Schönlein-Henoch Purpura Associated with Gastric Helicobacter pylori Infection

A 65-year-old man was admitted in January 1995 for numerous extensive papular and purpuric lesions located mainly on the lower limbs. However the trunk and upper limbs were also affected. Cutaneous biopsy revealed moderate leukocytoclasis vasculitis with fi-brinoid necrosis of the dermal capillaries. The purpuric rash was accompanied by abdominal pain, diarrhoea and melaena. Oesope-gastroduodenoscopy performed 2 days after onset of the rash revealed petechial haemorrhage of the stomach. Histologic examination showed atrophic chronic gastritis with haemorrhagic suffusions in the superficial chorion. This was associated with marked presence of Helicobacter pylori within the lumina of dilated antral foveolae. Bacteriological culture was not performed. Colonoscopy was performed because of persistent abdominal pain and rectorrhagia and showed diffuse purpuric involvement of the ileum and right bowel. Biopsies demonstrated acutely ulcerated and haemorrhagic ileitis. Histological examination showed focal oedematous and haemorrhagic lesions in the chorion.

Except for arthralgia and cutaneous and digestive involvement, clinical examination was normal. Blood cell count and biological tests (including creatinaemia 84 µg/l) were normal except for proteinuria (maximum 3 g/24 h), elevated C-reactive protein (53 mg/l) and increased IgA level (4.1 G/l, normal range 1.01-3.26). HCV, HBV and HIV serology tests were negative. Antinuclear antibodies were negative. Renal biopsy showed focal glomerular lesions with abundant mesangial IgA and fibrin deposition. The patient was treated with 2g/day amoxycillin and 1 g/day clarythromycin for 8 days, associated with 20
mg/day omeprazole for 1 month and 100 mg/day dapsone. Cutaneous lesions disappeared within 2 months. Creatinaemia remained within the normal range and proteinuria returned to normal within 6 months. One year later, clinical examination and biological tests showed no relapse.

Comments

SHP is a syndrome with multiple aetiologies. It has been reported after infectious diseases, but the search for non-infectious aetiologies of vasculitis is necessary. In our case only H. pylori was evidenced by histological examination in antral ulcerations. H. pylori is considered to play an important pathogenic role in duodenal ulcers and gastritis [1]. Treatment usually consists of omeprazole associated with antibiotics (amoxycillin alone or with macrolides or imidazoles). Another case of SHP associated with antral ulcerations, probably secondary to H. pylori infection, was recently reported [2]. In this case H. pylori infection was treated with omeprazole and amoxycillin and signs of SHP disappeared. A relapse occurred 10 months later, once again associated with H. pylori gastric infection. This case was strongly suggestive of a causative role of H. pylori in the occurrence of SHP. In addition, some cases of SHP have been reported after infection with related bacteria, Campylobacter jejuni [3,4].

Because abdominal pain is frequent in SHP, fibroscopic examination is not systematically performed: thus association of SHP with H. pylori infection may be underestimated. This may be of importance for the treatment and prevention of relapses of SHP, since eradication of H. pylori is possible [1].

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References


