

Unexpected etiology of massive lower gastrointestinal bleeding requiring blood transfusion: Crohn's disease, syphilis, or hematological malignancy?

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Dear Editor,

Crohn's disease (CD) is a chronic inflammatory bowel disease that may involve any segment of the gastrointestinal (GI) tract and typically presents with abdominal pain, diarrhea, weight loss, and occasionally hematochezia. CD is difficult to diagnose and can be confused with intestinal involvement in various infections, including tuberculosis.¹⁻⁴ Although mild GI bleeding is relatively common, massive lower GI bleeding requiring transfusion is rare⁵ and should prompt careful reconsideration of the initial diagnosis and evaluation for alternative or concomitant etiologies. Here, we present a patient initially diagnosed with CD who developed life-threatening lower GI bleeding, ultimately found to have infectious and hematological conditions contributing to the clinical picture.

A 60-year-old male presented to the emergency department with recurrent bloody defecation and lethargy. The patient appeared pale and was anxious. His medical history included hypertension. One week prior to admission, the patient had been evaluated for mucoid diarrhea and underwent colonoscopy, after which a diagnosis of CD was established and he was given azathioprine and prednisolone therapy by another gastroenterologist. Physical examination revealed normal cardiac and pulmonary functions. Cervical and axillary lymph nodes were palpated. Upon admission his blood pressure was 100/60 mmHg and his pulse was 86/minute. His initial laboratory evaluation revealed anemia and leukocytosis (hemoglobin level of 8.1 g/dl, hematocrit: 24.5%, WBC: 17.55 K/uL, monocyte count: 6,48 K/uL). Prothrombin time and C-reactive protein levels were within normal limits. Routine laboratory tests were within normal reference ranges except elevated urea (64 mg/dl) and albumin levels (3.2 g/dl). Because of lower GI bleeding, he was hospitalized, oral intake was stopped and transfused with two units of packed red blood cells. The following day, a colonoscopy was performed, which revealed deep ulcerations in the descending and transverse colon, accompanied by

two large adjacent clot formations (**Figure 1**). No active bleeding focus was identified at that time. Despite initial supportive management, the patient continued to pass bloody stools, necessitating transfusion of an additional four units of packed red blood cells. Because of this clinical picture, CT angiography of the abdomen was performed and it suggested an active bleeding source at the junction of the splenic flexure and transverse colon. A repeated colonoscopy again failed to localize an active bleeding site due to the presence of heavy blood contamination and blood clots. Immediately interventional radiology team was called to the hospital and two active bleeding vessels were successfully embolized by them, resulting in hemodynamic stabilization and cessation of overt GI bleeding. Given the patient's persistent monocytosis, which had been documented also prior to admission, low hemoglobin levels and the presence of generalized lymphadenopathy, and also he was consulted by the infectious diseases department. His serological testing revealed *Bartonella henselae* IgM positivity and a positive *Treponema pallidum* hemagglutination assay (TPHA). Azithromycin 250 mg P.O. once daily was given for 2 weeks for *Bartonella henselae* infection without organ involvement and to treat syphilis weekly 2.400 000 IU Penicillin G benzathine IM injections were initiated. Peripheral blood smear examination demonstrated dysplastic monocytes and approximately 10% circulating blasts. Subsequent bone marrow biopsy confirmed the diagnosis of acute myeloid leukemia (AML) with FLT3 mutation positivity, and the patient was referred to a bone marrow transplantation center for definitive management. In line with these clinical developments, the patient's initial pathology specimens were obtained and sent to an external center for examination by a pathologist experienced in the diagnosis of inflammatory bowel diseases. The pathologist's examination indicated that the pathology samples were not consistent with CD. The patient presented to our hospital with a bleeding disorder,

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so azathiopurin treatment was discontinued, and steroid treatment was also stopped by tapering due to the pathology preparations not being consistent with CD. Based on these developments, it was assessed that the lower GI bleeding was caused by syphilis or AML. After discontinuing the treatment for CD and using antibiotics for the infections, the patient's GI symptoms improved. A follow-up colonoscopy performed 4 months after the patient's emergency room visit, while still in remission from AML, was found to be completely normal (Figure 2).

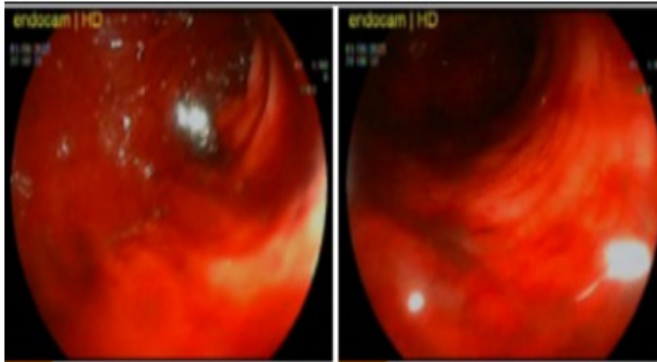


Figure 1. Endoscopic appearance of the colonic bleeding in descending and sigmoid colon

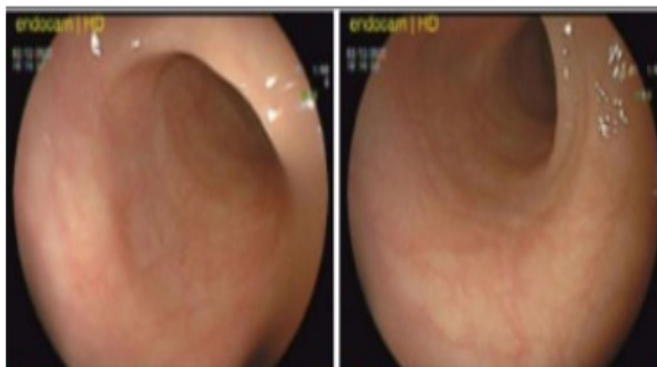


Figure 2. Follow-up colonoscopy four months later, showing complete mucosal healing with a normal colonic appearance

This case indicates several important clinical considerations. Although CD may rarely cause severe GI bleeding, deep ulcerations and transfusion-dependent hemorrhage should raise suspicion for alternative or additional pathologies. Infectious agents such as *Bartonella* species may involve the GI tract, particularly in immunosuppressed individuals,⁶ and may exacerbate mucosal injury causing mucosal ulcerations in the GI tract.^{6,7} Furthermore, ulcerations along the GI tract and lower GI bleeding caused by *Treponema pallidum* is documented and presented as literature information.^{6,8,9} Hematologic malignancies such as AML can also contribute to GI bleeding¹⁰ through mucosal fragility, leukemic infiltration, thrombocytopenia, and coagulopathy. Importantly, the initiation of immunosuppressive therapy based on an initial diagnosis of CD may have facilitated progression or unmasking of underlying infectious and malignant conditions in this patient. Therefore, clinicians should maintain a high index of suspicion for secondary causes in patients with presumed inflammatory bowel disease who exhibit atypical features such as persistent monocytosis, generalized lymphadenopathy, extensive deep ulcerations, or refractory and massive GI bleeding. In conclusion, this

case highlights the critical importance of comprehensive reassessment in patients with an established diagnosis of CD who demonstrate an unusual or aggressive clinical course. Early consideration of infectious and hematological etiologies may prevent diagnostic delay and guide appropriate, potentially life-saving interventions.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patient for the publication of this correspondence and any related clinical details.

Peer Review Process

This letter was externally peer-reviewed.

Conflict of Interest

The authors declare no conflicts of interest.

Financial Disclosure

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Author Contributions

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