A CASE REPORT

Henoch Schonlein Purpura: A Known But Often Forgotten Culprit in Gastrointestinal Bleeds

by Minesh Mehta, Richard P. Rood

INTRODUCTION

We describe an adult case of Henoch Schonlein Purpura presenting with abdominal pain and gastrointestinal bleeding. Colonoscopy revealed multiple erythematous, hemorrhagic, and ulcerated lesions throughout the colon. Biopsies of skin lesions were consistent with leukocytoclastic vasculitis and positive for IgA immunoflourescence.

Case Report

A 45-year-old Caucasian female with primary biliary cirrhosis presented with a 4-day history of nausea, vomiting, diarrhea and abdominal pain.

The abdominal pain started in the right lower quadrant but progressed into a more diffuse, generalized abdominal pain. She quickly developed mucoid, non-bloody diarrhea. Her appetite diminished and she subsequently developed nausea and vomiting. Within 24-48 hours after the onset of gastrointestinal (GI) symptoms, she developed a rash on both lower extremities that began on the feet and spread to the upper legs, groin and arms. The rash was painful and burning in nature but not pruritic. The patient denied recent medication changes, sick contacts, recent insect bites or any history of a similar rash. On review of systems she noted diffuse myalgias and arthalgias but denies fevers, chills and night sweats.

Her medications included albuterol, aspirin, ferrous gluconate, prozac, furosemide, omeprazole, oxycodone, potassium supplement, simvastatin, topiramate and ursodiol. Her past medical and surgical history included diabetes mellitus, depression, hyperlipidemia, primary biliary cirrhosis, lymphedema, meningioma with resection, an incisional hernia repair, debridement of abdominal wound, a laparoscopic cholecystectomy and a total abdominal hysterectomy during which a small bowel resection was done.

On admission her vitals were normal. Her exam was significant for multiple discrete coalescing 2-10mm erythematous and violaceous palpable, non-blanching, petechiae and purpura involving feet, legs, groins and arms. Her lower extremities also showed signs of chronic venous stasis with 2+ pitting edema. Her abdominal exam exhibited positive bowel sounds, mild tenderness to palpation and distension but there were no peritoneal signs.

Laboratory analysis showed anemia with (continued on page 72)
hemoglobin of 11.5 g/dL and mild hypokalemia.\textsuperscript{3,4} Alkaline phosphatase was elevated to 169 (baseline for her) however the rest of her liver panel was negative. Urinalysis revealed 12 RBCs and 30 mg/dL protein. Computed tomography (CT) scan of the abdomen revealed diffuse wall thickening of the colon and distal ileum with inflammatory stranding without evidence of bowel obstruction.

An infectious workup for the diarrhea was pursued including stool cultures, ova and parasites, Clostridium difficile toxin and stool white blood cells. Additionally, an autoimmune panel was sent since vasculitis was on the differential. Inflammatory bowel disease was considered, and the patient was scheduled for a colonoscopy. Dermatology was consulted for skin biopsy. The infectious workup was negative. The complete autoimmune panel was negative except for anti-mitochondrial antibodies. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were elevated to 90 and 8.5 respectively. She developed dark, bloody, sticky stools on her second day of admission with a concomitant drop in hemoglobin. She was scheduled for colonoscopy and started on IV solumedrol 60mg daily.

Biopsy of the skin lesions revealed a positive IgA immuno-fluorescence. Colonoscopy gross findings showed focally, edematous, erythematous, hemorrhagic, thickened and ulcerated lesions of the sigmoid, descending and transverse colon highly suggestive of vasculitis. Colonic pathologic findings showed chronic inflammatory infiltrate with a peri-vascular orientation. A few small vessels showed lymphocytes within their walls.

**DISCUSSION**

The combination of clinical presentation and positive IgA immuno-fluorescence on dermatopathology supports our diagnosis of HSP. HSP is a leukocytoclastic vasculitis involving IgA immune complex deposition in small vessels. It is classically a clinical diagnosis characterized by palpable purpura, abdominal pain, arthritis/arthralgias and hematuria. Biopsy of the skin and kidney play a confirmatory and prognostic role. In general, prognosis is good, except for patients with renal involvement.\textsuperscript{1} HSP has a peak incidence in the first and second decades of life with the annual incidence in children estimated at 14 per 100,000.\textsuperscript{3} The incidence of HSP in adults is significantly less at 1.3 per 100,000, with a mean age of presentation at 50 years old. Adults suffer higher rates of severe and atypical gastrointestinal complications.\textsuperscript{3} Mucosal gastrointestinal involvement typically affects the small bowel, however our patient had significant colonic involvement. Steroid therapy improves GI symptoms by decreasing intestinal wall edema and might prevent complications like intussusceptions.\textsuperscript{4}

**CONCLUSION**

We report a 45-year-old female with a history of primary biliary cirrhosis presenting with a GI bleed caused by HSP. In this patient, abdominal pain and bloody diarrhea were the initial symptoms followed by purpuric rash. While gastrointestinal involvement is common in HSP, the diagnosis is difficult when gastrointestinal symptoms precede cutaneous manifestations. Primary care physicians should consider HSP in a PBC patient that presents with abdominal pain. Identifying HSP early as the cause of GI bleed is important because management of HSP includes steroids. Initiating proper therapy early in the disease course can prevent complications like perforation, intussusception, massive GI bleed and bowel infarction requiring surgical intervention.

**References**