

Eosinophilic Fasciitis Induced by Fluvastatin

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A 65 - year- old woman without relevant diseases was evaluated for disseminated skin lesions after the beginning of fluvastatin therapy for hypercholesterolemia diagnosis.

After a week of fluvastatin treatment she developed generalized myalgia, starting by lower limbs and progressed rapidly involving also upper limbs so that fluvastatin was suspended. 4 months after the statin onset, showed severe swelling skin in arms demonstrating groove sign (Figure 1) and joint contractures which restricted normal movement at elbows, wrists, knees and ankles. Neither Raynaud phenomenon nor sclerodactilia was present [1-4].



Figure 1: Groove sign.

Laboratory data: Severe eosinophilia (30%) with normal count of leukocytes, red blood cells and platelets. Amylase, Lipase, aldolase, LDH, CK, LFTS, creatinine, urea, ions and total protein within the range of normalcy, ESR (54), PCR (1.33). Elevation of gamma globulin (1.79/dl). ANA, rheumatoid factor. ECA negative or normal. Proteinograma compatible with chronic inflammation. Evidence of respiratory function and DLCO normal. Glucocorticoidea therapy was begun.

It was requested skin biopsy, muscle and fascia, being informed by Pathological Anatomy as fasciitis and chronic myositis in evolution in muscle biopsy as well as dermal sclerosis in skin biopsy.

The Eosinophilic fasciitis is a syndrome of unknown etiology belonging to the sclerodermiform syndromes. It is characterized by inflammation and sclerosis of the deep fascia and the dermis, which are characterized by sclerodermiform injuries, peripheral eosinophilia, *Hypergammaglobulinemia* and increase of ESR. It is not known pathogenic mechanisms by which fibrosis appears in the fascia after such an active inflammatory process. The most frequent extra-articular manifestations are the joints. Skin biopsy including subcutaneous cellular tissue and fascia is necessary for the diagnosis. In the initial stages the inflammatory infiltrate is composed of plasma cells (Figure 2) and abundant eosinophils, which are more scarce or nonexistent in advanced lesions. Isolated fibrosis of miocitos can exist as well as areas of mild myositis. The presence of rimeed vacuoles (Figure 3) raises the differential diagnosis with inclusion body myositis, but in this entity, the facial and interstitial affectionation is not so striking in the aspect of fibrosis reparative.

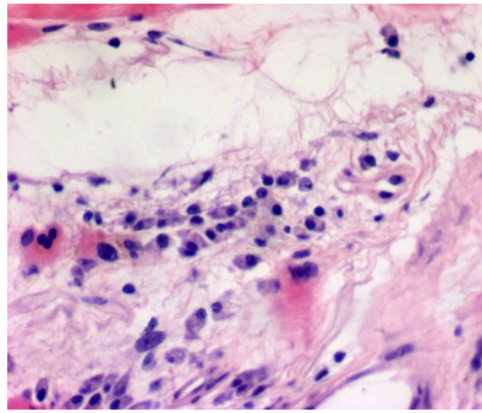


Figure 2: Hematoxylin- Eosine (x 40): severe interstitial infiltrate of plasmatic cells.

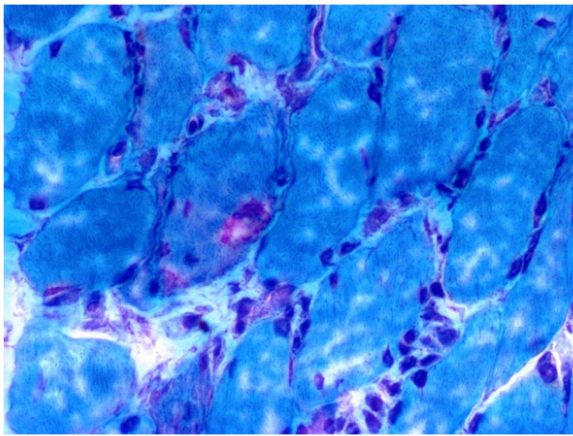


Figure 3: Rimmed Vacuoles.

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