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Recurrent Abdominal Pain with A Late Clue: Diagnosing Henoch-Schonlein Purpura After Skin and Joint Involvement

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ABSTRACT

Background: Henoch-Schonlein purpura (HSP) is the most common childhood vasculitis, primarily affecting children under 10 years. While typically self-limiting with a good prognosis, HSP can become life-threatening if multiple organs are involved. Early diagnosis was critical to prevent complications and ensure optimal outcomes. ***Objective:*** To highlight the diagnostic challenges of HSP when recurrent abdominal pain appeared with delayed onset of purpura and athralgia. ***Case:*** A 10-year-old male presented with abdominal pain, had been hospitalized, but after discharge, he developed red spots on his lower extremities and buttocks along with joint pain in his legs. As his abdominal pain recurred and worsened, he was readmitted. Physical examination revealed diffuse tenderness and palpable purpura on the lower extremities and gluteal area. Investigations revealed leukocytosis, neutrophilia, a high neutrophils/lymphocytes ratio, high Erythrocyte Sedimentation Rate, proteinuria, and a positive occult blood test. Inpatient therapy consisted of intravenous corticosteroids, intravenous Histamin-2 (H2) blockers, and oral nonsteroidal anti-inflammatory drugs. Upon discharge, all therapy was administered orally, with corticosteroids gradually tapered over 14 days. The patient showed clinical improvement without complications. ***Conclusion:*** HSP should be considered in the differential diagnosis of recurrent abdominal pain, even when of purpura and joint symptoms are absent initially. Early recognition can prevent diagnostic delays and reduce the risk of serious complications.

Keywords: henoch-schonlein purpura; abdominal pain; vasculitis; pediatric; delayed diagnosis

BACKGROUND

Henoch-Schonlein purpura (HSP), also known as immunoglobulin A (IgA) vasculitis, represents the most common form of childhood vasculitis. It predominantly affects small blood vessels and is characterized by the deposition of immune complexes. The etiology and pathophysiological mechanisms of HSP remain incompletely understood. However, it is widely believed to be the result of an aberrant inflammatory response to antigenic stimuli, particularly infectious agents, in genetically predisposed individuals [1]. The incidence of HSP ranged from 10-20 per 100.000 children per year, more than 90% of sufferers were under 10 years old, with a mean age of 6 years old [2]. The occurrence of HSP in infants is exceedingly rare. Among children, the condition shows a slight male predominance, with a male-to-female ratio of approximately 1.5:1, and its incidence declines progressively with increasing age [3].

A key clinical feature of HSP was a pressure or gravity-dependent nonthrombocytopenic purpuric or petechial rash. Other manifestations include abdominal pain, arthritis, and nephritis [4]. HSP can be confidently diagnosed in the presence of the characteristic purpuric rash; however, diagnosis becomes more challenging in patients who present with severe abdominal pain in the absence of cutaneous manifestations [5]. The prognosis was generally favorable, with most cases resolving without long-term complications. However, therapeutic intervention may be necessary during the acute phase, particularly in cases with gastrointestinal or renal involvement. Renal manifestations represent the most significant long-term complication and are responsible for approximately 1–2% of all cases of childhood end-stage kidney disease [3]. Although most cases of HSP were self-limiting, prompt diagnosis and appropriate management are crucial to reduce the risk of long-term complications. Careful monitoring

of evolving symptoms, as well as regular assessment of renal and gastrointestinal function, is essential to ensure optimal recovery for patients [6].

Here we report a case of HSP in a 10-year-old male who initially experienced recurrent abdominal pain that was not suspected to be HSP. The appearance of joint pain and purpura clarified the diagnosis; the patient recovered well after appropriate treatment. This case report aimed to highlight the diagnostic challenges of HSP when recurrent abdominal pain appeared with delayed onset of purpura and arthralgia.

CASE

A 10-year-old male initially complained of abdominal pain two days prior to hospital admission, accompanied by nausea, vomiting, and decreased appetite. There was no history of fever. His defecation and urination were normal. Two weeks before, he had experienced a cough and the flu. He had previously been treated at a general practitioner's clinic, but his symptoms did not improve. He had a history of asthma. The patient's delivery history was normal, the basic immunization history was complete, and the growth and development history were normal.

Clinical examination revealed a good nutritional status (BMI/age=+1.75 SD), moderately ill, alert, and the vital signs showed blood pressure 110/80 mmHg, pulse rate 90 times/minute, respiration rate 22 times/minute, temperature 36.6 degrees Celsius, SpO2 99% Room Air. On general status examination of the head, neck, thorax, and extremities were normal. For abdominal examination, on inspection, no distension was noted, auscultation of intestinal peristaltic sounds within normal limits, and on palpation found to be diffusely painful without signs of abnormal mass, hepatomegaly, or splenomegaly. Laboratory test results showed leukocytes 17.89 x 10^3/uL, hemoglobin 13.7 g/dL, platelets 344 x 10^3/uL, neutrophils 14.14 x 10^3/uL, lymphocytes 2.35 x 10^3/uL, monocytes 0.86 x 10^3/uL, neutrophils/ lymphocytes ratio 6.02; normal urinalysis (negative protein, nitrite, and ketone and normal sediment of erythrocytes and leukocytes). Abdominal ultrasound was in normal impression. Based on history, physical examination, and supporting examination, the patient was diagnosed with abdominal pain ec susp bacterial intestinal infection. The patient was treated for 4 days with the following medications: IVFD RL 20 gtt/min, then followed by D5 1/2NS 20gtt/min, cefotaxime 3x1 g intravenously, omeprazole 3x20 mg intravenously, ondansetron 3x4 mg intravenously, scopmaplus 2x1 caps, then replaced with paracetamol 3x400 mg orally, and antasida 3x5 ml orally. After gradually improving, the patient was discharged with sucralfate 3x5 ml, cefixime 2x200 mg, and paracetamol 3x400 mg orally.

After 4 days, he came for a check-up at the polyclinic, complaining of abdominal pain for 1 day before the check-up, along with not being able to defecate for 3 days, accompanied by reddish spots on his legs and buttocks and pain in the leg joints (ankle and knees), causing difficulty walking for 2 days before the check-up. Vital signs showed blood pressure 100/70 mmHg, pulse rate 92 times/minute, respiration rate 24 times/minute, temperature 36.5 degrees Celsius, SpO2 98% Room Air. On general status examination of the head, neck, and thorax, normal. Abdominal examination found diffuse tenderness. On his lower extremities and gluteus, we found non-itchy palpable purpura. The range of movement on his legs was limited. He was re-hospitalized to improve his condition. The patient underwent several laboratory tests with the following results: leukocytes 11.18 x 10^3/uL, hemoglobins 13.9 g/dL, platelets 346 x 10^3/uL, neutrophils 8.54 x 10^3/uL, lymphocytes 1.81 x 10^3/uL, monocytes 0.57 x 10^3/uL, neutrophils/lymphocytes ratio 4.72; urinalysis showed protein (+1), keton (+1), negative nitrit, and normal sediment of erythrocytes and leukocytes; urea 19 mg/dL, creatinine 0.5 mg/dL; Erythrocyte Sedimentation Rate 21mm/h; microscopic stool examination consist of leukocytes 2-4/hpf, erythrocytes 4-6/hpf; and Fecal Occult Blood Test was positive.

Based on history and various examinations, the patient was diagnosed with Henoch Schonlein Purpura and re-admitted with the following treatment: D5 ½ NS 15 gtt/min, metylprednisolone 3x12 mg intravenously, ranitidine 3x30 mg intravenously, Ibuprofen 3x360 mg orally, lactulosa 2x5 ml orally until the patient has defecated. The patient's condition improved after 4 days of treatment. After being discharged, he visited the polyclinic 3 times for evaluation of his condition. During the visit, his blood pressure was within normal limits. He underwent several examinations with the following findings: normal complete blood count and normal urinalysis. All his therapy was administered orally: ranitidine 3x150 mg, ibuprofen 3x360 mg, and methylprednisolone, which was gradually tapered over 14 days (3x6 mg for 7 days, 3x4 mg for 3 days, dan 3x2 mg for 4 days). The patient showed clinical improvement without complications.

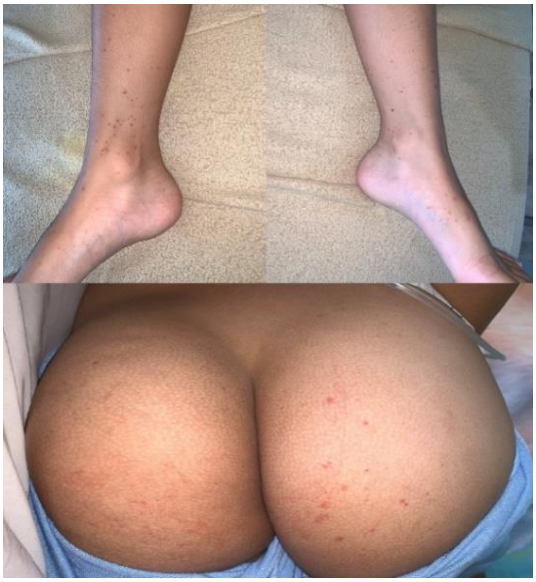

FIGURE 1: Palpable purpura on the lower extremities and gluteus area.

DISSCUSION

This report describes a 10-year-old male who initially presented with recurrent severe abdominal pain as the initial symptom, leading to a diagnosis other than HSP. However, once cutaneous and joint manifestations appeared, the patient was subsequently treated as HSP and demonstrated clinical improvement. HSP can affect individuals of any age, including adults, but it is predominantly seen in children, with an incidence ranging from 3 to 27 cases per 100,000 children. In childhood-onset HSP, approximately 90% of cases are diagnosed in children under the age of 10, and the peak incidence occurs between the ages of 4 and 6 years. The condition showed a slight male predominance, with a male-to-female ratio of approximately 1.5:1, and the incidence decreases with age [7].

HSP was a form of leukocytoclastic vasculitis driven by an antigen-induced elevation of immunoglobulin A (IgA) levels. This immune response results in the deposition of IgA-containing immune complexes within the vascular walls, which is most notable in the capillaries of affected organs and subsequent activation of the complement. Complement activation causes neutrophil recruitment and accumulation, culminating in inflammation and small-vessel vasculitis. The underlying IgA-mediated vascular injury is thought to arise from genetic predisposition and environmental triggers, including infections, pharmacologic agents, and vaccinations. Approximately 50–75% of patients diagnosed with HSP reported a history of upper respiratory tract infection, with *Streptococcus pyogenes* identified as the most frequently implicated pathogen.[8] In this case, the patient had an upper respiratory tract infection 2 weeks before his first admission, likely a risk factor for the patient to suffer from HSP.

The patient experienced diffuse abdominal pain accompanied by nausea, vomiting, and decreased appetite, without any history of fever or abnormalities in bowel or urinary habits. Gastrointestinal involvement was reported in 50–75% of pediatric patients with HSP. This may precede the appearance of purpura by a few days or a week in 10–20% of cases, and this can sometimes lead to clinical confusion until the rash appears. Abdominal pain represents the most frequent clinical manifestation and may present as a dull ache, but more commonly assumes a colicky quality. Although often diffuse, the pain may localize to the periumbilical or epigastric regions. Nausea and vomiting were frequent accompanying symptoms, while hematemesis and melena may also occur. Overt rectal bleeding is more likely when colonic vasculitis is present. These gastrointestinal manifestations arise from mesenteric vasculitis, which leads to visceral or peritoneal purpura and promotes leakage of blood and interstitial fluid into the bowel wall and lumen, occasionally resulting in bowel ischemia or infarction. Intussusception was the most common complication, occurring in approximately 3.5% of patients. Though uncommon, other complications include intestinal perforation, hemorrhagic ascites, acute acalculous cholecystitis, and acute pancreatitis [3,8].

Other signs that appeared later in our patient included non-itchy palpable purpura on his lower extremities and gluteus. A purpuric or petechial eruption was present in almost all patients with HSP. The rash constitutes the initial presenting feature in 50–75% of patients. The rash tends to localize to gravity-dependent and pressure-prone regions. Although the underlying mechanism remains uncertain, it has been suggested that gravitational forces promote the deposition of immune complexes in dependent areas, thereby triggering localized vascular inflammation [2]. In older children, the rash typically involves the ventral surfaces of the feet, ankles, and lower legs, whereas in younger children it more commonly affects the buttocks, back, and upper thighs. The eruption often begins as erythematous macules that evolve into petechiae or palpable purpura within 12–24 hours. The distribution is usually symmetrical. Characteristically, the lesions were palpable, may coalesce into larger plaques resembling ecchymoses. Pruritus or pain was typically absent. The color of the purpura progresses from red to purple and subsequently to brown tones as lesions resolve, a process that generally spans about 10 days. Blistering was uncommon, and hemorrhagic bullae or necrotic changes leading to ulceration occur rarely. Subcutaneous edema, reported in 35–70% of cases, is typically non-pitting and most frequently noted on the dorsal aspects of the hands and feet [8]. Besides palpable purpura, the patient felt joint pain in his knees and ankles, causing limited range of motion, which further supported the diagnosis of HSP. Arthralgia or arthritis occurs in approximately 65–85% of patients with the condition and represents the initial clinical manifestation in 17–25% of cases.

The likelihood of musculoskeletal manifestations shows an inverse relationship with age, with younger children exhibiting higher rates of joint symptoms. Large joints of the lower extremities, particularly the knees and ankles most frequently affected, whereas involvement of smaller joints, such as those of the fingers, as well as the spine, is relatively uncommon. The joint involvement was generally symmetrical and typically limited to fewer than four joints. Periarticular pain and swelling are common features, while erythema, warmth, and true joint effusion occur infrequently. Patients may experience a restricted range of motion in affected joints. Importantly, the arthralgia or arthritis is usually self-limited and does not result in long-term functional impairment [8].

Renal involvement develops in approximately 40–50% of patients, with 97% of affected individuals exhibiting renal manifestations within the first three months of disease onset. Hematuria, most often microscopic and typically asymptomatic, was the most frequent renal finding, underscoring the need for routine screening. Additional manifestations include proteinuria, nephrotic syndrome, and acute nephritic presentations accompanied by hypertension.

Renal colic has been described but remains a rare occurrence. Persistent hematuria and proteinuria are key predictors of progression to end-stage kidney disease. The risk factors for renal involvement include several clinical and laboratory features: severe abdominal pain with gastrointestinal bleeding, age > 5 years at onset, purpura persisting for more than one month, scrotal involvement, obesity, elevated serum IgA, heightened C-reactive protein (CRP), and reduced serum C3 levels. While nephropathy is generally mild in younger children, it tends to be more severe in older children (> 6 years) and adults [8]. Most cases of nephropathy in HSP were mild, with a favorable prognosis and complete recovery. Children who remain free of renal symptoms during the first six months after the initial presentation of HSP are unlikely to experience long-term renal sequelae [2]. In terms of signs and symptoms, our patient did not exhibit renal involvement. However, the patient had risk factors for nephropathy, including an age of 10 years and the presence of severe abdominal pain with occult gastrointestinal bleeding.

Although uncommon, Henoch–Schönlein purpura (HSP) may involve other organs. Neurological manifestations that typically emerge 2–4 weeks after disease onset. Patients with HSP may develop cerebral vasculitis. Headache, dizziness, and behavioral changes (irritability, hyperactivity, apathy, mood liability) develop in up to 30% of patients. Seizures, visual abnormalities, verbal disability, confusion, ataxia, and focal neurologic deficits are reported in only 2 to 8% of patients. Rarely, peripheral neuropathy, facial nerve palsy, myelopathy, intracranial hemorrhage, subarachnoid hemorrhage, subdural hematoma, cerebral venous thrombosis, Guillain-Barré syndrome, encephalopathy, and posterior reversible encephalopathy syndrome have been reported [2,8]. Pulmonary complications include diffuse alveolar hemorrhage, interstitial pneumonia, interstitial fibrosis, and pleural effusion. Cardiac manifestations, including myocarditis and myocardial necrosis/infarction, have rarely been reported in patients with HSP, which can be attributed to the vasculitis involving the myocardium. Ophthalmic manifestations such as engorgement of episcleral vessels, uveitis, and keratitis have rarely been reported in patients with HSP. Genital involvement, including orchitis, has been documented in 10–20% of boys with HSP. Clinical manifestations such as pain, tenderness, and swelling of the testis or scrotum may closely resemble the presentation of testicular torsion, necessitating careful diagnostic evaluation. Scrotal involvement is observed in approximately 20% of affected boys. Additional reported manifestations include penile edema, priapism, epididymitis, hematoma of the spermatic cord, testicular infarction, thrombosis of the spermatic veins, scrotal hematoma, and, in female patients was labial edema [8].

According to the classification criteria by the European League Against Rheumatism (EULAR), the Paediatric Rheumatology International Trials Organisation (PRINTO), and the Paediatric Rheumatology European Society (PRES), the diagnosis of HSP require the presence of a mandatory criteria: palpable purpura or petechiae with a predominance on the lower extremities, occurring in the absence of thrombocytopenia; accompanied by at least one of the following four features: (1) acute onset of diffuse and/or colicky abdominal pain; (2) arthritis (acute joint swelling or pain accompanied by restricted range of motion) or arthralgia (acute joint pain without restricted range of motion); (3) renal involvement characterized by hematuria (urine sediment showing >5 red cells per high-power field or red cell casts) and/or proteinuria (>0.3 g/24 hr; spot urine albumin to creatinine ratio >30 mmol/mg; or ≥2+ on dipstick); and (4) histopathological evidence of predominant IgA deposition [leukocytoclastic vasculitis with predominant IgA deposits (skin biopsy); or proliferative glomerulonephritis with predominant IgA deposits (kidney biopsy)]. These criteria demonstrate a sensitivity of 93% and a specificity of 89% [10]. Based on these criteria, our patient can be diagnosed with HSP, as the mandatory criteria of palpable purpura, along with acute diffuse abdominal pain and joint pain.

No single specific laboratory test that definitively establish the diagnosis of HSP. Serum IgA levels may provide supportive evidence, as elevations have been reported in approximately 50–70% of affected patients. Urinalysis should be performed in all patients, as it provides an important assessment of renal involvement. Because renal manifestations may emerge after other clinical features have resolved, continued urinary monitoring beyond the acute presentation is recommended [9]. Urinalysis may demonstrate dysmorphic erythrocytes, leukocytes, cellular casts, and/or proteinuria.

Elevated serum creatinine or blood urea nitrogen levels indicate renal impairment consistent with HSP-associated nephritis. Moreover, reduced serum total protein and albumin concentrations associated with proteinuria exceeding 1 g/m^2/day are suggestive of nephrotic syndrome [8]. At the time of readmission, our patient's urinalysis showed proteinuria (+1), while erythrocyte and leukocyte sediments were within normal limits. Renal function tests were also normal. Therefore, it can be concluded that this patient did not exhibit renal involvement.

The laboratory findings in our patient during hospitalization were leukocytosis, normal hemoglobin, normal platelet count, neutrophilia, high neutrophil-to-lymphocyte ratio, and high Erythrocyte Sedimentation Rate. A complete blood cell count and coagulation profile are required to exclude alternative causes of vasculitis and other conditions presenting with similar clinical features. Leukocytosis is frequently present. Hemoglobin levels are typically within normal limits, unless significant gastrointestinal, renal, or pulmonary hemorrhage occurs, a normochromic anemia may be observed.

A normal platelet count helps differentiate HSP from thrombocytopenic purpura, and normal coagulation parameters (prothrombin time, partial thromboplastin time, and bleeding time) distinguish HSP from primary hemorrhagic disorders. A study reported that elevated neutrophil counts and an increased neutrophil-to-lymphocyte ratio correlate with gastrointestinal involvement and nephritis. Additional research showed that leukocyte, neutrophil, and monocyte counts, as well as the neutrophil-to-lymphocyte ratio and C-reactive protein (CRP), were significantly elevated in patients with internal organ involvement relative to those without. Although CRP and Erythrocyte Sedimentation Rate (ESR) may be included as general inflammatory markers, they lack specificity and sensitivity for HSP. Elevated D-dimer and fibrin degradation products may reflect increased disease activity. Normal serum antinuclear antibody and complement (C3 and C4) levels help differentiate HSP from other vasculitides. In HSP, antinuclear antibody and rheumatoid factor tests yielded negative results, while complement levels (C3 and C4) were within normal limits [8].

Our patient had a positive fecal occult blood test. The result of abdominal ultrasonography was in normal impression. Stool testing for occult blood may be useful in patients with gastrointestinal symptoms. Abdominal ultrasonography was recommended for those with severe abdominal pain to rule out intestinal intussusception. Skin biopsy was generally unnecessary but may be considered in incomplete or atypical presentations. Renal biopsy is indicated in patients with severe proteinuria (>250 mg/mmol for at least four weeks, though shorter durations may still warrant consideration), persistent moderate proteinuria (100–250 mg/mmol), or reduced glomerular filtration rate [8]. The majority of HSP cases in children follow a self-limiting course. The principal objectives in the management of HSP: (1) alleviation of acute symptoms, (2) reduction of short-term morbidity, including abdominal complications that may necessitate surgical intervention, and (3) prevention of long-term renal insufficiency [11].

Skin involvement may require treatment when it presents with bullous lesions or necrotic rash. Recommendations for managing severe cutaneous manifestations with corticosteroids as first-line therapy and initiated promptly upon the appearance of bullae or concerning necrotic areas. Corticosteroids are administered orally at a dose of approximately 1 mg/kg/day. Quiet activities such as leg rest, along with optimal nutrition and sufficient hydration, were beneficial. Acetaminophen and nonsteroidal anti-inflammatory drugs (e.g., ibuprofen or naproxen) may be administered to relieve joint and soft-tissue pain. However, nonsteroidal anti-inflammatory drugs should be avoided in patients with active gastrointestinal bleeding or renal impairment due to their respective effects on platelet function and renal perfusion [8]. More severe cases have been reported in case series to respond to corticosteroid therapy at doses comparable to those recommended for severe cutaneous or gastrointestinal involvement. Additional immunosuppression is seldom necessary for musculoskeletal manifestations; however, immunosuppressive agents such as methotrexate, hydroxychloroquine, or dapsone may be considered when indicated [7]. Our patient had difficulty walking because of joint pain; therefore, NSAIDs (ibuprofen) were administered, with careful monitoring due to the presence of gastrointestinal involvement. Over the course of the illness, the patient demonstrated clinical improvement.

The role of corticosteroids in the management of HSP remains controversial. Current evidence does not support routine corticosteroid therapy for all patients with HSP, but selected clinical indications may warrant its use. Studies have demonstrated that corticosteroids can reduce the duration of abdominal pain and may help prevent serious complications, including gastrointestinal bleeding and intussusception. For gastrointestinal involvement cases, including acute abdomen or gastrointestinal bleeding, patients generally respond well to short-term oral prednisolone therapy. Prednisolone is administered at a dose of 1–2 mg/kg/day for one week, followed by a taper over the subsequent 2–3 weeks. Corticosteroids may also be indicated in cases of intrapulmonary hemorrhage, persistent nephrotic syndrome, marked scrotal edema, orchitis, or cerebral vasculitis. Nonetheless, the potential benefits of systemic steroid therapy must be balanced against its adverse effects. In line with the European SHARE (Single Hub and Access point for Paediatric Rheumatology in Europe) recommendations, systemic corticosteroids in combination with cytotoxic immunosuppressive agents (e.g., azathioprine, mycophenolate mofetil, cyclophosphamide) should be reserved for patients with organ- or life-threatening disease manifestations [3,8].

The patient in our case received corticosteroid therapy from the time HSP was diagnosed and subsequently demonstrated improvement in the acute symptoms. With monitoring throughout the course of therapy, no corticosteroid-related adverse effects were observed in our patient.

Overall, early initiation of oral corticosteroid therapy in patients with HSP has not been shown to prevent or attenuate the progression of renal disease. For children with severe nephritis or renal involvement accompanied by proteinuria lasting more than three months, the SHARE guidelines recommend considering angiotensin-converting enzyme inhibitors or angiotensin receptor blockers in addition to corticosteroids to prevent secondary glomerular injury. Immunosuppressive therapy may also have a role in patients with persistent or progressive proteinuria. The combination of corticosteroid and immunosuppressive regimens had higher recovery rates than those receiving corticosteroids alone. Additionally, combination therapy was significantly associated with remission of HSP nephritis [8].

While most patients with HSP exhibit renal involvement within the first three months of disease onset, a subset may develop renal manifestations at a later stage. Consequently, clinical follow-up should include weekly or biweekly urinalysis and blood pressure assessments during the initial months, with the frequency gradually reduced thereafter. As the disease begins to resolve, urinalysis and blood pressure monitoring should transition to monthly intervals, every other month for at least one year. Children who continue to demonstrate hematuria with or without proteinuria beyond six months require periodic evaluation of serum creatinine. For those with renal involvement, dietary sodium restriction was recommended to lessen the risk of hypertension [8]. After being discharged, our patient attended three follow-up visits, during which blood pressure remained within normal limits, and repeat complete blood count and urinalysis results were normal. With gradual tapering of corticosteroids, the patient showed good clinical improvement.

The prognosis of HSP was generally favorable, with renal involvement serving as a long-term complication, including chronic kidney disease (CKD), and the condition accounts for 1–2% of end-stage kidney disease [7]. The disease typically resolves within approximately four weeks, except in patients who develop persistent proteinuria or hematuria. Recurrence occurs in up to one-third of cases, particularly among those with renal involvement. Relapses most frequently arise within the first year following the initial episode, although they may occur several years later and may or may not be preceded by an upper respiratory infection. Long-term complications are uncommon but may include sustained hypertension and progression to end-stage kidney disease. A meta-analysis study identified several predictors of poor prognosis: older age at disease onset, reduced baseline glomerular filtration rate, initial presentation with nephrotic syndrome, nephritic–nephrotic syndrome, and crescentic nephritis on biopsy [8].

CONCLUSION

Henoch Schonlein Purpura should be considered in children presenting with recurrent abdominal pain, even in the absence of early purpura or joint manifestations. Timely recognition and appropriate management are essential to avoid diagnostic delays and to reduce the risk of potentially serious complications.

Conflict of Interest

The author declares that there is no conflict of interest related to the publication of this research article.

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Ethics

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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