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Clinical Spectrum of Adult Henoch-Schönlein Purpura: Diagnostic Challenges and Long-Term Prognostic Considerations

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Henoch-Schönlein Purpura, IgA Vasculitis, Diagnosis, Initial Management

Abstract

Immunoglobulin A vasculitis (IgAV) or Henoch–Schönlein purpura (HSP) is an immune complex-mediated systemic vasculitis that primarily affects small blood vessels in the skin, joints, gastrointestinal tract, and kidneys. Although more common in children, IgAV in adults tends to present with more severe symptoms and a higher risk of long-term kidney disease. IgAV is diagnosed based on clinical and histopathological criteria, with emphasis on non-thrombocytopenic palpable purpura, arthralgia/arthritis, abdominal pain, and/or kidney involvement, along with findings of leucocytoclastic vasculitis with IgA deposition on biopsy. Case Illustration: This case report describes a 20-year-old woman with palpable purpura on the lower legs and hands, arthralgia, a history of prior sore throat, and bronchial asthma with markedly elevated IgE. Laboratory findings showed elevated ESR and CRP; urinalysis was negative; skin biopsy revealed leucocytoclastic vasculitis. The patient was taking hormonal medication, methylprednisolone, and loratadine. This case demonstrates a typical presentation of IgAV in young adults, supported by a history of previous upper respiratory tract infection and atopic predisposition (bronchial asthma with high IgE). Clinical manifestations of palpable purpura on the extremities and arthralgia are highly consistent with IgAV. The leucocytoclastic vasculitis findings on skin biopsy are highly suggestive, although confirmation of IgA deposition remains ideal for definitive diagnosis. Management with corticosteroids and antihistamines aims to alleviate skin and joint inflammation. Long-term prognosis depends on close monitoring for potential development of IgAV nephritis.

INTRODUCTION

A 20-year-old woman came to the Immune Polyclinic with complaints of redness on her feet and hands. Redness complaints were first felt 2 weeks ago (15/04/2025). Initially, reddish spots appeared around the thighs. The next day the redness on the skin widened and increased in the legs and hands (Mistry et al., 2015; Organization, 2018). Complaints of redness on the body, stomach and head there are no similar complaints. The patient had previously tried to go to the GP practice and there was no improvement. On 4/21/25, the patient complained of stiffness, pain and swelling in the soles and ankles. Complaints of swelling and stiffness improve after soaking in warm water. In addition, a bluish bruise appears in areas that experience redness and subsequently turn faded and yellowish (Kostadinova-Petrova et al., 2017; Urakov, 2020). On 18/4/25, the patient experienced bumps in the arm after doing activities and sweating. The complaints improved after taking allergy medication (setirizin).

In early April, the patient had a strep throat and went to a general practitioner.

Riw. Previous Illness :

- Riw. Hypertension or Diabetes does not exist
- Riw. Heart disease is absent

- Riw. Systemic or autoimmune diseases are absent

Riw. Family :

- Parent (Mother) has a history of asthma
- Riw. Autoimmunity in the family is absent

Riw. Drug Use:

- Using hormonal drugs from an obgyn doctor because of menstrual disorders that have been consumed since 2 weeks
- Anti-inflammatory drugs (methylprednisolone) and allergy medications (loratadine) from an internal medicine doctor

Riw. Allergies :

- Food allergy : Seafood
- Drug allergy : None

Riw. Atopi :

- Asma Bronchial (+)

Riw. Social :

- A student

OBJECTIVES

Vital Signs :

Blood Pressure : 114/61
Pulse : 84 x/min
Pernapasan : 20 x/min
Temperature : 36.8 C
SpO2 : 98 % Room Air

Physical Examination :

Feed : Anemia -/- , Ichterik -/-
Cor: S1S2 tunggal, 314erivas, murmur (-)
Pulmo : Vesikular +/+, Rh ---/---, Wh ---/---
Abdomen : BU (+) Normal, Distension (-), Pressure pain (-)
Extremities : Warm +/+, CRT <2s, Edema --/--, Erythema -+/-+

LABORATORY EXAMINATION

Complete Blood :

WBC : 10.53 x 103/µL
NE# : 5.5 x 103/µL
LY# : 4.05 x 103/µL
MO# : 0.74 x 103/µL
EO# : 0.19 x 103/µL
BA# : 0.05 x 103/µL
Hb : 12.4 g/dl
HCT : 42 %
Thrombositis : 333 x 103/µL
LED : 68.0 mm/jam

CRP : 7.7 mg/L
Total IgE : 552.05 KUI/L
Kidney Function:
GOOD : 16.3 mg/dl
Creatinine : 0.81 mg/dl
eGFR : 105.29 ml/min/1.73 m2
Liver Function :
SGOT : 24
SGPT : 20
Viral Marker :
HbSAg : Non Reaktif
Anti HCV : Non Reactive
Urinalisa:

Specific Gravity	: 1.025
Turbidity	: Clear
pH	: 5.0
Leukosit	: Positive (++)
Nitrite	: Negative
Protein	: Negative
Glukosa	: Negative
Keton	: Negative
Blood	: Negative
Urobilinogen	: Negative
Bilirubin	: Negative
Color	: Yellow
Sedimen Urine	
Leucositol Sedimen	: 11
Erythrosit Sedimen	: 1
Sel Epitel Sedimen	: 18
Silinder	: 0.85
Bacteria	: 3196.3

PA Examination:

- Microscopy :
  The preparation of a piece of skin tissue consists of the epidermal layer, the dermal layer and subcutaneous fat.
  The epidermal layer contains keratinocytes. The dermis layer appears to contain mild perivasculous inflammatory cells in the form of lymphocyte cells, PMN neutrophils and some eosinophils in perivascular. It appears that the debris tissue is characterized by erythrocyte extravavaation.
- Conclusion :
  The morphological picture shows that the epidermis contains keratinocytes. The dermis contains inflammation of lymphocytes, PMN neutrophils and some perivascular eosinophils. Debris tissue with erythrocyte extroveration is visible.
  DD/ *Leukocytoclastic vasculitis*

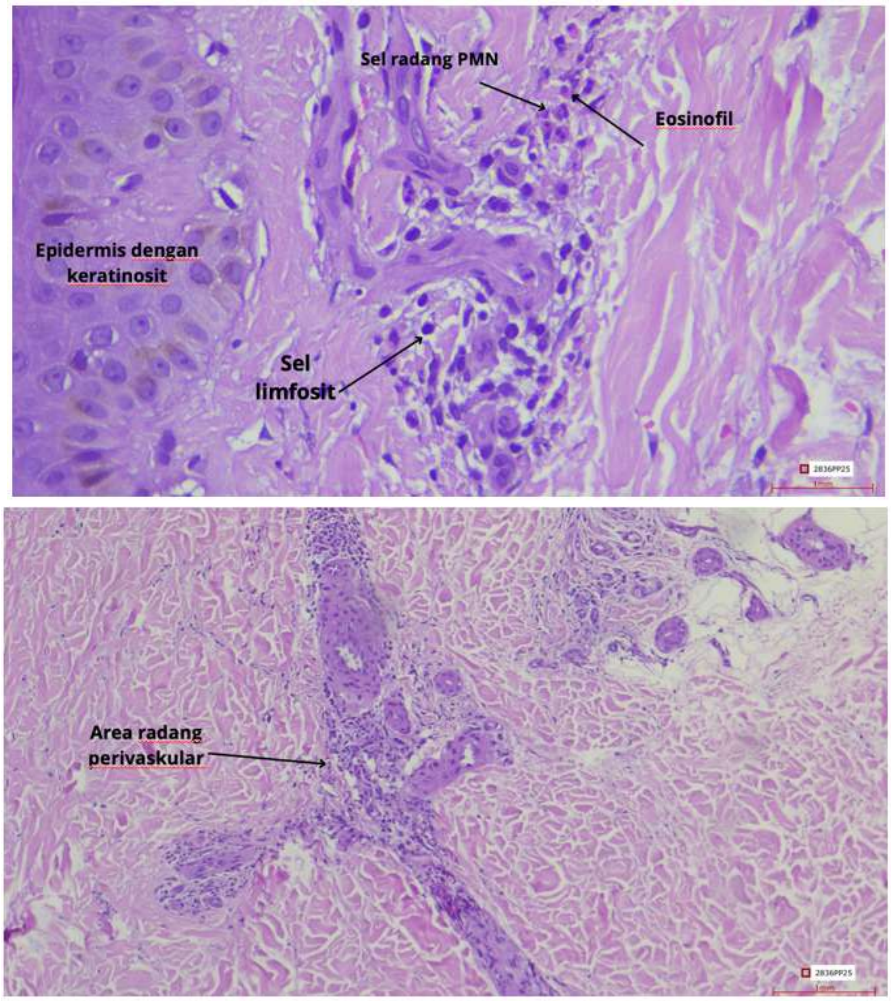

Figure 1. Microscopic histopathology image of skin biopsy
Source: Hospital Pathology Documentation, 2025

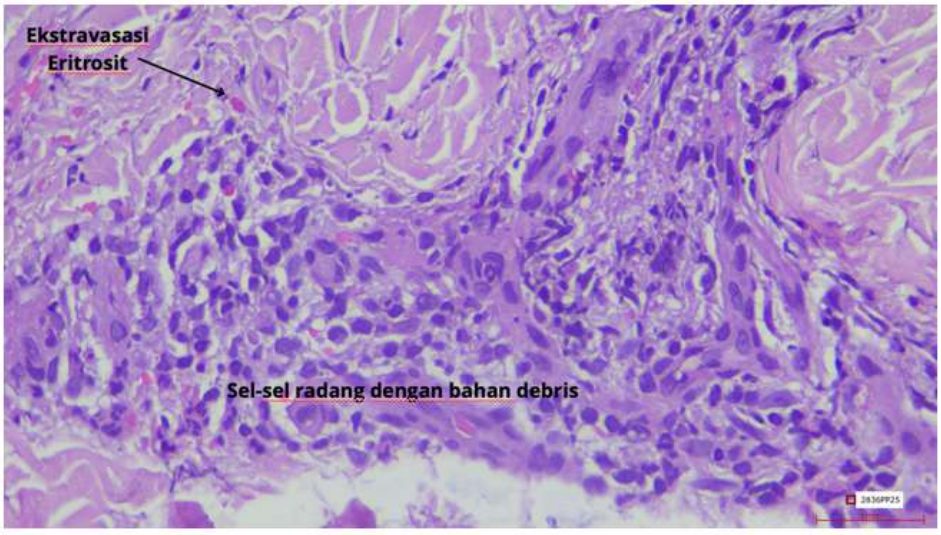

Figure 2. Additional microscopic histopathology image
Source: Hospital Pathology Documentation, 2025

Clinical Photo:

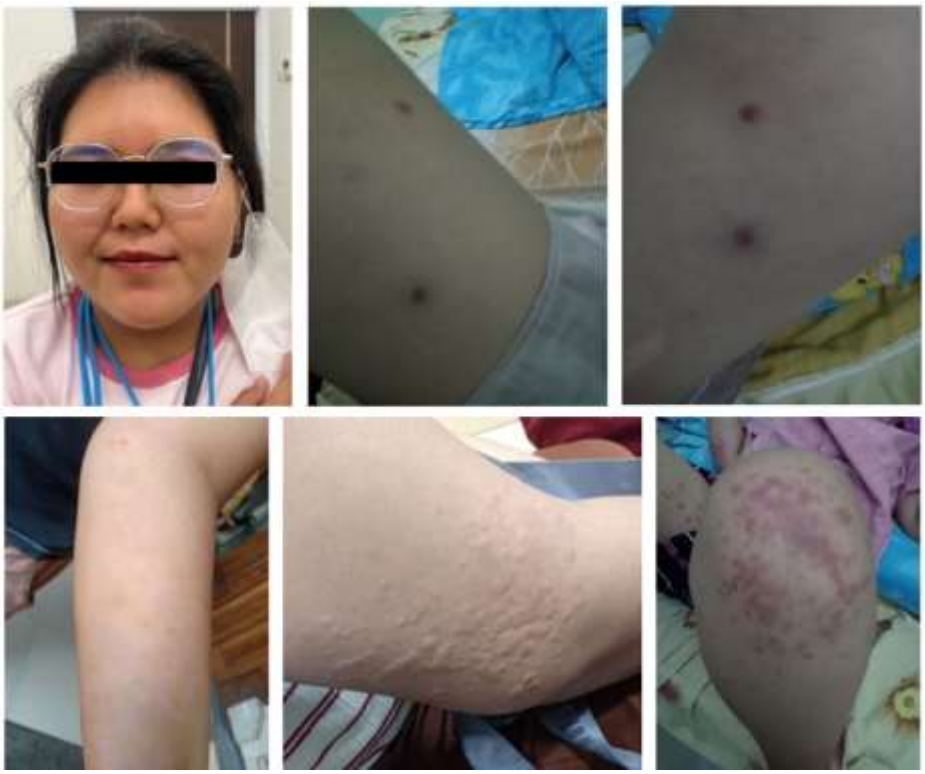

Figure 3. Clinical Photo (Palpable purpura on the patient's extremities
Source: Hospital Pathology Documentation, 2025

This case report aims to describe the clinical spectrum, diagnostic challenges, and long-term prognostic considerations of adult Henoch-Schönlein purpura through a detailed presentation of a 20-year-old female patient with typical cutaneous and articular manifestations (Ali, 2018; Koné-Paut et al., 2017; Mahmoud et al., 2016; Maritsi, 2017). The study also highlights the importance of skin biopsy in supporting the diagnosis and emphasizes the need for close monitoring of renal involvement given the higher risk in adults. The findings of this case report provide practical benefits for clinicians, particularly in primary care and internal medicine settings, by increasing awareness of the typical presentation of IgAV in young adults, aiding in early recognition, and guiding appropriate initial management. Furthermore, this report contributes to the existing literature by illustrating the interplay between atopic predisposition (bronchial asthma with high IgE) and preceding upper respiratory tract infection as potential triggers, thereby enriching the understanding of IgAV pathogenesis in adult populations.

METHOD

The research method used in this paper is a descriptive method with a case report approach. The study was conducted through clinical observation of a 20-year-old female patient who experienced complaints of redness in the legs and hands accompanied by pain, stiffness, and swelling of the joints. Data were obtained from anamnesis, physical examination, laboratory examination, and supporting examinations in the form of skin biopsy. Anamnesis is carried out to explore the history of the course of the disease, history of previous infections,

history of comorbidities, and the use of medications. The physical examination is focused on the identification of dermatological manifestations in the form of palpable purpura on the extremities. Laboratory tests include blood precipitation rate (LED), C-reactive protein (CRP), total IgE levels, and urinalysis to assess systemic involvement. The diagnosis was established based on histopathological results of skin biopsy showing a picture of leukocytoclastic vasculitis (Leukocytoclastic Vasculitis/LCV) with inflammatory cell infiltration and erythrocyte extravasation. All data were analyzed descriptively to describe clinical manifestations, risk factors, examination results, and patient management comprehensively.

RESULTS AND DISCUSSION

Definition and Risk Factors

Immunoglobulin A vasculitis (IgAV), formerly known as Henoch-Schonlein Purpura (HSP), is an immune complex-mediated systemic vasculitis that primarily affects small blood vessels in the skin, joints, gastrointestinal tract, and kidneys. The condition is characterized by the deposition of the dominant immune complex IgA in the walls of blood vessels (Castañeda et al., 2024; Luthfia Wardhani & Ariffatus Saroh, 2023). Although IgAV is the most common vasculitis in children, with an annual incidence of about 10 to 20 per 100,000 children under 10 years of age, the condition can also occur in adults and adolescents. In adults, IgAV tends to have a more severe presentation and a higher risk of causing long-term kidney disease (Roache-Robinson et al., 2025).

IgAV is generally characterized by clinical tetrads: non-thrombocytopenic palpable purpura, arthralgia/arthritis, abdominal (GI) pain, and renal involvement (hematuria/proteinuria). A typical histological picture is leukocytolastic vasculitis of small blood vessels with IgA deposition (and C3 complement components) on immunofluorescence. Therefore, the term "IgA vasculitis" more accurately describes its immunological pathogenesis (Castañeda et al., 2024).

Historically, the 1990 ACR criteria required purpura plus ≥2 from: age ≤20 years, intestinal/abdominal sounds, arthralgia, and histological findings of LCV (Hočevar et al., 2016). However, today the 2010 EULAR/PRINTO/PRES criteria are widely used: palpated purpura (dominant limb) must be accompanied by ≥1 of abdominal pain, arthralgia/arthritis, histological features of LCV and IgA, or evidence of renal (hematuria/proteinuria). The adult EULAR criteria also showed a sensitivity of 99–100% and a specificity of ~86–87%. The diagnosis of IgAV is both clinical and histopathological picture, but confirmation of IgA deposition via biopsy immunofluorescence is necessary for diagnostic certainty, as pure LCV can occur in other vasculitis (Castañeda et al., 2024).

This report discusses the case of a 20-year-old female patient who was diagnosed with suspicion of Henoch-Schonlein Purpura (IgA Vasculitis). The patient comes with a major complaint of redness in the feet and hands that has lasted for two weeks, accompanied by stiffness, pain, and swelling in the soles and ankles. The dominant palpable purpura on the lower limbs and hands, initially in the form of reddish spots that then spread and turn bluish like bruises, then fade to yellowish. This is a classic skin manifestation of IgAV, which is found in almost all patients (98-100%). Clinical images of patients strongly support these findings. Complaints of stiffness, pain, and swelling in the soles and ankles that improve with warm

water immersion are very consistent with arthralgia that occurs in 60-80% of IgAV patients. Joint involvement in IgAV is transient and does not cause permanent joint damage.

IgAV most affects children with an annual incidence of 3–26 cases/100,000 children per year, with a peak at 6 years of age. In adults, the incidence is much lower (~0.1–2/100,000 person-years) with a male:female ratio of approximately (1.5:1) (Castañeda et al., 2024; Reamy et al., 2020). In adult patients, the average onset ranges from 32–50 years. A large survey of the adult population in Slovenia even shows that IgAV is less rare than previously thought, with incidents 3–6 times greater than old reports (Hočevar et al., 2016).

Several risk factors and triggers have been identified. Upper respiratory tract infections (often caused by group A Streptococcus) or gastrointestinal infections usually precede the onset of IgAV. In pediatric studies in Taiwan, ~75% of HSP cases occurred after respiratory or GI infections. In addition, immunizations, medications (e.g., antibiotics, NSAIDs, and certain hormone therapies), or exposure to allergens are often reported as triggers. It is noteworthy that this patient had a history of strep throat infection a few weeks before the onset of purpura, according to the pattern of infection triggers. Cohort studies also show there is a genetic predisposition: HLA-KRIR polymorphisms (e.g., HLA-A03, B37) are associated with IgAV risk in some populations (Heineke et al., 2017). These cases strongly support the multifactorial etiology of IgAV. A history of strep throat a few weeks prior to the onset of purpura is strongly consistent with data that upper respiratory tract infections (especially by Streptococcus) are common triggers of IgAV. This infection may trigger an increased mucosal IgA response.

Atopic or allergic elements also appear as predisposing factors. A large population study in Taiwan showed that children with a history of asthma, allergic rhinitis, or atopic dermatitis have almost twice the risk of developing HSP compared to non-atopy (Chen et al., 2016). These data support the hypothesis that the disease is often triggered by an excessive allergic/mucosal immune response. These data support the hypothesis that the disease is often triggered by an excessive allergic/mucosal immune response. In this case, the 20-year-old patient had chronic bronchial asthma with a very high total IgE (~552 kUI/L) and the birth mother also had asthma. This atopic condition increases the vascular predisposition of IgA. The combination of infection history and atopic background supports the immunological relationship between allergic response and IgAV pathogenesis in patients today.

Pathogenesis of IgA vasculitis

IgAV pathogenesis involves the formation of excess IgA1 immune complexes and their deposition on the walls of small blood vessels. IgA vasculitis is an immune-complex disease of the IgA class. Basically, mucosal B cells produce galactose-deficient IgA1 (Gd-IgA1) due to glycosylation abnormalities or mucosal antigen stimulation. This IgA1 circulates and is stimulated to form autoantibodies (usually IgG) against the Gd-IgA1 epitope. Antibodies–antigens bind to form the IgA1–IgG immune complex. This immune complex circulates in the blood until it passes through the liver's cleansing system. Finally, the excess of this immune complex settles in small blood vessels, specifically the dermal capillaries, the renal glomerulus, and the gastrointestinal mucosa (Castañeda et al., 2024; Song et al., 2021).

It is this dominant IgA deposition that triggers complement activation. The IgA1 chain is able to activate alternative complement pathways and mannan-binding lectins. An increase in C3a, C5a, and terminal complement (C5b-9) fragments was found in IgAV skin and kidney

lesions, and their levels correlated with disease activity. Such complement proinflammatory derivatives act as chemokins attracting neutrophils and other inflammatory cells to the site of complex deposition (Castañeda et al., 2024). Activated neutrophils release proteolytic enzymes (collagenase, elastase) and inflammatory mediators (leukotrienes, IL-8) that cause endothelial damage and local vasculitis. The microscopic results are a picture of leukocytoclastic vasculitis (LCV): perivascular neutrophil infiltrates, nuclear dust, and erythrocyte extravasation. On immunofluorescence examination, granular deposits of IgA and C3 appear on the walls of small capillaries, an important confirmation for the definitive diagnosis of IgAV (Castañeda et al., 2024).

In addition to the immune complex, the role of T lymphocytes, pro-inflammatory cytokines (IL-6, IL-8, TNF-α), and mast cells also contribute, in particular to skin manifestations. In patients with this type of atopic, high allergic mediators (histamine, leukotriene cys-LT) can increase vessel permeability and worsen purpura (Chen et al., 2016). The disease also has a genetic linkage: some studies show associations with MHC class II genes and other hereditary factors (Heineke et al., 2017). Overall, IgAV is a complex immunological disease: infection or environmental antigens trigger anomalous IgA production, IC formation, complement activation, and systemic vascular inflammation (Castañeda et al., 2024).

Diagnosis of IgA Vasculitis in Adult Patients

The diagnosis of IgA Vasculitis (IgAV) is established on the basis of internationally agreed classification criteria, such as the criteria EULAR/PRINTO/PRES (Ruperto et al., 2010). This criterion requires the presence of palpable purpura or petekie (mandatory criteria) with a predominance in the lower limbs, plus at least one of four additional clinical manifestations: acute abdominal pain, histopathological findings indicating leukocytolastic vasculitis with dominant IgA deposition, arthritis or arthralgia, or kidney involvement (Ruperto et al., 2010).

Typical manifestations of IgAV (HSP) involve four main systems: the skin (palpated purpura), joints (arthritis/arthralgia), gastrointestinal tract, and kidneys (Castañeda et al., 2024). Almost all patients (98–100%) exhibit symmetrical palpable purpura in the hanging area (lower legs, buttocks, forearms). The following image shows the classic purpura skin lesion on the lower limb that the patient also experienced (redness that turns bluish and yellowish). These lesions initially appear in the form of red patches or urticaria that quickly become ptecic papules and striped purpura, especially on the extensors of the legs and buttocks. In this patient, the purpura is palpated with the dominance of the lower limbs (legs) and hands according to the general pattern. No similar rashes were found on the body or abdomen, consistent with the generality of distribution on the extremities. Skin lesions can be itchy or painful; in this case, a bruise-like blue color is seen that then fades (resolving phase), typical of the course of IgAV purpura in ~10 days (Castañeda et al., 2024; Reamy et al., 2020).

Arthralgia and arthritis are found in approximately 60–80% of IgAV patients. In children, joint complaints are often knees and ankles; adults are also more likely to involve the large joints of the lower extremities. It should be noted that NSAID therapy is commonly used for IgAV joint pain in the absence of GI/kidney involvement (Castañeda et al., 2024), but in these cases the patient is already taking oral corticosteroids that may also relieve arthralgia.

Abdominal pain (GI colic) or gastrointestinal manifestations occur in ~50–75% of IgAV patients. Complaints include colic abdominal pain, nausea, vomiting, and even gastrointestinal bleeding. In adult patients, abdominal complaints are more common than in children (Castañeda et al., 2024). This case does not experience abdominal pain or vomiting at all. Physical and laboratory examinations support the absence of GI involvement, so skin-articulation symptoms alone are dominant. This is not surprising because not all IgAV patients should have GI symptoms. Clinical reviews confirm that the absence of abdominal pain does not eliminate the diagnosis, especially since the criteria requirement requires only one manifestation other than purpura (Hočevar et al., 2016).

Renal involvement (IgA vasculitis nephritis) usually arises after initial manifestations, and is characterized by hematuria (microscopic or macroscopic) and/or proteinuria. Approximately 40–85% of adult patients have urinary manifestations (in contrast to ~30–40% of children) (Castañeda et al., 2024). The most common is transient microscopic hematuria (RBC casts are sometimes present). In this case, the initial urine test showed negative blood and protein (only 1 sedimentary erythrocyte cell). Thus, at the initial examination, there was no IgAVN. Platelet count is normal (333×103/μL) so that purpura is not due to thrombocytopenia. These findings support the diagnosis of IgAV with minimal or unemergent renal involvement, improving the long-term prognosis. There is also mild leukocytosis and increased LED (68 mm/h) which reflects an acute inflammatory process. Serum IgA levels are often not diagnostic reliable, and although they do rise frequently, they are not specific (Castañeda et al., 2024).

In these cases, the patient showed predominantly palpable purpura of the legs and hands, which met the mandatory criteria for the diagnosis of IgAV (Ruperto et al., 2010). In addition, complaints of stiffness, pain, and swelling of the soles and ankles indicated the presence of arthralgia, which is one of the additional criteria in favor of the diagnosis of IgAV (Roache-Robinson et al., 2025). The patient's skin biopsy results showed a picture of leukocytolastic vasculitis (LCV), which is consistent with the histopathological picture found in IgAV (Roache-Robinson et al., 2025).

However, it is important to emphasize that although the clinical and histopathological picture of LCV is highly suggestive, the definitive diagnosis of IgAV requires confirmation of IgA deposition on the blood vessel wall through direct immunofluorescence (DIF) examination on a skin biopsy (Froberg et al., 2025). LCV itself can be found in a variety of other types of vasculitis that are not associated with IgA deposition (Baigrie & Crane, 2025). Therefore, without specific confirmation of IgA deposition, the diagnosis of IgAV in this case is still a very promising diagnosis may however require further affirmation.

Table 1. Clinical manifestations of IgA Vasculitis and correlation with cases (frequency figures based on the literature) :3

Klinis manifestation	Frequency on IgAV	Found in Case
Purpura terpalpasi	~100% (lower limb dominance)	Yes (feet & hands)
Arthralgia / Arthritis	~60–80% (lutut/ankle)	Yes (ankle, recover)
Abdominal pain / GI bleeding	~50–75%	None
Hematuria / Proteinuria	~40–85% (adults)	None (UA negative)
Other complications	Hips, testicles, lungs rarely	None

Source: Adapted from Castañeda et al., 2024

Overall, the patient's clinical findings are very consistent with IgAV. A history of precedent airway infections (strep throat), atopic nature (bronchial asthma), and geographic patterns (leg purpura) support the diagnosis. Early therapy includes steroids and anti-allergies to treat skin and joint inflammation, as practiced (although the effects of steroids on the prevention of nephritis are debated) (Castañeda et al., 2024; Reamy et al., 2020). There are no severe manifestations (e.g., GI or kidney bleeding), so patients are on the mild–moderate disease spectrum with a good prognosis. The main long-term risk is the onset of IgA nephritis (even though the initial condition of the urine is clean) and the possibility of relapse. Therefore, periodic monitoring needs to be carried out.

Clinical Manifestations and Correlation with the Literature

Skin Engagement

Skin involvement is a universal manifestation in patients with IgAV and is often an early sign of the disease. Typical IgAV rashes often begin as erythematose, macular lesions, or urticaria that later develop into petekie and palpable purpura. These lesions are commonly found on extensor surfaces and dependent areas that are susceptible to pressure, such as the lower legs and buttocks, although they can extend to the upper extremities and torso (Roache-Robinson et al., 2025). In these patients, complaints of redness spots that initially appear on the thighs, then widen and multiply on the legs and hands, as well as a bluish discoloration like bruises before fading to yellowish, is very consistent with the palpable purpura image typical of IgAV.

Joint Involvement

Arthralgia or arthritis is reported to occur in 60-80% of IgAV patients and is often an early manifestation of the disease in adults. Large joints of the lower limbs, such as the knees and ankles, are most commonly affected (Roache-Robinson et al., 2025). Complaints of pain, swelling, and impaired function are hallmarks of joint involvement (Castañeda et al., 2024). In these patients, complaints of stiffness, pain, and swelling in the soles and ankles are well consistent with the pattern of joint involvement described in the literature. It is important to note that arthritis in IgAV is transient and does not cause permanent joint damage (Roache-Robinson et al., 2025).

Gastrointestinal (GI) Involvement

Gastrointestinal manifestations can occur in 10-40% of IgAV patients, with symptoms such as nausea, vomiting, abdominal pain colic, and gastrointestinal bleeding (Roache-Robinson et al., 2025). In adults, GI involvement is one of the leading causes of morbidity and mortality (Castañeda et al., 2024). Interestingly, these patients did not report any complaints of abdominal pain or other GI symptoms. The absence of GI complaints in this case is a finding that needs to be noted, given the potential severity of GI involvement in adult IgAV.

Kidney Involvement

Renal involvement, known as IgAV nephritis, occurs in about 50% of patients and is a leading cause of long-term morbidity and mortality, especially in adults and children over 10 years of age (Reamy et al., 2020). Renal manifestations include microscopic hematuria, proteinuria, or red blood cell casts (Roache-Robinson et al., 2025). In these patients, examination of renal function (BUN, creatinine, eGFR) is within normal limits. Urinalysis

showed positive leukocytes (++) and leukocyte sediments, however protein and blood were negative. Although the classic criteria of IgAV nephritis (hematuria, proteinuria) have not been met, the presence of significant leukocytosis indicates the presence of an inflammatory process in the urinary tract. Given that kidney involvement can develop several weeks to months after rash onset, and that adults are at higher risk for severe kidney disease, these findings require rigorous and periodic monitoring of kidney function, including re-urinalysis and quantification of proteinuria if they occur (Roache-Robinson et al., 2025; Reamy et al., 2020).

Laboratory and Pathology Findings

Recommended supporting tests include: complete blood (LED/CRP is usually elevated, normal/high leukocyte count, normal platelets), coagulation and biochemical profiles (renal/hepatic function), and complete urine tests (Reamy et al., 2020). In adult IgAV it is recommended to include electrolytes and serum IgA. Routine urinalysis is important for early detection of glomerulonephritis: microscopic hematuria or proteinuria may appear even without symptoms. In this patient, the urine is normal without blood or protein (blood (-), erythrocytes 1 cell), so there is no early IgA nephritis. If persistent hematuria/proteinuria is found, consider a renal biopsy for staging and therapy (Castañeda et al., 2024). Additional tests include throat swabs, skin stains, and other infection tests if suspected.

LED and CRP upgrades

An increase in Blood Depositation Rate (LED) of 68.0 mm/h and C-Reactive Protein (CRP) of 7.7 mg/L in these patients are indicators of systemic inflammation. These findings are commonly found in vasculitis-like conditions such as IgAV, reflecting the body's acute inflammatory response to the disease (Baigrie & Crane, 2025).

Total IgE Increase

The patient's total IgE increased significantly to 552.05 KUI/L (Chen et al., 2016). This is in line with the patient's history of atopy who is positive for bronchial asthma and food allergies. Although IgAV is pathophysiologically dominated by IgA 1 deposition, a significant increase in total IgE in patients, along with a history of atopy and allergies, is an interesting finding. The presence of multiple eosinophils on a patient's skin biopsy may indicate the presence of an allergic component or hypersensitivity that accompanies the vasculic inflammatory response. This requires consideration in the context of the overall immunological picture of the patient, although the available literature does not directly correlate high IgE with IgAV severity or prognosis.

Biopsi Kulit (Leukocytoclastic Vasculitis)

Microscopic images of the patient's skin biopsy showed the presence of mild perivasculitis with lymphocyte cells, PMN neutrophils, and some eosinophils on the perivascular, accompanied by erythrocyte extravasation. These findings are consistent with the picture of leukocytolastic vasculitis (LCV), which is a common histopathological feature in IgAV (Roache-Robinson et al., 2025). For definitive confirmation of IgAV, direct immunofluorescence (DIF) examination is strongly recommended to show dominant IgA deposition on the blood vessel wall, which is a diagnostic hallmark of IgAV (Froberg et al., 2025).

Drugs as Potential Triggers of IgAV

Patients take hormonal medications from an obgyn doctor for two weeks before symptom onset, as well as methylprednisolone and loratadine from an internal medicine doctor. Drug-

induced vasculitis is a known cause of leukocytolastic vasculitis (Baigrie & Crane, 2025). Although hormonal drugs and antihistamines are not explicitly mentioned as common triggers of IgAV in the current literature (which more often identifies vaccines, antibiotics, and TNF-α blockers as the cause) (Rasmussen et al., 2021), the possibility of drug-induced IgAV should always be considered, especially if the onset of symptoms coincides with the start of a new drug. A history of hormonal drug use for 2 weeks prior to symptom onset requires further evaluation of the temporal relationship. The drugs methylprednisolone and loratadine appear to be given to manage pre-existing symptoms, not as triggers.

Tatalaksana

General Governance Approach

IgAV treatment is adjusted to the severity of the clinical manifestations. The main approach is symptomatic and supportive. For cases with only mild skin and joint manifestations like this, the main therapies include rest, warm compresses, and analgesics. NSAIDs can be used to relieve arthralgia in patients without active renal or GI involvement. However, NSAIDs should be avoided if there is a risk of kidney deterioration or gastrointestinal bleeding (Castañeda et al., 2024). In these patients, prevention of renal ureticular scleritis is not necessary due to normal kidney function, so the use of corticosteroids is aimed at reducing inflammation of the skin and joints.

Corticosteroids are the first line of treatment in cases with severe joint/abdominal pain or internal organ complications (Castañeda et al., 2024; Reamy et al., 2020). Meta-analytical evidence suggests steroids do not prevent nephritis if prophylaxis is given, so it is not recommended for prevention alone. However, for severe joint/abdominal pain symptoms, high doses can accelerate symptom remission. In adult patients with inflammatory symptoms, the use of moderate doses of methylprednisolone (e.g. 0.5–1 mg/kg) may reduce joint pain and edema. If IgAV becomes severe (e.g., glomerulonephritis or GI hemorrhage), aggressive treatment is often considered: a combination of steroids with immunosuppressive agents (cytostatics). Recent guidelines recommend short-term corticosteroids (≤ 6 months) as an adjunct therapy for patients at high risk of progressive chronic kidney disease. Other immunosuppressive agents such as cyclosporine, mycophenolate mofetil, dapsone, and rituximab have shown success in cases that are resistant to steroids or with severe skin/kidney involvement (Chen et al., 2016).

Other treatments that have been reported to be useful (especially for mild chronic skin lesions and arthritis) include: colchicine and dapsone (refractory purpura rash only); methotrexate or azatioprin as steroid-sparing for recurrent arthritis; Mycophenolate mofetil, ciclosporin, or tacrolimus for mild–moderate nephritis. These drugs follow the IgA nephropathy protocol due to a similar immunopathological pattern. For progressive refractory IgAV, more aggressive therapies (cyclophosphamide, rituximab) may be considered, although data are limited. IVIG or plasmapheresis has been reported to be beneficial in severe cases with multiorgan failure (Castañeda et al., 2024).

In these patients, the use of methylprednisolone corresponds to a supportive approach to relieve joint and inflammatory symptoms. Given the potential risk of higher renal involvement in adult IgAV and the findings of leukoscituria in urinalysis, close monitoring of renal function and urinalysis is essential. If there is a progression towards obvious IgAV

nephritis, management approaches with corticosteroids and/or immunosuppressive agents should be considered individually, with proactive nephrological consultation (Chen et al., 2016).

Non-Pharmacological Management

Non-pharmacological management includes skin care (avoid trauma, maintain hygiene, compress ulceration); cold/warm compresses according to symptoms (this patient feels better with warm soaks); Surveillance: Blood pressure control (especially if the kidneys are involved) and periodic urinalysis of each visit. When proteinuria or hypertension appears, ACE inhibitors or ARBs are added for renal protection (Castañeda et al., 2024).

Prognosis

In general, IgAV is a disease that is usually self-limited. In children, 94% recover within 1–2 months (Reamy et al., 2020). In adults, about 10–30% of patients with IgAV may develop into CKD or ESRD in the long term. Risk factors for kidney failure include: the presence of crescents or glomerular sclerosis on biopsy, macroscopic proteinuria, and the onset of older adulthood. Severe gastrointestinal manifestations also affect the short-term prognosis (Castañeda et al., 2024). Non - core manifestations (neurological, testicle, lung) are rare but require attention when they arise.

IgAV relapse occurs in 30% of patients. Risk factors for ESRD include early impaired renal function and significant proteinuria (>1 or 1.5 g/day) (Castañeda et al., 2024; Ozen et al., 2019). Relapse is quite common, and gastrointestinal symptoms at diagnosis are the best predictors of relapse in adults. Usually relapses occur within the first few months after the first episode. Therefore, short-term monitoring is recommended—a minimum of 6–12 months. Recommended protocols include monthly screening: urinalysis (RBC and protein), renal function (urinary creatinine/albumin), and blood pressure (Reamy et al., 2020). This period targets early detection of recurrent or chronic nephritis. In this case, although there are no signs of kidney initially, close monitoring is important: checking urine every month for ≥6 months to make sure there is no latent hematuria or proteinuria. Rudimentary GI (guaiac stool) screening may be considered even if there are no symptoms.

In the long term, most adult patients with IgAV fully recover without any major sequela of the skin or joints. The prognosis is mainly determined by the involvement of the kidneys. If nephritis does not appear, the outcome is good. Patients need to be educated to recognize the symptoms of relapse (purpura appears again, arthralgia or new urine blood). In mild relapse (skin/joint only), supportive therapy may be repeated. Only if the relapse is accompanied by significant skin necrosis or hematuria, immunosuppressive therapy should be considered again.

CONCLUSION

This case shows a typical presentation of IgAV in young adults, supported by a previous history of upper respiratory tract infections and atopic predisposition (bronchial asthma with high IgE). Clinical manifestations of palpable purpura on the extremities and arthralgia are very consistent with IgAV. Although there was no significant gastrointestinal or renal involvement at initial presentation, the LCV findings on skin biopsy were highly suggestive, although confirmation of IgA deposition via direct immunofluorescence remains ideal for definitive diagnosis. Management with corticosteroids and antihistamines aims to relieve inflammation of the skin and joints. The long-term prognosis relies on close monitoring of the potential

development of IgAV nephritis, given the higher risk in adults, and patient education regarding the possibility of relapse is critical.

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