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CASE REPORT

Delayed diagnosis of abdominal Henoch-Schonlein purpura in children: A case report

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Abstract

BACKGROUND

For children with abdominal Henoch-Schonlein purpura presenting abdominal pain as an initial symptom and severe clinical manifestations, but without purpura appearance on the skin, the diagnosis and treatment are relatively difficult. This study summarized the characteristics of this group of patients by literature review and provided additional references for further refinement of glucocorticoid therapy in this vasculitis.

CASE SUMMARY

A 6-year-old girl presented mainly with repeated abdominal pain and had received short-term out-of-hospital treatment with hydrocortisone. On day 7 after onset, gastroscopy revealed chronic non-atrophic gastritis and erosive duodenitis without purpuric rash, and no obvious resolution of the abdominal pain was found after treatment against infection and for protection of gastric mucosa. On day 14 the inflammatory indices continued to rise and the pain was relieved after enhanced anti-infective therapy, but without complete resolution. On day 19, the patient presented with aggravated abdominal pain with purplish-red dots on the lower limbs, by which Henoch-Schonlein purpura was confirmed. After 5 d of sequential treatment with methylprednisolone and prednisone, abdominal pain disappeared and she was discharged.

CONCLUSION

Henoch-Schonlein purpura-related rash may appear after long-term abdominal pain, and should be distinguished from acute and chronic gastrointestinal diseases at the early stage without typical rash. For bacterial infection-induced Henoch-Schonlein purpura, glucocorticoid therapy alone without clearing the infection may not relieve symptoms.

Key Words: Henoch-Schonlein purpura; Delayed diagnosis; Rash; Abdominal pain; Gastrointestinal disease; Case report

Core Tip: Henoch-Schonlein purpura (HSP) is a common vasculitis in children. Abdominal pain is one of its characteristic features, but the presence of gastrointestinal symptoms alone without a rash is easily misdiagnosed. We report a case of HSP with abdominal pain as the first symptom, emphasizing the importance of differential diagnosis to avoid incorrect surgery.

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INTRODUCTION

Henoch-Schonlein purpura (HSP) is one of the most common vasculitis in childhood. The main pathogenesis is leukocytoclastic vasculitis and deposition of immunoglobulin A (IgA) immune complexes in small blood vessels[1]. HSP can affect the skin, joints, gastrointestinal tract, and kidney, with skin purpura as the initial symptom in most cases. Abdominal pain is a common symptom of HSP, mostly manifesting after rash[2]. EULAR/PRINTO/PRES 2008 proposed the diagnostic criteria for HSP as palpable purpura or petechia containing at least one of the following four conditions: (1) Acute diffuse abdominal pain; (2) leukocytoclastic vasculitis with IgA deposition revealed by biopsy; (3) arthritis or joint pain; and (4) renal involvement, including proteinuria or hematuria[3]. The rash usually appears on the compressed sites, especially the lower limbs and hip, but is not associated with thrombocytopenia.

Abdominal pain is a common symptom of HSP that presents mostly after the rash and rarely precedes it. Here, we highlighted an unusual case of a pediatric HSP patient with purpuric rash emerging after 19 d of persistent abdominal pain. Given the difficulty of making a clear diagnosis at the early stage when only abdominal pain without rash is present, a misdiagnosis of, for example, surgical acute abdomen or even a recommendation for surgery can result, bringing further pain and anxiety to children. To reduce the misdiagnosis rate of abdominal HSP and increase the clinical diagnostic accuracy, this study analyzed the clinical characteristics of this child in detail and summarized the characteristics of children with delayed abdominal HSP from reports that included a sample size of more than 50 cases published from 2000 to 2022.

CASE PRESENTATION

Chief complaints

A 6-year-old girl presented with intermittent abdominal pain and vomiting for 10 d.

History of present illness

Symptoms started 10 d before presentation with persistent dull pain around the umbilicus and upper abdomen, and non-ejective vomiting (gastric contents) 7-8 times a day with no coffee-like or bloody substances. There were no known sick contacts and no recent travel. The patient was seen at the local hospital and given hydrocortisone, 5 mg/kg/d and cefotaxime, 300 mg/kg/d for 6 d with no obvious remission. Gastroscopy on day 7 revealed chronic non-atrophic gastritis, hyperemia, and edema in the descending mucosa of the duodenum (Figure 1A).

History of past illness

Her past medical history was unremarkable.

Personal and family history

Family history for gastrointestinal diseases or HSP was negative.

Physical examination

On physical examination there was periumbilical tenderness, but no other abnormalities were found. There was no hepatomegaly or splenomegaly; Blumberg and Murphy signs were negative. There was no skin rash at admission.

Laboratory examinations

Blood tests showed increased inflammation indicators and platelets [white blood cells (WBC) 25.6 × 10^9/L, neutrophils (N) 81.7%, lymphocytes (L) 11.7%, C-reactive protein (CRP) 38 mg/L, platelets (PLT) 648 × 10^9/L]. Erythrocyte

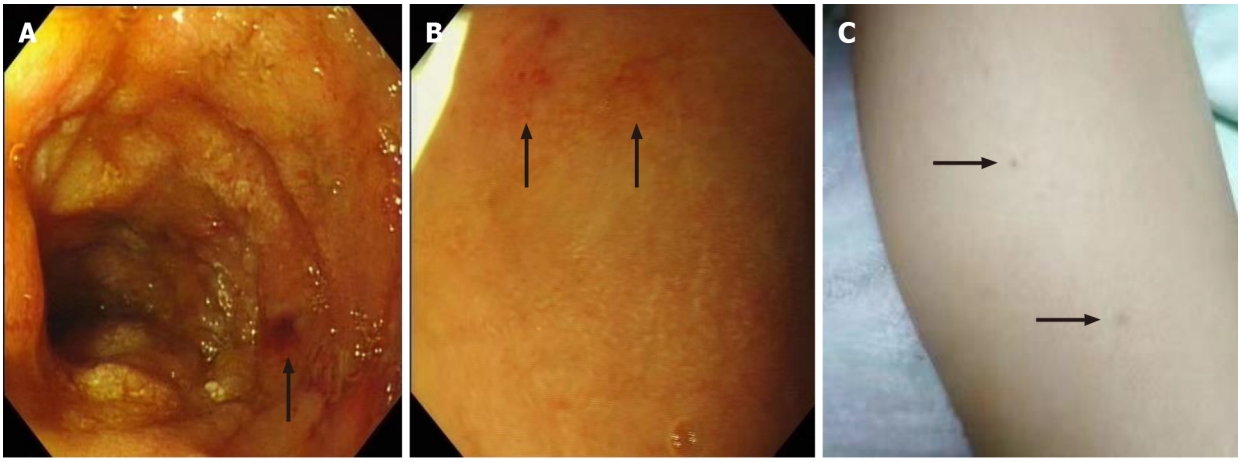

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Figure 1 Gastroscopy and other symptoms. A and B: Gastroscopy images of the child on the 7th (A) and 17th (B) days after the onset (arrows); C: On the 19th day after the onset, purplish-red hemorrhagic dots appeared on the skin of the child's lower limbs (arrows).

sedimentation rate, procalcitonin, transaminases, total and direct bilirubin, amylase, lipase, coagulation profile were within normal range. Stool culture, testing for rotavirus, adenovirus, aerobic bacteria, clostridium difficilis toxin and antigen were negative. Hemoccult was negative on three stools specimens. Urinalysis showed increased 24-h urine protein quantification (0.37 g/24 h) and α1- macroglobulin (6.01 mg/dL) Anti-Saccharomyces cerevisiae antibodies and anti-neutrophil cytoplasmic antibodies, were performed in the workout for inflammatory bowel diseases and resulted negative. Anti-nuclear antibody profile (anti-ds-DNA antibody, anti-SS-A antibody, anti-Ro52 antibody, anti-SS-B antibody, *etc*.) was negative to differentiate from systemic lupus erythematosus. On the other hand, in the immunological laboratory tests (IgG, IgA, IgM levels, IgG subclasses, and lymphocyte subpopulations), the level of IgA is elevated, while the other levels are normal.

Imaging examinations

An abdominal ultrasound scan showed no intussusception or appendiceal swelling. Abdominal computed tomography (CT) revealed obvious swelling in the wall of the segmental small intestine, peritonitis, and abdominal pelvic effusion.

FINAL DIAGNOSIS

Combined with the results of gastroscopy in another hospital, the diagnosis was as follows: (1) Acute peritonitis; and (2) chronic non-atrophic gastritis. The diagnosis of HSP was confirmed.

TREATMENT

No improvement in abdominal pain after anti-infective treatment with cefoperazone+tazobactam and protection of gastric mucosa with omeprazole and aluminum sulfate gel for 4 d. On day 14 after onset, inflammatory indicators continued to rise (WBC 31.4 × 10^9/L, N 86.8%, L 8.1%, CRP 75 mg/L upon routine blood examination). After 3 d of intensive anti-infection treatment with meropenem instead of antibiotics, abdominal pain was significantly relieved but did not completely resolve. Gastroscopy on day 17 after onset revealed chronic non-atrophic gastritis and erosive duodenitis (Figure 1B), and mild to moderate chronic inflammation in the mucosa with erosion, without indication of hemorrhagic rash in the gastric mucosa. On day 19 after onset, the abdominal pain was further aggravated by paroxysmal dull pain around the umbilicus, and purplish-red hemorrhagic dots appeared on the skin mainly on the lower limbs, without pruritus, bleeding, or ulceration (Figure 1C). Platelet count was normal.

After treatment with methylprednisolone, 2 mg/kg/d for 3 d and sequential oral prednisone, 0.75 mg/kg/d for 2 d.

OUTCOME AND FOLLOW-UP

The abdominal pain disappeared. Laboratory tests returned to normal: WBC 9.1 × 10^9/L, neutrophils (N) 40.1%, lymphocytes (L) 52.6%, CRP 4.0 mg/L. No abnormalities on fecal examination and urine analysis. The patient made a full recovery and was discharged. She continued to take prednisone, 0.5 mg/kg/d for a further 2 wk. There was no recurrence over 6 mo of follow-up.

DISCUSSION

HSP, also called immunoglobulin A vasculitis, characterized by generalized aseptic inflammation of small blood vessels as a pathological basis, is the most common vasculitis in childhood. The main clinical manifestations include skin purpura, abdominal pain and gastrointestinal hemorrhage, joint swelling and pain, and renal involvement. Rash and abdominal pain are the most common symptoms. Gastrointestinal symptoms, reported in 50%–85% of HSP patients, are possibly attributed as secondary to edema and hemorrhage in the gastrointestinal wall and mesentery, and include nausea and vomiting, vomiting of blood, and bloody stools. Among these gastrointestinal symptoms, 14%–36% occur before the rash appears[4]. We performed retrospective analysis of the literature published between 2000 and 2022 with delayed abdominal HSP with a sample size of more than 50 children in Table 1. In previous studies, the interval between abdominal pain and rash in HSP patients was reported as usually 2–10 d[5,6], the longest interval in patients without glucocorticoids being 28 d and the shortest interval 1 d[7-9]. In 2006, Sato *et al*[10] reported an elderly HSP patient with emergence of rash after 75 d of abdominal pain, but this patient was diagnosed with pancreatitis at an early stage. Thus, it could not be excluded that the early abdominal pain was caused by pancreatitis.

Table 1 Summary of delayed abdominal Henoch-Schonlein purpura in pediatric patients, %

Ref.	Calvo-Río *et al*[5]	Ji[19]	Chao *et al*[9]	Calviño *et al*[11]	Rubino *et al*[16]
Number	417	218	208	78	118
Sex (male/female)	240/177	140/88	116/92	42/36	66/52
Age (mo, yr)	8 mo-87 yr	2 yr 4 mo-13 yr	9 mo-15 yr	1 yr-13 yr	1 yr-17 yr
Median age	7.5 ry	-	6.4 yr	5.5 yr	7 yr
Etiologic factor	-	-	-	-	-
Infections	38	53	-	28	-
Drug intake	18.50	-	-	18.00	-
Allergy	-	21.10	-	-	-
Abdominal pain as initial manifestation	13.70	16.00	58.00	16.70	-
Time between abdominal pain and purpura (d)	6-15 d	1-12 d	1-13 d	1-10 d	-
Median d	10	-	4.8	3.3 d	-
Use of glucocorticoid	-	-	7.5 ± 2.8	-	-
No use of glucocorticoid	-	-	10.2 ± 3.6	-	-
Leukocytosis was present	36.7	46.8	47.5	52.6	-
Anemia	8.9	11.5	10.5	7.7	10
ESR was present	80.1	16.5	-	63.9	40
Increased IgA serum	31.7	-	-	57.1	27
ANCAs	0	-	-	-	-
Stool occult blood	-	16.10%	52/160 positive	12 positive cases	30 positive cases
CT or X-ray	-	-	42/82 positive		18/21 positive
Gastroscopy	-	-	15/27 positive	2/2 positive	3/5 positive
Treatment	-	-	-	-	-
Corticosteroids	35	90	99.50	23.10	68
Cytotoxic drugs	5	Use for some patients with renal impairment (87 patients)	-	Use for two children due to severe renal impairment	Use for one chid for steroid resistance
Nonsteroidal anti-inflammatory drugs	14	-	-	-	43
Dialysis	1	-	-	-	-

Outcome		-	-	-	-
Complete resolution	83.20	-	-	88.40	-
		-	-	-	100
Persistent nephropathy (renal sequelae)	7.70	-	-	11.60	-
Reoccurrence	31.90	-	10.10	14.50	21.00

ANCAs: Antineutrophil cytoplasmic antibodies; CT: Computed tomography; ESR: Electron spin resonance; IgA: Immunoglobulin A.

The child presented some specific clinical manifestations. First, gastrointestinal symptoms are reported in 50%–85% of HSP patients, commonly including nausea and vomiting, vomiting blood, bloody stool, and melena. Other rare gastrointestinal symptoms include intussusception, mesenteric ischemia, intestinal perforation, massive gastrointestinal bleeding, acute non-calculous cholecystitis, hemorrhagic ascites with peritonitis, pancreatitis, and biliary cirrhosis[11-16], but an association with chronic gastrointestinal diseases is rarely reported[17,18]. The patient in our study presented with abdominal pain and peritonitis in the early stage, associated with underlying chronic non-atrophic gastritis and erosive duodenitis, a complicated condition reported here for the first time based on our literature review.

Second, skin symptoms in HSP patients usually precede gastrointestinal symptoms, although the opposite occurs in 12%–19% of patients[4]. In a study including 208 HSP patients, Chen *et al*[14] found that 41 patients (25.3%) presented with gastrointestinal symptoms before the appearance of skin lesions, with an average interval of 4.8 d, while 18 patients had abdominal pain more than 1 wk before the skin symptoms[15]. The patient in the present report had abdominal pain that lasted for 19 d before the purpuric rash appeared at the typical site. Currently the longest interval between rash and abdominal pain (rash appearance first) in HSP patients is 75 d, but this patient was diagnosed with pancreatitis at an early stage, thus not excluding that early abdominal pain could have been caused by pancreatitis[10]. According to the literature we searched, the interval in most patients was 2–10 d[5,19]. With the exception of the present report, the longest interval was 12 d and the shortest interval was 1 d[7,8]. Our patient sustained rash onset after 19 d of abdominal pain, the longest interval reported to date.

Treatment with glucocorticoids and antibiotics in another hospital failed to control the disease. After admission to our hospital, CRP (an inflammation indicator) increased progressively, and abdominal pain was significantly relieved after intensive anti-infection treatment. Thus, the poor effect of glucocorticoids might be related to the failure of infection control. In our case, it is possible that a gastrointestinal infection triggered HSP. In fact, HSP commonly occurs after bacterial or viral infections (usually upper respiratory tract or intestinal infections), especially during the autumn season. Therefore, infection may play a role in the etiology of IgA vasculitis. The following hypotheses are proposed[20]: (1) The N-Acetylgalactosamine (GalNAc) present on the surface of the pathogen may contribute to the production of IgA and IgG antibodies that cross-react with Galactose deficient immunoglobulin A1 (Gd-IgA1); (2) Microbes may contain antigenic structures like the vascular wall, leading to the production of cross-reactive autoantibodies; (3) Mucosal infections result in upregulation of IL-6, which may contribute to the development of Gd-IgA1 by altering the glycosylation mechanism.

Currently, glucocorticoids are considered effective for HSP gastrointestinal and joint symptoms, and early initiation can effectively relieve abdominal and joint symptoms, significantly relieve abdominal pain, improve the relief rate within 24 h, as well as reducing the risk of intussusception and intestinal bleeding[19,21-25]. There is no evidence to prove that glucocorticoid therapy is effective in the regression and recurrence of rash[26], but we guess that early use of glucocorticoids may be associated with delayed appearance of rash in HSP. The patient in the present study was treated out of hospital with hydrocortisone, 5 mg/kg/d for 6 d, which may be an important reason for the delayed rash.

CONCLUSION

HSP is a relatively common childhood disorder that may be difficult to diagnose when the typical signs of rash, abdominal pain, arthralgias, and nephritis do not occur simultaneously. Our case serve as a reminder that abdominal pain can appear long before the rash.

FOOTNOTES

Author contributions: Wang ZL analyzed and interpreted the patient data regarding the Henoch-Schonlein purpura and was a major contributor in writing the manuscript; Tao Z collect related literature and complete partial manuscript content; Guo H revised and expanded the manuscript. All authors read and approved the final manuscript.

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