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Case Report

# Neurologic Manifestations of Eosinophilic Fasciitis: Literature Review and Case Report

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## Abstract

**Introduction:** Eosinophilic fasciitis is a rare fibrous disease of the conjunctive tissue, and its typical presentation includes scleroderma-like signs and joint contractures. Neurological complaints are rarely associated with this condition.

**Objective:** Describing a case of painful brachial monoparesis as an initial manifestation of aneosinophilic fasciitis condition and reviewing its main clinical and therapeutic aspects.

**Case presentation:** A 52-year-old black male construction worker has presented painful brachial monoparesis condition. The symptoms begun in the palmar region of his right hand evolving to the whole extension of his right upper limb together with pain and restriction of the flexion and pronation movements. The patient also presented edema in all four limbs. There was a progressive worsening of pain and movements impairment in all his limbs for over 10 months. The condition was exhaustively investigated with extensive laboratory exams, skull and spine magnetic resonance imaging, electroneuromyography, and peripheral nerves high-resolution sonography. Eosinophilic fasciitis diagnosis was confirmed by skin and soft tissues biopsy. The patient was treated with corticosteroids with full clinical remission of his symptoms.

**Discussion:** The rich symptomatology of eosinophilic fasciitis is of interest to many different fields of medical expertise. The diagnosis poses a challenge requiring skin biopsy and findings such as eosinophilia in peripheral blood, elevated sedimentation rate velocity and hypergammaglobulinemia. A high degree of suspicion is necessary in patients with scleroderma-like syndrome and peripheral eosinophilia. The corticosteroids still represent the main therapeutical option for the treatment.

**Conclusion:** Eosinophilic fasciitis a rare disease with few case reports in medical literature. Peripheral neuropathies or plexopathies are atypical initial manifestations of unknown frequency.

**Keywords:** Eosinophilic fasciitis; Peripheral neuropathy; Eosinophilia; High-resolution sonography of peripheral nerves

## Introduction

Eosinophilic fasciitis is a rare disease characterized by hardening of the skin and subcutaneous tissue, eosinophilia, polyclonal hypergammaglobulinemia and high Hemo Sedimentation Velocity (HSV) [1]. The disease was initially described by Shulman in 1975 [2], with few reports published since then. Its physiopathology is not entirely clear, but it is believed that an autoimmune mechanism exists with progressive deposition and infiltration of eosinophilsin the fascia and subcutaneous cellular tissue. As a result, there occurs a local inflammatory response, activation of IFN-gamma, IL-5, IL-10, and tissue fibrosis [1].

Clinically, the disease is manifested by pain, edema, and progressive thickening of the skin and soft parts of the upper limbs and trunk. One of the classical findings is referred to as groove sign, which consists in linear depression of the skin in the path of the superficial veins, which is apparent mainly during the elevation of the affected limb, when occurs the reduction of the peripheral venous pressure [3-5]. The hardening of the subcutaneous tissues may result in restriction of joint movement or movement of closing fists (prayer sign), muscle contractures, joint retraction, and impairment of peripheral nerves [1].

The diseases representing a differential diagnosis include systemic sclerosis, and other forms of scleroderma, such as morphea, other epidemic fasciitis syndromes, such as the ones caused by toxic agents (e.g., myalgia-eosinophilia syndrome and toxic oil syndrome), T-cells peripheral lymphomas, etc. The final diagnosis is given by wedge skin biopsy, which shows deep inflammation and thickening of the fascia with infiltration of inflammatory cells [1-3,6-15].

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A case of eosinophilic fasciitis is reported which was presented to the Neurology Service of the HUCFF/UFRJ as a painful brachial monoparesis emphasizing the importance of a high degree of suspicion in patients with scleroderma-like syndrome and eosinophilia, regardless of the condition presentation form.

## Case Presentation

The patient is a 52-year-old black male construction worker born in Rio de Janeiro who lives in the same city. In November 2018, he first experienced painful paresis in the palm of his right hand, also experiencing restriction to fingers flexion and fist extension. Such manifestations evolved to the proximal region of the right upper limb with pain and restriction to pronation movement. In association with this condition, the subject presented symmetric edema in upper and lower limbs. There has been a progression to the contra lateral limb for 10 months. The patient was hospitalized at the Neurology Service of the HUCFF/UFRJ in August 2019 for further investigation of the condition, and brachialplexopathy was suspected.

The neurological exam presented atypical gait without postural instability. There was no impairment of tonus, coordination, superficial and deep sensibility or of the cranial nerves. The patient presented vivid reflexes in the lower limbs and in the left upper limb. There was an amplitude decrease in bicipital, tricipital and brachioradial reflexes of the right upper limb and skin depression in venous territories. The groove sign could be seen bilaterally in the arms, being more apparent upon elevation of the right upper limb (Figure 1A). Edema on the hands was also observed (Figure 1B) as well as an important restriction of joint movements of the right hand, mainly finger flexion and thumb abduction.

Laboratory tests have shown elevation of HSV (87 mm), eosinophilia with 648 cells/$mm^3$ (VR 40-500 cells/$mm^3$) and polyclonal hypergammaglobulinemia at 2.18 g/dL (VR 0.7-1.6). Collagenosis markers and serology for the Human Immunodeficiency Virus (HIV), syphilis and hepatitis were negative.

ENMG of the right upper limb revealed a decrease in amplitude of muscle action potential composed of the median nerve. HRUS done with a 12 MHZ transducer showed focal thickening of the median nerve in the right upper limb and the right brachial plexus (Figure 2). Skull and spine MR showed no alterations that could justify the condition.

In face of a patient with cutaneous thickening, restriction of joint movements, eosinophilia, increased HSV, and increase diameter of nerves and brachial plexus, it was suggested the hypothesis of eosinophilic fasciitis. With the purpose of confirming this hypothesis, biopsy was performed in skin wedge, subcutaneous tissues, and right forearm fascia, revealing the presence of inflammatory cells, including eosinophils, between the fibers of the muscle tissue (Figure 3).

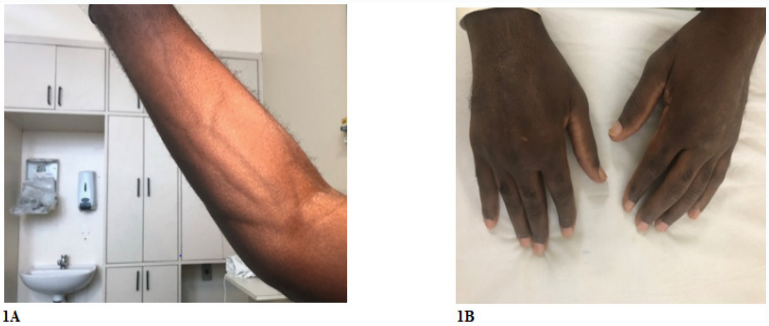
The patient was treated with prednisone tab initial dose of 1 mg/kg/day (80 mg/day total), with subsequent titration to 40 mg/day, and introduction of methotrexate (20 mg once a week). In a follow-up consultation after 3 months, the patient could perform movements of pronation, flexion, and extension of the limbs without difficulty. A mild edema remained in the upper limbs, and he reported a significant improvement in pain.

## Discussion

Eosinophilic fasciitis is a conjunctive tissue disease related to the deposition of eosinophils in the subcutaneous cellular tissue. The condition was described by Shulman [2] in 1975, with few published reports in contemporary literature since then. It is believed that the infiltration of eosinophils in the tissues results in a local inflammatory response with activation of cytokines and subsequent tissue fibrosis. As a result, skin and subcutaneous tissue hardening would occur, with accentuation of underlying venous territories [1-3]. Eosinophilic fasciitis etiology is not yet fully clarified, however, there are reports of the disease in patients with a history of high impact physical activity, infectious processes (Borreliaburgdorferi and Mycoplasmaarginini), toxicity by trichloroethylene and L-tryptophan, use of some medicaments (statins, lansoprazole and phenytoin) and hematological neoplasms such as lymphoma, leukemia and multiple myeloma [13,15,16].

The patient in question had a history of heavy work due to his job. In patients with suspected conditions, the report of physical activities or trauma is important since one of the mechanisms proposed as a triggering factor of the disease is the non-specific inflammation of an injured fascia, leading to an autoimmune response against antigens released by the tissue [11-14].

Clinically, the disease represents a differential diagnosis of scleroderma-like syndromes and collagenoses secondary to eosinophil infiltration, such as Gleich, and Churg-Strauss syndromes and the eosinophilia-myalgia syndrome associated with L-tryptophan intake [9]. The most characteristic semiological manifestations of eosinophilic fasciitis appear to be edema and skin hardening associated with depression in the superficial vascular territories ("groove sign") and lesions having an orange peel appearance ("peau d'orange") [3]. The skin hardening can cause tendons and joints to contract, with consequent paresis of the affected limb, including mimicking a primary neurological process. Systemic symptoms such as myalgia, asthenia and weight loss are also common [2,3,6].



1A 1B

**Figure 1**: Cutaneous signs of eosinophilic fasciitis. A) Groove sign. Depression of superficial vascular territoriesupon elevation of limb. B) Symmetric edemaon hand fingers.

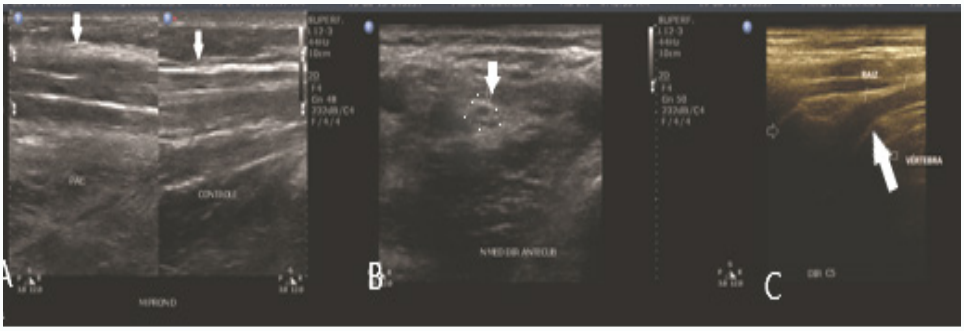


**Figure 2**: High-resolution ultrasound of soft parts, peripheral nerve, and nerve root. A) Fascia thickening of patient with eosinophilic fasciitis (left arrow) compared to the fascia of a subject without the disease (right arrow). B) Cross section of right median nerve in the forearm, with obvious increaseof someneural fascicles (arrow). C) Longitudinal section of thickened cervical nerve root.

Diagnostic criteria, severity score, and clinical guidelines for eosinophilic fasciitis were published in 2018 by Jinnin et al. [14] Currently, these guidelines serve as diagnostic criteria and reference to assess the severity of the disease (Tables 1 and 2).The most frequent laboratory characteristic is peripheral eosinophilia, present in up to 93% of patients [14]. It is usually transient and only occurs during the acute phase. It is believed that there is a correlation between the increase in eosinophils and disease activity since their levels decrease after beginning treatment. As eosinophilia is rarely seen in systemic sclerosis (about 7% of cases), this finding becomes useful information in differential diagnosis. Polyclonal hypergammaglobulinemia is less consistently present, having been reported in 3% to 72% of cases; in some patients it was correlated with disease activity [14,17]. However, an investigation by Seiboldet et al. [18], failed to demonstrate this correlation between hypergammaglobulinemia and disease activity in a significant way. High HSV, on the contrary, was found in approximately 29% to 80% of cases and was correlated with disease activity [18]. The enzyme creatine kinase generally has normal values, however, aldolase can rise to 60% of cases, and it has been reported that levels may drop after beginning treatment. Aldolase is considered an effective marker of disease activity by some authors [2,3,4-13].

MNR findings are valuable for the diagnosis of eosinophilic fasciitis and reveal thickening and hyperintensity of the superficial muscular fascia in T1, T2, and STIR sequences. After the administration of venous contrast, these findings are highlighted in T2 and STIR sequences. Most patients also present impairment of the deep muscular fascia [8,19]. MNR is the exam of choice in the differential diagnosis between eosinophilic fasciitis and myositis. In addition, it represents a technique of choice in selecting the biopsy site and treatment monitoring [8,14]. Soft tissue HRUS may show thickening of subcutaneous tissue and, eventually, of peripheral nerves, as in the case on display (Figure 2).

The final diagnosis is given by means of histopathological examination. In the disease initial stage, the typical findings are fascia and deep subcutaneous tissue edema with infiltration of several inflammatory cells such as lymphocytes, plasmocytes, histiocytes and eosinophils. As the disease progresses, the findings may proceed to epidermis atrophy, fascia thickening, and increased collagen bands in the subcutaneous tissue and deep layers of the dermis. Investigations from several research centers indicate that epidermal atrophy is seen in 16% of cases, including thickening of collagen bands (40% to 70%) and eosinophil infiltration (65% to 80%) [12-14]. In general, the fascia is thickened (2 to 15 times the normal size), well defined and adhered to the epimysium, with diffuse focal or perivascular inflammation with infiltration of lymphocytes and eosinophils [11,13,20].

One difference worth highlighting is that, while the dermis is the main location of fibrosis in patients with systemic sclerosis or localized scleroderma, in eosinophilic fasciitis fibrosis begins in the

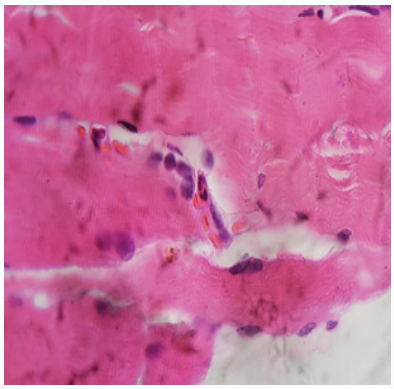


**Figure 3**: Biopsy in skin wedge, subcutaneous tissues, and right forearm fascia. The presence of eosinophils between the fibers of the muscle tissue can be noticed. Credits: Dr. Cynthia Bonacossa da Rocha Neves (Pathology Service of Clementino Fraga Filho Academic Hospital – HUCFF/UFRJ).

**Table 1:** Diagnostic criteria.

| Major criteria | Minor criteria |
|---|---|
| - Symmetric cutaneous thickening in plaques at the four ends. | - Histopathological examination with skin wedge biopsy showing subcutaneous tissue fibrosis, fascia thickening, and eosinophils and monocytes infiltration. |
| - Absence of Raynaud Phenomenon and exclusion of systemic sclerosis. | - Fascia thickening seen by means of imaging examinations such as magnetic resonance imaging. |
| The final diagnosis is made when the patient has one major criterion and at least one minor criterion. | |

**Table 2:** Severity score.

| | |
|---|---|
| Joint contraction (upper limbs) | 1 point |
| Joint contraction (lower limbs) | 1 point |
| Movement restriction (upper limbs) | 1 point |
| Movement restriction (lower limbs) | 1 point |
| Expansion and worsening of cutaneous rash (symptoms progression) | 1 point |
| A total of 2 or more points is scored as severe | |

fascia and subcutaneous cell tissue and only then extends to the deeper layers of the dermis. As such, an isolated skin biopsy that does not include deeper tissues may not close the diagnosis [14]. A report with six cases of eosinophilic fasciitis suggested that the presence of eosinophils in the fascia is restricted and transient, present only in the disease acute phase. Therefore, this histopathological finding is useful, but not essential for determining the disease diagnosis [21].

From a neurological point of view, there are few reports in the literature on peripheral neuropathies and polyneuropathy associated with eosinophilic fasciitis. The most frequently affected nerves are the median nerve and the posterior tibial nerve [10-12]. Cases of carpal tunnel syndrome by local tenosynovitic compression and tarsal tunnel syndrome due to impairment of the deep ankle fascia are also described. Some studies report the presence of eosinophilic fasciitis with an initial condition with lack of cutaneous changes and pain, but with painless progressive joint contractures. This presentation is accompanied by muscle weakness and may simulate myopathy [22]. Type 2A waist muscular dystrophy and Emery-Dreifuss muscular dystrophy may represent differential diagnoses in this context. Such a distinction is important because eosinophilic fasciitis is a treatable condition [22].

Regarding the impairment of the peripheral nerve, an unusual manifestation is multiple mononeuropathy. In patients with eosinophilic fasciitis there are few reports of this presentation, which is more commonly found in patients with eosinophilia-myalgia syndrome, particularly after consumption of preparations containing L-tryptophan [23]. In such cases, the severity of the neuromuscular pathology seems to be related to the level of ingestion of tryptophan. One of the proposed pathophysiological mechanisms is a direct neurotoxic effect of eosinophil infiltration into tissues or the action of eosinophil-derived neurotoxin [24,25]. Another proposed mechanism would be neurological damage by secondary vasculitis and impairment of vasa nervorum [9].

In the present case, in addition to symmetrical edema in the four limbs, hardening of the skin and peripheral eosinophilia, there was evidence of functional impairment of the median nerve and brachial plexus of the right upper limb. Such alteration was documented mainly by ultrasound, which showed an increase in diameter of these structures, possibly secondary to neural compression by the inflammatory process of supporting tissues. In this context, HRUS of peripheral nerves represented an important diagnostic aid.

A major review on the topic was published in 2016 by Wright et al. [6]. The authors stressed that, as it is a rare disease, it is common for the diagnosis to be a late one. The distinction from scleroderma is essential because corticosteroid therapy is considered a first-line treatment for eosinophilic fasciitis and should be used sparingly in scleroderma due to the risk of decompensating renal function through a scleroderma renal crisis [6,13]. Spontaneous remission can occur in up to 20% of patients after 2 to 5 years of illness [14]. However, there are several reports of symptoms recurrence after spontaneous resolution [26].

Treatment should include physiotherapy associated to immunomodulatory therapy. High doses of corticosteroids (immunosuppressive dose of prednisone 1 mg/kg) are described as the first line of treatment [6,27,28]. Since long-term use is necessary, corticosteroid-sparing medications, such as methotrexate, can be used [6] and, because of this, it was decided for the association of methotrexate, predicting that a prolonged use of the corticosteroid would be necessary. An effective response to pulse therapy with methylprednisolone has also been described [12,20].

In recurrent cases with incomplete response, hydroxychloroquine may be associated [6,13]. Other options include the combination of cyclosporine, D-penicillamine, and photochemotherapy [13,14]. Recently, TNF-alpha inhibitors, such as infliximab, have been tested in patient's refractory to corticosteroids and have shown beneficial effects. Furthermore, there are records of complete remission with cyclosporine in monotherapy and a combination of dapsone and prednisolone [18].

In several reports, pharmacological therapy was interrupted after improvement of skin lesions and serological tests, with complete remission of clinical symptoms [29,30]. Despite this, Lebeaux et al. [31] retrospectively investigated the clinical course of 34 patients with eosinophilic fasciitis and reported that 53% of those who were treated with oral corticosteroids and immunosuppressants did not experience recurrence of symptoms after discontinuation of medications. However, the others presented recurrence after decreasing the corticosteroid dosage. One report showed a 70% prevalence of recurrence after discontinuation of methotrexate, despite initial remission [32-35]. As a result, we conclude that, to date, there is insufficient evidence to guide the safe suspension of medication.

## Conclusion

Eosinophilic fasciitis is a rare fibrous disease, with few reports in national and international medical literature. A high degree of suspicion is necessary in patients with scleroderma-like syndrome, peripheral eosinophilia, polyclonal hypergammaglobulinemia and elevated inflammatory markers. Upon the institution of early treatment, there is a good chance of recovery.

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