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When the Spleen Speaks First: Aseptic Abscess as the Herald of Crohn's Disease

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Abstract

Background: Crohn's disease is a chronic inflammatory bowel disease (IBD) that may present with a range of extraintestinal manifestations, including cutaneous, hematologic, and hepatosplenic involvement. Aseptic splenic abscesses and pyoderma gangrenosum are rare initial presentations.

Case presentation: We present the case of a 31-year-old woman with chronic abdominal pain, fever, joint swelling, skin ulcerations, and melena. Extensive workup revealed aseptic splenic abscesses, colonic inflammation consistent with Crohn's disease, and pyoderma gangrenosum. Infectious and malignant causes were ruled out. She responded to corticosteroids and immunosuppressive therapy with Azathioprine.

Conclusions: This case highlights the need for high clinical suspicion for Crohn's disease in patients with unexplained systemic inflammation and splenic lesions. Aseptic splenic abscesses and pyoderma gangrenosum may be rare but significant extraintestinal features that warrant early recognition and immunosuppressive therapy.

Keywords: HCV Serologies, Hepatosplenic, Inflammatory Bowel Disease

Background

Crohn's disease can affect any part of the gastrointestinal tract and often presents with abdominal pain, diarrohea, and weight loss. Extraintestinal manifestations, including dermatologic and hepatosplenic findings, can precede gastrointestinal symptoms. Aseptic splenic abscesses are rare and

typically seen in systemic inflammatory disorders. Pyoderma gangrenosum, a neutrophilic dermatosis, is also associated with IBD but is rarely an initial sign. Early diagnosis and immunosuppression are crucial for avoiding unnecessary surgical interventions.

Case presentation

A 31-year-old lady, presented with an eight month history of dull, crampy abdominal pain localized to the peri-umbilical region. The pain was non-radiating, continuous in nature, worsened after food intake, and partially relieved with medications. Over the subsequent six months, she developed intermittent low-grade fever without chills or rash. Four months prior to presentation, she began experiencing joint pain predominantly involving the large joints, including the knees and elbows, later extending to the ankles. This was accompanied by joint swelling and erythema, without a clear relieving factor.

Approximately two months before presentation, the patient developed multiple wounds on her hands and feet. These started insidiously with pustular lesions on the right foot and subsequently involved the left foot and both forearms. The pustules spontaneously ruptured, discharging pus and resulting in ulcerative lesions. Concurrently, she had five episodes of melena, prompting blood transfusions multiple times in the past year. She also reported a weight loss of approximately 10 kilograms over a period of four months. There was no history of vomiting or diarrohea. Menstrual history was notable for amenorrhea lasting the past four months. She had no known comorbidities, and past surgical history included a lower segment cesarean section four years ago and appendicectomy two years ago.

On examination, the patient appeared moderately built and nourished, alert and oriented, with evident pallor, bilateral pedal edema, angular cheilitis, and multiple oral aphthous ulcers. A tender, erythematous nodule was noted on the right leg, and multiple punched-out ulcers were observed on the lateral aspects of both feet and forearms. Vitals were stable, though she was noted to be tachycardic. Abdominal examination revealed diffuse tenderness and significant splenomegaly.

Initial laboratory investigations revealed normocytic normochromic anemia with haemoglobin as low as 5.2 g/dL, persistent leukocytosis , elevated CRP , and ESR . Liver function tests showed hypoalbuminemia (3.2 g/dL), a markedly elevated alkaline phosphatase (315 U/L), and mildly elevated total bilirubin. Prothrombin time was prolonged (19 seconds) with an INR of 1.74. Stool occult blood was positive. ANA, HIV, HBsAg, and HCV serologies were negative. Cultures of blood and urine showed no growth.

Ultrasonography of the abdomen revealed splenomegaly with multiple hypoechoic lesions suggestive of abscesses, along with hepatomegaly, cholelithiasis, and portocaval and peripancreatic collaterals. CECT of the abdomen confirmed splenic abscesses with splenic vein thrombosis, thickening of the terminal ileum and ileocecal valve with lymphadenopathy, and mild ascites. Upper gastrointestinal endoscopy revealed a punched-out ulcer in the upper esophagus; biopsy demonstrated nonspecific esophagitis. Colonoscopy showed colonic inflammation, and biopsies from the transverse and sigmoid colon, as well as the ileocecal valve, revealed active colitis characterized by dense inflammatory infiltrates and lymphoid aggregates without granulomas or crypt distortion. Genexpert testing for tuberculosis was negative. A skin wedge biopsy from one of the ulcerated lesions on the limb revealed dense neutrophilic and lymphocytic infiltrate with signs of capillaritis,

consistent with pyoderma gangrenosum. Aspiration of the splenic abscess under ultrasound guidance yielded sterile fluid, and microbial workup including cultures and TB PCR was negative. An extensive infectious disease evaluation ruled out endocarditis, echinococcosis, bartonellosis, toxoplasmosis, babesiosis, and ehrlichiosis.

Despite broad-spectrum antibiotics including intravenous meropenem, the patient continued to have fever spikes and leukocytosis. Given the constellation of findings—non-caseating colitis, sterile splenic abscesses, and pyoderma gangrenosum—a diagnosis of Crohn's disease with extraintestinal manifestations was made. She was started on oral prednisolone 40 mg daily and azathioprine 50 mg daily, with folic acid, calcium-vitamin D supplementation, and nutritional support.

Following the initiation of immunosuppressive therapy, the patient showed marked clinical improvement. Fever resolved, abdominal pain subsided, melena did not recur, and skin lesions began to heal. At follow-up, she remained clinically stable with resolution of splenic lesions on imaging and was continued on azathioprine 100 mg daily and folic acid supplementation.

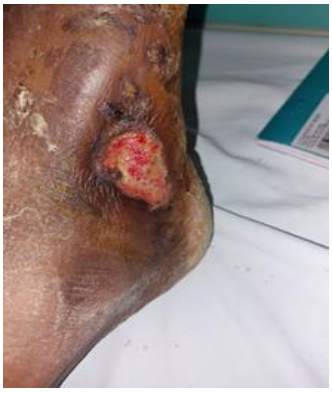

Figure 1: Punched out ulcer

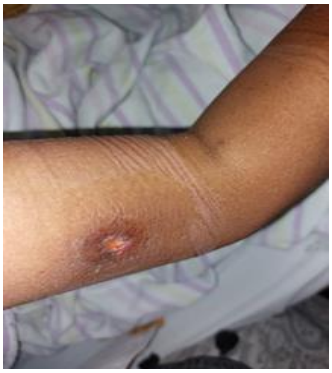

Figure 2: Punched out ulcer

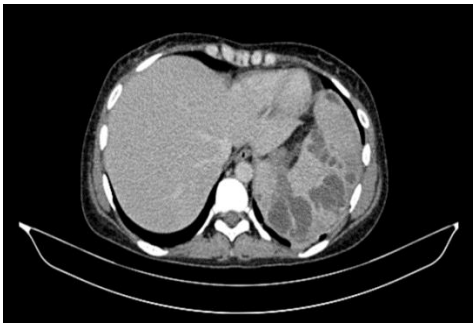

Figure 3

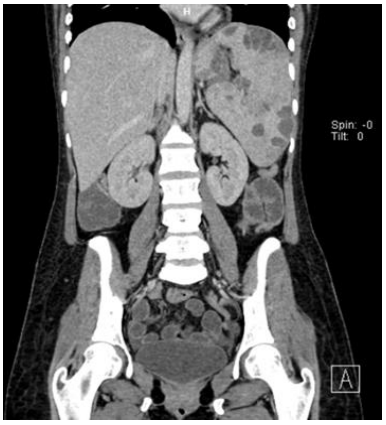

Figure 3, 4: Spleenic abscess

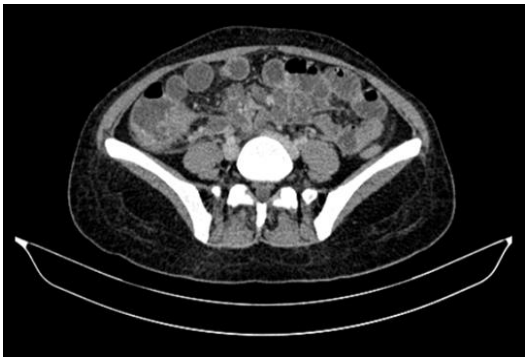

Figure 5: Ileocaecal junction thickening

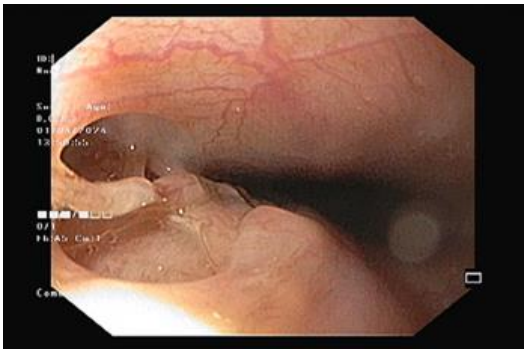

Figure 6: Esophageal ulcer

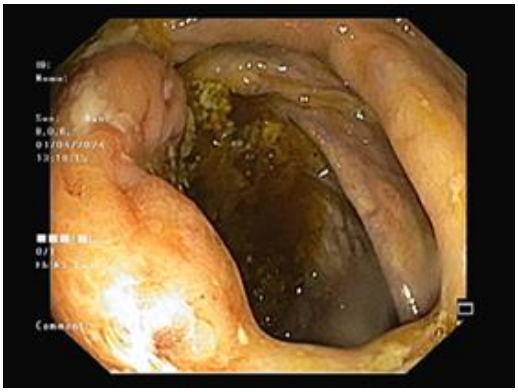

Figure 7

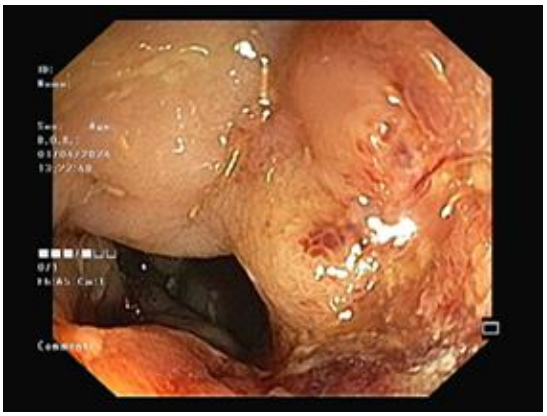

Figure: 7, 8: Platulous IC valce and erosions

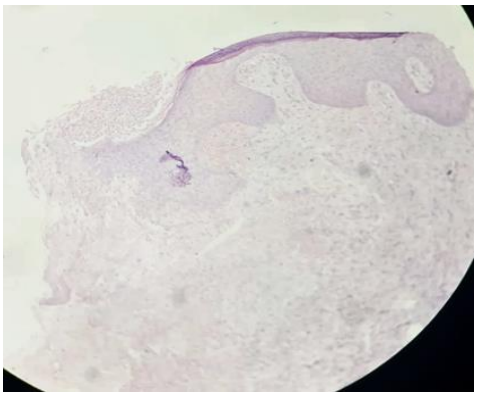

Figure 9

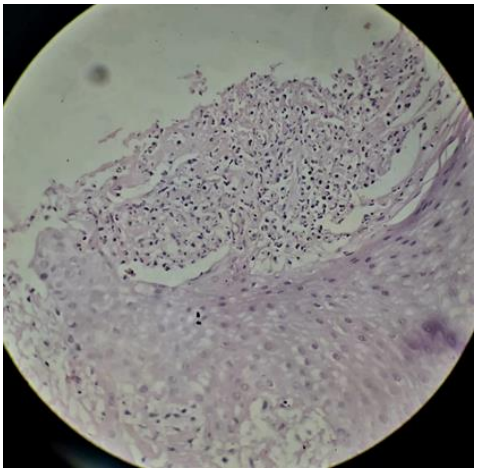

Figure: 9,10: Pyoderma gangrenosum

Discussion

Crohn's disease (CD), a subtype of inflammatory bowel disease (IBD), typically affects the gastrointestinal tract but can present with a wide range of extraintestinal manifestations (EIMs). These may involve the skin, joints, hepatobiliary system, and eyes. Approximately 25% of patients with CD will manifest EIMs during the course of their disease, and in rare instances, such features may precede gastrointestinal involvement, as in the present case.

Aseptic Splenic Abscesses

Aseptic abscesses (AAs) are sterile collections of neutrophils in visceral organs, most commonly the spleen and liver, associated with systemic inflammatory conditions like IBD, Behçet's disease, and neutrophilic dermatoses. Their pathogenesis is poorly understood but is thought to involve dysregulated neutrophil chemotaxis and cytokine activation. They are frequently misdiagnosed as bacterial abscesses, leading to inappropriate antibiotic use or even surgical intervention. The failure of antibiotics, lack of growth in cultures, and excellent response to corticosteroids in this patient favor an aseptic, immune-mediated etiology. Imaging often shows multiple hypoechoic or hypodense lesions, and aspiration reveals sterile pus. Notably,

splenic abscesses as an initial manifestation of CD are exceedingly rare, with only a handful of cases reported in the literature.

Pyoderma Gangrenosum

Pyoderma gangrenosum (PG) is a rare, ulcerative neutrophilic dermatosis associated with IBD, particularly ulcerative colitis, but also with CD. It typically begins as pustules or papules that ulcerate rapidly and exhibit pathergy. Histopathology is nonspecific but helps exclude other causes such as infections and vasculitis. In this case, the PG lesions on the patient's extremities were biopsy-proven and co-occurred with splenic abscesses and active colitis, suggesting a shared underlying inflammatory process. The rapid resolution of these lesions with corticosteroid therapy also supports an immune-mediated mechanism.

Diagnostic Challenges

The diagnosis of Crohn's disease in this patient was complicated by the lack of classic intestinal symptoms such as diarrhea. However, the presence of weight loss, melena, colonic ulceration, and biopsy findings of active colitis helped establish the diagnosis. The absence of granulomas on histopathology does not rule out Crohn's, as granulomas are found in only 30–50% of cases. Moreover, other conditions such as intestinal tuberculosis and infective endocarditis were appropriately ruled out with negative GeneXpert, sterile cultures, and normal echocardiography.

This case emphasizes the utility of a multidisciplinary approach involving gastroenterologists, dermatologists, radiologists, and infectious disease specialists in evaluating systemic inflammatory conditions.

Therapeutic Approach

Management of CD with EIMs involves immunosuppressive therapy, typically with corticosteroids, immunomodulators (e.g., azathioprine), or biologics (e.g., anti-TNF agents). This patient showed rapid improvement on corticosteroids and azathioprine, suggesting a steroid-responsive disease. Long-term immunosuppression may be required to prevent recurrence of both intestinal and extraintestinal manifestations.

Conclusion

This case illustrates an atypical and rare presentation of Crohn's disease where extraintestinal manifestations—specifically aseptic splenic abscesses and pyoderma gangrenosum—preceded gastrointestinal symptoms. Such presentations can be diagnostically challenging and are often mistaken for infectious or malignant processes, resulting in delays in appropriate therapy. The clinical response to corticosteroids and azathioprine reinforces the importance of recognizing these entities as part of the Crohn's disease spectrum. Early diagnosis and prompt initiation of immunosuppressive therapy are essential to mitigate morbidity and prevent unnecessary interventions. Clinicians should maintain a high index of suspicion for IBD in patients presenting with unexplained systemic inflammation and sterile visceral lesions.

Abbrevations

IBD, Inflammatory Bowel Disease; CD, Crohn's Disease; EIMs, Extraintestinal Manifestations; PG, Pyoderma Gangrenosum; AA, Aseptic Abscess; CECT, Contrast-Enhanced Computed Tomography; CRP, C-Reactive Protein; ESR, Erythrocyte Sedimentation Rate; ANA, Antinuclear Antibody; HIV, Human Immunodeficiency Virus; HBsAg, Hepatitis B Surface Antigen; HCV, Hepatitis C Virus; PCR, Polymerase Chain Reaction; TB, Tuberculosis; INR, International Normalized Ratio; TNF, Tumor Necrosis Factor.

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