LETTER TO THE EDITOR

A rare case of gastric Crohn's disease associated with immune thrombocytopenic purpura

Dear Sir,

Crohn's disease (CD) is a chronic idiopathic inflammatory disease characterized by the segmental, transmural involvement of the gastrointestinal tract. Ileocolonic and colonic/anorectal involvement is most common and account for 40% of the cases and involvement of small intestine in about 30%.1 Inflammatory Bowel Diseases (IBD), like CD, have been associated with various autoimmune conditions, with a cumulative prevalence of 8.2% to 10.5%.2 Concurrence of IBD with immune thrombocytopenia (ITP) is a rare phenomenon. Less than 20 cases have been reported in the medical literature and all of these patients had extensive colonic involvement. We are reporting a unique case, the first of its kind, of a patient with CD and isolated gastric involvement who was diagnosed with ITP.

A 57 yr old male with a four year history of dyspepsia, nausea, vomiting and pain in the stomach after eating, and weight loss, presented to our hospital for evaluation of acute thrombocytopenia that was detected on pre-treatment labs at his gastroenterologist's office. The patient had an upper and a lower endoscopy three months prior to admission. The esophagogastroduodenoscopy (EGD) (Fig. 1a) revealed multiple large friable, fungating, irregular, circumferential and pedunculated masses in the antrum of the stomach. The colonoscopy and capsule endoscopy exams were normal. Histopathologic examination of biopsies (Fig. 1b) from involved areas in the stomach was interpreted as chronic nonspecific granulating inflammation. Tests for helicobacter pylori were negative. The Prometheus IBD First Step Generation II test serological panel markers of anti-Saccharomyces cerevisiae antibody (ASCA) IgA assay was positive at 41.7, anti-Omp C (outer membrane porin from Escherichia coli) IgA 60.8, perinuclear anti-neutrophilic cytoplasmatic antibody (p-ANCA) antibody was 16.9 while ASCA IgG and antibody to CBir1 (anti-CBir1 flagellin) was normal confirming the diagnosis of gastric CD.

His physical exam on this admission was essentially unremarkable. He did not have a recent viral illness and he was not taking any medications. His stool was heme-positive. His labs revealed a mild anemia (hemoglobin of 9.3 g/dl) and thrombocytopenia (platelets 25 × 10^9/L) with giant platelets. The rest of his blood cell counts were normal. Erythrocyte sedimentation rate (ESR) was 60 mm/h. Serum iron profile, folic acid, and vitamin B12 were normal as were the remainder of the metabolic profile. Bone marrow aspiration revealed numerous immature megakaryocytes. The tests for Antinuclear antibodies, rheumatoid factor, Human Immunodeficiency Virus (HIV), cytomegalovirus, Epstein-Barr virus, viral hepatitis A, B and C virus were all negative. The patient had no family history for autoimmune disorders or IBD. An abdominal computerized tomography scan was normal. He was then managed with methylprednisone (2 mg/kg/day). His platelet count raised to 50 × 10^9/L and maintained on prednisone (80 mg/day) as an outpatient. His platelet counts remained between 400 and 500 × 10^9/L. Once his labs stabilized for one month, he was started on mesalamine for his CD. He now remains asymptomatic with normal platelet counts.

An association between IBD and ITP was first reported by Kosmo et al. in 1986.3 It has been suggested that antigenic mimicry is a possible pathogenetic mechanism by which increased mucosal permeability in active IBD caused enhanced exposure of the intestinal immune system to luminal antigens.4 Our patient had isolated gastric CD associated with ITP, unlike prior case reports of patients who had mainly ileocecal CD. Crohn's associated ITP should be treated first with glucocorticoids and in resistant or severe cases immune globulins may be administered, which is in accordance to recognized consensus guidelines on the management of ITP.5

References

Figure 1  a. Upper endoscopic findings of large, friable and fungating masses in the antrum of the stomach. b. Histopathology demonstrating chronic non-specific granulating inflammation.