Eosinophilic Fasciitis: A Case Report and Review of the Literature

Eozinofilik Fasiit: Bir Olgu Sunumu ve Literatürlerin Gözden Geçirilmesi

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Abstract
Eosinophilic fasciitis is a rare entity characterized by peripheral eosinophilia, fasciitis and "groove sign". The characteristic features of this inflammatory disease include scleroderma-like skin indurations, predominantly on the extremities, and peripheral blood eosinophilia. Systemic organs are generally not affected. In this paper, we present the clinical characteristics of a 35-year-old male patient who could not be diagnosed for a long period and was diagnosed as eosinophilic fasciitis following muscle biopsy. We also include herein the results of our literature survey regarding this disease. *(Turk J Rheumatol 2010; 25: 208-13)*
Key words: Eosinophilic fasciitis, groove sign

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Özet
Eozinofilik fasiit, periferik eozinofili, fasiit ve "Groove" işareti ile karakterize nadir görülen bir durumdur. Periferal kanda eozinofili ve özellikle ekstremitelerde skleroderma benzeri deri endurasyonları bu hastalığın karakteristik özellikleridir. Sistemik organlar genellikle etkilenmemiştir. Bu yazıda, uzun süre tanı konamayan ve kas biyopsisi sonrası eozinofilik fasit tanısı koyduğumuz 35 yaşındaki erkek olgunun klinik özellikleri ve hastalık hakkında yaptığımız literatür taramasının sonuçları sunulmuştur. *(Turk J Rheumatol 2010; 25: 208-13)*
Anahtar sözcükler: Eozinofilik fasiit, groove işareti
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Introduction

Eosinophilic fasciitis (EF) is a rare inflammatory disease of unknown etiology. This disease presents itself, just like scleroderma, with skin and connective tissue indurations in the extremities. It is usually seen in young males, rarely in elderly women and sporadically in children (1).

In some of the patients with EF, *Borrelia afzelii* antigen (2) and IgG antibody against *Borrelia burgdorferi* were found to be positive (3). Furthermore, nutrition with denatured oil (4) and predominantly L-tryptophan (5) and long-term use of lansoprazole (6) have been postulated to induce EF. Some of the cases are seen following bone marrow transplantation, and EF can also be seen in cases with Hashimoto thyroiditis and aplastic anemia, which bring into mind an autoimmune etiology (7).

Clinical signs of this disease have been reported as swelling, indurations and skin thickening in the legs and arms. During the course of the disease, there is edema in the extremities evolving into woody indurations characterized by orange-colored hyperpigmentation and skin tenseness. Typically the "groove sign" can be seen in the extremities. Some cases of localized morphea, defined as localized inflammation in the reticular dermis and superficial panniculus, have been reported. Synovitis and contractures can accompany the disease. Some patients also have signs such as muscle weakness, pain and rigidity in the extremities. EF shares similarities with many diseases; therefore, diagnosis is usually delayed and confused (8).

In this report, we aimed to present the clinical signs of an EF case with substantial limitation in extremity function.

Case Presentation

A 35-year-old male patient was referred to our polyclinic with complaints of rigidity and movement

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restriction in his arms and legs. These complaints had started four months ago with swelling in the dorsum of both hands and feet. In two months, the swelling had transformed to joint rigidity and movement restriction. The patient described that this muscle rigidity had gradually spread from the hands to the shoulders and from the feet to the hips. Personal history of the patient revealed cigarette smoking, 2-3 packets/day. Family history was negative for any allergic or rheumatic diseases. He had no known exposure to polyvinylchloride or L-tryptophan.

During examination, no rash was found on inspection. Systemic examination revealed blood pressure of 120/80 mmHg, pulse of 80 beats/min, respiration of 20/min, and a body temperature of 36.2°C. Examination of the musculoskeletal system showed sclerotic, thickened skin on legs and upper extremities. A groove sign was found on the right forearm (Figure 1). Neck and shoulder movements were moderately restricted and painless. Left elbow joint movement ranges were between 30-130 degrees. A 20 degree bilateral dorsiflexion was noted in the wrist joint. Bilateral metacarpophalangeal and proximal interphalangeal joints of the fingers had a flexion-extension movement range of 30 degrees. Moderate restriction was found in low back and hip motions. Both ankles were fixed at 90 degrees dorsiflexion. There was no sign of arthritis in the joints, and neurological examination of the patient was normal.

Laboratory tests revealed total peripheral blood leukocyte count of 11.000 with eosinophilia of 26.1% (n=1-3%). Peripheral blood smear showed eosinophilic predominance as well. Liver function tests were normal, and hepatitis markers were negative. Erythrocyte sedimentation rate (ESR) was 77 mm/hour and C-reactive protein (CRP) level 51 mg/dl. Rheumatoid factor and anti-nuclear antibodies (ANA) as well as salmonella and brucella agglutination tests were negative. Hypergammaglobulinemia was found in protein electrophoresis. The patient's tumor markers and thyroid function tests were within normal limits. No pathology was found in the microscopic and macroscopic examination of stool. Peripheral IgE level was within normal limits.

There was no pathologic sign in the lung X-rays, electrocardiographic examinations, abdominal ultrasonographic examination or in the electromyographic examinations of the upper and lower extremities.

In order to clarify peripheral eosinophilia and muscle rigidity, a subcutaneous and muscle biopsy was obtained from the calf. The histopathologic evaluation of the biopsy samples from the fascia and superficial striated muscle revealed an inflammatory infiltration comprising scarce eosinophilia, but a predominance of mononuclear cells and fibrosis in fascial soft tissue. Superficial spread of the infiltration into the muscle tissue was observed (Figure 2). These findings were reported as concordant with EF.

He was treated with fluocortolone 40 mg daily for two months, with no clinical benefit. Therefore, methotrexate (0.3 mg/kg/week) was added to fluocortolone treatment. With the aim of rehabilitation, stretching exercises were performed on the restricted joints. The patient was examined three months later and healing was observed in the rigidity of the upper and lower extremities, and joint movement range was almost complete. ESR and CRP levels were normal and there was no peripheral eosinophilia.

Discussion

Eosinophilic fasciitis (EF) is a rare disease, first identified in 1974 by Shulman (1). Despite the lack of studies on its incidence, we have screened a total of 309 studies on EF published in English in PubMed-MEDLINE from 1974 to the present. Among these publications, there were 223 cases diagnosed as EF. Of these, we could

Figure 1. Groove sign on the right forearm

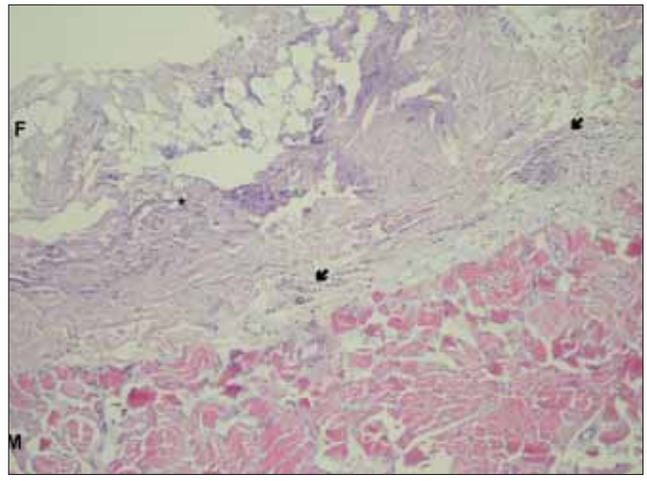

Figure 2. Inflammatory infiltration (arrow) with predominance of mononuclear cells and fibrosis (star) in fascial soft tissue (hematoxylin-eosin x200)

Table 1. Demographic and clinical characteristics of eosinophilic fasciitis cases in the literature (n=93)

Age [year; mean (SS)]		44.9 (17.7)
Min-max		6-80
Sex (F/M)		58/35
Hypergammaglobulinemia n (%)		
	Yes	42 (45.2)
	No	22 (23.7)
	Not reported	29 (31.2)
Elevated IgE level n (%)		
	Yes	2 (2.2)
	No	7 (7.5)
	Not reported	84 (90.3)
Peripheral blood eosinophilia n (%)		
	Yes	77 (82.8)
	No	6 (6.5)
	Not reported	10 (10.8)
Positive ANA n (%)		
	Yes	15 (16.1)
	No	46 (49.5)
	Not reported	32 (34.4)
Positive Scl-70 n (%)		
	Yes	2 (2.2)
	No	10 (10.8)
	Not reported	81 (87.1)
Positive Borrelia burgdorferi antibody n (%)		
	Yes	15 (16.1)
	No	10 (10.8)
	Not reported	68 (73.1)
Treatment n (%)		
	Prednisolone and methotrexate	10 (10.8)
	Cyclosporin	2 (2.2)
	OH-chloroquine	3 (3.3)
	Azathioprine and methylprednisolone	2 (2.2)
	Penicillin and prednisolone and vitamin E	3 (3.2)
	Prednisolone and cyclophosphamide	1 (1.1)
	Prednisolone	43 (46.2)
	Dexamethasone	1 (1.1)
	OH-chloroquine and methotrexate	1 (1.1)
	Prednisolone and OH-chloroquine	1 (1.1)
	Methotrexate and sulfasalazine	1 (1.1)
	Cyclosporin and methotrexate	1 (1.1)
	Methotrexate and OH-chloroquine and prednisolone	1 (1.1)
	PUVA and prednisolone	1 (1.1)
	Photochemotherapy	5 (5.4)
	Prednisolone and cimetidine and cyclosporin	1 (1.1)
	Dexamethasone and pamidronate	1 (1.1)
	Prednisolone and D penicillamine	5 (5.4)
	Methylprednisolone and interferon alpha	1 (1.1)
	Methylprednisolone	1 (1.1)
	Bone marrow transplantation	1 (1.1)
	D-penicillamine	1 (1.1)
	Methotrexate	1 (1.1)
	OH-chloroquine and prednisolone and cimetidine	1 (1.1)
	OH-chloroquine and prednisolone and fasciectomy	1 (1.1)
	Penicillamine and azathioprine	1 (1.1)
	Indomethacin	1 (1.1)
	Deflazacort	1 (1.1)
Response to treatment n (%)		
	Yes	78 (83.9)
	No	7 (7.5)
	Partial	8 (8.6)

F: Female, M: Male, ANA: Anti-nuclear antibody

reach only 93 cases with full text, and the clinical characteristics of these cases are given in Table 1 (8-65).

Cutaneous manifestations of EF have been described as progressive, with the earliest stage being edema of the extremities, followed by peau d'orange with hyperpigmentation, and finally induration (9). EF manifests itself with pain, edema, movement restriction, and skin thickening in the joints of the hands and feet. In some cases, these symptoms not only appear in the hands and the feet but can also comprise the extremities and the whole body. Rarely, conditions like splenomegaly, reactive hepatitis, esophageal dysmotility, peripheral neuropathy, autoimmune thyroiditis, hemolytic anemia, aplastic anemia, and thrombocytopenia can accompany the disease (9). In our case, there was movement restriction in the upper and lower extremities and thickening of the skin. The groove sign was seen on the right forearm. Based on our evaluation, there was no sign suggesting any systemic involvement.

Eosinophilic fasciitis can be seen along with other diseases. It has been reported along with collagen tissue diseases such as systemic sclerosis, Sjögren's syndrome, antiphospholipid syndrome, and systemic lupus erythematosus (10). Furthermore, EF has been reported as a paraneoplastic syndrome in some types of cancer such as colorectal carcinoma (11). In our case, there was no evidence of collagen tissue disease or cancer.

Laboratory findings of our patient revealed peripheral eosinophilia and IgE values within normal limits. Similar to our case's results, four of the five cases reported by Bobrowska-Snarska et al. (12) had normal IgE levels, and peripheral eosinophilia was reported in three of the five cases. Shulman (1) reported that hypergammaglobulinemia can be seen in these patients. Hypergammaglobulinemia was also observed in the protein electrophoresis of our case. In the 93 cases we evaluated, 45.2% had hypergammaglobulinemia and 82.8% had peripheral blood eosinophilia.

Muscle biopsy taken from the completely thickened skin area is the gold standard for diagnosis of EF. While there is eosinophilic and lymphocytic infiltration in the fascia, thickening due to the dense accumulation of collagen in the dermis is a typical pathological biopsy finding (9). Subcutaneous and muscle biopsy of our case also revealed inflammatory infiltration with eosinophilia and mostly a predominance of mononuclear cells as well as fibrosis in the fascial soft tissue.

Currently, there is no known effective treatment for EF. The best results have been observed with corticosteroid treatment. In cases resistant to corticosteroids, hydroxychloroquine, azathioprine (66) and methotrexate have been used (67). In some other studies, favorable results were obtained with cyclophosphamide, cyclosporine A and antithymocyte globulin application (13, 68, 69). Weaker effects have been reported for colchicine and D-penicillamine (67). Interestingly, good results in treatments with histamine receptor antagonists (cetirizine and cimetidine) have been observed as well (70). Favorable results were also obtained in some cases from UV-A photochemotherapy and psoralen treatment (14). In some other cases, dramatic improvements in clinical findings were observed with hydroxychloroquine treatment (15). On the other hand, in the treatment results of a series of 12 cases, Bischoff et al. (9) emphasized that 13 months of combination therapy with hydroxychloroquine and corticosteroids had no healing effect at all. Fluocortolone at a dose of 40 mg daily for two months was not effective in our case. Therefore, methotrexate (0.3 mg/kg/week) was added to the corticosteroid treatment. Clinical improvement was observed in a short period of combination therapy with corticosteroid and methotrexate. There are several studies reporting clinical improvement with corticosteroid and methotrexate combination therapy, similar to the situation in our case (16, 17). In 93 cases we evaluated from the literature, the treatment consisted mostly of prednisolone (46.2%) and prednisolone-methotrexate combination therapies (10.8%).

Most of the patients with EF are diagnosed as scleroderma due to skin indurations and fibrotic changes in tissues. Typically, in cases with EF, there are sensitive edematous and erythematous lesions in the extremities, with sudden onset. On the other hand, scleroderma is an insidious and slowly progressing disease. In contrast to scleroderma, sclerodactyly, Raynaud's phenomenon and visceral organ involvement are uncommon in EF patients. Different etiologic features and characteristics and different skin histologies differentiate these two diseases. EF can be differentiated from other diseases with capillary microscopy. Muscle weakness can be seen in cases with EF and can be confused with eosinophilic myalgia syndrome and myopathies. In cases with eosinophilic myalgia, pulmonary, cardiac, gastrointestinal, and neurologic signs are more acute and intense. In addition, increasing aldolase level, a muscle enzyme, is observed, differing from EF. The frequent confusion of EF with these diseases results in a delayed diagnosis (18-20). In our case as well, scleroderma was the suspected diagnosis, and EF was diagnosed through clinical findings and biopsy. However, aldolase levels could not be measured in our case.

In conclusion, the diagnosis of EF disease with routine clinical observation can be overlooked and therefore delayed. Good knowledge of the clinical features and early treatment can slow down the disease progression, which is substantially important regarding improvement in activities of daily life.

Conflict of Interest

No conflict of interest declared by the authors.

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