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Case Report

Henoch-Schönlein Purpura with Gastrointestinal Tract Involvement and Nephritis in Children in Limited Healthcare Settings

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Abstract

Henoch-Schönlein Purpura (HSP), or IgA vasculitis, is the most common systemic vasculitis in children, characterized by a clinical tetrad of palpable purpura, arthritis/arthralgia, abdominal pain, and renal involvement. Gastrointestinal manifestations may mimic surgical acute abdomen, while nephritis determines the long-term prognosis. This case report aims to present the management of HSP with multi-organ involvement in a limited-resource setting. An 8-year-old girl presented with nausea, vomiting, and severe abdominal pain, preceded by the appearance of a reddish rash on the extremities and buttocks one week prior. Physical examination revealed diffuse palpable purpura and epigastric tenderness. Investigations showed leukocytosis, microscopic hematuria, and mild proteinuria. Abdominal ultrasound revealed positive sonographic tenderness at McBurney's point without signs of acute appendicitis or intussusception. The patient was diagnosed with HSP with gastrointestinal involvement and nephritis. The patient was treated conservatively with intravenous hydration, antibiotics, gastric mucosa protectors, and systemic corticosteroids. Significant clinical improvement in abdominal pain and skin rash was achieved following corticosteroid administration, and the patient was discharged with a prednisone tapering regimen. Early diagnosis of HSP is crucial to avoid unnecessary surgical intervention in patients with severe abdominal pain. Corticosteroids are effective in relieving acute gastrointestinal symptoms. Long-term monitoring of renal function is mandatory given the risk of chronic kidney disease progression, even in cases with mild initial symptoms.

Keywords: Abdominal Pain, Corticosteroids, Henoch-Schönlein Purpura, IgA Vasculitis, Nephritis.

1. Introduction

Henoch-Schönlein Purpura (HSP), now known as IgA vasculitis, is the most common small vessel vasculitis syndrome found in the pediatric population (Hamdan & Barqawi, 2008; Wardhani & Saroh, 2023). This disease is an autoimmune disorder mediated by the deposition of Immunoglobulin A (IgA) immune complexes in target organs, resulting in systemic inflammatory vasculitis (Jaszczura et al., 2018; Shin, 2023). Epidemiologically, more than 75% of HSP cases occur in children under 10 years of age, with peak incidence at 4-6 years, and it has a higher prevalence ratio in males compared to females (Reid-Adam, 2014; Sugianti et al., 2016). Although the exact incidence in Indonesia is not yet known, data from tertiary referral hospitals indicate a tendency toward an increase in new cases over the past few decades (Putri & Awalia, 2022; Sugianti et al., 2016).

The classic clinical manifestations of HSP include a tetrad of symptoms consisting of non-thrombocytopenic palpable purpura, arthritis or arthralgia, abdominal pain, and renal

involvement (Ghazanfar et al., 2024; Patel, 2023). The main clinical challenges often arise from gastrointestinal (GI) and renal involvement. GI involvement is reported to occur in 50-75% of patients, ranging from mild colicky abdominal pain to life-threatening complications such as massive gastrointestinal bleeding, intussusception, and intestinal perforation (Çağlayan, 2025; Jarasvaraparn et al., 2016). Diagnostic difficulty often occurs when gastrointestinal symptoms precede skin manifestations, which occur in approximately 10-15% of cases, so they frequently resemble other surgical acute abdomen conditions (Rajalakshmi & Srinivasan, 2015).

In addition to acute manifestations in the gastrointestinal tract, renal involvement or HSP Nephritis (HSPN) is the most important determinant of long-term prognosis. Nephritis occurs in 30-50% of patients, with a clinical spectrum ranging from isolated microscopic hematuria to progressive crescentic glomerulonephritis (Reid-Adam, 2014). The pathogenesis of this condition involves an abnormal immune response to viral or bacterial infections, in which IgA1 with abnormal glycosylation forms immune complexes that deposit in the renal mesangium and vascular walls, triggering complement activation and tissue damage (Chen & Mao, 2015).

Management of HSP is generally supportive because the disease tends to be self-limiting. However, in cases with significant organ involvement, such as severe abdominal pain or risk of nephritis, the use of corticosteroids becomes an important consideration, although its effectiveness in preventing chronic kidney disease remains debated (Kurnia, 2019; Oni & Sampath, 2019). In healthcare facilities with limited resources, the ability to recognize clinical signs of HSP early particularly distinguishing vasculitis abdominal pain from surgical acute abdomen and detecting subclinical nephritis is key to preventing unnecessary surgical intervention and mitigating long-term organ damage. This case report discusses the clinical management of a child with HSP accompanied by severe gastrointestinal involvement and nephritis in a limited healthcare facility, highlighting the importance of careful clinical diagnosis and close monitoring.

2. Case Study

On October 14, 2025, an 8-year-old girl was brought by her father to the Emergency Department (ED) with a chief complaint of nausea and vomiting experienced since 4 days before hospital admission, accompanied by weakness and decreased appetite. History-taking revealed that the complaint began one week earlier with the appearance of red spots (purpura) on both hands and feet, extending to the abdomen and buttocks, but not involving the face. Two days after the appearance of the rash, the patient complained of abdominal pain. Over the following three days, the intensity of vomiting decreased, but the red spots on both lower extremities did not subside and instead spread further, while complaints of abdominal pain and joint pain were felt intermittently. Associated symptoms such as fever at the time of hospital admission were denied; however, there was a history of cough and cold preceded by fever two weeks earlier that improved after 5 days with ibuprofen and ambroxol therapy. Complaints of bloody bowel movements and bloody urination were denied.

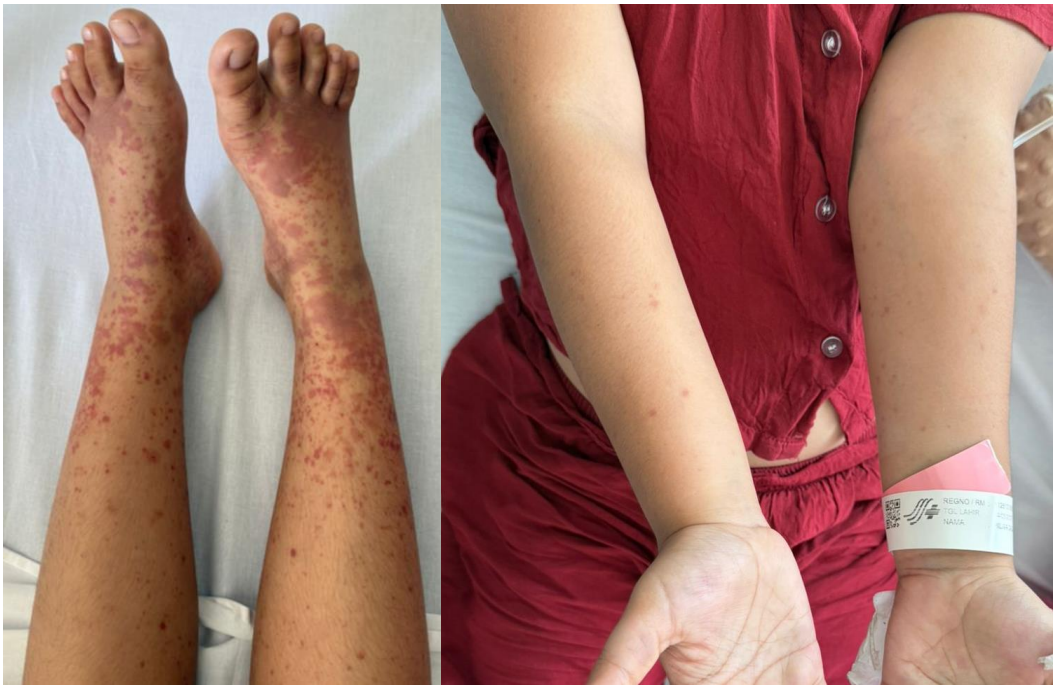

Figure 1. Clinical photographs of the patient

Physical examination at the time of admission showed a generally adequate condition with a Glasgow Coma Scale of E4M6V5 (compos mentis). Vital signs revealed a blood pressure of 110/70 mmHg, tachycardia with a pulse rate of 156 beats per minute, temperature of 36.6°C, respiratory rate of 30 breaths per minute, and oxygen saturation of 96% on room air. The patient's nutritional status was classified as obese (weight-for-height 26%) with a body weight of 35 kg (weight-for-age at the 95th CDC percentile) and height of 116 cm (height-for-age below the 5th CDC percentile). Specific examination findings included signs of dehydration in the form of sunken eyes and decreased skin turgor, as well as tenderness in the epigastric region. Cutaneous manifestations consisting of multiple palpable *purpura* were found on both upper and lower extremities with lesion diameters ranging from 1-2 cm. Based on the history and physical examination, the patient was given a working diagnosis of Henoch-Schönlein Purpura (HSP) and was admitted to the pediatric ward of Mitra Plumbon Indramayu Hospital.

Written informed consent for participation in this case report and for publication of the accompanying clinical photographs (Figure 1) was obtained from the patient's parent/legal guardian prior to submission. This study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Ethics Committee of Mitra Plumbon Indramayu Hospital.

Table 1. Results of Hematology (Blood) & Clinical Chemistry Examinations

Examination	Result	Unit	Reference Range
Routine Hematology			
Hemoglobin	12.7	g/dL	11.5 ~ 14.5
Hematocrit	38.50	%	35.00 ~ 47.00
Leukocytes	18,810*	/uL	4,500 ~ 11,000
Platelets	447,000	/uL	150,000 ~ 450,000
Erythrocytes	4.9	juta/uL	3.80 ~ 5.80
Erythrocyte Indices			
MCV	78.90	fL	76.0 ~ 90.0

Examination	Result	Unit	Reference Range
MCH	26.00*	pg/mL	31.00 ~ 36.00
MCHC	33.00	g/dL	31.00 ~ 36.00
Leukocyte Differential Count			
Basophils	0.2	%	0.0 ~ 1.0
Eosinophils	0.3*	%	1.0 ~ 6.0
Band Neutrophils	0.0*	%	3.0 ~ 5.0
Segmented Neutrophils	74.3*	%	40.0 ~ 70.0
Lymphocytes	19.0*	%	30.0 ~ 45.0
Monocytes	6.2	%	2.0 ~ 19.0
Clinical Chemistry			
Carbohydrates			
Random Blood Glucose (Rapid)	92	mg/dL	DM : >= 200

Table 2. Results of Urine Examination (Complete Urinalysis)

Examination	Result	Unit	Reference Range
Macroscopic Urine			
Color	Yellow		Yellow
Turbidity	Slightly Turbid		Clear
Urine Chemistry			
Urine Specific Gravity	>1.030*		1.005 - 1.030
pH	6.0		5.0 ~ 8.0
Erythrocytes	1+	/uL	Negative
Urine Bilirubin	Negative		Negative
Urine Urobilinogen	Normal	mg/dL	Normal
Urine Ketones	2+	mg/dL	Negative
Glucose	Negative	mg/dL	Negative
Urine Protein	Negative		Negative
Nitrite	Negative		Negative
Urine Leukocytes	2+	/uL	Negative
Mikroskopis Urine			
Sediment Erythrocytes	10-14*	/LPB	0 ~ 3
Sediment Leukocytes	16-25*	/LPB	0 ~ 8
Urine Epithelial Cells	2-3		
Casts	Negative	/uL	Negative
Urine Bacteria	Positive	LPB	Negative
Crystals	Negative	/uL	Negative
Yeast Cells	Negative	/uL	Negative
Mucous Threads	Negative	/uL	Negative
Other Urine Findings	-		

Table 3. Results of Stool Examination (Routine Feces)

Examination	Result	Unit	Reference Range
Macroscopic Feces			
Color	Yellow		
Consistency	Soft		Soft
Mucus	Negative		Negative
Blood	Negative		Negative

Microscopic Feces			
Leukocytes	1-2	/LPB	< 5
Erythrocytes	0-2	/LPB	< 2
Helminth Eggs	Negative		Negative
Amoeba	Negative		Negative
Others	-		Negative

3. Case Presentation and Discussion

3.1. Case Presentation

Ancillary investigations were performed to assess systemic organ involvement. A complete blood count on October 13, 2025 showed hemoglobin 12.7 g/dL, hematocrit 38.50%, platelets 447,000/µL, and erythrocytes 4.9 million/µL. Leukocytosis was present with a leukocyte count of 18,810/µL (see Table 1). Erythrocyte indices showed MCV 78.90 fL, decreased MCH at 26.00 pg/mL, and MCHC 33.00 g/dL. The leukocyte differential count showed a predominance of segmented neutrophils (74.3%) with lymphocytopenia (19.0%), as well as basophils 0.2%, eosinophils 0.3%, band neutrophils 0.0%, and monocytes 6.2%. Clinical chemistry examination for random blood glucose yielded a result of 92 mg/dL. Routine stool examination on October 15, 2025 showed macroscopically yellow stool with soft consistency, without mucus or blood. Microscopically, leukocytes 1–2/HPF and erythrocytes 0–2/HPF were found, and no helminth eggs or amoeba were detected (Table 3).

Renal involvement was evaluated through urinalysis on October 14, 2025. Macroscopically, the urine was yellow and slightly turbid. Urine chemistry examination showed specific gravity >1.030, pH 6.0, ketones positive 2+ (+2), erythrocytes positive 1+ (+1), and leukocytes positive 2+ (+2) (see Table 2). Protein, glucose, bilirubin, urobilinogen, and nitrite parameters showed negative results. Microscopic sediment examination revealed hematuria and pyuria, marked by erythrocytes 10–14/HPF and leukocytes 16–25/HPF. Epithelial cells 2–3/HPF and bacteria positive were also found, however casts, crystals, yeast cells, and mucous threads were negative.

Radiological examination by Whole Abdomen Ultrasound showed the liver with normal size but increased parenchymal echogenicity (regular surface, sharp edges, no nodules), giving the impression of fatty liver, differential diagnosis inflammatory changes (see Figure 2). In the McBurney region, the appendix was not visualized (non-visualized) however positive sonographic tenderness was present (McBurney pain +), pointing toward the suspicion of retrocecal appendicitis. No invagination pattern (bowel within bowel) was observed. Other intraabdominal organs including the gallbladder, pancreas, spleen, both kidneys, urinary bladder, and uterus were within normal limits without structural abnormalities, stones, or masses. No free intraabdominal fluid was found.

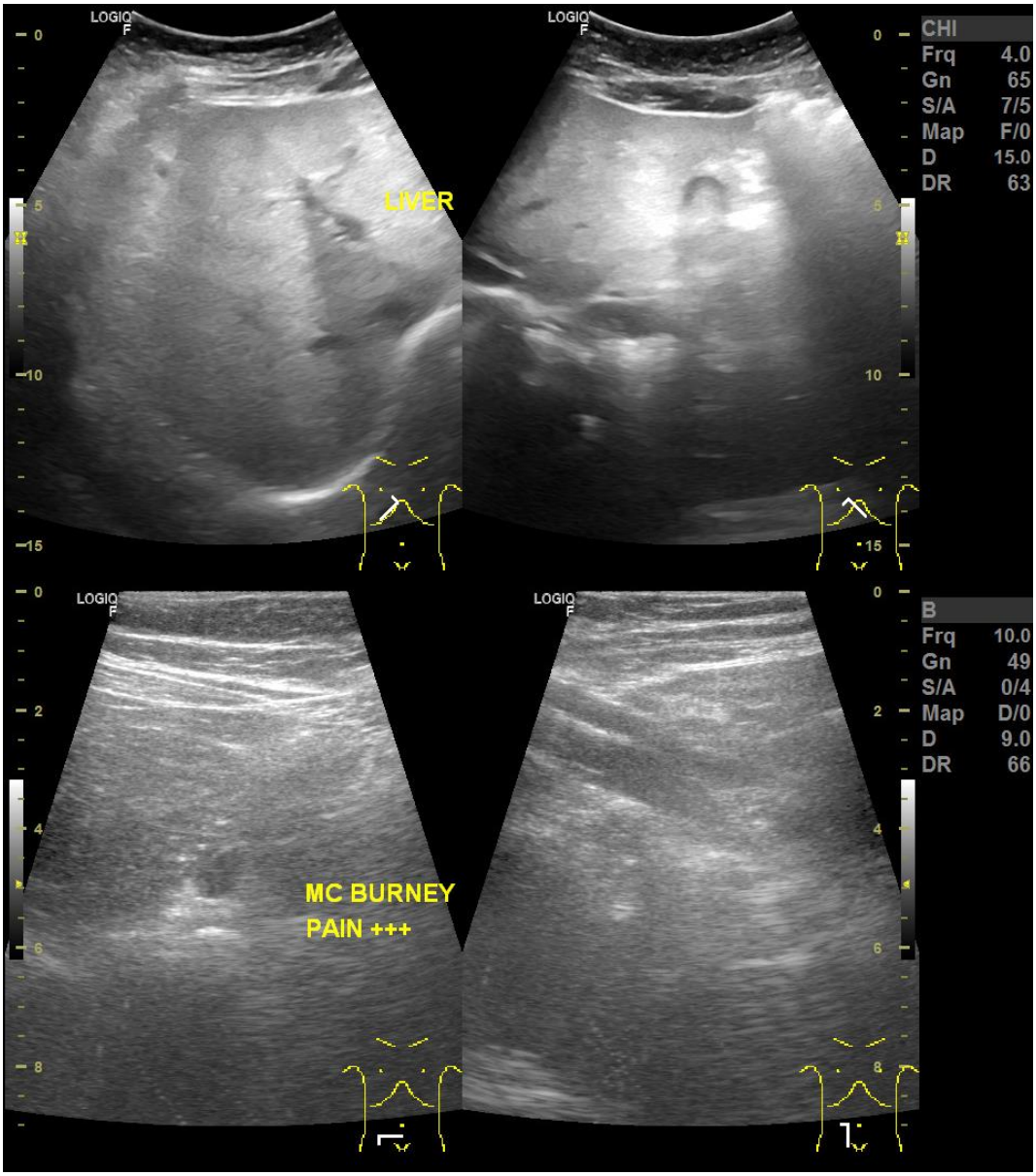

Figure 2. Radiological examination, Whole Abdomen Ultrasound

Initial management in the ED included a loading dose of Ringer's Lactate 350 cc followed by maintenance fluids of 1500 cc/24 hours, intravenous omeprazole 40 mg/24 hours, and intravenous ondansetron 4 mg/12 hours. During ward admission, the patient received antibiotic therapy with intravenous ceftriaxone 2x1 gram, supplementation with vitamin D3 1x1000 IU and Cal-95 1 tablet/24 hours, and the gastric mucosal protector sucralfate 3x5 cc. To address inflammation and pain, ibuprofen 5-10 mg/kg body weight per dose and pulse methylprednisolone 10-30 mg/kg body weight/day, maximum 1 gram, were administered for 3 days, which was then continued with tapering off prednisolone 1-2 mg/kg body weight/day for 1 week. The patient was hospitalized for 5 days until the clinical manifestations improved and the red spots decreased significantly. At the outpatient follow-up on October 24, 2025, no red spots were found, and the patient continued the prednisolone tapering program on an outpatient basis.

3.2. Discussion

Henoch-Schönlein Purpura (HSP), or IgA vasculitis, is the most common systemic vasculitis found in children, characterized by inflammation of small blood vessels and leukocyte infiltration. The disease involves the skin, joints, gastrointestinal tract, and kidneys (Hamdan & Barqawi, 2008; Wardhani & Saroh, 2023). In this case, the patient is an 8-year-old girl, falling within the common age range for HSP occurrence. Literature states that more than 75% of patients are under 10 years of age, with peak incidence at 4-6 years, although statistically there is a predominance in boys with a ratio of 2:1 (Reid-Adam, 2014). The diagnosis in this patient was established based on the 2010 European League Against Rheumatism (EULAR) and Paediatric Rheumatology European Society (PRES) criteria, in which palpable purpura (non-thrombocytopenic) was found accompanied by abdominal pain and renal involvement in the form of hematuria and proteinuria (Reid-Adam, 2014).

The exact etiology of HSP is not yet fully understood, however the disease is thought to be mediated by IgA immune complexes and is often triggered by infection (Saulsbury, 1987). In this patient, there was a history of fever, cough, and cold two weeks before the appearance of the rash. This is consistent with the literature stating that HSP is often preceded by upper respiratory tract infection. Various pathogens such as Group A beta-hemolytic Streptococcus, Mycoplasma pneumoniae, and viruses such as Adenovirus are frequently associated as triggers (Reid-Adam, 2014; Wardhani & Saroh, 2023). The underlying pathogenesis involves elevated levels of IgA1 with abnormal glycosylation (galactose deficiency) that forms immune complexes with IgG. These complexes then deposit in target organs such as the skin, gastrointestinal tract, and renal mesangium, triggering complement activation and inflammatory response (Chen & Mao, 2015).

Gastrointestinal (GI) manifestations in this patient were very prominent, consisting of nausea, vomiting, and severe abdominal pain. GI involvement occurs in 50-75% of HSP cases and is caused by vasculitis of the intestinal wall blood vessels resulting in edema and intramural hemorrhage (Rajalakshmi & Srinivasan, 2015). The patient experienced leukocytosis (18,810/µL), which according to studies is an independent risk factor for severe gastrointestinal involvement (Yahyaoui et al., 2018). On abdominal ultrasound examination, McBurney tenderness was found (positive sonography) with a non-visualized appendix, as well as an impression of fatty liver, differential diagnosis inflammatory changes. This is relevant to the literature which states that GI symptoms in HSP can mimic acute abdomen such as appendicitis, and ultrasound is very important for ruling out surgical complications such as intussusception which occurs in up to 3.5% of cases (Amelia et al., 2025; Çağlayan, 2025). In this patient, intussusception was not found (negative target sign), so conservative management could be continued.

Renal involvement (HSP Nephritis/HSPN) is the most important determinant of long-term prognosis. This patient showed signs of nephritis in the form of cloudy urine, microscopic hematuria (erythrocytes 10-14/HPF), and mild proteinuria. Approximately 30-50% of HSP patients experience renal involvement, which generally manifests within the first month of disease (Reid-Adam, 2014). The mechanism of renal damage involves deposition of galactose-deficient IgA1 immune complexes in the mesangium that triggers mesangial cell proliferation (Chen & Mao, 2015). Although most cases of hematuria will improve, the presence of proteinuria, even if mild, requires close monitoring due to the risk of progression to chronic kidney disease (CKD). The risk of CKD is higher in patients with severe initial symptoms (nephritic/nephrotic syndrome), however patients with mild symptoms also retain a long-term risk (Davin & Coppo, 2014).

Management in this patient included fluid rehydration, antibiotics (ceftriaxone), gastric protection, and corticosteroids (methylprednisolone continued with tapering off prednisolone). The use of corticosteroids in this patient provided a good clinical response to abdominal pain and joint complaints. This is supported by various studies showing that corticosteroids effectively reduce the intensity and duration of abdominal pain and arthralgia in the acute phase (Kurnia, 2019; Oni & Sampath, 2019). However, it should be noted that based on current evidence, including the Cochrane review and KDIGO guidelines, the use of corticosteroids has not been proven to prevent the occurrence of nephritis or improve long-term renal outcomes in mild cases (Oni & Sampath, 2019). Therefore, the main purpose of administering steroids in this case was to address severe gastrointestinal vasculitis, not as a preventive measure for renal abnormalities.

The prognosis of this patient is generally good, marked by clinical improvement and disappearance of the rash at outpatient follow-up. Nevertheless, HSP has a recurrence rate of approximately 16.4% and risk of renal complications that can appear later (Ekinci et al., 2020). Given that chronic kidney disease can develop years after onset, even in patients with initially mild symptoms, periodic monitoring of blood pressure and urinalysis is crucial for at least 6-12 months post-diagnosis (Davin & Coppo, 2014). Education to parents regarding warning signs of recurrence and the importance of regular follow-up is an integral part of long-term management in limited healthcare facilities.

4. Conclusion

Based on this case report, it can be concluded that Henoch-Schönlein Purpura (HSP) in children requires high clinical vigilance, particularly when accompanied by severe gastrointestinal manifestations and renal involvement. In this case, careful diagnosis at a limited healthcare facility successfully spared the patient from unnecessary surgical intervention, despite the clinical symptoms and initial sonographic findings resembling acute appendicitis. Systemic corticosteroid administration was proven effective in providing rapid clinical improvement of abdominal pain complaints and skin manifestations in the acute phase, consistent with literature supporting its use for severe gastrointestinal symptoms. Nevertheless, the presence of renal involvement in the form of hematuria and proteinuria, even if mild, demands strict long-term monitoring. Given that corticosteroid use has not been proven to prevent the progression of nephritis, education for parents regarding the importance of regular blood pressure monitoring and urinalysis is key to long-term management for the early detection of potential chronic kidney disease.

For clinicians practicing in resource-limited hospitals, this case highlights several practical recommendations. First, a standardized urinalysis protocol should be applied at admission and repeated at 1, 3, 6, and 12 months post-diagnosis to detect subclinical renal involvement early, even in patients with initially mild urinary findings. Second, clear referral pathways to pediatric nephrology or a higher-level facility should be established for cases presenting with worsening proteinuria, hypertension, or declining renal function, where renal biopsy or immunosuppressive therapy may be warranted. Third, a structured follow-up checklist including blood pressure measurement, dipstick urinalysis, and assessment for rash recurrence can be implemented at the primary or district hospital level without requiring advanced laboratory infrastructure. These measures, if systematically adopted, have the potential to reduce delayed diagnosis of HSP nephritis and mitigate long-term renal morbidity in pediatric patients managed outside tertiary care centers.

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