

# Henoch-Schönlein Purpura: A Rare Cause of Recurrent Acute Pancreatitis

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To the Editor: Henoch-Schönlein purpura (HSP) is a generalized vasculitis that can cause a large variety of symptoms in different organs.<sup>[1,2]</sup> It usually presents as acute-onset palpable purpura mainly located on the lower extremities. Acute pancreatitis (AP) is a potentially life-threatening inflammatory disorder and rarely presents as a complication of HSP. AP secondary to HSP is extremely rare,<sup>[3]</sup> and it is even rarer as an initial presenting feature of HSP before the occurrence of characteristic palpable purpura. We herein described a patient whose underlying HSP became clinically apparent after the development of AP. To the best of our knowledge, this is a very rare report of recurrent AP induced by HSP.

A 57-year-old man was transferred to our pancreas center after 1 week of treatment for AP in a local hospital. He had severe colic and intermittent abdominal pain on admission. His blood pressure was 120/70 mmHg (1 mmHg = 0.133 kPa), body temperature was 36.8°C, and pulse was regular at 72 beats/min. No skin rash was observed. Abdominal examination revealed rebound tenderness in all quadrants. There was no hepatosplenomegaly, and bowel sounds were normal. There was no history of trauma, medication, chronic illness, or alcohol intake. The patient's family history was unremarkable.

Laboratory tests showed the following: hematocrit, 41.9%; hemoglobin, 147 g/L; white cell count,  $11.1 \times 10^9/L$ ; neutrophilia, 87.3%; and platelet count,  $487 \times 10^9/L$ . The erythrocyte sedimentation rate and C-reactive protein concentration were significantly high at 30 mm/h and 31.8 mg/L, respectively. Serum testing showed the following: creatinine, 66.5  $\mu\text{mol/L}$  (reference range, 44–132  $\mu\text{mol/L}$ ); blood urea nitrogen, 5.89 mmol/L; calcium, 2.16 mmol/L; total cholesterol, 3.19 mmol/L; and triglycerides, 1.17 mmol/L. The serum amylase and lipase concentrations were 70 U/L (reference range, 0–110 U/L) and 260 U/L (reference range, 0–60 U/L), respectively. The serum immunoglobulin A concentration was slightly higher (4.99 g/L; reference range, 0.7–4.0 g/L). Abdominal computed tomography revealed effusion around the pancreatic head, thickened anterior fascia of the right kidney, and edematous swelling in the descending duodenum [Figure 1a]. He began conservative treatment for

AP. On day 8, the patient developed an extended purpuric rash over his upper and lower extremities [Figure 1b and 1c]. He then underwent colonoscopy and gastroscopy because of a positive fecal occult blood test. Diffuse purpura was observed in his stomach, small intestine, and colon [Figure 1d–1f]. The patient was diagnosed with HSP complicated by AP. He began therapy with intravenous methylprednisolone (80 mg/d) for 3 days followed by oral prednisone (60 mg/d). The patient's clinical status quickly improved, and he was discharged on the 10<sup>th</sup> day of treatment. Three months later, we discovered that the patient had been admitted to a local hospital because of recurrent AP and HSP. Unfortunately, the details of his admission were unavailable except that he was discharged 7 days after hospitalization.

AP is a disease with a broad spectrum of etiologies.<sup>[4]</sup> Although most cases are secondary to biliary stones or alcohol abuse, other potential causes such as toxins, drugs, surgery, metabolic or autoimmune conditions, and infections should be considered once the two most common etiologies have been excluded. Interestingly, HSP is a rarely described cause of AP. Although the details remain unknown, it is suggested that inflammation of the small blood vessels surrounding the pancreas is involved in the development of AP.

In the case, the patient showed classic signs of HSP, including nonthrombocytopenic purpura over his upper and lower extremities and gastrointestinal symptoms. Diagnosis of AP was easily established based on the presence of abdominal pain, an elevated serum lipase concentration, and peripancreatic fluid collections on abdominal enhanced computed tomography. Other conditions,

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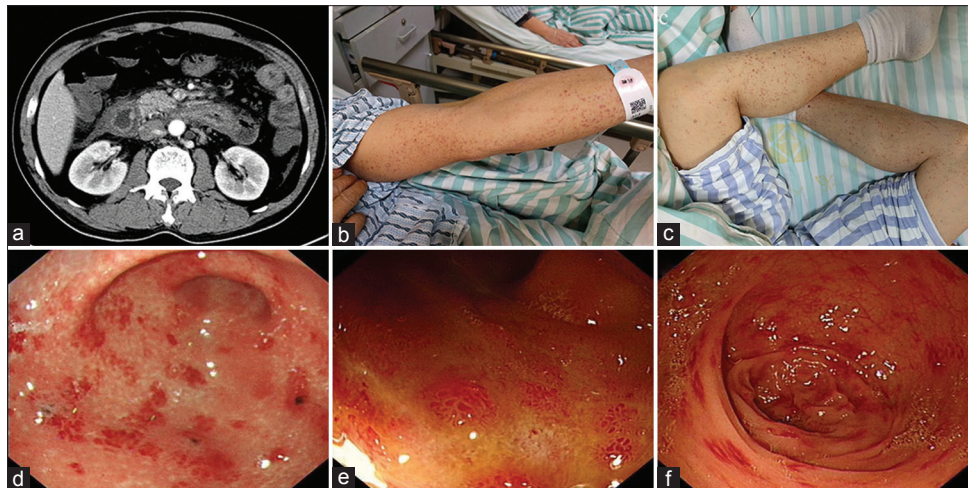
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**Figure 1:** (a) Contrast-enhanced axial computed tomography image reveals peripancreatic inflammatory changes (surrounding pancreatic head) suggestive of acute pancreatitis. Palpable purpura involves the (b) bilateral arms and (c) legs on day 8. Gastroscopy and colonoscopy show diffuse purpura in the (d) stomach, (e) small intestine, and (f) colon.

such as biliary tract disease, alcohol, hypertriglyceridemia, trauma, infections, drugs, and hereditary pancreatitis, were inconsistent with the clinical and laboratory presentations in our patient. Accordingly, HSP was regarded as the cause of his AP.

For HSP-associated pancreatitis, steroids should result in prompt resolution.<sup>[5]</sup> Our patient's AP did not improve with supportive treatment. However, his symptoms of pancreatitis disappeared quickly after using steroids. Notably, the patient developed recurrence of AP, and it was highly possible that early drug discontinuation contributed to this.

In conclusion, HSP is a rare and benign cause of AP, which could occur before the characteristic rash and present as the initial manifestation of HSP. Steroids could improve the outcome of HSP in patients with secondary AP.

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### Conflicts of interest

There are no conflicts of interest.

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