

Case report

Eosinophilic Fasciitis Following SARS-CoV-2 Infection: A Case Report and Review of Temporal Associations

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Running title: Eosinophilic Fasciitis after COVID-19 Infection

Abstract

Background: Eosinophilic fasciitis is a rare scleroderma-like disorder characterized by inflammation and thickening of the fascia, typically presenting with skin induration, peripheral eosinophilia, and absence of systemic organ involvement. Its etiology remains unclear, although immune-mediated mechanisms and various triggers have been proposed. **Case presentation:** We report a case of a 49-year-old woman who developed progressive pain, stiffness, and skin induration of the upper extremities shortly after COVID-19 infection. Laboratory evaluation revealed eosinophilia, acute phase reactants and negative autoimmune serology. Magnetic resonance imaging demonstrated fascial involvement without muscle pathology. Histopathological examination confirmed eosinophilic fasciitis, showing inflammatory infiltrates and fibrosis within the fascia. The

Received: Apr 1st 2026
Accepted: Apr 15th 2026
Published: May 1st 2026
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How to cite: Markov, M., Kosturkova, M., Dimova, M., & Velikov, V. (2026). Eosinophilic Fasciitis Following SARS-CoV-2 Infection: A Case Report and Review of Temporal Associations. *Rheumatology (Bulgaria)*, 34(1), 65-76. <https://doi.org/10.35465/b54ksv66>



patient was treated with high-dose glucocorticoids and steroid-sparing agent – methotrexate, with subsequent clinical and laboratory improvement. At follow-up, complete resolution of symptoms was observed, reaching drug-free remission after approximately 18 months. Conclusion: This case highlights eosinophilic fasciitis as a potential post-infectious immune-mediated condition following COVID-19. Early recognition, appropriate imaging, and confirmatory biopsy are essential for timely diagnosis, appropriate treatment and favorable outcomes.

Keywords: Eosinophilic fasciitis; COVID-19; post-infectious immune-mediated disease; case report

1. Introduction

Eosinophilic fasciitis (EF) is a rare scleroderma-like disorder involving inflammation and thickening of the fascia, often accompanied by eosinophilia and hypergammaglobulinemia. It was first described by Shulman in 1974 [1], and after the first big clinical cohort was reported in 1988 [2], EF was largely accepted as a separate entity. Unlike systemic sclerosis, it typically spares the face and internal organs and is not associated with Raynaud's phenomenon or specific autoantibodies. It presents clinically with rapidly progressive skin induration and limb stiffness, sometimes leading to contractures. Diagnosis is based on histological evidence of fascial inflammation and thickening [3].

The exact etiology of eosinophilic fasciitis remains uncertain, although an immune-mediated mechanism is strongly suspected. This assumption is supported by the frequent presence of hypergammaglobulinemia, occasional detection of autoantibodies, and the generally favorable response to corticosteroid therapy [4,5]. Various predisposing factors have been described, including local tissue injury, exposure to certain medications, infectious agents, especially viruses, underlying hematological or solid malignancies, and coexisting autoimmune disorders.

We present a case of eosinophilic fasciitis following COVID-19 infection, emphasizing the potential role of viral-triggered immune dysregulation in disease pathogenesis.

2. Case Presentation

A 49-year-old woman presented with progressive pain and stiffness of the shoulder girdle, upper limbs, abdominal muscles, and hips, beginning in

January 2024 2 weeks after COVID-19 infection. Symptoms initially involved painful, restricted shoulder movement, later extending to the arms and forearms. Within a week, she developed right arm swelling with forearm and lateral elbow skin induration, followed by milder similar changes on the left. Her past medical history was unremarkable.



Figure 1. Clinical photograph of the right forearm demonstrating localized skin induration and hyperpigmentation along the ulnar aspect, corresponding to the area of maximal involvement. The skin appears thickened and firmly attached to the underlying tissues, also demonstrating the “groove sign” —a longitudinal depression of the skin along blood vessels. A biopsy was obtained from this region for histopathological confirmation.

On admission, the patient was afebrile and in good general condition. Examination revealed painful restriction of both active and passive shoulder movements and painful active movement of the right wrist. Firm induration was present along the ulnar aspect of the right forearm and right lateral epicondyle, with milder involvement of the left forearm. No peripheral joint swelling was observed. During follow-up, induration extended to right forearm and elbow with hyperpigmentation and induration.

Laboratory evaluation revealed inflammatory activity and eosinophilia: ESR 50 mm/h (normal <20), CRP 38.1 mg/L (normal <5), leukocytes $13.73 \times 10^9/L$ (normal 4–10), platelets $540 \times 10^9/L$ (normal 150–400), eosinophils 10% (normal <5), and elevated LDH (491 U/L), while creatine kinase remained within the normal range. Liver and renal function were normal. ANA was positive at low titer (1:80, granular), while extended autoantibody

panel was negative. Complement and immunoglobulin levels were within normal ranges. Infectious screening (HBV, HCV, QuantiFERON-TB) was negative, and no hematologic malignancy was identified.

MRI of the right forearm demonstrated increased T2 signal intensity along the fascial planes, with contrast enhancement following administration of contrast medium. No fluid collections were observed. The adjacent muscle groups showed preserved signal intensity, without evidence of myositis. These findings were consistent with isolated fascial inflammation and supported the diagnosis of eosinophilic fasciitis (Figure 2).

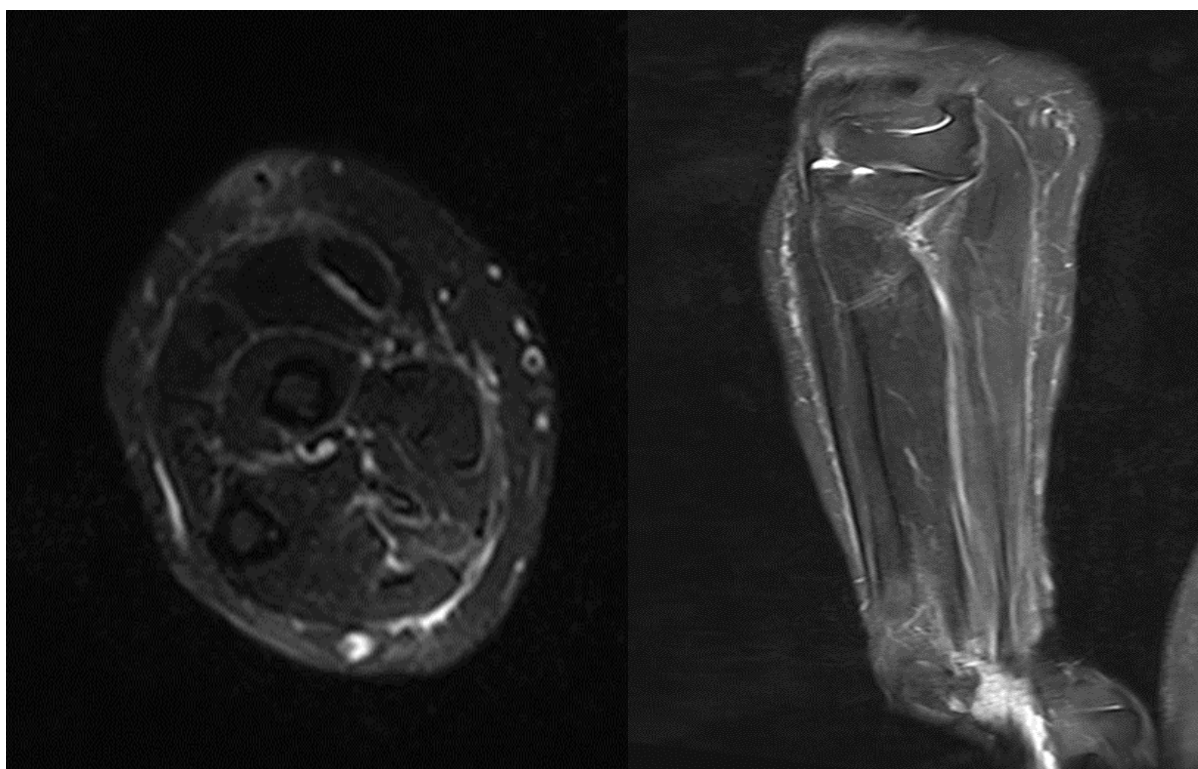


Figure 2. Axial T2-weighted fat-suppressed magnetic resonance imaging (MRI) of the right forearm demonstrating increased signal intensity and thickening along the fascial planes, consistent with inflammatory involvement of the fascia. The adjacent muscle groups show preserved signal intensity, without evidence of myositis or fluid collections. These findings are in keeping with eosinophilic fasciitis.

An incisional biopsy of skin, subcutis, and fascia from the right arm (12 April 2024) showed fibrotic changes with sparse lymphocytic infiltrates in the dermis and subcutis, and mixed inflammatory infiltrates in the fascia (lymphocytes, plasma cells, neutrophils, eosinophils) with focal necrosis and fibrosis, consistent with eosinophilic fasciitis.

The diagnosis of diffuse eosinophilic fasciitis was established based on the clinical presentation, peripheral eosinophilia, negative autoimmune serology, MRI evidence of fascial involvement, and confirmatory histology. Systemic sclerosis and inflammatory myopathy were excluded due to absence of Raynaud's phenomenon, sclerodactyly, internal organ involvement, and normal muscle enzymes.

Treatment was initiated with intravenous methylprednisolone at approximately 1.5 mg/kg/day, resulting in rapid clinical improvement. The patient was subsequently discharged on oral methylprednisolone 64 mg/day with gradual tapering. Methotrexate (15 mg/week) was introduced early as a steroid-sparing agent, together with folic acid supplementation. By October 2024, inflammatory markers and eosinophil count had normalized, with partial functional recovery under tapering glucocorticoids. Subsequent follow-up showed continued improvement, allowing discontinuation of glucocorticoids and gradual methotrexate taper.

At the latest follow-up (March 2026), complete clinical remission was achieved, with full recovery of range of motion, resolution of skin induration, normal laboratory parameters, and no evidence of relapse off therapy (Figure 3).

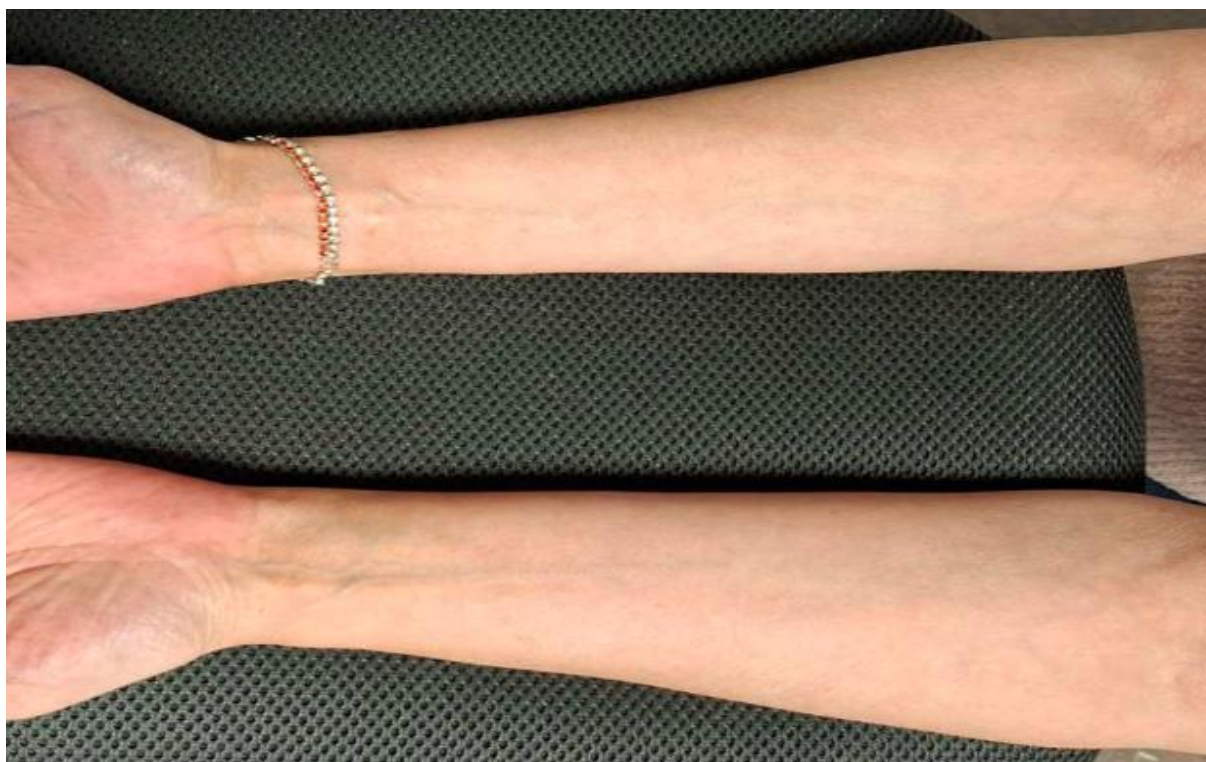


Figure 3: Clinical appearance of both forearms at follow-up showing complete resolution of previous skin induration. The skin is soft, with normal contour and mobility, without residual thickening, hyperpigmentation, or contractures, indicating full clinical remission

Given the close temporal relationship between symptom onset and recent COVID-19 infection, together with the absence of other evident causes, COVID-19 was considered a possible triggering factor.

3. Discussion

Although no universally accepted diagnostic criteria for eosinophilic fasciitis exist, several classification approaches have been proposed, including those by Bennett et al., Endo et al., Pinal-Fernandez et al., and Jinnin et al. [5,8-10] Despite some differences in structure, these frameworks consistently emphasize a characteristic pattern of limb or truncal induration, exclusion of systemic sclerosis, peripheral eosinophilia or inflammatory abnormalities, imaging evidence of fascial involvement, and histopathological confirmation by full-thickness biopsy. Our patient fulfilled these core diagnostic elements. Given the rarity of the disease, none of the sets was validated in larger cohorts.

Since the first described patients in the late 1970s, temporal associations with possible etiological/triggering implications were reported for many factors (Table 1).

Table 1. Reported precipitating and associated factors in eosinophilic fasciitis, with the typical level of supporting evidence and representative key references.

Factor	Typical supporting evidence	Key references
Vigorous physical exercise	Repeatedly reported across reviews, cohorts, and case reports; often temporally linked by days to weeks	[2], [11], [12], [13]
Trauma or mechanical stress	Recurrent association in reviews and clinical summaries; evidence is observational rather than controlled.	[11], [13]
Hematologic malignancy	Systematic review identified 104 malignancies among 1,703 EF patients, with many hematologic cancers; multiple case reports support temporal linkage.	[15], [16]
Solid tumors	Included in malignancy systematic review, but less frequent than hematologic cancers.	[15], [16]
Specific autoimmune comorbidities	Co-reported conditions include lupus , primary biliary cirrhosis, and thyroid autoimmunity, mostly from case-based literature.	[17], [18], [19]
Immune checkpoint inhibitors	PD-1/PD-L1 blockade – nivolumab, pembrolizumab	[20], [21], [22]

Other medications	Reported drugs include statins, heparin, phenytoin, carbidopa, anti-TNF agents – certolizumab [12]; mostly case reports or small clusters.	[23]
Borrelia burgdorferi / Lyme disease	Reported in some reviews and case reports, but causal inference is limited and may vary by endemic area.	[24]
Radiation therapy	Reported in review summaries and isolated case-based literature.	[25]
Burns / local tissue damage	Based mainly on isolated reports and review mentions.	[26]
Graft-versus-host disease / post-transplant state	Reported in small numbers; difficult to separate from sclerodermoid GVHD or malignancy-related disease.	[27]
COVID-19 infection	Case-report and case-series	[7], [28], [32]
EBV infection	Case report	[29]
Idiopathic	Modern reviews and series still note that many patients have no clear precipitating factor.	[2], [11]

EF: Eosinophilic Fasciitis, PD-1: Programmed cell death protein 1, PD-L1: Programmed death-ligand 1, anti-TNF: Anti-tumor necrosis factor, GVHD: Graft-versus-host disease, COVID-19: Coronavirus disease 2019, EBV: Epstein-Barr virus.

A consistent body of evidence indicates that eosinophilic fasciitis is frequently preceded by mechanical triggers, particularly strenuous physical exertion or localized trauma. These observations suggest that repetitive fascial microinjury may initiate an aberrant immune response characterized by eosinophil recruitment and subsequent fibrotic remodeling of the fascia.

Other triggers, described in the scientific literature, are malignancies, autoimmune conditions, drug-induced cases, with evidence supporting strongly association with immune check-point inhibitors, specifically PD-1 and PD-L1 blockers, and radiation therapy. Given the known association between eosinophilic fasciitis and malignancy, hematologic evaluation was performed and did not reveal evidence of a hematologic disorder. In addition, the patient had no B symptoms, no hepatosplenomegaly, and no suspicious findings on chest radiography or abdominal ultrasonography. Therefore, no further oncological work-up was considered clinically indicated at that stage.

Viral infections have been increasingly implicated as potential triggers of eosinophilic fasciitis, particularly in the context of SARS-CoV-2. Reviews and case-reports implicate also boreliosis and EBV-infection but the data is scarce and causal relationship cannot be estimated.

Accumulating epidemiological evidence supports a potential association between SARS-CoV-2 infection and the subsequent development of autoimmune and autoinflammatory diseases [30, 31].

From a clinical care perspective, our case highlights a potential pitfall in the evaluation of patients after COVID-19. Early symptoms such as pain, stiffness, and fatigue are frequently attributed to post-viral or nonspecific inflammatory syndromes, which may lead to diagnostic delay. In contrast, the presence of progressive skin induration, symmetrical limb involvement, and eosinophilia should be considered red flags prompting targeted investigation. In this setting, MRI is particularly valuable for identifying fascial inflammation and guiding biopsy, especially when clinical findings are subtle or evolving.

Moreover, available evidence indicates that prognosis in eosinophilic fasciitis is primarily determined by disease stage at presentation and timeliness of treatment initiation. Multiple cohort studies have consistently identified diagnostic delay as one of the strongest predictors of poor outcome, reflecting progression from an early inflammatory phase to irreversible fibrosis [33, 34]. Additional adverse prognostic factors include truncal involvement [9], coexistence of morphea-like lesions [9], elevated inflammatory markers, and histological evidence of dermal fibrosis [9]. Recent multicenter analyses further suggest that a fibrotic disease phenotype and persistent eosinophilia are associated with relapse and long-term sequelae, whereas patients presenting in an early edematous stage have a more favorable course [35].

Consistent with these findings, our patient, who received an early diagnosis, demonstrated a sustained and complete response to systemic glucocorticoids followed by methotrexate, with full clinical remission at long-term follow-up.

4. Conclusions

Eosinophilic fasciitis is a rare immune-mediated condition that may occur following SARS-CoV-2 infection. This case highlights the importance of considering eosinophilic fasciitis in patients presenting with compatible

clinical features after COVID-19, even in the absence of classical triggers. Early recognition based on clinical findings, supported by imaging and histopathology, is essential for prompt initiation of immunosuppressive therapy and favorable outcomes.

Declarations

Author Contributions

Conceptualization: M.K.; Methodology: M.K., M.M.; Formal analysis: M.K., M.D.; Investigation: M.M., M.K.; Writing: original draft preparation: M.M., M.K.; Writing: review and editing: M.D., M.M., M.K., V.V.

Funding

This article received no external funding.

Declaration

The case report was conducted in accordance with the Declaration of Helsinki.

Informed Consent Statement

Informed consent was obtained from all subjects involved in the study.

Data Availability Statement

The data presented in this study are available on reasonable request from the corresponding author.

Conflicts of Interest

The authors declare no conflict of interest.

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Еозинофилен фасцит след инфекция със SARS-CoV-2: клиничен случай и обзор на времевите асоциации

Резюме

Въведение: Еозинофилният фасцит е рядко заболяване от групата на склеродермоподобните състояния, характеризиращо се с възпаление и задебеляване на фасцията, клинично проявяващо се с кожна индурация и периферна еозинофилия. Етиологията му остава неясна, като се предполага имунно-медиран механизъм и участие на различни отключващи фактори. Клиничен случай: Представяме случай на 49-годишна жена с прогресираща болка, скованост и кожна индурация на горните крайници, възникнали скоро след преболедуване от COVID-19. Лабораторните изследвания установиха еозинофилия и повишени възпалителни маркери при отрицателен аутоимунен профил. Магнитнорезонансната томография демонстрира засягане на фасцията без ангажиране на мускулатурата. Хистологичното изследване потвърди диагнозата еозинофилен фасцит. Проведено бе лечение с високи дози глюкокортикостероиди и метотрексат като стероид-спестяващ агент, с последващо клинично и лабораторно подобрение. При проследяване се установи пълна клинична ремисия без поддържаща терапия. Заключение: Представеният случай подкрепя възможна връзка между еозинофилния фасцит и прекарана SARS-CoV-2 инфекция като потенциален отключващ фактор. Ранното разпознаване, образната диагностика и хистологичното потвърждение са от ключово значение за навременното лечение и благоприятния изход.

Ключови думи: еозинофилен фасцит; COVID-19; постинфекционно имуномедирано заболяване; клиничен случай