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Henoch Schonlein Purpura: A Case Report

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Abstract

IMMUNOGLOBULIN A (Ig A) VASCULITIS formerly known as HENOCH-SCHONLEIN PURPSURA (HSP) is a multisystem disorder affecting the skin, joints, gastrointestinal tract and kidneys. It is a self-limited, systemic, immune complex mediated small vessel leukocytoclastic vasculitis characterized by non-thrombocytopenic palpable purpura, abdominal pain and arthritis. It is the most common vasculitis in children. Glomerulonephritis and gastrointestinal bleeding are the most commonly associated complications. Etiology of HSP is unclear but is associated with infections (bacterial, viral, parasitic), medications, vaccination, tumors, alpha-1-antitrypsin deficiency, familial Mediterranean fever. A 13-year-old female patient was admitted with chief complaints of rashes over both lower limbs along with abdominal pain for 10 days. Rashes are non-pruritic in nature. The patient was treated with tablet OMNACORTIL 35mg OD. The patient was discharged after recovering symptomatically.

Keywords: Henoch schonlein purpura; Arthritis; Immune mediated; Purpura

Introduction

Immunoglobulin A (IgA) vasculitis [1] formerly known as Henoch-Schonlein purpura is defined as a systemic, immune complex mediated, small vessel leukocytoclastic vasculitis characterized by nonthrombocytopenic palpable purpura, abdominal pain, and arthritis [2]. HSP is a multisystem disorder affecting the skin, joints, gastrointestinal tract and kidneys, although involvement of other organs can occur rarely [3]. It is vasculitis with IgA deposition in vessel walls leading to symptoms involving the skin, joints, intestines, and kidneys [1]. It is the most common vasculitis in children. IgA vasculitis is typically self-limited, but a subset of patients experiences a remitting-relapsing course [4]. Its etiology is unclear but is associated with infections (bacterial, viral, parasitic), medications, vaccination, tumors (non-small cell lung cancer, prostate cancer, and hematological malignancies), alpha-1-antitrypsin deficiency and Familial Mediterranean Fever [3]. Although the cause is unknown, HSP is often preceded by an acute infectious illness and has a seasonal pattern (non-summer months), providing strong evidence for an infectious trigger [1]. Numerous studies have linked disease predisposition, severity, and long-term morbidity with genes on portions of the HLA alleles. Glomerulonephritis and gastrointestinal bleeding are the most commonly associated complications. IgA vasculitis should be suspected in patients presenting with palpable purpura who also develop arthralgias (75%) and abdominal pain (50%-65%). The differential diagnosis includes immune thrombocytopenic purpura, bleeding disorders, medication reactions, senile purpura, meningococcal sepsis, familial Mediterranean fever, rocky mountain spotted fever, acute leukemia, bone marrow failure syndromes, and other vasculitides [2]. Laboratory investigations included a hemogram, serum electrolytes, blood urea and creatinine, total and differential serum proteins, serum cholesterol, complement (C3), antinuclear factor (ANF), antistreptolysin-O (ASO), C-reactive protein (CRP), coagulogram and timed urinary protein estimation. Skin biopsy from the involved skin was subjected to light microscopy and direct immunofluorescence studies. Percutaneous kidney biopsy was done in patients who had major renal involvement (nephrotic or nephritic illness) [4]. Treatment of HSP is self-limiting in 94% of children and 89% of adults. Symptomatic treatment will be sufficient for symptoms such as rash and arthritis. Acetaminophen and nonsteroidal anti-inflammatory drugs can be used. Aspirin should be avoided in children. Oral steroids are

indicated in patients with severe rash, edema, severe colicky abdominal pain (without nausea, vomiting), renal, scrotal, and testicular involvement. Plasmapheresis or high dose intravenous immunoglobulin therapy may be recommended for worsening renal function, and haemorrhage in the lungs and brain refractory to steroids and immunosuppressive drugs [5].

Case Report

A 13-year-old female patient was admitted with chief complaints of rashes over both lower limbs along with abdominal pain for 10 days. Rashes are non-pruritic in nature. She had a history of blood in stools for 5 days which was subsided. Bilateral knee joint pain was present which is associated with difficulty in walking. On physical examination diffuse erythema and pedal edema were present. Pedal edema was non pitting in nature. No other co morbidities were present and no previous history of drug allergy or food allergy either.

Diagnosis

Physical examination and laboratory tests were performed. Her laboratory investigations revealed hemoglobin 10.1g/dl, hematocrit 37.3%, neutrophills 66%, Platelet count 377 L, ESR 45mm/hr, serum creatinine 0.69mg/dl. Urine examination showed albumin ++, RBC 1-2 cells. C^3, C^4 levels decreased. CRP positive and tachycardia with pulse rate up to 130bpm. ANA profile and RA factor were negative. PT and APTT are in normal range. USG abdomen and Peripheral smear was normal. Urine for culture showed dysmorphic RBC. No history of chest pain or SOB or vomiting or red eye. On examination A^0, J^0, Cy^0, CL^0, PE^+, L_0.

Treatment

The patient was treated symptomatically and initiated corticosteroid therapy Tab. OMNACORTIL 35mgOD. The patient recovered symptomatically.

Conclusion

Henoch-Schonlein purpura is defined as a systemic, immune complex mediated, small vessel leukocytoclastic vasculitis characterized by nonthrombocytopenic palpable purpura, abdominal pain, and arthritis. HSP is a self-limiting disease in most cases. Treatment for HSP is symptomatic in mild cases with rash and arthritis which can be cured with NSAIDs. In severe cases with renal and other organ involvement corticosteroids and immunosuppressants are used.

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