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Case Report

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Leukocytoclastic Vasculitis as Presenting Manifestation of Chronic Hepatitis B Virus Infection

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Abstract

Leukocytoclastic vasculitis, also called leukocytoclastic cutaneous angiitis, is the most common form of vasculitis. Although most cases are idiopathic, etiologic agents include infectious agents, connective tissue diseases, drug hypersensitivity reactions, and solid or hematologic malignancies. Although infectious processes are a known cause of leukocytoclastic vasculitis, hepatitis B virus (HBV) infection is very rare. We present a 47-year-old woman, with no previous pathologic history, who consulted for arthralgias in the knees and ankles, myalgia's and purpuric rash with mild pruritus in both lower limbs, of one month evolution. Skin biopsy of lower extremity lesions was compatible with leukocytoclastic vasculitis. Hepatitis B serology was positive, so she started antiviral treatment with tenofovir and prednisone with good evolution of her skin lesions.

Keywords: Vasculitis, Hepatitis B virus

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Introduction

Leukocytoclastic vasculitis, also called leukocyocytoclastic cutaneous hostitis according to the revised nomenclature of the 2012 Chapel Hill consensus conference, is characterized by the inflammation of the blood vessels of small caliber, usually post-channel venules. The inflammatory infiltrate can be neutrophilic, lymphocyte or granulomatous. Neutrophils suffer degeneration or leukocytoclasia giving rise to the so-called nuclear dust. It is the most common form of vasculitis with an approximate incidence of 45 million annual cases the Worldwide, it is more frequent in adults and slightly more in women [1].

Although 45 - 55% of cases are idiopathic, among the etiological agents that we can name are infectious agents (15 - 20%), connective tissue diseases (15 - 20%), hypersensitivity reactions to medications (10 - 15%), or solid or hematological neoplasms (5%) [2]. While infectious processes are a known cause of leukocytoclastic vasculitis, HBV is very uncommon.

Case Presentation

A 47-year-old non-smoking woman, without previous pathological history, consulted for arthralgia on knees and ankles, myalgia in twins and purple rash with mild pruritus in both lower members, of a month of evolution (Figure 1).

Hematological, kidney parameters, acute phase reactants, thyroid hormones and urine sediment were normal. Hepatic enzymes were slightly increased oxalacetic glutaminase (TGO): 55.9 IU/L (< 34 IU/L), pyruvic glutaminase (TGP): 122.7 IU/L (10 - 41 IU/L), alkaline phosphatase: 157.4 UI /l (70 - 290 IU/L).

The autoantibodies including rheumatoid factor, antinuclear antibodies, ANCA, lupus inhibitor, anticardiolipin antibodies, anti β2 antibodies glycoprotein 1 and cryoglobulins were negative, as well as VDRL. The complement levels were slightly decreased C3: 91 mg/dl (103 - 145), and C4: 17 mg/dl (20 - 50).

Viral serologies for hepatitis C and HIV viruses were negative. However, the serology of hepatitis B reported the following Hbeag which is not reactive with Hbeac (anti-e) reagent. HBV-DNA viral load 7,120,000 IU/ml.

The cutaneous biopsy of lower member lesions was compatible with leukocytoclastic vasculitis. An abdominal ultrasound was normal, but fibro scan reported a F2 stadium compatible with liver fibrosis (F0 without fibrosis to F4 cirrhosis), correlating with chronic hepatitis. It initiated antiviral treatment with tenofovir 300 mg/day and prednisone 15 mg/day, the latter in decreasing dose, with good evolution of its skin lesions, persisting pigmented macules as a sequel.

Discussion

The World Health Organization (WHO) in 2015 estimated that 257 million people (3.5% of the population) lived with chronic infection due to the HBV. On the other hand, in the same year it was estimated that the viral hepatitis was the cause of death of 1.34 million people. Without treatment, HBV and C virus infections are the main causes of liver cirrhosis (720,000 deaths) and hepatocarcinoma (470,000 deaths). These chronic complications are the cause of 96% of deaths due to viral hepatitis [3].

Both acute and chronic forms of hepatitis B are associated with different extra hepatic complications, which can precede or be after liver manifestations. The pathophysiological mechanism is the deposit of immunocomplexes caused by the immune response against the virus. It is considered that up to a fifth of patients with chronic hepatitis B have extra hepatic manifestations, among which we can mention arthritis, glomerulonephritis and Raynaud phenomenon. In relation to

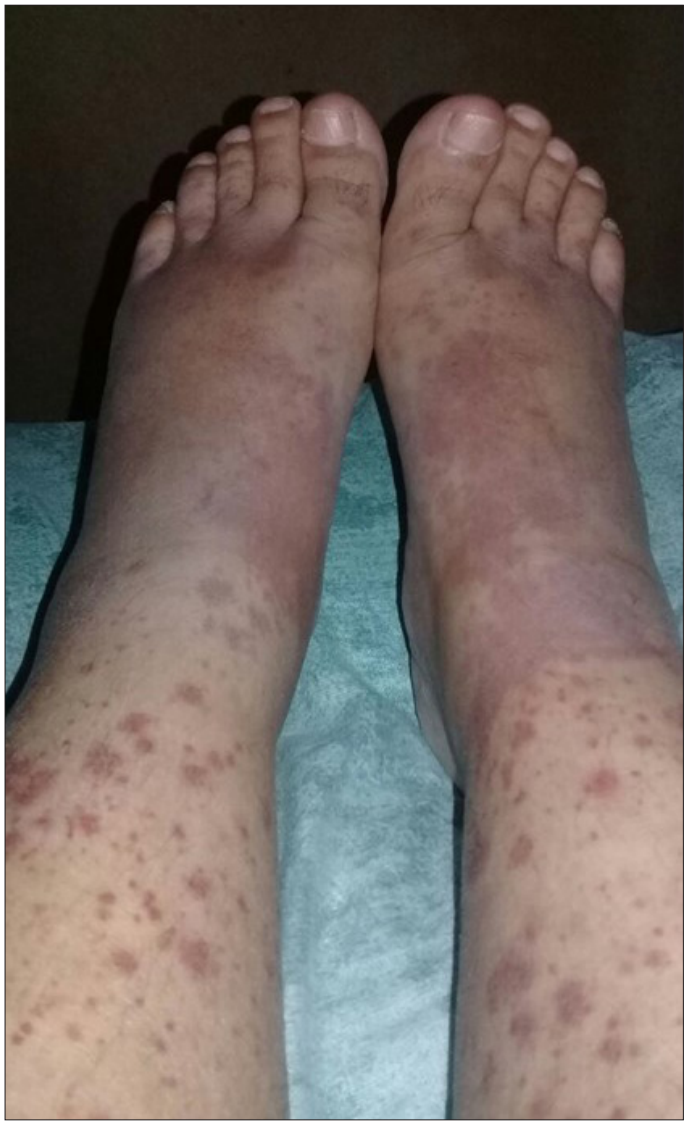

Figure 1: Purpuric rash compromising both lower members.

vasculitis, different types of them can be found, among which are nodous polyarteritis, cryoglobulinemic vasculitis and skin vasculitis. The latter are an infrequent manifestation, with an approximate incidence of 1%. Sometimes you can find HBB antigens in skin lesions [4].

In our patient the first manifestation of chronic hepatitis B infection was its skeletal and cutaneous muscle symptoms. An exhaustive search did not evidence systemic or clinical compatible with body polyarteritis. In our knowledge there is only one previous case in which isolated leukocyocytoclastic vasculitis was the form of presentation of chronic hepatitis, since in the other reported cases patients had systemic manifestations such as cryoglobulinemia [5].

The treatment includes antivirals and immunosuppressants in order to control on the one hand the viral load and on the other the formation of immunocomplexes. In our patient, the tenofovir association and low doses of prednisone resulted in the disappearance of symptoms.

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None.

Conflict of Interest

None.

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