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CASE REPORT

IgA vasculitis and acute hemorrhagic edema of infancy: a comparative analysis of two case reports

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Abstract

Introduction: Acute hemorrhagic edema of infancy (AHEI) is sometimes considered a milder variant of immunoglobulin A vasculitis (IgAV). This paper presents two clinical cases, aiming to explore the overlaps and differences between these two pathologies. **Case report:** A four-year-old initially presented with odynophagia, fever, and arthralgia. The diagnosis of IgAV was established following the development of purpura. A nontoxic five-month-old girl, initially diagnosed with viral exanthema, presented with fever, mild respiratory symptoms, and a maculopapular rash on the torso. Gradual progression to purpuric lesions and acral edema led to the diagnosis of AHEI. **Discussion:** While IgAV and AHEI share some clinical and histological features, their immunofluorescence patterns differ significantly. IgA deposition is observed in nearly 100% of IgAV cases but only in 10–35% of AHEI cases. This disparity may be attributed to the natural immaturity of the immune system in younger infants, potentially explaining the differences between these two diseases.

Keywords: Iga vasculitis. Henoch-Schöenlein purpura. Acute hemorrhagic edema. Pediatrics. Purpura. Leukocytoclastic vasculitis.

Vasculite por IgA e edema hemorrágico agudo da infância: uma análise comparativa de dois relatos de caso

Resumo

Introdução: O Edema Hemorrágico Agudo da Infância (EHAI) e a Vasculite de Imunoglobulina A (VIgA) parecem representar um espectro distinto da mesma condição. Pretende-se explorar as semelhanças e diferenças entre essas patologias a partir de casos clínicos. **Relato do caso:** Um menino de quatro anos com oligoartite foi diagnosticado com VIgA após o aparecimento de púrpura. Uma lactente com bom estado geral, febre, sintomas respiratórios e exantema maculopapular foi inicialmente diagnosticada com exantema viral e, após progressão das lesões cutâneas para equimoses e aparecimento de edema acral, com EHAI. **Discussão:** Apesar da VIgA e o EHAI partilharem características clínicas e histológicas, a imunofluorescência difere. A deposição de IgA é observada em cerca de 100% dos casos de VIgA e em 10–35% dos casos de AHOI. Esta disparidade pode ser atribuída à imaturidade do sistema imunitário em idades mais jovens, podendo explicar algumas diferenças entre estas duas patologias.

Palavras-chave: Vasculite a IgA. Púrpura Henoch-Schonlein. Edema hemorrágico agudo da infância. Pediatria. Púrpura. Vasculite leucocitoclástica.

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Keypoints

What is known

- Immunoglobulin A vasculitis (IgAV) and acute hemorrhagic edema of infancy (AHEI) are forms of leukocytoclastic vasculitis that affect small vessels.
- IgAV primarily affects older children (four to seven years of age), presenting with palpable purpura, arthralgia, abdominal pain, and nephritis.
- AHEI typically occurs in infants under two years of age, presenting with fever, limb edema, and purpuric lesions.
- Both conditions involve immune complex-mediated vasculitis, with IgA deposition being prominent in IgAV and varying in AHEI.
- Histologically, both exhibit leukocytoclasia and vessel wall immune deposits; IgA deposition is more consistent in IgAV. AHEI shows distinctive C1q deposition.

What is added

- Detailed case reports providing insights into the varied presentations of IgAV and AHEI.
- Comprehensive comparison of IgAV and AHEI, highlighting their similarities and differences.

Introduction

Immunoglobulin A vasculitis (IgAV), formerly known as Henoch-Schönlein purpura, and acute hemorrhagic edema of infancy (AHEI), also referred to as Finkelstein or Seidlmayer disease, are forms of leukocytoclastic vasculitis that affect small vessels[1]. The incidence of IgAV is three to 27 cases per 1,000,000 children, with some experts suggesting that AHEI might be a milder form of IgAV, comprising 2.5%-7% of IgAV cases. The relationship between the two conditions remains unclear[2-4].

The classic presentation of IgAV includes palpable purpura, articular complaints, abdominal pain, and nephritis while AHEI typically presents with fever, limb edema, and purpura or ecchymosis in nontoxic patients[1,4].

This paper aims to explore the similarities and differences between IgAV and AHEI through the presentation of two clinical cases.

Case report

Immunoglobulin A vasculitis

A four-year-old boy presented with a two-day history of fever and odynophagia and was clinically diagnosed with acute tonsillitis. He received empiric treatment with oral amoxicillin/clavulanic acid and the symptoms resolved within 24 hours. Four days later, he developed tibiotarsal joint pain, resulting in a limp. At the pediatric emergency department (ED), the physical examination and laboratory studies, including complete blood count (CBC) and C-reactive protein (CRP), were normal. His condition improved with oral ibuprofen over a few hours and he was discharged.

Two days later, he returned to the ED with recurring knee joint and lower back pain. Mild periarticular swelling was noted in both knees, but there were no signs of joint effusion. The parents reported a liquid bowel movement three days earlier but said there were no further gastrointestinal (GI), urinary, or other symptoms. Bloodwork showed a mildly elevated erythrocyte sedimentation rate (ESR) (39 mm/1st hour) and CRP (0.970 mg/dL), while the CBC, serum urea, and creatinine levels, as well as the urinalysis, were normal. Partial pain relief was achieved with oral ibuprofen and the patient was admitted for further investigation and surveillance.

Antibiotic treatment was discontinued due to concerns regarding serum sickness disease and treatment with ibuprofen led to the rapid improvement of the arthralgia overnight. Additional articular imaging was deemed unnecessary. On the first day after admission, scattered petechiae were observed on the lower limbs (Fig. 1A) and tibiotarsal region (Fig. 1D). Ecchymotic lesions and palpable purpura were noted in the popliteal region (Fig. 1C), tibiotarsal joints (Fig. 1B), and wrist. A diagnosis of IgAV was considered. Over the next 48 hours, the rash expanded slightly before gradually resolving. He had a normal daily urinalysis, a max ESR of 43 mm/1st hour, a mildly elevated immunoglobulin A (IgA) titer (230 mg/dL), elevated Antistreptolysin O titer (ASOT) (341 UI/ml), and a normal coagulation study. He was discharged home within three days on oral nonsteroidal anti-inflammatory drugs (NSAIDs).

The patient has been followed up for over a year with no recurrence of symptoms, maintaining a normal urinalysis and blood pressure.

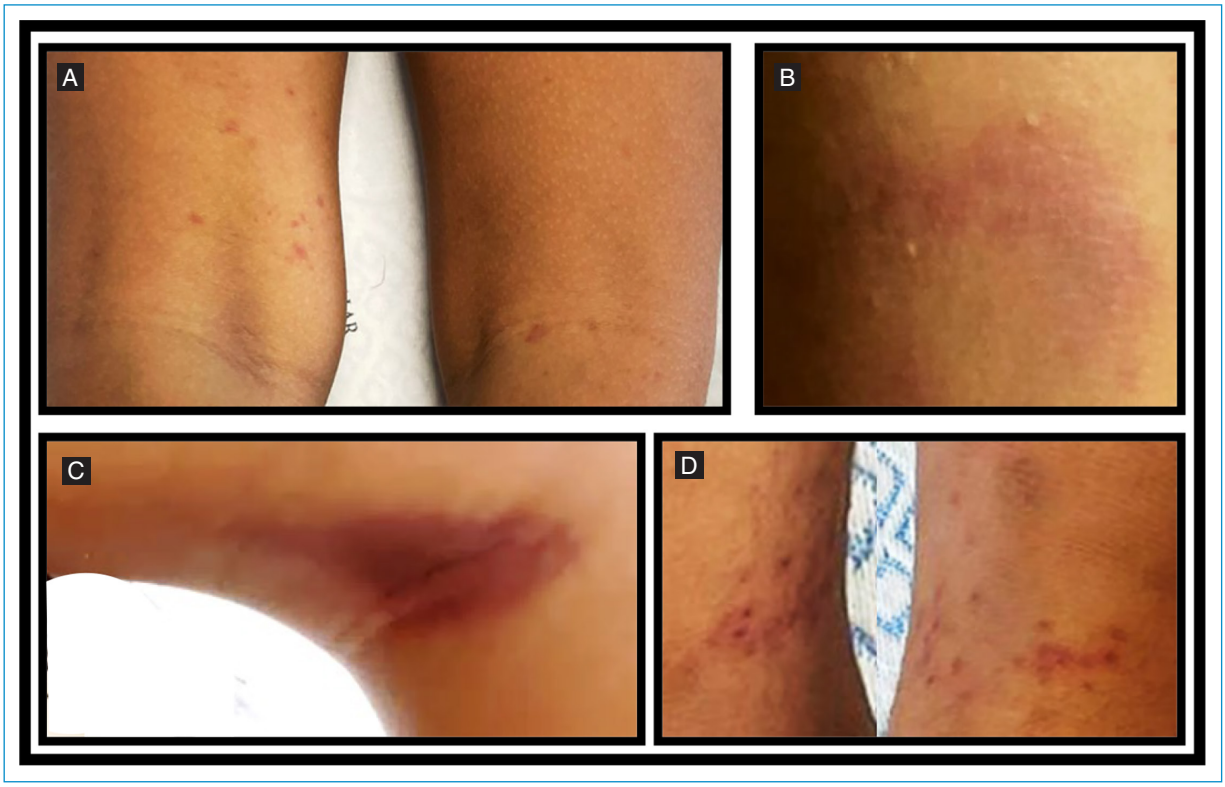

Figure 1. Cutaneous manifestations of IgA vasculitis. Petechia in the lower limbs (**A**) and tibiotarsi region (**D**). Ecchymosis in the tibiotarsi (**B**) and popliteal region (**C**).

Acute hemorrhagic edema of infancy

A five-month-old girl presented with low-grade fever, mild upper respiratory symptoms, and a maculopapular rash on the torso. She was diagnosed with an acute viral infection at a local pediatric ED and discharged with symptomatic treatment. Four days later, she was reevaluated due to persistent symptoms. Laboratory tests, including CBC and inflammatory markers, were unremarkable and she was again discharged with symptomatic treatment.

The following day, due to persistent fever, she was brought to our ED. On admission, she had a fever (axillary temperature 38.3 °C), a nontoxic appearance, and a maculopapular rash with purpuric lesions involving the face, arms, and legs, along with acral and facial edema (Figs. 2A and 2B). Laboratory tests were repeated due to the persistent fever, showing negative inflammatory markers and normal serum urea and creatinine levels. She was diagnosed with AHEI and influenza infection and admitted overnight for observation.

During admission, she maintained a good clinical status and became afebrile. The cutaneous manifestations improved rapidly, with lesions almost completely resolved within 24 hours, making further investigation unnecessary. She was discharged the following day, with complete clinical resolution achieved within five days.

Discussion

Etiology and pathophysiology

IgAV is characterized by the presence of aberrantly glycosylated IgA, which forms immune complexes (ICs) either with IgA itself or with antiglycan antibodies. These ICs can deposit in the glomerular mesangium and vascular walls, triggering an inflammatory response mediated by inflammatory mediators and complement activation[5–7]. This mechanism is certainly relevant to the first case presented, where elevated IgA levels were noted.

While the exact cause remains unclear, various infectious and chemical triggers have been implicated. Approximately half of IgAV cases are preceded by infections, with *Streptococcus spp* being identified in around 17% of cases[2,8–10].

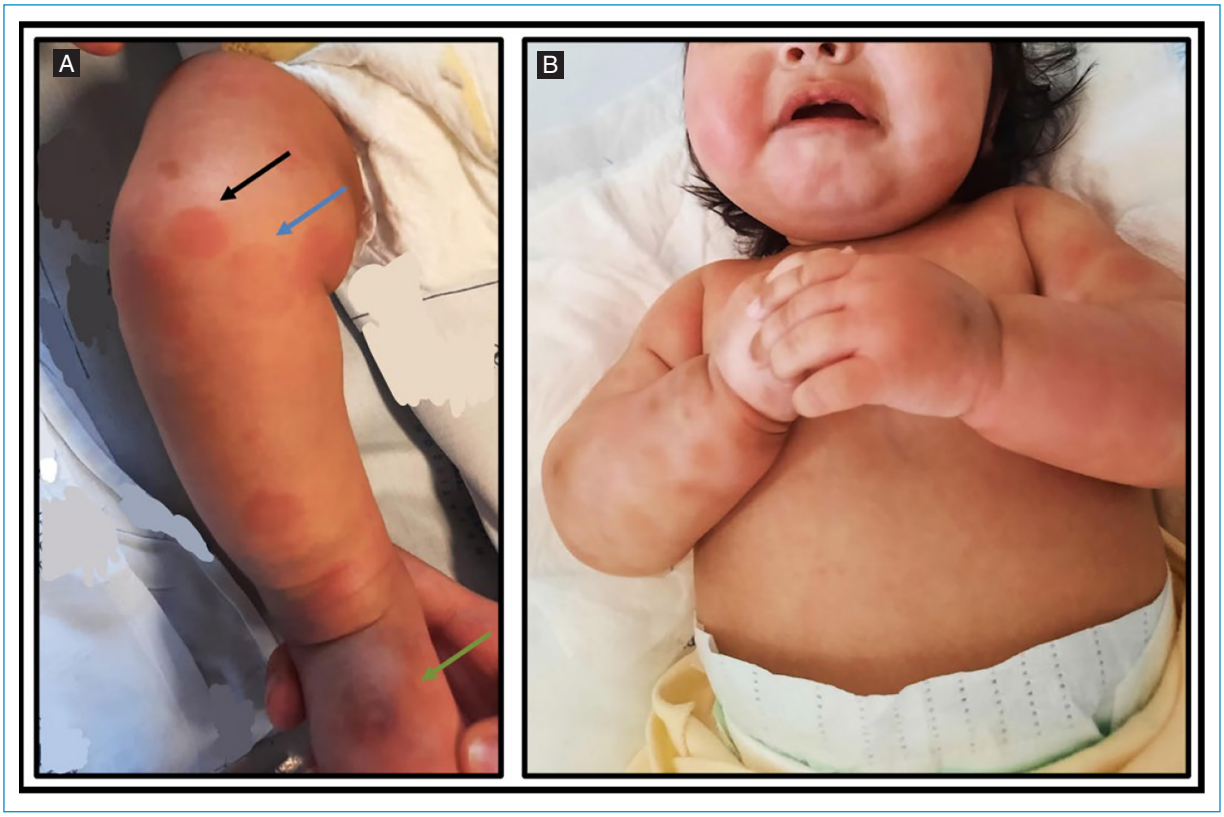

Figure 2. Cutaneous manifestations of acute hemorrhagic edema of infancy. **A**: macular lesions (black arrow), targetoid lesions (blue arrow), and purpuric lesions (green arrow) in the right lower limb. **B**: macular, rosette-like lesions and ecchymosis on the face, upper limbs, and torso; edema of the face and distal aspect of the upper limbs.

Similarly, AHEI involves immune-complex mediated mechanisms and is frequently associated with preceding infections. Rates of infectious triggers have been reported in up to 75% of cases[11]. Indeed, in both cases reported an upper respiratory tract infection functioned as a triggering factor.

Histology and immunofluorescence

In this case of IgAV, the patient exhibited petechiae and palpable purpura and although a biopsy was not conducted, IgAV is known to correlate histologically with key features, including infiltration of neutrophil granulocytes in small superficial vessels, leukocytoclasia, and erythrocyte extravasation into the surrounding dermis. The progression of the rash during hospitalization was likely associated with inflammatory processes such as leukocytoclasia. Similar histological findings are observed in AHEI, affecting capillaries and post-capillary venules in the upper and middle dermis. The transient nature of the rash in AHEI patients, as seen in the case of the five-month-old girl presented, may indicate a less severe inflammatory response compared to IgAV. Both conditions can exhibit fibrinoid necrosis[12-14].

Immunofluorescence typically reveals strong IgA deposition in IgAV, whereas IgA deposits are detected in only 10 to 35% of AHEI biopsy specimens, suggesting a milder or differently mediated immune response compared to IgAV[15]. This discrepancy contributes to the ongoing debate regarding whether AHEI represents a mild variant of IgAV or a distinct entity[3,13,15]. This could explain the rapid resolution of cutaneous symptoms and lack of systemic involvement in the AHEI patient. Perivascular deposits of IgM, fibrinogen, and C3 can be present in both conditions, but C1q deposition is unique to AHEI. In IgAV, vessel wall immune deposits include IgA1, C3, C3c, C3d, and varying amounts of IgG and IgM, with minimal or absent C1q or C4[6,7,16]. The pathophysiology of these conditions supports shared features, such as purpuric lesions as presented by the patients described, however, the unique deposition of C1q in AHEI could reflect an alternative immune pathway. Some authors propose that

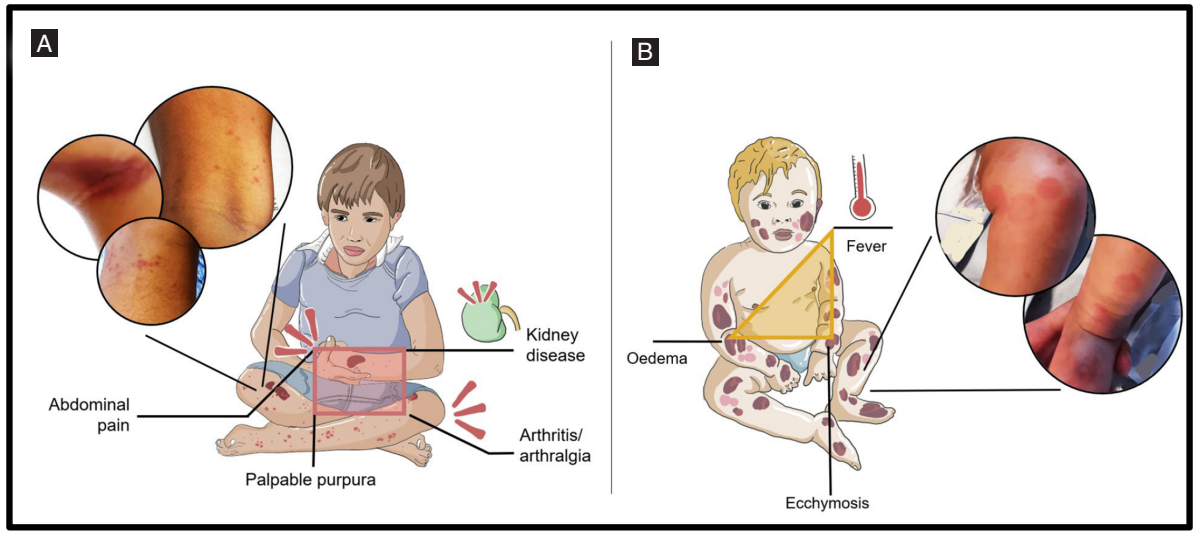

Figure 3. A: the classic presentation of immunoglobulin A vasculitis includes a tetrad of symptoms: palpable purpura with no thrombocytopenia or coagulopathy, acute-onset arthritis/ arthralgia, abdominal pain, and kidney disease. **B:** the clinical presentation of acute hemorrhagic edema of infancy typically consists of a triad of fever, edema, and rosette-shaped, annular, or targetoid-shaped purpura/ecchymosis in a nontoxic appearing patient.

these differences may stem from the immaturity of the immune system, particularly in IgA production. IgA levels in younger children are notably lower, reaching only 19 and 25% of adult levels by 12 and 24 months, respectively, which aligns with the typical onset of AHEI before the age of two years, as in the case presented. Interestingly, there is a documented overlap between IgAV and AHEI in children aged two to four years, which may indicate a progressive maturation of the immune system[17].

Clinical presentation and diagnosis

IgAV and AHEI predominantly affect pediatric patients. While AHEI typically presents in children under two years of age, IgAV peaks between four and seven years of age, which is consistent with the cases reported. Although the case of AHEI presented involved a female patient, the literature reports a male predominance in both AHEI and IgAV[2].

Figure 3 illustrates the classical presentations of IgAV and AHEI[1,2,4]. The four-year-old patient in this case exhibited three of the four hallmark symptoms of IgAV: skin involvement, gastrointestinal involvement, and arthralgia. Despite initial cutaneous presentation being characteristic petechiae and palpable purpura, the cutaneous symptoms emerged later than usual, complicating the diagnosis as they are seen in roughly three-quarters of patients at the time of diagnosis[18]. It should be noted that IgAV may initially manifest with erythematous, macular, or urticarial lesions that evolve into ecchymoses, petechiae, and palpable purpura, often symmetrically distributed in gravity-dependent areas[1]. Facial involvement, which was absent in this case, is rare and does not occur in isolation. The presence of arthralgia in the patient with IgAV aligns with existing literature, which indicates that arthralgia or arthritis affects 70-90% of patients. When arthritis is present, it typically exhibits a transient, oligo-articular pattern, with a predilection for the lower limbs, as observed in this case[2]. In our report, arthralgia was the sole initial presenting symptom of IgAV and can occur in about 15% of patients[2]. Skin edema can mimic a swollen joint. Gastrointestinal manifestations are frequent, occurring in up to 72% of patients. It is usually mild, as in the case described, usually manifesting by colicky abdominal pain but acute life-threatening GI bleeding and intussusception can occur[2]. Renal involvement, seen in 40–50% of IgAV cases, is typically asymptomatic and requires active screening, with microscopic hematuria and proteinuria being common findings[2]. Other manifestations include orchitis and neurologic symptoms[2].

In contrast, AHEI initially resembles a nonspecific viral exanthem and progresses with monomorphic lesions on the extremities, face, and scrotum. The initial

maculopapular rash observed in the AHEI case described exemplifies the typical progression from a "viral-like" exanthem to the characteristic cutaneous manifestations of AHEI, highlighting the challenges associated with differential diagnosis. Localized edema of affected areas is more frequent in AHEI than in IgAV, as noted in the patients presented[19]. Unlike in IgAV, noncutaneous involvement is rare in AHEI and the lack of systemic disease was classically considered a clinical criterion for diagnosis. However, some authors have reported arthritis, GI bleeding, and orchitis in AHEI[4].

The diagnosis of both IgAV and AHEI primarily relies on clinical presentation. In atypical cases, a skin biopsy may be warranted[2]. Laboratory tests such as platelet count and coagulation studies help rule out other causes of cutaneous hemorrhagic diseases like immune thrombocytopenia. In IgAV, serum IgA levels are elevated in 50-70% of patients and the initial urinalysis is typically normal, with proteinuria and/or hematuria potentially developing later[2,8].

Laboratory findings in AHEI are generally nonspecific, although leucocytosis, thrombocytosis, and eosinophilia may be observed[20]. The characteristics of the two diseases are summarized in Table 1.

Differential diagnosis

Regarding the patient with IgAV, poststreptococcal reactive arthritis could have been considered as there was prior acute tonsillitis, an elevated ASOT and lower limb joint complaints in a symmetrical distribution. However, the prompt overnight response to NSAIDs and further cutaneous manifestations made this condition less likely[21]. A serum sickness-like reaction was also contemplated due to the presentation of arthralgias occurring within two to seventeen days following amoxicillin/clavulanic acid administration. Despite this temporary correlation, typical features such as lymphadenopathy, urticarial rash, or fever were absent[22]. Additionally, a viral etiology, specifically parvovirus B19 infection, could have been considered given its potential to manifest primarily with arthropathy. In children, parvovirus B19 infection can present with symmetric arthralgia affecting the knees and ankles, similar to the pattern observed in this case. The absence of an erythema infectiosum rash made this diagnosis less likely[23]. The papular-purpuric "gloves and socks" syndrome typically presents with erythema and a petechial or purpuric rash but classically affects the hands and feet, which contrasts with the observed distribution in this patient.

In the case of the five-month-old with AHEI, consideration was given to the possibility of a nonspecific viral exanthem, which has been termed the "great imitator" by K. Nicole et al. The progression of clinical features over time played a crucial role in the diagnosis of AHEI.

Given the predominantly cutaneous manifestations of these conditions, it is crucial to consider child abuse in the differential diagnosis, as bruising is the most commonly reported injury associated with abuse in infants[24]. Additionally, vigilance for serious hematologic disorders, such as hematological malignancies, is essential.

Treatment

Supportive care plays a pivotal role in the management of both IgAV and AHEI, as illustrated by the treatment approaches in the clinical cases reported. Both the boy with IgAV and the girl with AHEI benefited from vigilant monitoring and analgesic/antipyretic support for symptom relief[5].

For the patient with IgAV, scheduled analgesia with NSAIDs was a cornerstone of treatment, as NSAIDs are the mainstay for managing arthralgia and arthritis. While corticosteroids may be considered for the symptomatic relief of severe abdominal pain and IgAV nephritis, these were not applicable in this case. Moreover, corticosteroids do not appear to prevent the onset of renal disease or abdominal complications, nor do they alter recurrence rates, and should not be used for empirical prophylaxis of kidney or GI complications[25,26]. In more severe cases, therapy with corticosteroids seems to reduce the severity and duration of abdominal pain during the first two weeks of treatment, but it does not reduce the risk of abdominal surgery[25]. Intravenous methylprednisolone could be administered in life-threatening situations, when the oral route is not tolerated or if there is no response to oral therapy. Renal involvement grants a multidisciplinary approach involving a pediatric nephrologist[25].

Given the typically self-limited and benign nature of AHEI, the primary focus of treatment is supportive care[20]. In the case presented, there was only a need for on-demand paracetamol.

Hospital admission should be decided on an individual basis, however Masarweh et al. propose six criteria that should be considered when evaluating the need for admission: 1) orchitis; 2) moderate to severe abdominal pain; 3) arthritis in three or more joints; 4) proteinuria; 5) inability to ambulate independently; 6) evidence of gastrointestinal bleeding[27]. The patient with IgAV was admitted before a definitive diagnosis was established, for further investigation and monitoring while the five-month-old girl was admitted for parental reassurance and observation.

Table 1. Clinical characteristics, diagnosis, and management of immunoglobulin A vasculitis and acute hemorrhagic edema of infancy

Characteristic	IgA vasculitis	Acute hemorrhagic edema of infancy
Epidemiology	Male predominance	
	Common in children aged two to 11 years	Typically affects children aged four to 24 months
Pathophysiology	Immune-complex mediated mechanisms	
Histology	Neutrophil infiltration of vessels, leukocytoclasia, erythrocyte extravasation, and fibrinoid necrosis can occur	
Immunofluorescence	Strong IgA deposition Minimal or absent C1q deposition	IgA deposits in 10 to 35% of biopsies C1q deposition
Clinical Presentation	Classical clinical tetrad: palpable purpura with no thrombocytopenia or coagulopathy, acute-onset arthritis/ arthralgia, abdominal pain, and kidney disease. Orchiditis may be present	Classical clinical triad: fever, acral edema, and rosette-shaped, annular, or targetoid-shaped purpura/ecchymosis in a generally nontoxic appearing patient. Limited systemic symptoms
Triggers	Often follows an upper respiratory tract infection or other viral illnesses	
Diagnosis	Mainly clinical diagnosis	
Treatment	Supportive care; NSAIDs for pain management Corticosteroids may be considered in severe cases	Supportive care
Prognosis	Generally favorable prognosis Renal involvement is linked to long-term morbidity and mortality	Self-limited disease with spontaneous and complete resolution
Recurrence	25% of patients	Typically a one-time occurrence

Follow-up

For the patient with IgAV presented, follow-up should include a urinalysis and blood pressure assessment repeated weekly during the first month, every two weeks in the second and third month, every month from the fourth to sixth month and then on months nine and 12[28]. Recurrence is seen in 25% of patients. Long-term renal complications, such as chronic kidney disease (CKD), a cause of morbidity and mortality among children with IgAV, functions as a prognostic factor[28,29]. A lower glomerular filtration rate at presentation and nephritic or nephrotic syndrome in the acute period of IgAV are poor prognostic features, with the risk of progressing to CKD being 41% in those patients vs. 15% in those with microscopic urine abnormalities[2].

Despite the rapid progression of cutaneous lesions, AHEI is a self-limited disease with spontaneous and complete resolution occurring in one to three weeks. Patients rarely have recurrent attacks and complications[30].

The differing outcomes between the cases reported (the boy with IgAV requiring long-term follow-up and the girl with AHEI who recovered quickly without the need for prolonged follow-up) underscore the importance of differential diagnosis in pediatric vasculitis. Neither of the patients presented experienced a recurrence of the disease.

Author contributions

All authors: conception and design of the study, report, review or other type of work or paper; acquisition of data either from patients, research studies, or literature; analysis or interpretation of data either from patients, research studies, or literature; drafting the article; critical review of the article for important intellectual content; final approval of the version to be published; agreement to be accountable for the accuracy or integrity of the work.

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Conflicts of interest

None.

Ethical considerations

Protection of humans and animals. The authors declare that no experiments involving humans or animals were conducted for this research.

Confidentiality, informed consent, and ethical approval. The authors have followed their institution's confidentiality protocols, obtained informed consent from patients, and received approval from the Ethics Committee. The SAGER guidelines were followed according to the nature of the study.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing of this manuscript.

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