Henoch-Schonlein Purpura: Unusual Manifestation of Hepatitis A

Sellouti M*, El Kassimi I², Radi A¹, Ourrai A¹, Hassani A¹, Abilkassem R¹, and Agadr A¹

1Department of Pediatrics, Mohammed V Military Teaching Hospital, Morocco
2Department of internal medicine, Mohammed V Military Teaching Hospital, Morocco

Abstract

Henoch-Schonlein purpura is a systemic IgA vasculitis that affects small vessels. It is characterized by cutaneous purpura associated with joint, abdominal and/or renal involvement. It is common in children and its etiology remains the result of immune complex reaction to various antigens, its association with viral hepatitis A unusual.

We report the case of a 12-year-old girl who developed cholestatic jaundice secondary to viral hepatitis A. Two weeks after her admission she presented with slender purpuric tasks of the lower limbs associated with arthralgia and abdominal pain. Histological study of skin biopsy confirmed the diagnosis of Henoch-Schonlein purpura. The evolution was spontaneously favorable.

This case highlights the fortuitous association of Henoch-Schonlein purpura and hepatitis A.

Keywords: Henoch-Schonlein purpura; Cholestatic jaundice; Hepatitis A

Introduction

Henoch-Schonlein purpura is the most common systemic vasculitis of children that affects small vessels [1]. It is characterized by a predominant purpura in sloping areas, accompanied by arthralgia and/or arthritis of the large joints, abdominal pain and renal involvement [2]. The cause is not well, but is thought to result from a immune complex response to various antigenic stimuli. The combination of Henoch-Schonlein purpura and viral hepatitis A infection has been described exceptionally in childhood [3,4]. The relationship between these two conditions, however, remains unclear: accidental association, “trigger” role or etiopathogenic factor: We report an observation of Henoch-Schonlein purpura that occurred during an acute infection with viral hepatitis A.

Case Presentation

A 12-year-old girl, admitted for acute abdominal pain with no particular medical history. She reported vomiting, anorexia, fatigue, dark urine, and jaundice. Clinical exam found normal anthropometric measurements. The patient was afebrile, with a blood pressure at 115/56 mm Hg, and heart rate at 77 beats/min. The abdominal examination was normal; the liver and the spleen were not palpable. The rest of the physical examination was normal.

Biological assays showed hyperleukocytosis at 15203/mm³ (59% lymphocytes and neutrophils at 41%), hemoglobin at 9g/dl, red blood cells at 4.1 10⁶/mm³, VGM at 70.2 μ3, 21.9μg TCMH, 31.9% MCHC, and platelet count at 544 10³/mm³. Hepatic cytolysis with SGOT at 1299UI/l and SGPT at 880UI/l. Total and direct bilirubin level respectively at 155 and 110μmol/l, alkaline phosphatase at 290UI/l, a prothrombin level at 42.9%; kaolin cephalin time at 30 seconds (control at 25 seconds); blood glucose level at 4.9 mmol/l; a cholesterolemia at 4 mmol/l; hypertriglyceridemia at 3.45mmol/l, ura at 4.2 mmol and creatininemia at 71μmol/l.

Hepatitis A serology was positive for IgM (IgM anti-HAV). Serology of Hepatitis B (antigen HBS, antibodyHBC) Hepatitis C (antibodyHCV) and EBV (IgM VCA and IgG VCA) were negative. The diagnosis of hepatitis A was retained. Two weeks later, the patient expressed purpuric and infiltrated lesions of the lower limbs (Figure 1) intensified by orthostatism associated to arthralgia and abdominal pain. The urine test strip didn't find proteinuria nor hematuria. The cryoglobulinemia test was negative. Skin biopsy showed a leukocytoclastic vasculitis.

After a follow-up of 6 months, the patient did not present other episodes of Henoch-Schonlein purpura or any renal impairment.

Discussion

In our case report, the diagnosis of Henoch-Schonlein purpura is retained on the basis of clinical characteristic (purpura, arthritis, and digestive manifestations) and histological findings (leucocytoclastic vasculitis on the cutaneous biopsy). The recent character of the viral hepatitis A infection is, for its part, authenticated by the presence of IgM controlled twice. The first case of Henoch-Schonlein purpura associated with viral hepatitis A has been reported in the literature by Garty [3]. Four other cases (three boys and one girl) have been reported since that date [4,5].
The mean age of these cases was 10.4 years and all had symptoms of viral hepatitis A associated with cutaneous manifestations of Henoch-Schönlein purpura. Extrahepatic manifestations during viral hepatitis occur in 15 to 20% of patients and take variable aspects. These events are reported much more frequently with hepatitis B than with hepatitis A and C [6]. Their physiopathology is not yet known but they would, for most authors, be the clinical expression of an autoimmune disorder.

The combination of Henoch-Schönlein purpura with hepatitis B or C is known despite its rarity [7,8]. However, the association of Henoch-Schönlein purpura with hepatitis A remains exceptional since only five cases are reported to date.

This case report highlights the unusual association of Henoch-Schönlein purpura and viral hepatitis A. The diagnosis of Henoch-Schönlein was retained on the basis of the American College of Rheumatology (ACR) criteria: age < 20 years, palpable purpura, acute abdominal pain, polymuclear infiltration of the vascular wall and perivascular at biopsy. These criteria do not include immunofluorescence data. This one typically shows during the Henoch-Schönlein a deposit of IgA in vessel walls. But, during hepatitis A, there is usually a deposit of IgM [4,9]. IgA vascular deposits found in other cases of Henoch-Schönlein purpura associated with hepatitis B or C are rare. Indeed, the deposits in cases associated with hepatitis B correspond to immune complexes containing the HBS antigen, anti-HBs IgMs, IgGs, complement fractions C3 and C4 [10,11].

**Conclusion**

In summary, we believe that hepatitis A infection may be associated with Henoch-Schönlein purpura. Hepatitis A essay should be established in every children with elevated transaminase levels especially in developing countries where hepatitis A is common.

**References**