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CASE REPORT

Severe gastrointestinal involvement in adult-onset Henoch–Schönlein purpura associated with clarithromycin-resistant *Helicobacter pylori* infection

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Clarithromycin;
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Abstract *Background:* Henoch–Schönlein purpura (HSP) is an uncommon vasculitis in adults. Gastrointestinal involvement is part of the classical tetrad and can present as bleeding. *Helicobacter pylori* infection in the setting of HSP has been reported a few times in the literature and may be involved in the pathogenesis of this disease as a triggering agent.

Case report: A 48-year-old man presented to the emergency department with 9 days of acute symmetric additive polyarthritis, 2 days of palpable purpura involving lower limbs, recent-onset intense mesogastric pain and hematochezia. *H. pylori* was detected in gastric tissue and triple therapy (clarithromycin, amoxicillin and omeprazole) was started. Gastrointestinal bleeding and other symptoms stopped 24 h after steroid initiation and he was later discharged on prednisone (1 mg/kg) and azathioprine (100 mg/day). Shortly after discharge he was readmitted with hematochezia and clarithromycin-resistant *H. pylori* infection was suspected. Bleeding stopped following reinstitution of corticosteroids and a second-line scheme (levofloxacin, amoxicillin and omeprazole) was introduced. Corticosteroids were gradually tapered and he remained on azathioprine. Nine months later he was doing fine. The pertinent literature is briefly discussed, highlighting the previous cases of concurrent diagnosis in adult patients.

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Conclusion: To the best of our knowledge, this is the first report describing resistance to clarithromycin-containing triple therapy in a *H. pylori*-infected adult patient with HSP. Gastrointestinal bleeding remains one of the most feared manifestations of HSP. These patients may benefit from *H. pylori* screening, as this might positively affect their prognosis. Further studies in adults are nevertheless needed to clarify this association and its therapeutic impact.

1. Introduction

Henoch–Schönlein purpura (HSP) is an uncommon vasculitis in adults, with an annual estimated incidence of 8–18 cases per million [1]. Gastrointestinal involvement is part of the classical tetrad and occurs in one in ten adult patients at disease onset and in more than half of the cases when the disease is fully established [2,3]. Hemorrhage is one of the most severe manifestations of gastrointestinal involvement [4]. *Helicobacter pylori* infection in the setting of HSP has been reported a few times in the literature, but its role in the course of this disease remains controversial [5]. Here, we describe the case of a male patient presenting with gastrointestinal bleeding, as a manifestation of HSP, in whom *H. pylori* infection was also diagnosed, and then we discuss the pertinent literature.

2. Case report

A 48-year-old man with a medical history notable for newly diagnosed type 2 diabetes presented to the emergency department with 9 days of acute symmetric additive polyarthritis, 2 days of palpable purpura involving lower limbs and recent-onset intense mesogastric pain. On further questioning, he revealed that he recently suffered from constipation that required manual disimpaction. Soon after that, he developed hematochezia, which was self-limited and without hemodynamic compromise. He was febrile and had a heart rate of 117 bpm. His abdomen was soft with no peritoneal irritation signs. Initial laboratory workup showed no significant alterations in complete blood count. His serum creatinine was 0.6 mg/dL and C-reactive protein 98.5 mg/dL (upper normal limit: 6 mg/dL). No abnormalities were found on urinalysis. Fecal immunochemical test was positive for occult blood. ELISA for human immunodeficiency virus, hepatitis B surface antigen and anti-hepatitis C virus were all negative. Huddleson's reaction was also negative. He was positive for perinuclear anti-neutrophil cytoplasmic antibodies (pANCA) with a titer of 1:640. Antinuclear, anti-MPO and anti-PR3 antibodies, as well as rheumatoid factor, were all negative. No lesions were found in the contrast-enhanced abdominal computerized tomography scan. Under the suspicion of vasculitic process, a skin biopsy was performed on the same admission day. He remained under observation until his second inpatient day when he had hematochezia and was thus started on prednisone (1 mg/kg).

Ringed esophagus, esophageal mucosal erythema, gastric subepithelial hemorrhage, duodenal erythema with an ulcer <1 cm were noted on upper endoscopy. Descending colonic and rectal mucosal erythema, and rectal edema and subepithelial hemorrhage were found on rectosigmoidoscopy. Histopathology of biopsy specimens revealed chronic esophagitis compatible with reflux disease, active chronic gastritis, acute ulcerated duodenitis, focal active colitis, and edema and congestion of the rectal lamina propria. Immunofluorescence was not performed. *H. pylori* was detected in gastric tissue and he was therefore started on triple therapy (clarithromycin, amoxicillin and omeprazole).

Leukocytoclastic vasculitis was confirmed by skin histopathology and immunofluorescence demonstrated granular vascular deposits of IgA (+2), confirming the presumptive diagnosis of HSP. Bleeding and other symptoms stopped 24 h after steroid initiation and he was later discharged on prednisone and azathioprine (100 mg daily).

He skipped some doses of his medications and, shortly after discharge, was readmitted with hematochezia. Bleeding stopped following reinstitution of corticosteroid therapy. One month after finishing triple therapy, urea breath test (UBT) was performed and interpreted as positive, so we started him on a second-line scheme (levofloxacin, amoxicillin and omeprazole). He completed his treatment regimen and, four weeks later, UBT was repeated and tested negative. Corticosteroid therapy was gradually tapered and he remained on azathioprine. Nine months later, on the follow-up, he was doing fine and reported no symptoms.

3. Discussion

Gastrointestinal bleeding is considered a severe gastrointestinal manifestation of HSP and, in the largest unselected series to date, occurred in about 23 percent of patients, either as occult (10.3%) or overt hemorrhage (12.9%) [3,4]. However, it is worth noting that children were included in the previous figures and also that gastrointestinal involvement was more frequent in children than adults (67.3% *vs* 57.4%; $p < 0.05$) [3]. Gastrointestinal symptoms may precede the skin involvement and have been ascribed to immune complex deposition in vessel walls which lead to edema and hemorrhage [2]. pANCA with negative ELISA-ANCA has been previously detected in HSP patients with gastrointestinal symptoms and this may indicate that the ANCA target antigens may be different in HSP. These patients had higher disease activity [6]. Gastric and duodenal endoscopic appearance of HSP was first described in a 14 year-old girl by Akdamar et al. in 1973 [7]. Colonoscopic findings have been reported more rarely and were first described in two adult patients more than a decade later by Di Febo et al. [8,9]. The main findings include redness, petechiae, erosion, nodular changes, ulceration and strictures. These lesions are predominantly distributed in the second part of the duodenum, terminal ileum and rectosigmoid colon [10,11]. Concurrent involvement of upper and lower gastrointestinal tracts may not be uncommon as long as both upper endoscopy and colonoscopy are performed [11].