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Superior mesenteric artery syndrome in a 13-year-old girl with henoch schonlein purpura: A case report

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Abstract
Superior Mesenteric Artery (SMA) syndrome is an uncommon gastrointestinal blockage caused by the compression of the third section of the duodenum between the abdominal aorta and the superior mesenteric artery. Henoch Schönlein Purpura (HSP), or IgA vasculitis, is the most common childhood vasculitis, marked by palpable purpura, arthralgia, gastrointestinal symptoms and renal involvement. The coexistence of both conditions is exceptionally rare. We report a 13-year-old girl who presented with persistent epigastric pain and bilious vomiting, followed by cutaneous purpura and mild hematuria. Imaging and histopathology confirmed SMA syndrome and HSP respectively. Management with nutritional support and corticosteroids led to complete resolution. This case underscores the importance of recognizing dual pathologies and the role of steroids in treating SMA syndrome secondary to HSP.

Keywords: Superior mesenteric artery syndrome, henoch schönlein purpura, IGA vasculitis, corticosteroid therapy, pediatric gastrointestinal obstruction

Introduction
SMA syndrome, also referred to as Wilkie's syndrome, results from narrowing of the aortomesenteric angle (typically < 25°) and reduced aortomesenteric distance, resulting in mechanical duodenal obstruction [1]. Its incidence is rare, estimated between 0.013% and 0.3%, particularly unusual in children [2]. Presentations include abdominal fullness, nausea, vomiting and weight loss. Diagnostic confirmation relies on imaging such as CT angiography showing reduced angle and distance [3].
HSP (IgA vasculitis) manifests with palpable purpura, arthralgia, abdominal pain, and often renal involvement [4]. Although gastrointestinal involvement in HSP is common, SMA syndrome as a complication is rarely reported. The overlap between HSP and SMA syndrome is poorly documented. A survey of the literature indicates just one case report and one case-control study by Harada *et al.* that outlined duodenal irregularities in HSP resembling SMA syndrome [5, 6]. This indicates a possible, though overlooked, connection between the two conditions.
This case report details a 13-year-old patient with HSP who experienced SMA syndrome due to inflammation of the duodenum. Through this case sharing, we intend to highlight this uncommon yet important association and offer perspectives on handling these situations.

Case Presentation
A 13-year-old previously healthy girl presented with a seven-day history of fever and crampy epigastric abdominal pain, followed by recurrent bilious vomiting for three days. She also reported two episodes of melena and hematuria over the preceding 24 hours along with mild weight loss of approximately five percent. During her hospital stay, she developed a non-blanching, progressive purpuric rash that initially appeared over the feet and later spread to involve the entire lower limbs and buttocks (Figure 1A and 1B). She also complained of mild bilateral ankle pain without associated swelling.
On examination, the patient was thin-built and mildly dehydrated, with stable vital signs. Abdominal examination revealed mild distension with non-radiating epigastric tenderness and hyperactive bowel sounds, without guarding, rigidity or organomegaly. Cutaneous examination showed palpable purpura over both lower limbs and buttocks.

There was mild tenderness over both ankles, but no swelling or restricted joint movement.

Laboratory investigations showed a hemoglobin level of 11.6 g/dL, total leukocyte count of 20,160/mm³ (with 91% neutrophils and 3.9% lymphocytes) and platelet count of 3, 44, 000/mm³. The erythrocyte sedimentation rate (ESR) was 16 mm/hr and C-reactive protein (CRP) was 13 mg/L. Renal and liver function tests were within normal limits except for mild hypoalbuminemia (3.2 g/dL). Serum amylase was 105 U/L, while lipase was markedly elevated at 765 U/L. Urinalysis revealed trace hematuria and mild proteinuria. Both antinuclear antibody (ANA) and antineutrophil cytoplasmic antibody (ANCA) tests were negative. Coagulation profile showed a prothrombin time of 21.7 seconds, an INR of 1.64, and an activated partial thromboplastin time of 29 seconds. The mantoux test was negative.

Ultrasonography of the abdomen demonstrated a distended urinary bladder with few free-floating internal echoes and normal wall thickness. Chest radiograph was normal. Contrast-enhanced computed tomography (CT) of the abdomen with oral contrast revealed gastric and proximal duodenal distension with a reduced superior mesenteric angle (20°-25°) and aorto-mesenteric distance (< 8 mm), findings consistent with superior mesenteric artery (SMA) syndrome. There was also mild extrinsic compression of the proximal segment of the celiac artery by the right diaphragmatic crus, resulting in approximately 50% luminal narrowing with minimal post-stenotic dilatation, suggestive of median arcuate ligament syndrome (Figure 2).

A skin biopsy from the purpuric lesions demonstrated leukocytoclastic vasculitis with IgA deposition on immunofluorescence, confirming the diagnosis of Henoch-Schönlein Purpura (HSP). Based on the combined clinical, imaging, and histopathological findings, a final diagnosis of concurrent superior mesenteric artery syndrome and Henoch-Schönlein Purpura was established an exceedingly rare association in pediatric patients.

The patient was managed conservatively. She was kept nil per oral solids with nasogastric decompression and received intravenous fluids and nutritional support through a nasojejunal feeding tube aimed at gradual weight gain. Intravenous methylprednisolone at a dose of 1 mg/kg/day was initiated to treat HSP and the presumed inflammatory duodenitis contributing to SMA compression. Over the subsequent seven days, her vomitings subsided, and oral feeding was gradually reintroduced. Within three weeks, she showed significant weight gain, and repeat imaging demonstrated improvement in the aorto-mesenteric angle and restoration of normal duodenal passage. Corticosteroids were tapered over the next four weeks. During the follow-up, the patient was asymptomatic, with full disappearance of the rash and no signs of kidney involvement.

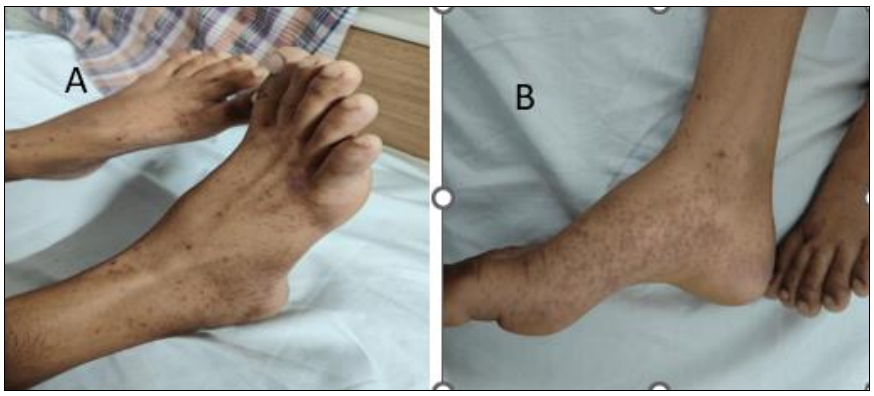

Fig 1: A and B: Non blanching purpuric rash

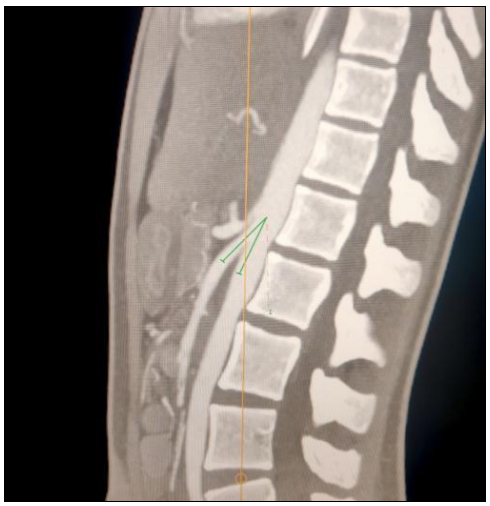

Fig 2: Image showing superior mesenteric angle (18°) and aorto-mesenteric distance (5 mm)

Discussion

The coexistence of SMA syndrome and HSP is extraordinarily rare. HSP-related duodenal inflammation may induce local edema or fat pad alteration, leading to a secondary SMA compression a mechanism considered in the eight-year-old girl previously reported. Given that SMA syndrome is typically linked to weight loss or catabolic states, the presence of duodenal inflammation in HSP suggests an alternative pathophysiology. Furthermore, steroid therapy mainstay for HSP may simultaneously alleviate SMA compression by reducing inflammation, as seen in our patient and the case by Shayah *et al.* [7]. In management, distinguishing the dual pathology is critical. If SMA syndrome alone is presumed, management often begins with decompression and nutritional rehabilitation with surgery reserved if conservative measures fail [8]. In contrast, when HSP is present, early corticosteroid intervention is essential and may render surgical interventions unnecessary.

SMA diagnosis necessitating a strong clinical suspicion because of non-specific symptoms such as nausea, vomiting, epigastric pain and weight loss. Consequently, SMA may be one of the reasons for the duodenitis frequently observed in HSP [9]. Indeed, the clinical presentation of our patient aligned with the diagnoses of both HSP and SMA. In an earlier investigation of SMA cases identified through ultrasonography, gastroduodenoscopy, hypotonic duodenography and contrast-enhanced computerized tomography, the angle between the aorta and SMA was <25 degrees (normal values 25-60), and the space between them measured <8.0 mm (normal values 10-28 mm) [10]. In our case, the CT scan of the abdomen with oral contrast showed distension of the stomach and proximal duodenum, along with a decreased superior mesenteric angle (< 20-25°) and aorto-mesenteric distance (< 8 mm), indicating superior mesenteric artery (SMA) syndrome.

HSP is mainly a clinical diagnosis reliant on the presence of palpable purpura. Histopathological examination reveals leukocytoclastic vasculitis with mainly IgA deposits in skin or kidney biopsy [11]. In the current situation, skin biopsy from the purpuric lesions confirmed HSP, showing leukocytoclastic vasculitis with IgA deposition on immunofluorescence, thus validating the diagnosis of Henoch-Schönlein Purpura (HSP).

Our case emphasizes the significance of identifying underlying conditions such as HSP that could lead to SMA syndrome. Timely application of steroid treatment and total parenteral nutrition was crucial in the patient's recuperation, promoting weight gain and the capacity to handle oral diet. Prompt administration of steroids not only relieves extrarenal symptoms like joint and abdominal pain but also affects the progression of kidney involvement in patients with HSP [12]. In our case, we have started intravenous methylprednisolone at a dosage of 1 mg/kg/day to address HSP and the suspected inflammatory duodenitis that is causing SMA compression. After initiation of steroid and nutritional support, her vomiting decreased and oral feeding was slowly reintroduced. In three weeks, she exhibited considerable weight gain and follow-up imaging indicated enhancement in the aorto-mesenteric angle and return to normal duodenal passage.

Conclusion

This report describes a rare but instructive case of SMA syndrome secondary to HSP in a 13-year-old girl. Timely recognition and appropriate combined management with nutritional support and corticosteroids led to full resolution without surgical intervention. Clinicians should remain vigilant for SMA syndrome in HSP patients with atypical or persistent GI manifestations and consider early steroid therapy to address both conditions.

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Declarations

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- **Conflict of interest:** None declared
- **Ethical approval:** Not required

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