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Case Report

Paediatrics Section

IgA Vasculitis with Central Nervous System Involvement in a Child: A Case Report

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ABSTRACT

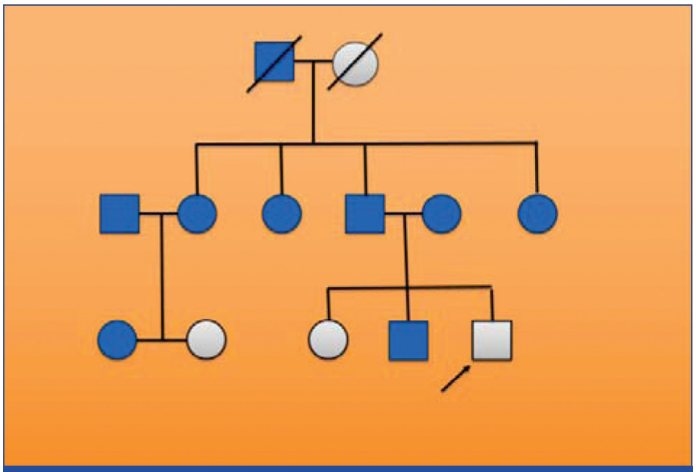
Henoch Schönlein Purpura (HSP), now termed IgA vasculitis, is a small vessel vasculitis classically characterised by palpable purpura, abdominal pain, arthralgia and renal involvement. Neurological manifestations such as seizures or encephalopathy are distinctly uncommon and may precede the appearance of palpable rash, leading to significant diagnostic confusion with acute Central Nervous System (CNS) infections. This report discusses a 13-year-old boy who presented with continuous fever, severe colicky abdominal pain, recurrent vomiting and multiple episodes of generalised tonic clonic seizures, in the absence of early purpura or altered sensorium. Antenatal, birth and developmental histories were unremarkable; however, family history was notable for seizure disorder in multiple paternal relatives. Clinical examination revealed fever, mild dehydration and diffuse abdominal tenderness, without focal neurological deficits or meningeal signs. Initial investigations showed neutrophilic leucocytosis, elevated inflammatory markers, abnormal electroencephalography and Magnetic Resonance Imaging (MRI) of the brain demonstrating focal cerebritis with micro haemorrhages. Cerebrospinal fluid analysis was not suggestive of bacterial meningitis. Urinalysis revealed microscopic haematuria with trace albuminuria. Despite broad-spectrum antimicrobial therapy, the child had persistent fever and abdominal symptoms. Subsequently, non-blanching palpable purpura developed over all extremities and the genital region, accompanied by transient hypertension. After exclusion of infectious, autoimmune and post-infectious inflammatory causes, skin biopsy demonstrated leucocytoclastic vasculitis, confirming IgA vasculitis. Initiation of corticosteroid therapy resulted in rapid resolution of fever, abdominal pain, seizures and rash. This case underscores the diagnostic challenges of CNS-predominant IgA vasculitis and emphasises the importance of recognising evolving dermatological signs, which may be delayed or masked by early steroid administration as a part of meningitis protocol potentially misleading towards alternative diagnosis.

Keywords: Encephalitis, Henoch-Schönlein purpura, IgA vasculitis, Leucocytoclastic, Meningitis, Salmonella infections

CASE REPORT

A 13-year-old boy presented to the paediatric emergency department with a two-week history of continuous fever without chills or rigours, associated with acute onset colicky abdominal pain of moderate to severe intensity (Numeric Rating Scale score 8/10) which was diffuse, continuous, postprandially aggravated, and intensified on passing stools and he had around 3-4 episodes of non-bilious, non-projectile vomiting for 10 days. This illness was followed by four episodes of generalised tonic clonic seizures occurring within 24 hours. Each episode lasted approximately 5-10 minutes and was associated with uprolling of the eyes. Patient was initially treated with intravenous broad-spectrum antibiotics (amikacin 15 mg/kg/day, metronidazole 25-30 mg/kg/day, and ceftriaxone 50 mg/kg/day) and antiepileptic measure (levetiracetam 9 mg/kg/day and injection phenytoin 14 mg/kg/day) in outside sector initially. The seizures had focal onset involving the left lower limb, followed by secondary generalisation to other limbs. Post-ictal confusion persisted for around 30 minutes. There was no history of preceding headache, visual disturbances, head trauma, or prior seizures. Birth and developmental histories were normal. Family history was significant for seizure disorder in an elder sibling who developed seizures at the age of 12 years, characterised by generalised tonic-clonic episodes, and is currently on medication with good control, and patient's paternal grandmother, paternal cousin, also has history of seizures detailed information regarding the type and treatment could not be ascertained [Table/Fig-1]. There was no consanguinity.

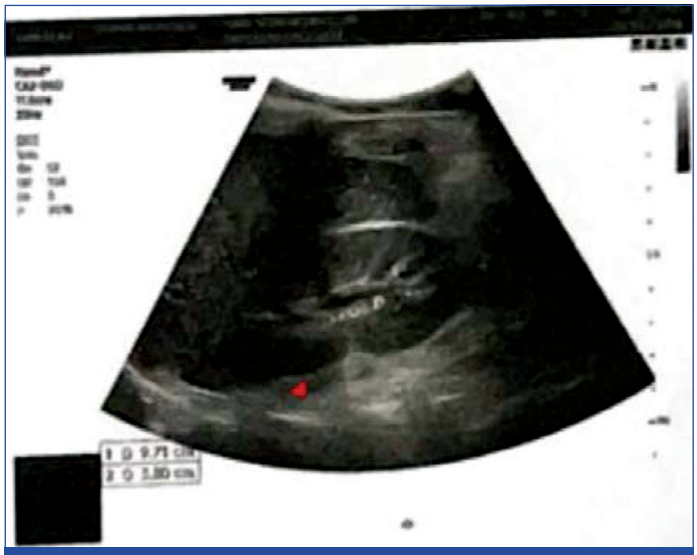
On admission, the child was febrile (101.7°F), his pulse rate was 98 beats per minute, respiratory rate was 17 cycles per minute, peripheral oxygen saturation was 99% on room air, and blood pressure was 110/70 mmHg, mildly dehydrated, and postictal, with a Glasgow Coma Scale score of 15/15. No rash was evident initially. Abdominal examination revealed diffuse mild tenderness in epigastric and right iliac region without guarding or rigidity. Neurological examination showed no focal deficits or meningeal signs. The patient was initially managed with standard antiepileptic measures. A loading dose of levetiracetam 20mg/kg was administered, followed by maintenance therapy at 20 mg/kg/day in divided doses every 8 hourly.



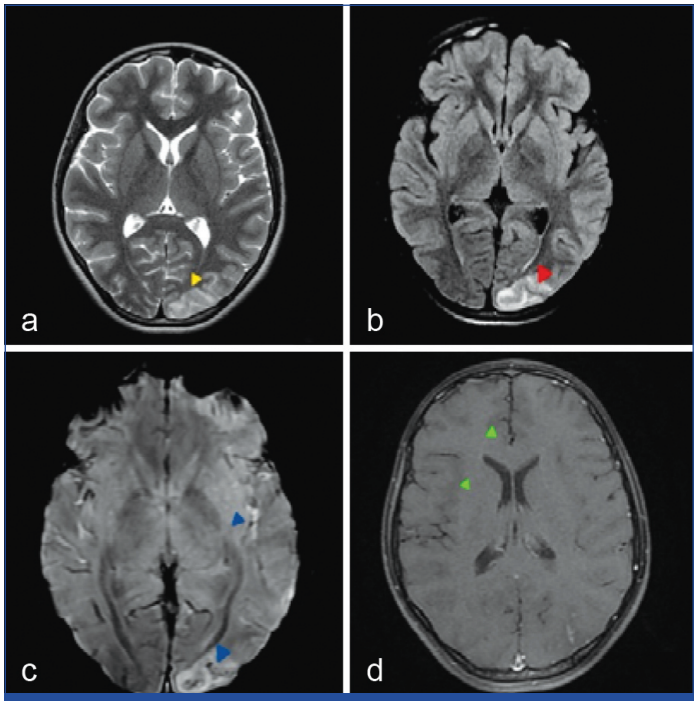
[Table/Fig-1]: Pedigree chart demonstrating seizure disorder affecting across three generations on the paternal side. A diagonal slash through a symbol denotes deceased individuals. The probed (patient) is indicated by an arrow. The pedigree demonstrates multiple affected members across generations on the paternal side, including the paternal grandmother, a sibling, and a cousin, suggesting a familial predisposition to seizure disorder.

Laboratory investigations demonstrated neutrophilic leucocytosis (total leucocyte count 16,110/mm^3; with neutrophils 77.6%),

thrombocytosis (platelet count 5.25×10^5/mm^3), and elevated inflammatory markers with rising C-reactive protein levels (76.0, 90.1, and 92.6 mg/L) and erythrocyte sedimentation rate of 20 mm/h (normal range 0-10). Peripheral smear showed leukocytosis with thrombocytosis. Cerebrospinal fluid analysis and culture were not suggestive of CNS infection. Ultrasonography of the abdomen revealed minimal free fluid in the lower abdomen [Table/Fig-2]. Widal test was positive for Salmonella typhi O antigen with a titre of 1:320. MRI of the brain with magnetic resonance angiography (plain and contrast) showed focal areas of gyral swelling with cortical and subcortical hyperintensities on T2/FLAIR sequences, along with multiple microhaemorrhages on susceptibility-weighted imaging [Table/Fig-3].

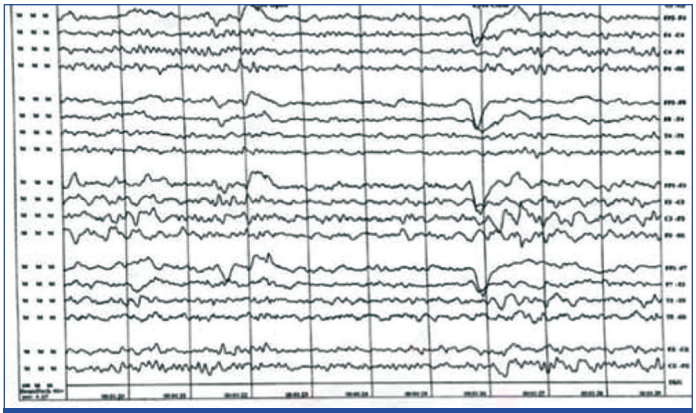


[Table/Fig-2]: Ultrasonographic image showing minimal anechoic free fluid in the lower abdomen.



[Table/Fig-3]: a) Magnetic Resonance Imaging (MRI) of the brain (T2-weighted sequence) occipital and parietal cortical thickening with subcortical oedema (yellow arrow); b) T2/ FLAIR hyper intensities noted in the left occipital lobe(red arrow); c) Few tiny SWI hypointense micro bleeds (blue arrows) noted within. No evidence of diffuse restriction; d) Post-contrast study showing linear leptomeningeal enhancement (green arrow).

Electroencephalography revealed abnormal sharp waves in the left cerebral hemisphere [Table/Fig-4]. In view of ceftriaxone allergy, the patient was started on intravenous inj. meropenem 120 mg/kg/day i.v. in three divided doses (q8h), inj. vancomycin 60 mg/kg/day i.v. in four divided doses (q6h), and inj. acyclovir as per weight-based

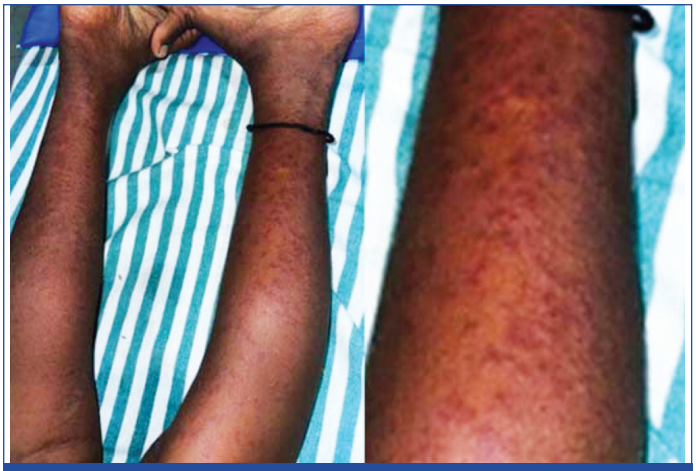


[Table/Fig-4]: EEG recording revealed abnormal background activity characterised by posterior dominant theta rhythm (7 Hz) along with sharp wave discharges localised to the left cerebral hemisphere, indicating focal cortical irritability.

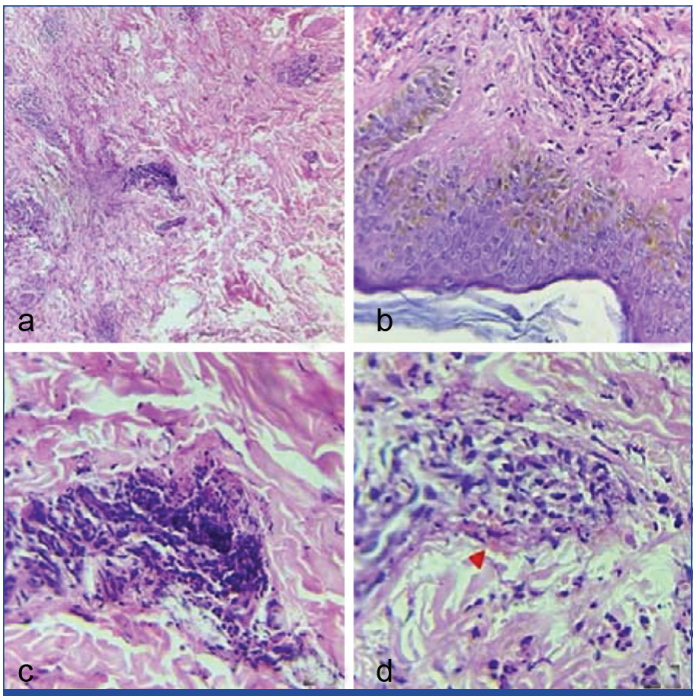
dosing 20 mg/kg/dose i.v. (q8h) for 14 days. During this period, the patient developed a transient purpuric rash over the extensor surface of the right hand, which resolved within two days following initiation of Dexamethasone which was administered at a dose of 0.2 mg/kg/dose intravenously every eight hourly as per meningitis protocol. Acute encephalitic syndrome was initially considered but later ruled out despite persistent abdominal pain. Further evaluation showed negative Mantoux test, CBNAAT of gastric aspirate, ruling out tuberculosis. Blood and urine cultures were negative ruling out infective aetiology. Liver and renal function tests, electrolytes, ferritin, serum amylase, and lipase were within normal limits, excluding metabolic and surgical causes. Routine urine examination revealed microscopic haematuria with trace albuminuria, white blood cell casts and urine porphobilinogen was negative. Antistreptolysin O titres, Antinuclear Antibodies (ANA) and Extractable Nuclear Antigens (ENAs) panel, and complement levels (C3, C4) were normal, ruling out post-streptococcal glomerulonephritis, acute intermittent porphyria, and other autoimmune disorders. During the hospital course, the patient developed two episodes of transient hypertension (140/80 mmHg) associated with severe abdominal pain. Renal doppler and repeat abdominal ultrasonography were normal, excluding renovascular pathology. Two doses of oral nifedipine (10 mg) were administered. Despite broad-spectrum antibiotics and supportive therapy, the child continued to have persistent fever and abdominal pain. On day 29 of hospitalisation, non-blanching palpable purpura appeared over the upper limbs and subsequently involved both lower limbs up to the thighs [Table/Fig-5]. Two days later an erythematous rash over the penile shaft was also noted. D-dimer levels were elevated. Skin punch biopsy from a recent lesion showed leucocytoclastic vasculitis [Table/Fig-6]. Based on clinical features and histopathology, the patient fulfilled the American College of Rheumatology 1990 criteria and the EULAR/PRINTO/PRES 2010 classification criteria for IgA vasculitis, with one mandatory criterion and three supportive criteria [1]. A diagnosis of Henoch–Schönlein purpura (IgA vasculitis) was established. Oral prednisolone was initiated at 2 mg/kg/day for two weeks, resulting in complete resolution of fever, abdominal pain, seizures and rash. With sustained clinical improvement, the dose was tapered stepwise to 1.5 mg/kg/day for one week, one mg/kg/day for one week, 0.5 mg/kg/day for one week, and then 0.5 mg/kg on alternate days for one week before discontinuation. The patient was discharged with counselling regarding disease course, risk of relapse and the need for regular follow up. At one-month follow-up, the child remained seizure free and clinically stable, with no recurrence of rash or abdominal pain. The prescribed medications had been completed, and a mild weight gain was noted as a transient side effect during the treatment period.

DISCUSSION

IgA vasculitis is the most common systemic vasculitis in children and is characterised by IgA immune-complex deposition in small vessels, predominantly affecting the skin, gastrointestinal tract, joints, and kidneys



[Table/Fig-5]: Clinical photograph showing progression of non-blanching palpable purpura over both lower limbs, consistent with HSP.



[Table/Fig-6]: Skin biopsy with Haematoxylin & Eosin (H&E) stain: a) Low-power view (×40) showing perivascular inflammatory infiltrate in the dermis. b) Medium-power view (×100) demonstrating leucocytoclastic vasculitis with fibrinoid necrosis of vessel walls, along with mild spongiosis and parakeratosis of the epidermis; c) High-power view (×200) showing dense neutrophilic infiltrate with prominent leucocytoclasia (nuclear dust); d) High-power view (×400) highlighting extravasation of red blood cells (red arrow) and fragmented neutrophilic nuclei; dermis also shows mixed inflammatory infiltrates.

[2-4]. Neurological involvement is uncommon, reported in fewer than 5% of cases, and may manifest as seizures, headache, encephalopathy, or rarely intracranial haemorrhage [5,6]. Diagnostic difficulty arises when neurological manifestations precede the appearance of palpable purpura, as seen in this case. Early administration of dexamethasone as part of acute encephalitic syndrome management may be associated with further masking of developing rash, which may delay the diagnosis [7]. The present case is notable for its atypical presentation with seizures and radiological evidence of cerebral micro haemorrhages before the development of characteristic cutaneous lesions. Similar observations have highlighted that CNS involvement may reflect cerebral vasculitis secondary to IgA immune-complex deposition [6].

MRI findings such as parietal and occipital hyper intensities and micro bleeds, as observed in our patient, have been previously reported in IgA-related cerebral vasculitis and are thought to represent small-vessel inflammation and vascular fragility [8]. The clinical manifestations and severity of the disease depend on the location of the involved vessels, the calibre of the affected vasculature, the degree of vessel wall damage, and the underlying pathological mechanism [9]. In HSP, inflammation of the vessel walls increases their fragility and promotes a pro-thrombotic state. Along with the generation of antiphospholipid antibodies and additional disturbances in coagulation pathways, these factors predispose patients to both bleeding and thrombotic complications [5]. The prolonged febrile illness, abdominal pain, positive Salmonella serology, and raised inflammatory markers initially suggested an infectious aetiology. However, persistence of symptoms despite appropriate antibiotics and the later emergence of palpable purpura prompted reconsideration of the diagnosis. Severe infections are known to dysregulate immune responses and may act as triggers for leucocytoclastic vasculitis [10-12]. Salmonella species have been reported as potential precipitants of IgA vasculitis by inducing excessive IgA-mediated immune responses [10].

During an extensive review of the existing literature, we identified a growing body of evidence linking vitamin D deficiency with HSP and other immune-mediated inflammatory conditions, suggesting a potential immunomodulatory role of vitamin D. In view of these observations, vitamin D serology was performed in our patient and revealed a significant deficiency, supporting a possible association between hypovitaminosis D and immune dysregulation. This finding is consistent with the observations of Yeganeh MH et al., who demonstrated a significant relationship between low vitamin D levels and the severity and type of complications in HSP [13]. Penile skin involvement, as noted in this patient, is a recognised but under-reported manifestation of acute IgA vasculitis and is strongly associated with active disease [14]. Histopathological confirmation remains the gold standard in atypical cases. Demonstration of leucocytoclastic vasculitis on skin biopsy was decisive in establishing the diagnosis [1,15]. Corticosteroids are recommended in patients with severe gastrointestinal symptoms, neurological involvement, or refractory disease. Evidence suggests that steroids result in faster symptom resolution, although they do not significantly alter long-term renal outcomes [9,16]. The rapid clinical improvement following steroid therapy in this patient supports their role in severe extra-renal manifestations [16].

Neurological involvement in IgA vasculitis is rare but increasingly recognised in the literature, with manifestations ranging from seizures and Posterior Reversible Encephalopathy Syndrome (PRES) to intracerebral haemorrhage and focal neurological deficits. Ha TS and Cha SH first described cerebral vasculitis associated with Henoch–Schönlein purpura using sequential MRI, demonstrating reversible multifocal cerebral lesions suggestive of vasculitic involvement [17]. PRES has been reported in several pediatric cases, including those by Dasarathi M et al., Sivrioglu AK et al., and Funken D et al., where patients presented with seizures, headache, cortical blindness, or focal neurological symptoms with characteristic reversible occipito-parietal MRI abnormalities [18-20]. Interestingly, PRES occurred both in hypertensive patients with renal involvement and in normotensive children without an identifiable precipitating factor, suggesting endothelial dysfunction and cerebral vasculitis as potential pathogenic mechanisms [18-20]. Intracerebral haemorrhage, although uncommon, has also been documented by Imai T et al., Ng CC et al., and Misra AK et al., with neuroimaging revealing large parieto-occipital or intracerebral hematomas in association with seizures and focal deficits [21-23]. Imai T et al., additionally proposed reduced factor XIII activity as a possible contributing factor for haemorrhagic complications [21]. Shen H et al., further highlighted the spectrum of encephalopathy and CNS dysfunction in children with IgA vasculitis, emphasising that neurological sequelae may occasionally persist despite treatment [24]. Overall, most reported patients demonstrated favourable neurological and radiological recovery following supportive care, corticosteroids, antihypertensive management when indicated, and antiepileptic therapy [17-24].

This report underscores the importance of early recognition of CNS manifestations in IgA vasculitis and prompt neuroimaging evaluation in patients presenting with seizures, altered sensorium, or focal neurological deficits. A comparison of the findings of the present study with previously reported cases is shown in [Table/Fig-7] [17-24].

Author (Year)	Place of Study	Age / Sex	Neurological Presentation	Radiologic CNS Abnormality	Associated Co-infection / Conditions
Ha TS & Cha SH (1996) [17]	South Korea	5-year-old Female	Generalised seizures	MRI: bilateral multifocal cerebral lesions compatible with non-haemorrhagic vasculitic involvement; resolved on follow-up	Not specified
Dasarathi M et al., (2012) [18]	India	11-year-old Girl	PRES with headache, vomiting, neurological signs preceding rash	MRI: features consistent with PRES; resolved with supportive care	Normotensive PRES; no hypertension or documented infection
Sivrioglu AK et al., (2013) [19]	Turkey	5-year-old Girl	PRES with seizures, cortical blindness, headache	MRI: bilateral occipito-parietal and right frontal cortical–subcortical oedema consistent with PRES	Acute renal failure and hypertension; probable post-URI
Imai T et al., (2002) [21]	Japan	7-year-old Girl	Massive intracerebral hemorrhage with focal neurological deficits	CT/MRI: extensive parieto-occipital intracerebral hemorrhage; reduced factor XIII activity	No hypertension; suspected factor XIII deficiency
Ng CC et al., (1996) [22]	Likely Hong Kong /China	5-year-old Child	Large intracerebral hematoma with seizures	CT: large left parietal ICH (no further MRI provided) resolved on follow-up, no neurological sequelae	No specific infection reported
Misra AK et al., (2004) [23]	India	12-year-old Boy	Convulsions with focal neurological signs	CT/MRI: large right occipital haematoma	No infection reported; recovered with conservative management
Shen HJ et al. (2017) [24]	China	Multiple children (3 patients)	Seizures and CNS dysfunction; one with residual deficit	Neuroimaging abnormalities present in all cases; variable patterns	No specific infection reported
Funken D et al., (2021) [25]	Germany	7-year-old Girl	Diplopia and afebrile focal seizures preceding purpura	MRI: PRES with focal bilateral parietal T2/FLAIR hyperintensities	No hypertension or secondary precipitating factor

[Table/Fig-7]: Comparison of different CNS domains in Henoch–Schönlein purpura (HSP) [17-19,21-24].

CONCLUSION(S)

IgA vasculitis may rarely present with predominant neurological manifestations before the appearance of palpable purpura, leading to diagnostic delay. Persistent fever and abdominal pain despite appropriate antibiotic therapy should raise suspicion of vasculitis. MRI-detected cerebral micro bleeds may indicate CNS involvement. Awareness of atypical presentations, repeated clinical reassessment, and timely histopathological confirmation are essential for early diagnosis. Corticosteroids are effective in managing severe or atypical manifestations of IgA vasculitis.

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