He was on maintenance adalimumab. QFT-G TB was positive on routine testing for medication renewal. Chest x ray and repeat PPD test were negative. He gave no exposure history. Patient was continued on adalimumab and was not started on treatment for LTBI. Discussion: In the three cases above, the two with an extensive travel history and exposure risks were treated for LTBI. Case number 3 was not treated due to low pretest probability for TB but observed and continued on biologic treatment. Quantiferon test (QFT) became the first IGRA approved by FDA in 2003 as an aid for diagnosing M. tuberculosis infection. Presently there is no “gold standard” to confirm a diagnosis of LTBI or culture-negative active tuberculosis. In persons with untreated, culture-confirmed TB, the sensitivity of QFT-G for detecting M. tuberculosis infection is approximately 80%, but the sensitivity in specific subgroups, such as young children and immunocompromised patients, is still unknown. For LTBI, QFT-G sensitivity might be less than that of the skin testing, but the lack of a confirmatory test makes this difficult to evaluate as in the three patients above. Conclusion: When faced with a disparity of testing results, clinicians caring for patients with IBD on biologics and who order a QFT-G in lieu of repeat PPD testing, must be aware of how to interpret the results. Application of this interpretation to individual patients will have significant clinical impact on therapy.

Methods: n/a
Results: n/a
Conclusion: n/a

CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease
PRESENTATION TYPE: Oral or Poster

ACG Research Grant Support: No
Supported by Industry Grant: No
Commercial Products or Services: No
Initiated Research: Investigator
Financial Relationships: No
FDA Approval: Yes
Designed Study: Investigator
Abstract Author: Investigator

AUTH DESIG: ACG Membership Status <font color="red">*</font>
Leon Kundrotas : ACG Member
Fnu Rajlakshmi : ACG Non-Member
(No Image Selected)
(no table selected)

AVERAGE SCORE: 3.25
Purpose: Chronic inflammation in the terminal ileum (TI) suggests a cause for the patient’s symptoms, especially when the clinical suspicion is Crohn’s disease. Non-steroidal anti-inflammatory drugs, lymphoid hyperplasia, intestinal infections, lymphoma, infections and ulcerative colitis (UC) are some of the other causes of terminal ileitis. This is an unusual case of a 41-year-old multiparous female with past medical history of small bowel obstruction and colitis who presented with a 5 day history of crampy abdominal pain, nausea, vomiting and diarrhea. On the CT scan of the abdomen she was found to have small bowel obstruction with segmental enteritis in close proximity to the terminal ileum. She was started on IV antibiotics and steroids, kept NPO and NG tube was placed to relieve the obstruction. Due to little improvement in the obstruction, she underwent exploratory laparotomy with resection of the narrowed small bowel segment with side to side anastomosis and appendectomy. Patient was started on temporary parenteral feeding, and continued on IV steroids for management of suspected inflammatory bowel disease. Strangely, the pathology results for the small bowel resection specimens showed presence of endometriosis in both the appendix and terminal small bowel segment without any evidence of inflammatory bowel disease. Her steroids were soon tapered off and with tolerance of oral feeds patient was discharged from the hospital with outpatient gynecology follow-up for management of endometriosis. Ileal endometriosis should be carefully considered in the differential diagnosis of Crohn’s disease in menstruating females, especially the ones who are nulliparous and have dysmenorrhea, dyspareunia, dyschezia, menometrorrhagia or other peri-menstrual symptoms. This case exemplifies the importance to not mistake endometriosis for Crohn’s disease as they each have different therapeutic management.

Methods: N/A
Results: N/A
Conclusion: N/A
Purpose: Background: Perforation occurs in less than one percent of cases undergoing diagnostic colonoscopy. Similar perforation rate is reported in inflammatory bowel disease (IBD) patients, although there is disagreement in current literature. Severe disease on endoscopy and chronic steroid use have been associated with higher risk of perforation in IBD patients. This case describes colonic perforation after a random cecal biopsy during a surveillance colonoscopy in a well-controlled IBD patient previously exposed to years of chronic steroids. We speculate the needle in the middle of standard biopsy forceps may have punctured susceptible thin-walled cecum, resulting in perforation.

Case: 39-year-old female with long-standing ileocolonic Crohn's disease since the age of 14 without upper tract or perianal involvement maintained on asacol and imuran underwent diagnostic colonoscopy. Her colonoscopy 5 years prior was complicated by perforation due to endoscope trauma at the splenic flexure requiring diverting ileostomy and subsequent reversal. Chronic steroids resulted in right ankle bone graft approximately 15 years prior. Her Crohn’s disease has been well-controlled for the last 10 years without further exposure to steroids. She was asymptomatic and inflammatory markers were normal prior to surveillance colonoscopy. Carbon dioxide was used for insufflation and the terminal ileum was intubated without difficulty. Colonoscopy revealed scattered pseudopolyps throughout the colon. A random cecal biopsy was taken using a Radial Jaw ® 4 Standard Biopsy Forceps with Needle (Boston Scientific, Natick, MA). Immediately after this biopsy, perforation was recognized and three endoclips were placed. Pneumoperitoneum was confirmed on abdominal x-ray. The patient was admitted for observation with intravenous antibiotics and fluids. Twenty-four hours after the procedure, the patient was asymptomatic and discharged without the need for surgery. Biopsies showed quiescent colitis, negative for dysplasia. Conclusion: Chronic steroid use is a known risk factor for perforation during diagnostic colonoscopy in IBD patients. This case illustrates such risk from chronic steroids may linger for many years. Endoscopists should consider needleless biopsy forceps in cases of IBD patients on chronic steroids requiring multiple surveillance biopsies.
REVIEWER RECOMMENDATION CODE DESCRIPTION: None

REVIEWER COMMENTS:
Purpose: Pyoderma Gangrenosum is a neutrophilic dermatosis that presents with painful, ulcerating lesions. Over 50% of patients with this disorder will have an underlying systemic disease, including inflammatory bowel disease, inflammatory arthritis or hematologic malignancy. Here we present a patient who had been managed in the outpatient setting over several years with what was presumed to be recurrent MRSA lesions. Her ulcerative lesions progressed to large painful ulcers with purulence and required hospitalization. Upon examination, they were highly suggestive of Pyoderma Gangrenosum (Figure 1). Biopsy showed a necrotic epidermis and dermis with underlying inflammatory cells. Rare abscess formation was noted. History noted significant recurrent abdominal cramping with occasional tenesmus and episodic bloody stool. High dose steroid therapy was administered. The patient underwent a colonoscopy which showed ulceration of the distal colon to the splenic flexure. Biopsy proven ulcerative colitis was diagnosed. Pyoderma gangrenosum is a well-associated skin manifestation of inflammatory bowel disease. Typically associated with female patients such as ours, it does not seem to correlate with underlying colon inflammation. It is often mis-diagnosed for weeks to months. There are several different presentations of PG. Classically it is associated with a very painful deep skin ulceration including the dermis and epidermis with a clearly defined border. Size can range from several cm to encompassing an entire extremity circumference. There also is a pustular and bullous variant. These are less frequent than the classic version but have their namesake characteristic appearance. There are also reports of peristomal pyoderma associated only with an abdominal stoma. Treatment typically includes aggressive immune suppression with steroids or other immune modulating agents such as TNF inhibitors. Often, the disease will improve significantly over days with resolution of the patient’s symptoms. The lesions, however, may take weeks or months to fully heal.

Methods: NA

Results: NA

Conclusion: NA

CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease

AVERAGE SCORE: 4.25

REVIEWER FLAG: (none)

REVIEWER RECOMMENDATION CODE DESCRIPTION: None

REVIEWER COMMENTS:
Purpose: A 48-year-old man with no significant past history was evaluated by Gastroenterology three years ago for a history of acute diarrhea, chronic perirectal fistula and abscess. Colonoscopy showed extensive pancolitis with innumerable aphthous ulcers and pseudopolyps. No fistulous tracts were noted in the rectum. Small bowel follow-through (SBFT) was normal. Findings were consistent with a diagnosis of Crohn’s disease. He was initiated on treatment with sulfasalazine, short course of metronidazole and achieved a clinical remission. Over the next few years, symptoms remained well-controlled with the exception of two episodes of recurrent perirectal abscess and fistulous drainage. He received a few courses of antibiotics. A repeat colonoscopy showed moderate patchy pancolitis with relative sparing of the rectum; however, an inflamed area was noted in the last 2 cm of the rectum close to the anal verge. Rectal biopsies showed ulcerated moderately differentiated adenocarcinoma. Pre-operative staging showed no evidence of metastatic disease and he underwent abdominoperineal resection (APR) with colostomy placement. Pathology showed signet cell adenocarcinoma with extensive mucin secretion. Staging showed T3N1M0 and he underwent twelve treatments of adjuvant chemotherapy. Repeat colonoscopy one year later showed continued mild active disease throughout the large bowel but biopsies were negative for dysplasia. Currently, he remains asymptomatic without evidence of recurrence of colorectal cancer. Mucinous adenocarcinoma is a rare variant of colorectal cancer, accounting for approximately 5% to 15% of cases. Diagnosis is based on histological assessment, with more than 50% of the lesion containing extracellular mucin. A diagnosis of signet ring adenocarcinoma is made if greater than 50% of the cells are identified as signet ring cells. Malignant transformation of perirectal fistulas in Crohn’s disease is even rarer, with only 61 cases reported in the literature from 1950 to 2008. According to a systematic review by Thomas et al, most individuals with Crohn’s disease who developed malignancy arising from a perianal fistula were females (61%) with a shorter duration of inflammatory bowel disease (IBD) than males (18 years vs. 24 years). As in our patient, the most common histology noted was adenocarcinoma (59%), with the most common fistula location being in the rectum (59%). Our hypothesis is that malignant transformation may occur as a result of chronic inflammation and bacterial infiltration of the inflamed fistulous tract. Malignancy should be kept in the differential diagnosis of any patient with recurrent fistulizing disease, especially if it is refractory to medical therapy.

Methods: N/A
Results: N/A
Conclusion: N/A

AUTH DESIG: ACG Membership Status <font color="red">*</font>:
Stephanie Judd : ACG Member
Suhag Patel : ACG Non-Member
Fadi Antaki : ACG Member
(No Image Selected)
(no table selected)
AVERAGE SCORE: 3.5
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Haritha Avula: [No Comments]
James Buxbaum: [No Comments]
Raquel Davila: [No Comments]
Brian Weston: [No Comments]
Purpose: A 70-year-old male presents with a week-long course of severe non-bloody watery diarrhea and is discovered on admission to also have a large poorly-healing lower extremity ulceration (Figure 1) for which he has had chronic wound debridement for the past 6 months. He denies any diarrhea prior to a week ago, or constitutional symptoms. His last colonoscopy was within the past year which was normal. His leg wound initially presented six months ago as a red bump on his right lower leg which rapidly enlarged and ulcerated over the course of one month. Initially this wound was presumed to be infectious but failed to respond to antibiotics. Vascular studies were normal. Over the next five months multiple biopsies were obtained but were nonspecific and the differential of pyoderma gangrenosum was raised. On presentation the patient was afebrile and hemodynamically stable. Laboratory evaluation was significant for ESR elevated at 75. A comprehensive stool evaluation for C. diff, enteric pathogens, and parasites were negative. CT enterography showed some subtle narrowing of the terminal ileum suggestive of inflammation. Colonoscopy revealed granularity in the transverse colon and by pathology revealed mild active chronic colitis in random biopsies throughout the colon and terminal ileum. The patient was diagnosed with inflammatory bowel disease and started on mesalamine. Within one week his diarrhea had resolved and at four months followup his leg ulceration was healing (Figure 2). He continues on mesalamine. Pyoderma gangrenosum is a rare inflammatory ulceration usually of the lower extremities affecting 3-10 individuals per one million people annually. It is an uncommon manifestation of systemic inflammatory disorders and as in our case may be the rare primary manifestation of underlying inflammatory bowel disease.

Methods: N/A
Results: N/A
Conclusion: N/A

AUTH DESIGN: ACG Membership Status <font color="red">*</font>: Daniel Chan : ACG Non-Member
Christopher Aakre : ACG Non-Member
Rebecca Adair : ACG Non-Member
Jason Post : ACG Non-Member
Siva Ketha : ACG Non-Member

Figure 1: Leg Wound at Time of Presentation
Figure 2: Leg Wound at 4-Months of Treatment

IMAGE CAPTION: Figure 1: Leg Wound at Time of Presentation Figure 2: Leg Wound at 4-Months of Treatment

AVERAGE SCORE: 4.5

REVIEWER FLAGS: (none)

REVIEWER RECOMMENDATION CODE DESCRIPTION: None

REVIEWER COMMENTS:
Unusual Case of Febrile Pleuropericarditis – A Potentially Life-threatening Side Effect of Balsalazide

ABSTRACT BODY:

Purpose: Ulcerative colitis (UC) is an idiopathic chronic inflammatory disorder that involves the rectum and the colon. The treatment typically involves inducing remission followed by the administration of maintenance medications to prevent relapse. One class among the drugs used for both remission and maintenance is the 5-aminosalicylates (5-ASAs). We present a rare case of febrile pleuropericarditis, a potentially life-threatening side effect of 5-ASA derivatives, which resolved after cessation of balsalazide. A 36-year-old Caucasian male with UC presented with two-week history of fever (as high as 102°F), fatigue, shortness of breath and worsening right-sided pleuritic chest pain. His most recent UC flare had been 6-8 weeks prior to admission, treated with a prednisone taper and maintained on balsalazide. Upon admission, CT chest revealed right pleural effusion with possible underlying infiltrate. The initial diagnosis was community acquired pneumonia and the patient was started on levofloxacin. The patient continued to complain of chest pain and developed daily fevers as high as 103.2°F despite broadened antimicrobial coverage. An extensive work up, including thoracentesis and pericardiocentesis, was initiated to evaluate for infectious, autoimmune/inflammatory and malignant conditions. All cultures, cytology, and laboratory results were negative. For his symptomatic pleuropericarditis, the patient was started on indomethacin which did not improve his symptoms. At this point, it was believed that his symptoms were secondary to balsalazide use and this was discontinued. He rapidly and completely defervesced, and his symptoms significantly improved. He was re-challenged later on with balsalazide which resulted in reoccurrence of fever and pleuritic chest pain within several days. Pericarditis and pleuropericarditis have been reported as rare, but potentially life-threatening side effects of 5-ASA therapy. As a 5-ASA prodrug, balsalazide might be expected to share the same class effect, but to date has only been reported in association with afebrile pericarditis in one case report. We believe we have identified a very rare case of febrile pleuropericarditis associated with balsalazide therapy. Our patient had partial and temporary relief of chest pain and dyspnea with fluid removal from the chest and pericardium, and only minimal response to indomethacin. His symptoms and high fevers did not resolve until cessation of balsalazide. In conclusion, this case illustrates a rare side effect of balsalazide, easily overlooked, which any physician should consider in his/her differential diagnosis on a patient with IBD on 5-ASA derivatives.

Methods: NA
Results: NA
Conclusion: NA

CURRENT CATEGORY: G. Clinical Vignettes/Case Reports
CURRENT SUB-CATEGORY: I. Inflammatory Bowel Disease
PRESENTATION TYPE: Poster Only
ACG Research Grant Support: No
Supported by Industry Grant: No
Commercial Products or Services: No
Initiated Research: Investigator
Financial Relationships: Not Applicable
FDA Approval: No
Designed Study: Investigator
Abstract Author: Investigator

AUTH DESIGN: ACG Membership Status <font color="red">*</font>:
Roxana Coman : ACG Non-Member
Altin Gjymishka : ACG Non-Member
Sarah Glover : ACG Member
(No Image Selected)
AVERAGE SCORE: 2.75
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Haritha Avula: [No Comments] 
James Buxbaum: [No Comments] 
Raquel Davila: [No Comments] 
Brian Weston: [No Comments]
Purpose: Fever of unknown origin is a common presentation to the Emergency Department and often results in an admission. The differential can be extensive, and often the corresponding signs and symptoms influence our path in the clinical course. Case: A 59-year-old man presented with one week of fevers and diffuse abdominal pain. He denied diarrhea or hematochezia. Medical history included hypertension, hyperlipidemia, and coronary artery disease. Home medications are aspirin and metoprolol. Physical exam was unremarkable. The differential diagnosis included Crohn’s colitis, infection, vasculitis and lymphoma. Significant laboratory values were ALT 57, AST 82, alkaline phosphatase 53, bilirubin 89, white blood cell count 8.8, eosinophilia of 21%, lactate dehydrogenase (LDH) 336. Computed tomography (CT) of abdomen and pelvis showed cystic liver lesions, inflammatory changes in the distal small bowel, cecum and ascending colon. He received ciprofloxacin and metronidazole. Stool cultures for Salmonella, Shigella, Campylobacter, EHEC, Giardia, cryptosporidium, ova and parasites returned negative. Serological tests for parasites, Ehrlichia, Saccharomyces cerevisiae and strongyloides, ANCA, ANA and ASCA antibodies and blood cultures were negative. PPD was non-reactive. Colonoscopy revealed granulomatous inflammation. An inguinal lymph node and bone marrow biopsies were unremarkable. He was discharged on prednisone after empirically being treated with Ivermectin, and scheduled for follow up to repeat colonoscopy and serologic testing for CD. A four week follow up revealed that the patient was fever and eosinophilia free. Discussion: This case represents an atypical presentation of Crohn’s disease, mimicking clinical features seen in infectious and lymphomatous conditions. Hepatic cysts on abdominal CT, eosinophilia and fever are concerning for parasitic infection. Hodgkin and aggressive B-cell Non-Hodgkin lymphoma may present with eosinophilia, elevated LDH and systemic B symptoms. Suspicion for Crohn’s disease remained high in spite of the atypical presentation. While a mucosal biopsy is often sufficient, an operative specimen may be required when the diagnosis of Crohn’s is equivocal. Our patient declined surgery. A high suspicion that involvement of the deeper muscularis layer characterized this patient’s Crohn’s disease. Conclusion: Our case illustrates that Crohn’s can present without classic history and may overlap with other disease processes. It is important to rule out infection and malignancy before coming to a diagnosis and proceeding with treatment in these particular patients. Mucosal biopsies may not be sufficient in some cases and full thickness specimens may be required for diagnosis.
AVERAGE SCORE: 4.5
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Purpose: Introduction: Infliximab was approved for the treatment of ulcerative colitis in the fall of 2005. Common side effects of anti-tumor necrosis factor therapy include fevers, rash, anaphylactoid reactions, as well as hematological manifestations. We report a case of an infliximab allergic reaction causing compressive optic neuropathy. Case report: A 41-year-old female with diagnosed with ulcerative colitis 21 years ago, hypothyroidism, and Grave’s disease was started on infliximab for ulcerative colitis therapy. She received three injections of infliximab with successful induction of remission. During the fourth infusion she developed right hand and forearm edema, both known reactions to infusion, with no further complications. Prior to the sixth infusion, the patient had sutures removed from her right upper eyelid from a recent right upper eyelid lengthening procedure due to exophthalmos. She had followed ophthalmology for Grave’s disease optic neuropathy in the past. Fifteen minutes into the sixth infusion, she was noted to have worsening of right optic neuropathy with associated facial edema. The infusion was stopped and the patient went to the ER, where a CT scan of the orbits showed interval increase in edema of extraocular muscles causing compression of the optic nerve. The reaction was deemed to be secondary to infliximab and she was started on steroids with successful resolution of symptoms. However, due to concern of worsening ulcerative colitis and after discussion with her ophthalmologist, infliximab was attempted again four months after the reaction. She was pre-medicated with prednisone a day prior to the infusion as well as hydrocortisone and diphenhydramine on the day of infusion. During this infusion of infliximab, the patient again had a similar reaction and therapy was halted. Discussion: Infliximab has a well-known side effect profile, which includes facial/hand edema, rashes, and even fevers as common reactions. However, to our knowledge this is the first report associating infliximab with allergic compressive optic neuropathy. This current vignette highlights the importance of recognizing compressive optic neuropathy as a consequence of a delayed allergic reaction to the infliximab infusion.

Methods: N/A

Results: N/A

Conclusion: N/A
Purpose: Isotretinoin is a medication used for the treatment of acne. Inflammatory bowel disease (IBD), is a common condition affecting young adults between the ages of 15 and 30 years of age. A multitude of case reports and litigation dating back over two decades dealing with isotretinoin have occurred and calls into question its relationship to inflammatory bowel disease. We present a case of a female patient who developed ulcerative colitis with cyclical flares after taking isotretinoin. A 35-year-old female presented with recent onset of bloody diarrhea, abdominal pain, cramping and tenesmus of 3 weeks duration. The patient states she has had flares before but not in about 2 years. She states these symptoms began 6 years prior and she has had flares every 2-3 years in a similar fashion. She admits to a 12-pound weight loss and severe fatigue. She denies taking any medications including chronic NSAIDs, and has no family history of IBD. On further questioning the patient admits to taking isotretinoin 6 years prior and notes her initial symptoms started approximately 3-6 months after completing the course of the anti-acne medication. She had a colonoscopy with biopsies consistent with left sided ulcerative colitis and was started on steroids. The patient symptoms improved dramatically. She began to gain weight, and her bowel functions returned to baseline; she is currently doing well and has not had another flare. Patients with abdominal complaints and having a history of isotretinoin exposure should raise IBD on the list of potential differentials. One mechanism for the role of isotretinoin is through retinoic acid effects in lymphocyte migration and immunomodulation in the gut. Retinoic acid may bring about the expression of α4β7 and CCR9, which attracts T cells; whilst also attracting activated B cells to the gut mucosa. Existing literature is conflicting regarding an association between isotretinoin and IBD; however we feel that patients should be informed of the risk of developing IBD. Patients should be advised to stop the medication if abdominal symptoms occur. We now include regularly the history of acne and its treatment in all patients with IBD.

Methods: na
Results: na
Conclusion: na
Isotretinoin and risk for inflammatory bowel disease: a nested case-control study and meta-analysis of published and unpublished data.

Etminan M, Bird ST, Delaney JA, Bressler B, Brophy JM.

The results of this study do not suggest an increase in the risk for IBD, including UC or CD, with use of isotretinoin.
Purpose: Crohn’s disease affects patients and with a spectrum of symptoms they individually classify as their “flare”. Classically, a Crohn’s disease flare presents with abdominal, bloody diarrhea, mild tenesmus and symptoms of malabsorption such as steatorrhoea or vitamin deficiencies. We present a case of a male patient who presented with profound refractory hypomagnesaemia as a sign of his Crohn’s flare. A 71-year-old male presented with weakness, dizziness, lightheadedness, and an unsteady gait of 1 week duration. He also admitted to difficulties in concentrating and remembering things recently. The patient stated it had become so difficult to walk that he fell and sustained injuries 3 days prior to presentation. The patient denies any fever, chills, chest pain or palpitations but did admit to non-bloody diarrhea and generalized abdominal pain that began on the day of admission. The patient stated he had never felt these symptoms before and were unlike his previous presentations. His history was significant for Crohn’s disease, hypertension, hyperlipidemia and CABG 10 years prior. On physical exam his abdomen was soft and notable for only mild epigastric tenderness. A working diagnosis of ischemia or neurologic insult was entertained but the EKG was significant for sinus tachycardia without ST changes and troponins were negative. A CT of the head demonstrated no intracranial mass or hemorrhage. However a CT scan of the abdomen reveal thickening of the distal and terminal ileum suggestive of Crohn’s disease. Additionally his labs demonstrated profound hypomagnesaemia of 0.4 mg/dl and hypocalcemia of 6.0 mg/dl with a normal albumin requiring a monitored setting. The patient also had a normal WBC count without left shift. The patient was not on any medications that would cause low magnesium, denied alcohol use, and had no evidence of renal disease. These low values persisted despite aggressive repletion through both IV and oral means. However through supportive care his flare subsided and the values of both electrolytes improved dramatically. The patient is currently doing well and has had no further bouts of Crohn’s disease. His final magnesium level was 2.0 mg/dl on discharge. Hypomagnesaemia may be an early sign of Crohn’s disease, as presented in this case; symptoms may precede the onset of a flare. The majority of magnesium absorption is in the small bowel; specifically in the distal ileum. Inflammation in this region prevents the transcellular transporter receptor channel melastatin TRPM 6 and 7 from absorbing magnesium. We recommend a prospective analysis of magnesium levels in patients with Crohn’s flare to further correlate these findings.
Purpose: A 59-year-old woman with Crohn’s ileocolitis complicated by small bowel obstruction from stricture status post ileocecectomy with postoperative recurrence who received three induction doses of infliximab after intolerance to steroids and mesalamine presented with multiple systemic complaints for the past 3 months. These include hair loss, headaches, cough, pleuritic chest pain, exertional dyspnea, orthopnea, myalgias, arthralgias, morning stiffness, proximal muscle stiffness, fatigue and weight gain of 20 pounds in addition to baseline non bloody diarrhea. The patient had stopped all medications due to weight gain after her last dose of Infliximab, however her symptoms persisted. She denied fever, chills or rashes. On exam, she was hemodynamically stable with no visible rashes. Her musculoskeletal exam was notable for mild right wrist synovitis, bilateral proximal muscle weakness as well as tenderness in all limb joints. Her labs were notable for weakly positive antihistone antibodies, positive ANA of 1:160 and positive dsDNA with an otherwise negative rheumatologic and endocrine panel – suggestive of Infliximab induced lupus. The patient refused to begin a trial of steroids and preferred to tolerate her symptoms without medications. TNF-alpha inhibitors represent an important modality of targeted immunotherapy in treating auto inflammatory illnesses. However, induction of autoantibodies such as anti-ANA and anti-dsDNA as well as the rare incidence of autoimmune disease such as systemic lupus erythematosus (SLE), psoriasis and vasculitis have been documented as side effects. The development of a lupus-like syndrome secondary to Infliximab differs from typical drug induced lupus by the presence of dsDNA and lower incidence of anti-histone antibodies. This patient met the revised ACR criteria for drug-induced lupus; however failure of these symptoms to resolve on discontinuation of Infliximab is unusual. Although the patient’s refusal to take steroids to treat her lupus complicates the diagnosis, this prompts the possibility that she may have had a developing SLE that evolved into complete lupus once triggered by Infliximab. Consequently, studies are needed to justify screening for autoimmune antibodies and disease before beginning treatment with TNF alpha inhibitors so that treatment plans can be modified accordingly. References: 1. Almoallim H, et al. Anti-tumor necrosis factor-α induced systemic lupus erythematosus. Open Rheumatol J. 2012;6:315-9. 2. Ramos-Casals M, et al. Autoimmune diseases induced by TNF-targeted therapies: Analysis of 233 cases. Medicine (Baltimore). 2007 Jul;86(4):242-51. 3. Williams EL, et al. Anti-TNF-induced lupus. Rheumatology (Oxford). 2009;48(7):716-20.

Methods: N/A
Results: N/A
Conclusion: N/A

For more information, please visit the provided references.
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Haritha Avula: [No Comments]
James Buxbaum: [No Comments]
Raquel Davila: [No Comments]
Brian Weston: [No Comments]
**Title:** Streptococcus acidominimus and Portomesenteric Venous Gas: A Rare Presentation of Crohn's Disease

**Presenter:** Ryan McCaffrey

**Presenter (Institution Only):** New York Hospital Queens

**Presenter (Country Only):** United States

**Abstract Body:**

**Purpose:**
Background: *Streptococcus acidominimus* is a viridans streptococci that rarely causes infections in humans; review of the literature only lists 10 reported cases. We report an unusually severe initial presentation of Crohn’s disease involving superimposed infection of this species, causing severe ileitis, portomesenteric gas, venous thrombosis, and septic emboli to the liver. Case description: A 39-year-old male with no PMH presented complaining of nausea, watery diarrhea with mucus, and mild abdominal cramping. On physical exam the patient had a high grade fever with mild epigastric tenderness but an otherwise unremarkable abdominal exam. A CT abdomen demonstrated severe inflammation of a loop of the distal ileum with thrombosis and gas visualized in the portal venous system and branches of the SMV, as well as multiple sub-centimeter liver abscesses. Labs revealed mild leukocytosis, transaminitis and direct hyperbilirubinemia. GGT, lipase, and lactic acid were normal. All blood culture bottles grew *Streptococcus acidominimus* sensitive to all beta-lactam antibiotics. An antibiotic course with ampicillin-sulbactam was given with adequate clinical response. Further workup revealed a high ASCA titer, negative p-ANCA, and protein C deficiency. A colonoscopy was performed and biopsy of the terminal ileum showed chronic inflammation. With improving clinical condition, the patient was initiated on mesalamine therapy for Crohn’s and anticoagulated with coumadin. Discussion: *Streptococcus acidominimus* is frequently described in the veterinary literature. In the few cases reported in humans, the bacterium has been associated with severe infections with abscess formation. Brain abscesses, endocarditis, acute petrositis, and multiloculated empyema have been described. Besides severe ileitis and liver abscesses, our patient presented with portomesenteric vein gas which is typically associated with mesenteric ischemia. Such findings have only rarely been attributed to Crohn’s disease, presumably caused by ulceration and/or sloughing of the epithelial lining of the mucosa, allowing air to enter the venous system. As it is not typical for Crohn’s disease to present initially with such severe findings, it is likely that superimposed infection with *S. acidominimus* played a key role in the disease course. Presumably, an insidious and antecedently subclinical chronic inflammatory process weakened the mucosa in our patient allowing invasion of this normally benign bacteria, which led to cascading inflammation and eventually portomesenteric vein gas, thrombosis, bacteremia, and septic emboli in the liver. This hypothesis is substantiated by the patient’s quick clinical response to antibiotic therapy alone.

**Methods:** N/A

**Results:** N/A

**Conclusion:** N/A

**Current Category:** G. Clinical Vignettes/Case Reports

**Current Sub-Category:** I. Inflammatory Bowel Disease

**Presentation Type:** Poster Only

**ACG Research Grant Support:** No

**Supported by Industry Grant:** No

**Commercial Products or Services:** No

**Initiated Research:** Investigator

**Financial Relationships:** No

**FDA Approval:** No

**Designed Study:** Investigator

**Abstract Author:** Investigator

**AUTH DESIGN: ACG Membership Status $<font color="red">*</font>$:**

Ryan McCaffrey : ACG Non-Member
Jose NajulSeda : ACG Non-Member
Cristina Gutierrez : ACG Non-Member

(No Image Selected)

(no table selected)
AVERAGE SCORE: 4.25
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Sita Chokhavatia: [No Comments]
Waqar Qureshi: [No Comments]
Bo Shen: [No Comments]
Marc Zuckerman: [No Comments]
Purpose: Giant inflammatory polyposis (GIP) is a benign and rare sequela of ulcerative colitis or colonic Crohn's disease and can mimic colorectal carcinoma. Inflammatory polyps >15 mm in size are termed "giant". We present a rare case of GIP of the rectum in a patient with indeterminate colitis which was successfully treated with endoscopic polypectomy and adalimumab therapy. A 40-year-old white male with 10 year history of ulcerative proctitis presented with rectal bleeding, abdominal gas/bloating and diarrhea while on oral and topical mesalamine therapy. He had a history of recurrent calcium oxalate renal stones and was noted to be iron and B12 deficient. Colonoscopy showed a sessile polypoid growth about 3 cm in size and a cluster of polyps measuring 1.5 to 2 cm in size in the distal 12 cm of the rectum. Endoscopic appearance was highly suspicious for malignancy. Mucosal and snare biopsies, however, were consistent with inflammatory polyps with mucin lakes and edematous stroma. There was herniation of non-dysplastic glandular epithelium/mucin into the stroma ("pseudoinvasion") and reactive bizarre stromal cells without any evidence of dysplasia or malignancy. The edema, hemosiderin and foreign body giant cells suggested torsion/twisting as the mechanism for the herniation of lobular arrangements of glands into the stroma. The remainder of the colon biopsies were unremarkable. Terminal ileal biopsies showed mild crypt distortion without any active inflammation. An esophagogastroduodenoscopy, small bowel follow-through and capsule endoscopy were negative. He was noted to be perinuclear antineutrophil cytoplasmic antibody negative and anti-Saccharomyces cerevisiae antibodies IgA and IgG positive. Patient was started on prednisone taper along with infliximab infusion therapy (5mg/kg body weight). He continued to be symptomatic and repeat colonoscopy 6 months later showed persistent severe proctitis with inflammatory polyposis without dysplasia. An endoscopic ultrasound showed hypoechoic, heterogeneous thickening of mucosa with intact muscularis propria in the involved part of the rectum. Piecemeal endoscopic snare polypectomy was performed to remove the majority of the polypoid tissue. Patient was switched to adalimumab 40 mg biweekly SQ therapy. On 2-year follow-up, he remains in clinical remission on adalimumab therapy with no evidence of recurrent polyposis on endoscopic evaluation. This case highlights the need for clinical, endoscopic and histopathological correlation to diagnose GIP. Recognition of this rare entity will prevent unnecessary radical surgical resection for presumed carcinoma. Adalimumab therapy in combination with endoscopic polypectomy appears promising in the treatment of GIP.

Methods: n/a
Results: n/a
Conclusion: n/a

AUTH DESIG: ACG Membership Status <font color="red">^</font>: Justin Hartke : ACG Member
Josh Wilson : ACG Member
Salma Akram : ACG Member
(No Image Selected)
AVERAGE SCORE: 5.25
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
Sita Chokhavatia: [No Comments]
Waqar Qureshi: [No Comments]
Bo Shen: [No Comments]
Marc Zuckerman: [No Comments]
Purpose: Background: Inflammatory bowel disease (IBD) is known risk factor for venous thromboembolism. Proximal small bowel involvement is an uncommon pattern of Crohn’s disease. We are reporting a case of unexplained recurrent venous thromboembolism, diagnosed with proximal Crohn’s disease. Case: A 39-year-old female presented with epigastric pain and indigestion. The patient is known to have an unexplained hypercoagulable state with recurrent DVTs, failed coumadin treatment, and on chronic Enoxaparin injections with Greenfield filter placement for more than one year. Upper endoscopy showed multiple distal duodenal aphthous ulcers. Biopsy showed duodenitis with no granuloma and a negative H. pylori staining. Gastrin level ordered and was normal. Suspicion of Crohn’s disease led to colonoscopy which showed normal colon with scattered ileal aphthous ulcers. The endoscopic features supported diagnosis of Crohn’s disease and the patient was started on prednisone and mesalamine with good response. Video capsule endoscopy was done later and showed multiple aphthous deep and irregular ulcers from distal duodenum to ileum, mainly in the jejunum with multiple strictures. Discussion: Crohn’s disease is a well-known risk factor for venous thromboembolism. Previous studies have shown that increased risk of venous thromboembolism in inflammatory bowel disease is multifactorial, which may include endothelial dysfunction, increased platelet aggregation, hyperhomocysteinemia, antiphospholipid antibodies, and can involve all components of the clotting system. Our patient was diagnosed with unexplained resistant hypercoagulable state with recurrent DVTs. The patient was found to have extensive proximal Crohn’s disease from distal duodenum to the terminal ileum. This could be the underlying cause of her unexplained hypercoagulable state. She has proximal Crohn’s which is uncommon and could be related to the severity of her hypercoagulable state. Conclusion: Crohn’s disease and IBD in general should be investigated in any case of unexplained hypercoagulable state. There could be an association between the pattern of Crohn’s disease and the severity of hypercoagulability.

Methods: N/A
Results: N/A
Conclusion: N/A
REVIEWER COMMENTS:
Sita Chokhavatia: [No Comments]
Waqar Qureshi: [No Comments]
Bo Shen: [No Comments]
Marc Zuckerman: [No Comments]
Purpose: Introduction: Sweet’s syndrome (SS) is also known as acute febrile neutrophilic dermatosis and is characterized by constellation of features that include tender erythematous skin lesions (papules, nodules, and plaques), fever, neutrophilia and pathologic evidence of dense neutrophilic infiltrate without leukocytoclastic vasculitis. SS is divided into idiopathic (or classic), malignancy-associated and drug-induced sub-types based on etiology. Classical SS constitutes majority of cases and is associated with pregnancy, infections and inflammatory bowel disease (IBD). SS is most commonly associated with IBD but incidence is very rare and less than forty cases are reported so far in literature. SS associated with IBD tends to occur more commonly in females unlike other skin manifestations which have no sex predilection. It is more commonly associated with Crohn's disease (CD) than ulcerative colitis (UC). Case description: We are presenting a case of a 51-year-old male who came to our hospital with complaints of hematochezia, painful, nodular, erythematous skin rash which started on arms and later involved lower extremities. He also had asymmetric oligoarthritis (right wrist, left wrist and elbow), recurrent mouth ulcers and painless red eye. He had low grade fever and on labs was found to have leucocytosis, elevated ESR and CRP. Autoimmune and infectious work up was negative. He underwent colonoscopy which showed shallow ulcers throughout colon but more in right colon with involvement of ileocecal valve. Ophthalmology was consulted for red eye and they diagnosed it as episcleritis. Pathology from the skin biopsy revealed granulomatous and nodular neutrophil dermatitis extending to subcutis without leukocytoclastic vasculitis suggestive of SS. Subsequent colon biopsy revealed chronic active colitis with granulation tissue suggestive of likely CD. He responded well to prednisone therapy with resolution of episcleritis and skin rash in few weeks. Conclusion: We conclude that SS is an uncommon phenomenon that is associated with IBD and must be considered as differential diagnosis in patients presenting with skin lesions. SS mimics other skin manifestations like Pyoderma gangrenosum and Erythema nodosum (EN) which are also associated with IBD. In our case EN was considered as differential but it usually involves legs in asymmetrical distribution and biopsy should have shown panniculitis without inflammatory infiltrates. Usually SS presents after the diagnosis of IBD or along with it but rarely precedes it. Therefore any patient presenting with SS should be worked up for underlying IBD. Treatment with steroids obtains rapid clinical response with healing of dermal lesions without leaving any sequelae.

Methods: N/A
Results: N/A
Conclusion: N/A
Radhika Kothakota : ACG Non-Member
Warren Piette : ACG Non-Member
Senseng Carmencita : ACG Non-Member
Juan MunozPena : ACG Non-Member
Dinesh Gaddam : ACG Non-Member
(No Image Selected)
(no table selected)
AVERAGE SCORE: 4.75
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS:
ABSTRACT BODY:
Purpose: A 62-year-old man with history of ulcerative colitis presented with scrotal pain and swelling. Patient denied pneumaturia, abdominal pain, diarrhea, fever/chills, or overt GI bleeding. Past medical history was significant for type 2 diabetes and ulcerative colitis. He reported multiple perineal fistulas with recent Fournier gangrene requiring serial debridements over the last 2 months. He was afebrile with stable vitals. Abdominal exam was notable for suprapubic tenderness, multiple open ulcers and discharging fistulas in the perineum. Several scars from prior fistulas were noted. MRI showed horse-shoe shaped perirectal abscess and multiple perineal fistulas. Colonoscopy showed anal stenosis and a fistulous opening in the rectum. Remaining colon appeared normal. Rectal biopsies showed mildly increased plasma cells and eosinophils within lamina propria with mild architectural distortion, and patchy mild acute inflammation without granulomas. Based on clinical presentation and findings, patient was diagnosed with fistulizing Crohn’s perianal disease with rectal involvement. He underwent surgical debridement with suprapubic catheter placement and was started on metronidazole, ciprofloxacin and infliximab with good response. Discussion: Perianal Crohn’s disease affects 25-40% of all Crohn’s patients and includes anal fissures, perianal fistulas, anorectal abscesses, and anal stenosis. Perianal fistula affects 20-30% of all Crohn’s patients. Diagnostic tests include: exam under anesthesia with probing, fistulography, barium studies, CT, pelvic MRI, and anorectal EUS. Oral metronidazole for 6-12 months followed by gradual taper can be used as initial therapy for mild to moderate disease. For severe or refractory disease, anti-TNFs agents such as infliximab or adalimumab, have been shown to reduce the number of draining fistulas and even achieve complete closure. Response is rapid with median time to response of 2 weeks. Immumodulators such as 6-MP or azathioprine have also been shown to achieve complete healing or decrease discharge but response is much slower. Only 3 cases of IBD complicated with Fournier’s gangrene have been reported in the literature. Of these 2 had undiagnosed Crohn’s disease and one had ulcerative pancolitis who developed Fournier’s gangrene after perianal surgery. Management involves serial debridements, broad spectrum antibiotics, anti-inflammatory therapy for IBD, and diversion colostomy in severe cases.
REVIEWER COMMENTS:
Sita Chokhavatia: [No Comments]
Waqar Qureshi: [No Comments]
Bo Shen: [No Comments]
Marc Zuckerman: [No Comments]
Purpose: Introduction: Thromboembolism is an uncommon but a known phenomenon in both types of inflammatory bowel disease (IBD). Thrombosis usually involves the peripheral veins, less commonly the pulmonary and cerebral veins. Arterial thrombosis and thrombosis at multiple sites is extremely rare. We describe a case of ulcerative colitis with recurrent arterial and venous thrombosis. Case report: A previously healthy 47-year-old male presented initially with pain and numbness in the right leg and a few days of bloody diarrhea. Duplex imaging revealed thrombotic occlusion of the right common femoral and popliteal arteries for which he was treated with thromboembolectomy. Colonoscopy was suggestive of ulcerative colitis involving the rectum and descending colon and histopathology revealed chronic active colitis. He was discharged on warfarin and sulfasalazine, however he was poorly compliant to warfarin due to intermittent rectal bleeding. He presented a year later with gangrene from complete occlusion of the left superficial femoral artery. He underwent left below-knee amputation. Long term anticoagulation was initiated with warfarin. Almost another year later, he was admitted for a flare of ulcerative colitis. He was started on prednisone and azathioprine. Venous duplex done for a swollen right lower limb revealed extensive deep vein thrombosis involving the femoral, popliteal, posterior tibial and peroneal veins. Enoxaparin was started and warfarin was continued. Discussion: In addition to microvascular thrombotic activity in the intestinal mucosa, the occurrence of systemic thromboembolism is a recognized extra-intestinal manifestation in IBD. Lifetime incidence of thromboembolic episodes in IBD varies from 1.2% to 6% in various studies. The development of thrombosis is known to parallel disease activity in Crohn's disease but not in ulcerative colitis. Majority of these patients do not have any known underlying thrombophilic state. Levels of platelets, factor V, factor VIII, fibrinogen and fibrinopeptide have been demonstrated to be elevated during disease flares of IBD, though the cause-effect relationship remains unclear. It still needs to be determined whether hypercoagulability is a secondary phenomenon in IBD from the chronic inflammation or an underlying factor that contributes to disease pathogenesis. A few case series and reports have shown benefits of both heparin and warfarin. The optimal anticoagulation strategy in patients with IBD and whether more aggressive treatment of the underlying inflammation would play a role in secondary prevention of thrombosis remains unclear.

Methods: N/A
Results: N/A
Conclusion: N/A

AUTH DESIGN: ACG Membership Status <font color="red">*</font>:
Karthik Gnanapandithan : ACG Non-Member
Ramprakash Devadoss : ACG Non-Member
Curuchi Anand : ACG Member
(No Image Selected)
(no table selected)
AVERAGE SCORE: 4.5
REVIEWER FLAGS: (none)
REVIEWER RECOMMENDATION CODE DESCRIPTION: None
REVIEWER COMMENTS: