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Case Report

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Navigating the Purpura Puzzle: Insights into Henoch-Schonlein Purpura – A Rare Case Presentation

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Abstract

Henoch-Schonlein Purpura (HSP) also known as IgA vasculitis, is an immune complex-mediated small vessel vasculitis that primarily affects children below 10 years old. Adult onset HSP or IgA vasculitis, is indeed rare compared to childhood onset. While HSP predominantly affects children especially aged 4 to 6, adult cases account only for about 10-20% of all HSP cases. What makes adult-onset HSP particularly noteworthy is not just its rarity but also its more severe clinical course. Renal involvement is significantly more frequent and severe in adults. With up to 50% or more shows signs of nephritis, and some progressing to renal insufficiency. Adults often require more aggressive treatment and have longer hospital stays (average of 10.2 days vs 4.3 days in children). The prognosis is generally less favorable in adults, with slightly lower complete recovery rate (90% in adults vs nearly 99% in children). So, a 26-year-old female presenting with HSP is not only uncommon but also clinically significant due to the higher risk of complications especially renal. The treatment includes intravenous antibiotics and injection dexamethasone and oral prednisolone.

Keywords: Henoch-Schonlein Purpura (HSP); Vasculitis; Nephritis; Arthritis

Abbreviations

HSP: Henoch-Schonlein Purpura; IgA: Immunoglobulin A; GI: Gastrointestinal; ROS: Reactive Oxygen Species; PReS: Pediatric Rheumatology European Society; ACR: Urine Albumin To Creatinine Ratio; ANA: Antinuclear Antibody; C-ANCA: Cytoplasmic Antineutrophil Cytoplasmic Antibody; P-ANCA: Perinuclear Antineutrophil Cytoplasmic Antibody; CRP: C-reactive Protein, ESR: Erythrocyte Sedimentation Rate; PT: Prothrombin Time; INR: International Normalized Ratio; LFTs: Liver Function Tests.

Introduction

Henoch-Schonlein Purpura (HSP) is an Acute Immunoglobulin A (IgA) mediated disorder characterized by generalized vasculitis involving small vessels of the skin, gastrointestinal (GI) tract, kidneys, joints, and rarely the lungs and the central nervous system. The majority of HSP cases are preceded by an upper respiratory tract infection suggesting potential infection, which triggers the formation of immune-complex mediators. Most common pathogens are streptococcus, staphylococcus, and parainfluenza. The immune complex formation enters circulation and deposits in the small vessels of skin, GI tract, joints, and kidneys, which triggers the release of inflammatory mediators like C3a and C5a and recruitment of neutrophils, monocytes, and release of cytokines and reactive oxygen species (ROS). Common clinical manifestations include tetrad of purpura, arthritis, abdominal pain, and nephritis. A typical prodrome of HSP includes headache, anorexia, and fever. The key steps in diagnosing HSP include clinical evaluation, blood tests, urine tests, biopsies, imaging tests, and the Pediatric Rheumatology European Society (PReS) Criteria, if it involves children [1-3].

Case Report

A 26-year-old female with no known comorbidities presents with complaints of loose stools for one day, reddish purple rash over legs, buttocks, and forearms for two days, abdominal pain and vomiting for two days, generalized myalgia with joint pain for one week which was worsening. The patient was apparently normal one week before, after which she developed a fever with a sore throat, which subsided spontaneously within three days. The patient developed joint pain involving wrists, shoulders, and knees for seven days, progressively worsened, non - migratory type with no specific aggravating and relieving factors, no diurnal variation with mild swelling with no small joint involvement. She also complained of generalized myalgia for one week. The patient noticed reddish purple rash (over legs, buttocks, thighs, and forearms) for two days, gradually increased, and was not associated with any itching or tenderness. She also complains of abdominal pain for two days (diffuse, continuous, non- progressive) with 10 episodes of vomiting (non-projectile, non-bilious, and not blood stained). She also has a history of loose stools for one day, 2 episodes, watery, not blood stained [4,5].

Her past medical history was not significant. She has no known comorbidities, not on any regular medications. No history of any drug allergy in the past. Her social and family history was unremarkable. Her menstrual history was normal. Her obstetric history includes P2, L2; normal vaginal delivery, last childbirth was three years ago.

During general examination, patient was conscious, oriented, afebrile with mild dehydration. No pallor, icterus, cyanosis, clubbing, lymphadenopathy, pedal edema. Her blood pressure was 130/80 mmHg. and pulse rate 84/minute, respiratory rate of 18 cycles/minute, and temperature 97.4 F and SpO2: 99% in room air. On the local examination, raised, palpable, non-tender reddish-purple spots present over buttocks, thighs, legs, forearm, and hands [6].



Figure 1: Systemic examination, cardiovascular examination S1, S2 heard; no murmurs heard. Respiratory system, bilateral air entry present. No added sounds. Central nervous system, GCS 15/15. Her abdominal examination showed diffuse mild tenderness on deep palpation at epigastric and umbilical region with no guarding, and no palpable masses.

Her investigations include

Parameter	Result	Normal Range
WBC count	19,590	4-11x109/L
Hemoglobin	11.8	11.5 – 16.5 g/dL
Platelet	3,43,000	150000-400000 cells/cu.mm
Sr. Creatinine	0.6	0.5 – 1.2 mg/dL

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Sr. Urea	12	15-40 mg/dL
CRP	>130	> 0.3 mg/dL
ESR	27	0-20 mm/hr
PT/INR	14.2/1.06	11-13.5s/0.8-1.1
Electrolytes	Normal	
LFTs	Normal	

Table 1: C-reactive protein (CRP), Erythrocyte sedimentation rate (ESR); Prothrombin time (PT); International normalized ratio (INR); Liver function tests (LFTs).

Parameter	Result	Range
Urine RBC	22-24	Less than 4
Urine albumin to creatinine ratio (ACR)	40	Less than 25 mg/g
ANA	Negative	Less than 1:40
C-ANCA	Negative	20 – 25 AU/ml
P-ANCA	Negative	Less than 5 IU/ml
IgA Levels	Normal	61-356 mg/dL
Complement level	Normal	41-90 hemolytic units

Table 2: Urine albumin to creatinine ratio (ACR); Antinuclear antibody (ANA); Cytoplasmic antineutrophil cytoplasmic antibody (C-ANCA); Perinuclear antineutrophil cytoplasmic antibody (P-ANCA); Immunoglobulin A (IgA).

All routine investigations were carried out and found to be normal. The stool occult blood was positive, and her urine RBC was 22-24, which should be below 4. Her urine albumin to creatinine ratio was found to be 40, which was slightly elevated. Her ANA, C-ANCA, P-ANCA were negative. IgA levels and complementary levels were within normal limits. The CT abdomen with contrast showed diffuse wall thickening of D3 and D4 segments of duodenum, proximal and mid jejunal loops with adjacent fat stranding and surrounding enlarged lymph nodes. Features favoring duodenal enteritis and possibility of Henoch - Schonlein Purpura. The upper gastrointestinal endoscopy was done, which showed ? Gastroduodenal manifestation of vasculitis and the presence of hiatus hernia. Histo-pathological examination of duodenal mucosa showed the above findings. The skin biopsy of the left forearm showed features compatible with leukocytoclastic vasculitis [7,8].

Premedication	UPPER GLENDOSCOPY REPORT
Esophagus	· Normal
OG Junction	32 cms, Hiatal opening at 35cms. GEFV Hill Type 2.
Stomach	· ·
Fundus	1 Normal
Body	> Normal
Antrum Biospy(B) taken.	: Erythematous mucosa with haemorrhagic spots seen.
Pylorus	: Normal
	i Normal
D2	r Erythematous nodular friable mucosa. Biopsy(A) taken.
Impression	Hiatus Hernia. Gastro duodenal manifestation of vasculitis.
	Figure 2: Upper GI Endoscopy Report.

		Upper GI Endoscopy
remedication		LV PROPOFOL
sophagus		Normal
G Junction		40CMS
tomach		
Fundus	1	Normal
Body		Normal
Antrum		Normal
Pylorus		Normal
Duodenum		
D1/		
D3		Narrowing noted in D3. Scope could not be passed beyond.
Biopsy		Not taken
Impression		: D3 NARROWING.
		Figure 3: Upper GI Endoscopy.

Course in the Hospital/Treatment:

The patient came with the above-mentioned complaints. On day 1, the patient was symptomatically managed with IV proton pump inhibitors, anti-emetics and IV fluids. Rheumatology opinion was obtained in view of reddishpurple spots over bilateral lower limbs who suspected IgA vasculitis and advised for C-ANCA, P-ANCA, complement levels, IgA levels, and skin biopsy. The results of C-ANCA and P-ANCA were found to be negative. Complement levels and IgA levels were within normal limits. Dermatology opinion was sought in view of skin biopsy and was suggestive of leukocytoclastic vasculitis. As per Rheumatology, the patient was started on Inj. prednisolone 20 mg on day 2 [9].

The patient was treated with inj. Metronidazole 500 mg intravenous with inj. Amikacin 500 mg intravenous, inj. Cepheperazone with Sulbactum 1.5 g intravenous in view of anti-biotic prophylaxis.

On day 4, the patient experienced severe abdominal pain with painful bilious vomiting, for which general surgery and medical gastroenterologist opinion was obtained, as per his guidance CT abdomen with contrast was done, which showed diffuse thickening of the duodenum and jejunum. An upper gastrointestinal endoscopy was also done, which showed erythematous nodular friable mucosa in D2 segment, hiatus hernia, and? gastroduodenal manifestations of vasculitis. The patient started on dexamethasone 8 mg intravenous once a day with cap. On day 6, dexamethasone tapered to 6 mg per day and the patient continued with the other medications as directed. On day 8, dexamethasone dose further tapered to 4 mg once a day. The other medications were continued. On day 9, the antibiotics were stopped. The patient was given inj. Dexamethasone 4 mg, and she was discharged with tab. Wysolone 20 mg [10-12].

Discussion

Henoch-Schonlein Purpura (HSP) is a disease that inflames small blood vessels. The inflammation causes small blood vessels in the skin, intestines, kidney, joints to start leaking. The main symptom is rash in the legs and buttocks. Although people of any age can get HSP, the disease mostly affects children between the ages of 3 and 10. It is more common in boys than girls. Adults with HSP tend to have more severe diseases than children. Usually, IgA vasculitis goes away on its own after 4 weeks. Within 6 months, the disease comes back in about 1/3 of patients, but it usually does not cause any long-term complications. Rarely does it cause kidney disease. So, regular checkups are required [13].

The disease triggers due to genetics or environmental factors or after infections with streptococci, hepatitis B, herpes simplex virus, parvovirus, measles, mumps, rubella, adenovirus, H. pylori, where complexes of IgA and C3 (complement component) are deposited on

arterioles, capillaries, and venules. This eventuality leads to the production of inflammatory mediators like vascular prostacyclin, which play a major role in the development of organ specific clinical manifestations.

The clinical diagnosis is based on the presence of tetrad of symptoms, Purpura (95-100 %) hall mark of the disease, arthritis or arthralgia (60–85%), abdominal pain (35–85%), and renal disease. Purpura, symmetrically distributed over extensor surfaces of lower limbs, buttocks, and forearms. Arthritis/ Arthralgia - oligoarticular with large joints of the lower extremities (knee, ankle, hip) commonly affected. Abdominal Pain, which is diffuse, increases after meals and is associated with nausea and vomiting. Complications include gangrene of bowel; bowel perforation; and massive hemorrhage.Renalinvolvement includes isolated microscopic hematuria; proteinuria; and nephrotic syndrome [11].

Laboratory reports are complementary in assessing renal involvement like urinalysis, urine microscopy, and serum creatinine. Imaging studies are useful in evaluating any abdominal involvement with any potential complications. In patients with incomplete or unusual presentation, biopsy of the affected organ (skin, kidney) confirms the diagnosis. The management includes supportive care, symptomatic therapy, immunosuppressive therapy. Supportive therapy includes management of good hydration, symptomatic pain relief, and monitoring the development of complications [14].

Symptomatic treatment includes NSAIDS for pain management for arthralgia, and arthritis, early glucocorticoid treatment for shortened duration of abdominal pain, and decreased risk of intussusception or surgical intervention. Indications for glucocorticoid therapy are persistent nephrotic syndrome, crescent in more than 50% of the glomeruli, severe abdominal pain, substantial gastrointestinal hemorrhage, severe soft tissue edema, severe scrotal edema, neurological involvement, and intrapulmonary hemorrhage. In this patient, per gastroenterologist and rheumatologists advise, corticosteroid tapering dose was started. Immunosuppressive treatment in HSP nephritis is used in patients with severe renal involvement. Common drugs include azathioprine, cyclophosphamide, cyclosporine, hydroxy chloroquine. But in this patient, there is no kidney involvement, hence no immunosuppressives were started. Regular follow-up of urinalysis, blood pressure measurement, kidney function to avoid worsening renal impairment. In the majority of patient's outcome of HSP is excellent with spontaneous resolution.

Conclusion

HSP is typically a self-limiting small vessel vasculitis with an excellent prognosis in most cases. Management is primarily

supportive, focusing on symptom relief with analgesics with hydration. In moderate to severe presentations, particularly with gastrointestinal involvement, systemic corticosteroids such as dexamethasone or oral prednisolone may be warranted. Most patients recover fully without sequelae; however, vigilant follow-up is crucial, especially in cases with renal involvement, to monitor for potential complications such as persistent hematuria or proteinuria. In this case, histopathological examination of the duodenal mucosa supported the diagnosis, and skin biopsy from the left forearm confirmed the leukocytoclastic vasculitis consistent with IgA - mediated pathology.

Author Contribution

Conceptualization: Dr. Arun Kumar (M.D), Dr. Jayakumar KT (M.D) and Dr. Kumar JS (M.D). **Writing:** Dr. Abhirami Etican and Sujatha Sharmila Govind.

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Patient Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

References

- 1. Sood R, Parekh P, Raj N, Saani I (2022) A Case Report on an Adult Presentation of Henoch-Schonlein Purpura. Cureus 14(6): e26385.
- Kotrashetti VA, Bainade KS, Nair S, Yewale S, Sonawane VB (2020) Henoch-Schonlein Purpura in child: A case report and review of literature. International Journal of Contemporary Pediatrics 8(1): 193-196.
- Sohagia AB, Gunturu SG, Tong TR, Hertan HI (2010) Henoch-Schonlein Purpura A Case Report and Review of Literature. Gastroenterology Research & Practice 2010: 597648.
- 4. Song N, Zhang L, Kou L, Xu J, Li G (2025) A case report of Henoch-Schonlein Purpura in the elderly complicated by multisystem involvement. Medicine 104(1): pe41163.
- 5. Dhanju AS, Narang N, Kaur H, Singh R, Kaur M (2023) Henoch-Schonlein Purpura in adults: A case report. International journal of Advances in Medicine 10(6).
- 6. Camacho LC, Rivera M (2023) Schonlein-Henoch purpura

in adults: Report of four cases. Revista Colombiana de Reumatologia 30(1): 72-77.

- 7. Meltam A, Yasar K, Isiyel E, Bahar B, Fidan K, et al. (2017) Cerebral Vasculitis in Henoch-Schonlein Purpura: A case report. Arch Rheumatology 32(3): 264-267.
- 8. Ruslan LA, Putu DAS (2024) Adult onset Henoch-Schonlein Purpura: A case report. Bali Dermatology Venerology and Aesthetic Journal 7(2).
- 9. Alexander KCL, Benjamin B, Kim FL (2020) Henoch-Schonlein Purpura in children: An updated review. Current Pediatric Reviews 16(4): 265-276.
- Yong YH, Chuang YHL, Wang IC, Huang HY, Gershwin ME, et al. (2008) The immunobiology of Henoch-Schonlein Purpura. Autoimmunity Reviews 7(3): 179-184.

- 11. Almeida JL, Campos LM, Luciana BP, Leone C, Koch VH, et al. (2007) Renal involvement in Henoch-Schonlein Purpura: A multivariate analysis of initial prognostic factors. J Pediatr (Rio J) 83(3): 259-266.
- 12. McPartland K, Wright G (2019) Acute Abdominal Pain: Henoch-Schonlein Purpura Case in a young adult, A rare but important diagnosis. Clinical Medicine 19(1): 77-79.
- 13. Bhimma R, Craig B Longman (2023) IgA Vasculitis (Henoch-Schonlein Purpura). Drugs & Diseases.
- 14. Xuehong W, Yongmei Z, Laiqiang G, Shuyue W, Youyou Z, et al. (2016) Henoch-Schonlein Purpura with Joint involvement: Analysis of 71 cases. Pediatric Rheumatology 14: 20.