Late diagnosis of Henoch–Schönlein purpura following detection of jejunal ischemia on conventional endoscopy

A 40-year-old man was admitted to our hospital for acute-onset, colicky abdominal pain and vomiting. He had undergone gastrojejunostomy for gastric ulcer bleeding 18 years ago. On physical examination, the patient appeared dehydrated and his abdomen was soft and diffusely tender without peritoneal signs. Laboratory investigations revealed a leukocyte count of 14890/mm³ with 91% in segment form. The initial computed tomography (CT) scan of the abdomen showed focal concentric wall thickening of the jejunum in the left upper abdomen (Fig. 1). Enteritis was suspected and the patient was managed with bowel rest, intravenous fluid replacement, and antibiotics. After 7 days, the patient reported slight reduction of the abdominal pain, however, the inflammatory indices were gradually deteriorating (white blood cell count: 27230/mm³ and C-reactive protein: 11.9 mg/dL). A follow-up CT scan showed markedly aggravated wall thickening and extension of the inflamed area of the jejunum (Fig. 2). Endoscopic examination demonstrated severe segmental ischemia in the efferent loop of the jejunum (Fig. 3).

We decided to carry out laparotomy to resect the ischemic intestine and confirm the diagnosis, however, the patient refused surgical intervention because of the improvement in his symptoms. With the patient’s informed consent, conservative management was continued. The patient’s symptoms gradually improved and the inflammatory indices started to show a decrease on the twelfth day of admission. A follow-up endoscopy revealed a marked reduction in the mucosal ischemia in the efferent loop of the jejunum (Fig. 4). At 3 weeks following admission, the patient developed a purpuric rash, which covered his lower legs. Urinalysis revealed hematuria (20–30 red blood cells/high-power field) and marked proteinuria (3.5 g/day). Light microscopic examination of the renal biopsy samples revealed focal mesangial and endocapillary proliferation (Fig. 5a) and strong positivity for immunoglobulin A (IgA, Fig. 5b) on immunofluorescent staining. A diagnosis of Henoch–Schönlein purpura (HSP) was subsequently made. The patient was discharged after 4 weeks of admission and made a good recovery. HSP is a systemic vasculitis of unknown etiology and is thought to be related to an IgA-mediated autoimmune phenomenon [1]. Clinical diagnosis is made on the basis of the presence of physical findings such

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**Fig. 1** Initial abdominal computed tomography (CT) scan in a 40-year-old man admitted for acute-onset, colicky abdominal pain and vomiting. There is focal concentric wall thickening and prominent mucosal enhancement around the efferent loop of the jejunum (arrow).

**Fig. 2** Follow-up abdominal CT scan showed aggravation of the wall thickening and an increase in the inflamed area in the jejunum (arrowheads).

**Fig. 3** Conventional upper endoscopic findings (approach via the gastrojejunostomy area): there is severe segmental ischemia of the efferent loop of the jejunum.
as purpuric rash, arthralgia, abdominal pain, and renal complications [2]. The gastrointestinal system is one of the most frequently involved systems in children with HSP and common gastrointestinal-related complaints include abdominal pain, nausea, and bloody stool. These symptoms are caused by the inflammation of the vessels, which leads to edema and hemorrhage in the intestinal wall. Severe cases (1.5 – 4.6%) proceed to necrosis, intussusception, and perforation of the intestine, which may require surgical intervention [3].

Severe jejunal ischemia in adult HSP is rare and might easily be missed, especially in the absence of dermatological findings. However, the upper gastrointestinal endoscopic findings that are suggestive of vasculitis-induced mucosal ischemia might be considered as a manifestation of HSP [4, 5], especially in previously healthy adults with acute-onset symptoms. Therefore, special attention should be paid to gastrointestinal manifestations and the decision to undertake surgical intervention should depend on the patient’s symptoms rather than the laboratory results or imaging studies.

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