Case Report

RARE CASE OF HENOCH–SCHÖNLEIN PURPURA IN A 34-YEAR OLD MALE

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Abstract:
Henoch-Schönlein purpura (HSP), also known as IgA vasculitis, primarily affects children but can also occasionally present in adults. The symptoms are thought to result from the deposition of IgA in the walls of blood vessels within various organs, most commonly the skin, gastrointestinal tract, joints, and kidneys. Here, we report a case of a 34-year-old man who developed severe abdominal pain resembling acute appendicitis two weeks after a viral gastrointestinal infection and administration of cefoperazone-sulbactam antibiotic. The diagnosis of HSP was established based on a progression of symptoms, including severe abdominal pain, arthralgias, melena, and a distinctive non-blanching rash on the trunk and lower extremities. This report will detail the diagnostic workup and treatment approach that resulted in symptom resolution in this unusual adult presentation of HSP.

Key Words: IgA vasculitis, Henoch-Schönlein purpura; adult case; abdominal pain; non-blanching rash; melena

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INTRODUCTION

Immunoglobulin A (IgA) vasculitis is a sudden-onset systemic condition characterized by inflammation of small blood vessels due to the buildup of IgA-containing immune complexes around them and the activation of neutrophils. infectious agents such as viruses or bacteria, as well as certain medications or toxins.[1] The hallmark features of Henoch-Schönlein purpura (HSP) typically consist of four key symptoms: raised purplish skin rashes (palpable purpura (100%), joint discomfort or arthritis (60–75%), abdominal pain and bloody stools (50-65%) and nephropathy (20–55%). The order in which symptoms appear can differ among patients; some may develop the characteristic rash first, while others might experience abdominal pain up to two weeks before the rash emerges. In 10–40% of patients, gastrointestinal manifestations may precede the onset of skin purpura.[2] Abdominal pain in IgA vasculitis is often diffuse, colicky, or angina-like in nature, and may localize to the periumbilical area. It is frequently associated with symptoms such as nausea, vomiting, diarrhea, rectal bleeding, or melena as demonstrated in our case report as well. In certain cases, the clinical picture can resemble an acute surgical abdomen. A fecal occult blood test is recommended to help identify subclinical gastrointestinal involvement.[3] The diagnostic criteria for HSP/IgA vasculitis include the Ankara 2008 classification criteria for IgA vasculitis (IgAV), supported by both the European Alliance of Associations for Rheumatology (EULAR) and the Pediatric Rheumatology European Society (PReS). According to these criteria, the

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presence of purpura is essential for diagnosis, accompanied by at least one of the following: (a) abdominal pain, (b) biopsy-confirmed IgA deposits, (c) joint pain or arthritis, or (d) kidney involvement, such as hematuria or proteinuria. These criteria were assessed using data from 827 patients diagnosed with IgAV and 356 with other vasculitis's, demonstrating a sensitivity of 100% and specificity of 87% in pediatric cases. Initially made for pediatric population, these criteria hold true for adult population as well after numerous trials showing both increased specificity and sensitivity in adult population.[4] Management of milder symptoms is usually symptomatic involving hydration and analgesics. NSAIDs are beneficial in arthralgias but should be avoided in case of renal and gastrointestinal system involvement due to potential worsening of symptoms as demonstrated in our case report as well. In case of severe clinical manifestations especially in adults, glucocorticoids alone or with an immunosuppressive agent are the first choice of treatment. They are especially beneficial during the early and acute phases of the disease, owing to their potent anti-inflammatory properties and quick therapeutic effect. However, the effectiveness of these treatment approaches remains a subject of ongoing debate, with no clear consensus established.[5] This case report underscores the uncommon presentation of Henoch-Schönlein purpura (HSP) in an adult male, a condition that predominantly affects the paediatric population. It also illustrates how the patient's severe abdominal pain, initially suggestive of acute appendicitis on ultrasound, was accurately diagnosed through appropriate investigations. Prompt initiation of moderate-dose corticosteroid therapy led to a successful resolution of symptoms.

CASE SUMMARY

A 34-year-old male (identifier withheld), presented on 5 August 2025 with a 10-day history of fever, abdominal pain, palpable purpura, and melena. The illness began on 26 July 2025, when he developed diffuse colicky abdominal pain after consuming shawarma and a soft drink. The pain persisted despite oral and intravenous analgesics and empiric antibiotics. Over the following days, the pain localized to the right iliac fossa with rebound tenderness, leading to a provisional diagnosis of acute appendicitis. On 3 August, he developed melena, and by 5 August, multiple palpable non-blanching purpuras appeared on his lower limbs, prompting hospital admission. He also reported fever, arthralgias, myalgias, nausea, and vomiting. His past history included a recent viral illness and two unspecified "SUM" injections prior to symptom onset. During hospitalization, proteinuria, microscopic hematuria, and a decline in GFR were noted, consistent with renal involvement. On admission, the patient was alert and oriented with a Glasgow Coma Scale (GCS) of 15/15. His vital signs were stable, with blood pressure of 118/76 mmHg, pulse 88 bpm, temperature 37.2 °C, and normal oxygen saturation. Examination revealed multiple palpable purpuric lesions on both lower limbs (*Figure 1 shows purpura on the patient's feet; Figure 2 shows purpura on the back; Figure 3 shows purpura on the arm*). The abdomen was soft but tender in the right iliac fossa, with normal bowel sounds and no guarding or rigidity. Cardiovascular and respiratory examinations were normal, with audible S1 and S2 and vesicular breath sounds, respectively. Neurological examination showed no focal deficit. Laboratory evaluation revealed markedly elevated inflammatory markers with CRP >100 mg/L, lactate 10.4 mmol/L, and D-dimer >100, while procalcitonin was normal. Liver and renal functions were initially preserved, and electrolytes were within normal limits. Immunological studies showed normal IgA (207 mg/dL) and anti-dsDNA elevated normal levels (51 IU/mL), with normal rheumatoid factor, C3, and C4 levels. HbA1c was 6.2%. Stool microscopy revealed numerous red blood cell 12 pus cells per high power field, and was positive for occult blood. CT abdomen suggested possible mesenteric venous involvement, but a mesenteric

venogram demonstrated patent superior mesenteric, portal, and inferior mesenteric veins, excluding thrombosis. Renal investigations later showed reduced GFR with proteinuria and hematuria. Based on the clinical trial of abdominal pain, gastrointestinal bleeding, and palpable purpura, supported by elevated IgA and renal involvement, a diagnosis of IgA vasculitis (Henoch–Schönlein purpura) was established. Initial management included intravenous tramadol and Toradol for analgesia, metoclopramide for nausea, and proton pump inhibitor infusion. Broad-spectrum antibiotics were commenced, including intravenous meropenem paracetamol IV and metronidazole (500 mg TDS). Owing to refractory abdominal pain, persistent melena, renal involvement, and systemic symptoms including arthralgias, myalgias, nausea, and vomiting, corticosteroid therapy was initiated with intravenous methylprednisolone (125 mg IV stat, followed by 80 mg IV BD). The regimen was subsequently tapered and switched to oral prednisolone (Deltacortil 5 mg, 6 tablets BD). Following steroid therapy, the patient demonstrated gradual improvement, with resolution of abdominal pain, reduction of purpura, stabilization of gastrointestinal bleeding, and improvement in renal function.

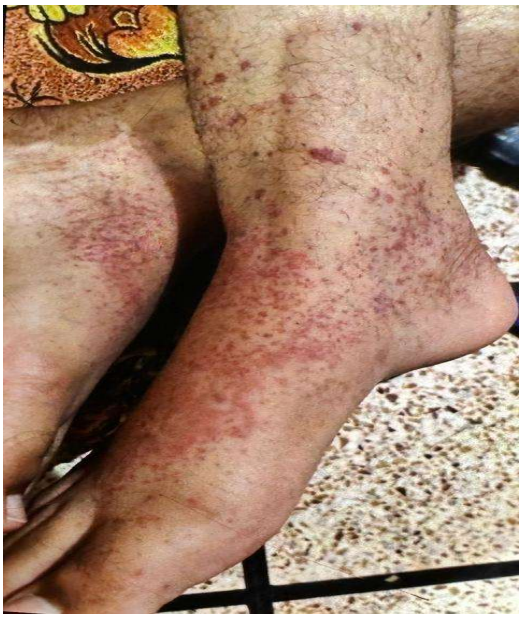

Figure 1: Purpura on patient's feet

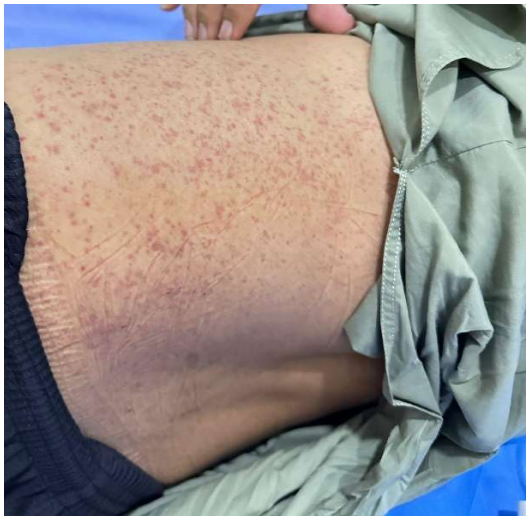

Figure 2: Purpura on patient's back

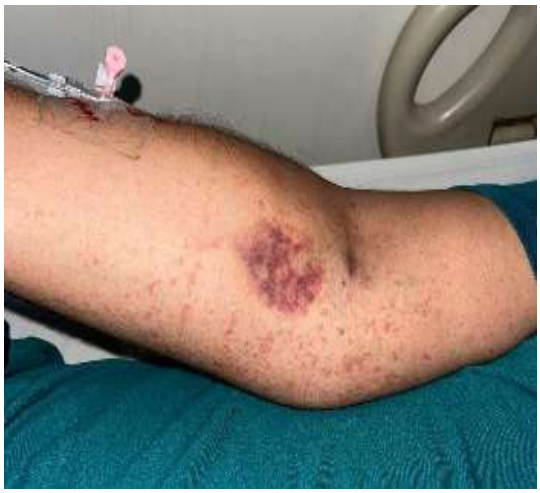

Figure 3: Purpura on patient's arm

DISCUSSION

IgA vasculitis, previously known as Henoch–Schönlein purpura (HSP), is a small-vessel vasculitis predominantly seen in children, with a frequency of 3–26.7 per 100,000, while adult cases are relatively uncommon (0.1–1.8 per 100,000). [1,3,4,5] In adults, the disease is usually more severe with poorer outcomes, as 20–80% [2] develop glomerulonephritis (IgAVN)[5], and 30–40% may progress to end-stage kidney disease (ESKD) within 20–30 years of symptom onset.[3,5] Our patient, a 34-year-old male, presented with the classic tetrad of gastrointestinal involvement (abdominal pain

and melena), palpable purpura as shown in *Figures 1-3*, arthralgia, and renal involvement, which developed during hospitalization. In adults, the frequency of these features is as follows: palpable purpura in 100%, arthralgia in 60–80%, gastrointestinal manifestations in 50–75%, and renal involvement in 40–85% of cases.[3] This presentation aligns with the diagnostic frameworks of the Ankara Criteria 2008, American College of Rheumatology (ACR, 1990), the European League Against Rheumatism/Pediatric Rheumatology International Trials Organization/Pediatric Rheumatology European Society (EULAR/PRINTO/PRES, 2010), and the revised Chapel Hill International Consensus (2012).[2,3,4,5] Although originally developed for children, these criteria remain valid in adults, as no specific adult criteria exist. The EULAR/PRINTO/PRES classification is generally preferred, with 12.4% higher sensitivity and 5% higher specificity compared to the ACR criteria.[3,5] A notable feature in this case was the atypical sequence of symptom onset. The patient initially experienced diffuse abdominal pain localizing to the right iliac fossa, closely mimicking acute appendicitis. Gastrointestinal symptoms may precede cutaneous manifestations in up to 40% of cases[2], often creating a diagnostic dilemma and risking unnecessary surgical intervention. The differential diagnoses include HSR vasculitis, cryoglobulinemic vasculitis, microscopic polyangiitis, and polyarteritis nodosa.[3] The later appearance of melena, palpable purpura, and proteinuria helped confirm the diagnosis, underscoring the importance of considering IgA vasculitis in adults with unexplained abdominal pain and gastrointestinal bleeding. Multiple agents have been implicated as triggers for IgAV. Drugs such as quinolones, fluoroquinolones (levofloxacin, ofloxacin, ciprofloxacin), ACE inhibitors, angiotensin II receptor antagonists (losartan), clarithromycin, and some NSAIDs have been reported.[1] Infectious triggers include *Parvovirus*, *Hepatitis B*, *Parainfluenza virus*, *Influenza virus*, *Adenovirus*, *Respiratory Syncytial Virus (RSV)*, Coronavirus, *Helicobacter pylori*, Group A *Streptococcus*, *Haemophilus parainfluenzae*, and *MRSA*.[1,2,5] The most common association is with upper respiratory tract infections, which stimulate IL-6 and alter IgA glycosylation, leading to galactose-deficient IgA1 and subsequent immune complex deposition in small vessels and the renal mesangium.[3,5] Genetic predisposition is also important [2], with associations reported for HLA-DQA1, HLA-DQB1, HLA-DRB1[1,3], and HLA-B41:02.[5] Incidence varies by ethnicity, being higher in Asians compared to Caucasians, and lowest in Africans, Caribbeans, and South Asians. Familial aggregation further supports a genetic component.[5] In this patient, a recent viral illness was the likely trigger. Renal involvement emerged during hospitalization, with proteinuria, microscopic haematuria, and reduced eGFR. Persistence of skin lesions and melena for more than two months is considered a risk factor for glomerulonephritis;[3] however, IgAVN developed in this patient within a week of the classical triad. Histologically, renal biopsies in IgAVN and IgA nephropathy (IgAN) are indistinguishable, both showing mesangial IgA deposition.[3,5] While microscopic hematuria may also occur in nephritic syndrome, nephrotic syndrome, or renal failure, the clinical context and biopsy findings confirm IgAVN.[3,5] Children rarely progress to CKD and are often diagnosed on clinical grounds without histopathology.[3,5] The absence of crescents and glomerular sclerosis in the biopsy favored a good prognosis.[3] Early diagnosis and treatment prevented disease progression, resulting in complete remission.[1] Generally, the short-term prognosis of IgAV depends on gastrointestinal involvement, while long-term outcomes are determined by renal disease.[3] Diagnosis remains clinical, as no confirmatory test exists.[5] In adults, evaluation relies on the 2010 EULAR/PRINTO/PRES pediatric criteria.[5] Other causes of purpura should be excluded through platelet counts and coagulation studies.[1,5] Renal function tests (serum creatinine, eGFR) and urine studies (hematuria, dysmorphic RBCs, RBC casts,

proteinuria quantification) are recommended.[5] Renal biopsy is indicated for impaired renal function, persistent proteinuria >1 g/day despite RAAS inhibitors, nephrotic/nephritic syndrome, or rapidly progressive glomerulonephritis.[5] A skin biopsy typically shows leukocytoclastic vasculitis in postcapillary venules, and direct immunofluorescence within 48 hours of onset demonstrates IgA and C3 deposition.[2,5] However, IgA deposits are not pathognomonic, as they may occur in other vasculitis's, and serum IgA is neither diagnostic nor prognostic despite possible elevation.[2,5] Importantly, IgAV in adults carries a stronger association with malignancies, particularly solid tumors (lung, prostate) and hematological cancers, necessitating age- and sex-appropriate cancer screening.[2,5] Several histological classifications exist: ISKDC (for children only), Oxford 2009 (MEST-C system with clinical risk factors), Haas (linking IgA subclass to renal survival), and the Semi-Quantitative Classification (SQC) by Koskela, 2017 (scoring glomerular, tubular, interstitial, and vascular biopsy findings).[5] Among these, the SQC offers the best predictive value, followed by the Oxford system.[5] Treatment aligns with KDIGO 2021 guidelines, which recommend symptomatic management.[2,5] The patient received comprehensive supportive care, including analgesics (IV tramadol, ketorolac), antiemetics (metoclopramide), PPIs for gastrointestinal protection, and antibiotics (IV meropenem, metronidazole) for infection coverage. Given the severity of his presentation persistent abdominal pain, gastrointestinal bleeding, systemic symptoms, and renal involvement glucocorticoids were initiated. Intravenous methylprednisolone followed by oral prednisolone with a gradual taper led to rapid improvement in purpura, abdominal pain, gastrointestinal bleeding, and renal function. Although evidence for steroids in altering long-term renal outcomes remains inconclusive, their role in acute severe disease is supported by clinical reports, particularly for rapid symptom relief.[1,2,3,5] This case underscores the importance of recognizing IgA vasculitis in adults presenting with abdominal pain and gastrointestinal bleeding, as this awareness may prevent unnecessary surgical intervention. It also highlights potential triggers such as viral infection and prior antibiotic exposure, consistent with reports of infections and medications precipitating IgAV in genetically predisposed individuals.[1,5]

CONCLUSION

This case emphasizes the diagnostic and therapeutic challenges of adult-onset IgA vasculitis. Clinicians should maintain a high index of suspicion in adults with abdominal pain and gastrointestinal bleeding, particularly when followed or accompanied by purpura. The atypical sequence of symptoms in our patient, with abdominal pain and gastrointestinal bleeding preceding cutaneous manifestations, mimicked acute appendicitis and posed a diagnostic dilemma. Early recognition, thorough investigation, and prompt corticosteroid therapy were crucial in preventing complications and achieving remission. While corticosteroids are effective for acute systemic and renal manifestations, long-term renal monitoring remains essential. Early intervention in adults with unexplained abdominal pain and purpura can significantly improve prognosis.

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AUTHOR'S CONTRIBUTIONS

OF: Concept, Article Writing
IMI: Abstract, Introduction
IMR: Case Description
MOR: Data Collection
AA: Data Collection
ZA: Data Collection
FA: Data Analysis, Critical Approval

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