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Henoch-Schönlein Purpura (HSP): Case Report

Dr. Thenmozhi. P[1], Soumya. C[2]

[1]Professor and HOD, Department of Medical and Surgical Nursing, Saveetha College of Nursing,
[2]M.Sc (Nursing), Nurse Practitioner in Critical Care II Year, Saveetha College of Nursing,
[1,2]Saveetha Institute of Medical and Technical Sciences, Chennai, Tamil Nadu, India

ABSTRACT

An acute immunoglobulin A (IgA) - mediated condition known as Henoch-Schönlein purpura (HSP) is characterized by a widespread vasculitis affecting the small blood vessels of the skin, GI tract, kidneys, joints, and, in rare cases, the lungs and central nervous system (CNS). Henoch-Schönlein purpura is characterized by the "classic triad" of purpura, arthritis, and stomach pain. Antigen-antibody (IgA) complexes activate the alternative complement pathway in this systemic illness, causing inflammation and small vessel vasculitis. Mild illness resolves on its own, and symptomatic treatment is all that is required. For HSP that ranges from mild to severe, systemic steroids are advised. The level of renal involvement affects the prognosis, thus close monitoring is necessary. Early detection - as in the case of our adolescent patient—and the right kind of management can help to slow the progression of the illness and prevent organ damage.

KEYWORDS: *Henoch-Schönlein purpura (HSP), IgA vasculitis, steroids*

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INTRODUCTION

IgA vasculitis, also known as Henoch-Schönlein purpura (HSP), is a condition that most frequently affects children and affects the skin, mucous membranes, and occasionally other organs. The illness results in palpable purpura (small, elevated spots of blood beneath the skin), which frequently coexists with joint and gastrointestinal pain- the "classic triad ". Small amounts of blood and protein may be lost in the urine as a result of renal involvement (hematuria and proteinuria), although this usually remains undetected; in a small percentage of instances, the kidney involvement progresses to chronic kidney disease. The illness was first documented in the 1860s by Johann Lukas Schönlein (1793–1864), a teacher of Eduard Heinrich Henoch (1820–1910), a German pediatrician. Henoch was also responsible for its naming. Schönlein and Henoch correlated the purpura to arthritic involvement, while Henoch related the purpura to gastrointestinal issues. William Osler was the first to recognize the underlying allergic mechanism of HSP. It occurs more often in children than in adults, and usually follows an upper respiratory tract infection. The most prevalent form of vasculitis in children is HSP, which has a frequency of roughly 20 per 100,000 children each year. Based on the constellation of symptoms, a diagnosis is established. Blood tests may show elevated creatinine and urea levels (in kidney involvement), raised IgA levels, and raised C-reactive protein (CRP) or erythrocyte sedimentation rate (ESR) results. It can be distinguished from conditions where low platelet counts are the origin of the purpura, such as idiopathic thrombocytopenic purpura and thrombotic thrombocytopenic purpura, by the possibility of an increased platelet count.

We report a unique case of Henoch–Schönlein purpura, a 14-year-old male.

Case description

A 14-year-old adolescent male presented with fever and sore throat for 3 days. He was initially given oral antibiotics. Within a day, he began to experience an erythematous, non-pruritic rash that spread from his feet to his thighs, upper limbs, and involving his palms and soles. Later, the feet became swollen and

had moderately severe (7/10) intense pain that was made worse by walking. On physical examination, there was pharyngeal erythema, petechiae on the soft palate, cervical lymphadenopathy, a nodular, and non-tender, non-blanching purpuric rash involving both upper and lower extremities with non-pitting pedal edema (Figures 1 and 2). Two days later, the patient experienced right and left upper quadrant abdomen pain that was persistent, colicky in nature, 8/10 in intensity, and made it worse after eating and associated with hematemesis and watery stools. Laboratory tests showed leukocytosis (WBC: 18,700); Hb: 13.5 g/dL; Hct: 41.2%; BUN: 17 mg/dL; Serum Creatinine: 0.8 mg/dL; Urinalysis: no hematuria or proteinuria; ESR: 58 mm/Hr; CRP: 6.1 mg/dL; Antistreptolysin O titer: 823 IU/L; ANA: Negative; EBV-VCA IgM: Negative; Anti-HAV IgM: Negative; HbsAg: Negative; Anti-HBc IgG: Negative; Mono spot test: Negative; c-ANCA: 0.2 units; p-ANCA: 0.2 units; Stool for occult blood: positive. Multiple erosions in the duodenum and antrum were visible during esophagogastroduodenoscopy (Figure 3). The small bowel's histopathology (Figures 4 and 5) revealed intact villous architecture, neutrophilic and eosinophilic infiltrates, and leukocytoclastic vasculitis. There was no evidence of epithelioid granuloma. Colonoscopy showed erythema and inflammation in the terminal ileum and cecum. Cecal biopsy also showed leukocytoclastic vasculitis. A skin biopsy revealed pustular leukocytoclastic fibrinoid vasculitis with a micro abscess (Figure 6). The patient was diagnosed with Henoch-Schönlein purpura. He was treated with intravenous fluids. He was started on oral prednisone 20 mg twice a day, with resolution of his symptoms and a decrease in ESR and CRP.

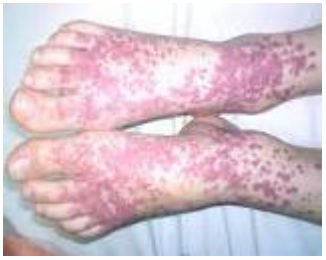

Fig: 1

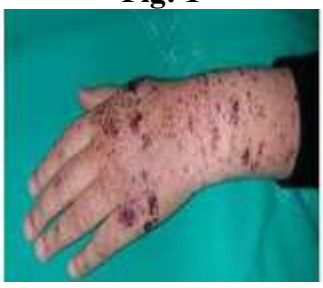

Fig: 2

Figure 1 & 2 showing clinical picture of palpable purpura involvement of upper and lower extremities

Fig: 3

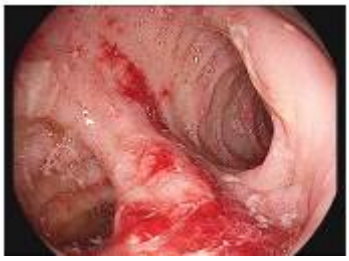

Figure 3 - Endoscopic findings showing inflammation, submucosal hemorrhage and ulceration.

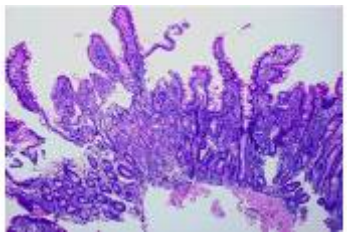

Fig: 4 Small bowel biopsy showing neutrophilic and eosinophilic

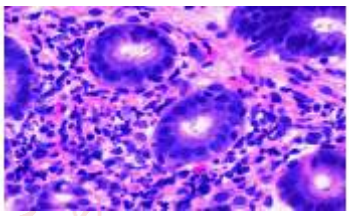

Fig:5 Histopathology of small bowel showing micro abscess infiltrates

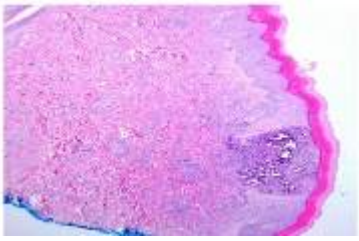

Fig: 6 Histopathology of skin

Discussion

An uncommon inflammatory condition of the small blood vessels (capillaries), Henoch-Schönlein purpura (HSP) is typically a self-limited condition. It causes inflammatory alterations in the tiny blood vessels and is the most prevalent type of juvenile vascular inflammation (vasculitis). Headache, fever, loss of appetite, cramping, abdominal pain, hives, bloody diarrhea, and joint pain are among the symptoms of HSP that typically start out unexpectedly. Typically, red or purple blotches will form on the skin (petechiae). Joints, kidneys, the digestive system, and, in rare instances, the brain and spinal cord can also experience HSP-related inflammatory alterations. Although the precise origin of HSP is unknown, evidence suggests that immune system malfunction may be a contributing factor (i.e., increased IgA immune complexes). It could be challenging to diagnose HSP. Skin lesions and/or joint discomfort,

in addition to laboratory tests, skin biopsies, and other factors are used to diagnose the condition. There is no specific treatment, although the majority of patients experience a short course of the condition, which gives them a fair chance of recovering. Some patients may receive treatment with glucocorticoids (steroid medications) if non-steroid anti-inflammatories are unable to improve symptoms.

Conclusion

With respect to the patient's clinical, laboratory tests and histopathology findings, patient was treated with Intravenous fluids and oral prednisone. The main aim of the treatment is to prevent multiorgan involvement. Prompt diagnosis and multidisciplinary intervention can lead to appropriate management and mitigate potential complications, as illustrated in this case.

Conflict of Interest

None

Funding

None

Consent for publication

Informed consent was obtained from the parents of the patients to publish this case in medical journal.

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