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Rheumatica Acta: Open Access

DOI CC By

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Dates: Received: 21 December, 2016; **Accepted:** 02 January, 2017; **Published:** 4 January, 2017

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Keywords: Eosinophilia; Eosinophilic fasciitis; Infections; Myalgia; Hepatitis C virus

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Case Report

Eosinophilic Fasciitis in a Patient with Hepatitis C Virus Infection: Coincidence or Association?

Abstract

Eosinophilic fasciitis is a rare connective tissue disease characterized by symmetrical and painful swelling with a progressive induration and thickening of the skin and soft tissues. Its etiology is unknown, but possible causes or associated conditions include drugs, physical exercises, autoimmune diseases, neoplasia and infections.

Herein, we report, at the first time in the literature, the eosinophilic fasciitis in a patient with hepatitis C virus infection.

Introduction

Eosinophilic fasciitis is an uncommon disorder characterized by acute or subacute symmetric swelling of the skin and the subcutaneous tissues. The flanks, upper legs and forearms are usually affected, while the face and hands are spared [1-4].

The etiology of eosinophilic fasciitis is unknown, but possible causes or associated situations include drugs, extenuated physical exercises, autoimmune diseases, neoplasia and also infections [1,5-8].

However, until the present moment, there have no described cases of eosinophilic fasciitis in patient with hepatitis C virus infection.

Case Report

A 58 year old Caucasian male presented for evaluation of a three months history of diffuse and progressive myalgia associated with painful limitation of mobility. It evolved with lower limb edema and thickening of the forearms symmetrically (Figure 1), abdomen, and anterior neck. There was no history of Raynaud's phenomenon, dysphagia, muscle weakness, dyspnea, sclerodactyly, ulcer cutaneous nor L-tryptophan ingestion. Patient was sedentary.

At hospital admission, the vital signs were normal. Initial laboratory tests showed peripheral blood leukocytosis ($31.8x10^9$/L) with 45.2% of white blood cells being eosinophils; creatine phosphokinase of 34U/L (reference value: 24-173U/L); aldolase of 9.6U/L (<7.5U/L); erythrocyte sedimentation rate of 23mm/1st hour (6.2-13.2mm/1st hour); C-reactive protein of 131.0mg/L (<5.0mg/L); absence of hypergammaglobulinemia; negative anti-nuclear antibody test.

Magnetic resonance imaging with evidence of thickening, symmetric edema and enhancement of the deep and superficial fascia of the thigh and legs compartments (Figure 2), without collections. Vastus lateralis muscle biopsy was performed, showing an intense area of eosinophilic inflammatory infiltration in the perimysium region, with sparse foci of inflammation in the endomysia region and necrosis of muscle fibers.

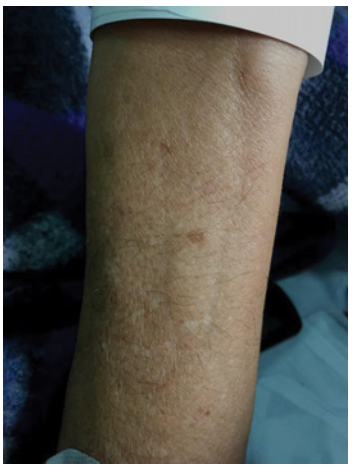

Figure 1: Typical groove sign (depressed veins aspect) of the forearm of a patient with eosinophilic fasciitis.

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Neoplastic and parasite causes were excluded.

Additionally, patient had positive hepatitis C virus serology (HCV PCR 4355 (3.64 log), genotype 1b). The liver enzyme levels were slightly elevated and liver ultrasound was homogeneous.

Initiated prednisone 1mg/kg/day and, after 14 days, there was significant improvement of myalgia and normalization of eosinophil fraction. Subsequently, as prednisone tapering, cyclosporine was initiated at 2mg/kg/day. Concerning to the virus C infection, he was followed up on an Outpatient basis by a hepatologist.

Discussion

We report the case of a patient with eosinophilic fasciitis and hepatitis C virus infection.

Eosinophilic fasciitis is a scleroderma-like syndrome that was first described by Shulman in 1974 [1-3]. Its onset it typically acute and findings include erythema, swellivng and induration of the extremities, usually accompanied by eosinophilia, polyclonal hypergammaglobulinemia and elevated erythrocyte sedimentation rate [9-11]. Typical histologic findings include chronic inflammatory infiltration affecting deep fascia with lymphocytes, histiocytic, and occasionally eosinophils [1]. Magnetic resonance imaging can be used for the diagnosis of eosinophilic fasciitis [14-16]. The classical is the presence of fascial thickening and signal abnormalities in patients at the time of diagnosis [14,15], similarly to the present case report.

The possible causes or associated situations include drugs, extenuated physical exercises, autoimmune diseases, neoplasia [1,5-8]. Concerning to infections, there are description of eosinophilic fasciitis associated to intestinal parasitic infection [17], *Mycoplasma arginini* (18), tuberculosis (19) and borrelia [5,20,21]. However, until the present moment, there have no described cases of eosinophilic fasciitis in patient with hepatitis C virus infection.

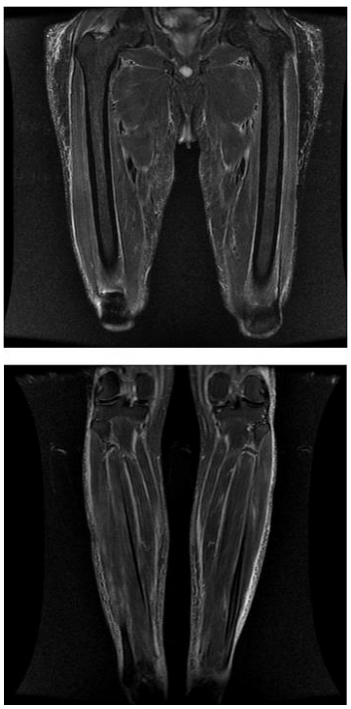

Figure 2: Magnetic resonance imaging of the thighs and legs demonstrated superficial and deep fascial thickening with T2 hyperintensity.

The association of eosinophilic fasciitis with infectious disease [5,17-21], especially hepatitis C virus, raises the question of a real link or a fortuitous association between the conditions. We suggest that in some patients, perhaps genetically predisposed, infections may be at the origin of fasciitis.

The glucocorticoid can be considered as the first-line treatment of eosinophilic fasciitis and are usually effective in more than 70% of cases. Other treatments included non-steroid anti-inflammatory drugs, chloroquine, cimetidine, azathioprine, cyclosporine A, infliximab, PUBA and UVA-1 [12,13]. In the present case, the patient had improvement of muscle symptoms with prednisone.

In summary, we reported for the first time an eosinophilic fasciitis patient with positive serology to hepatitis C virus. Basing on other studies that describe eosinophilic fasciitis and different infections [5,17-21], the hepatitis C virus may be at the trigger of fasciitis in our patient, who must have been genetically predisposed. However, more studies with reports or a series of cases will be necessary to evaluate the exact relationship between these two entities.

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