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Unilateral Calf Swelling and Skin Induration During Pembrolizumab-Based Neoadjuvant Therapy for Triple-Negative Breast Cancer: A Case Report

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Abstract

Eosinophilic fasciitis (EF) is a rare fibro-inflammatory disorder that has been reported following immune checkpoint inhibitor (ICI) therapy. We report the case of a woman in her 70s with locally advanced triple-negative breast cancer (TNBC) who developed acute unilateral calf pain, erythema, swelling, and woody skin induration approximately four months after commencing neoadjuvant pembrolizumab-based therapy. Investigations demonstrated peripheral eosinophilia and a normal creatine kinase (CK) level. Pembrolizumab was withheld because of initial concern for ICI-associated myositis, and oral prednisolone was commenced at 60 mg daily (approximately 1 mg/kg). Magnetic resonance imaging (MRI) performed 19 days after corticosteroid initiation showed mild subcutaneous soft-tissue edema without evidence of myositis or characteristic fascial abnormalities. Pain, erythema, and swelling improved within days, and the eosinophil count fell during corticosteroid treatment, although skin induration persisted. Rheumatology assessment supported a clinical working diagnosis of suspected EF. Biopsy was not pursued in view of clinical improvement. Pembrolizumab was permanently discontinued after multidisciplinary discussion. Prednisolone was gradually tapered and discontinued over approximately four months without the need for additional immunosuppression. At last review, five months after symptom onset, she remained off corticosteroids, with resolution of pain, erythema, and swelling and only residual skin induration. Paclitaxel-associated sclerosis and morphoea profunda remained competing explanations, and attribution to pembrolizumab was possible rather than established. This case highlights the importance of specialist assessment of unexplained limb swelling and woody skin induration during combination anticancer therapy. Normal CK levels and corticosteroid responsiveness do not establish EF.

Categories: Rheumatology, Oncology
Keywords: eosinophilic fasciitis, immune checkpoint inhibitor, immune-related adverse event, immunotherapy, pembrolizumab, triple-negative breast cancer

Introduction

Immune checkpoint inhibitors (ICIs) have transformed the treatment of several solid malignancies and are now an integral component of neoadjuvant therapy for patients with high-risk early triple-negative breast cancer (TNBC). The phase III KEYNOTE-522 trial demonstrated improved event-free survival with neoadjuvant pembrolizumab plus chemotherapy followed by adjuvant pembrolizumab, compared with the corresponding placebo-containing regimen, in patients with stage II-III TNBC [1].

The expanding use of ICIs is accompanied by a broad spectrum of immune-related adverse events (irAEs). Rheumatic and musculoskeletal irAEs include inflammatory arthritis, polymyalgia rheumatica-like syndromes, inflammatory myopathies, vasculitis, and, rarely, eosinophilic fasciitis (EF) [2,3]. Early recognition is important because these toxicities can cause significant morbidity and may interrupt potentially curative anticancer therapy.

EF is a rare fibro-inflammatory disorder characterized by inflammation and progressive fibrosis of the deep fascia [4,5]. Patients typically present with symmetrical limb swelling, pain, woody skin induration, and progressive limitation of joint mobility, often accompanied by peripheral eosinophilia. Characteristic findings include peau d'orange changes and the groove sign, reflecting linear depressions along superficial veins. The fingers and face are typically spared, which can help distinguish EF from systemic sclerosis. Fascial abnormalities on magnetic resonance imaging (MRI) or full-thickness biopsy can support the diagnosis [4,5]. EF has been reported following ICI therapy, particularly with programmed cell death protein 1 (PD-1) inhibitors such as pembrolizumab and nivolumab [6,7]. Its pathogenesis remains incompletely understood. T-cell activation, eosinophilic inflammation, and profibrotic pathways have been implicated, and checkpoint blockade may contribute through loss of inhibitory control over immune responses [5-7].

The clinical features of EF may overlap with those of cellulitis, deep vein thrombosis, and ICI-associated myositis. We report a case of unilateral calf swelling and woody skin induration during neoadjuvant pembrolizumab-based therapy for TNBC. The report examines the challenges of evaluating an atypical unilateral presentation of suspected EF without diagnostic confirmation, assessing drug attribution during combination therapy, and managing a suspected adverse reaction that interrupted potentially curative treatment. Its educational contribution lies in distinguishing uncertainty about the diagnosis from uncertainty about the responsible drug when making treatment decisions.

Case Presentation

A woman in her 70s with an Eastern Cooperative Oncology Group (ECOG) performance status of 1 presented with a breast mass. Core biopsy confirmed invasive TNBC, clinically staged as cT2 cN1 cM0. Her medical history included atrial fibrillation, for which she was treated with flecainide 50 mg twice daily, edoxaban 60 mg once daily, and bisoprolol 2.5 mg once daily. These were her only longstanding regular medications. She had no previous history of autoimmune disease, dermatological disorders, or muscle disease. Her baseline absolute eosinophil count was 0.24×10^9/L.

Neoadjuvant pembrolizumab, carboplatin, and weekly paclitaxel commenced on November 20, 2025, as part of a KEYNOTE-522-based regimen. She completed four cycles of pembrolizumab with carboplatin and all 12 planned weekly paclitaxel doses, with some delays related to fatigue and a short holiday. The final paclitaxel dose was administered on February 19, 2026, followed by the first cycle of pembrolizumab with epirubicin and cyclophosphamide on February 26, 2026. The next cycle was delayed because of fatigue and feeling generally unwell. No further systemic anticancer treatment was administered before calf symptom onset.

On April 1, 2026, 132 days after treatment initiation, 41 days after the final paclitaxel dose, and 34 days after pembrolizumab with epirubicin and cyclophosphamide, she developed acute-onset pain, erythema, swelling, and tenderness affecting the left calf.

She was assessed on April 3, 2026. On examination, the skin over the lower left calf was edematous and tight, with marked tenderness and woody induration. The groove sign was absent, and there was no involvement of the contralateral lower limb or upper limbs. Ankle movement was restricted predominantly by pain. Pain limited assessment of muscle strength, and definite objective weakness or fixed contracture could not be established. Formal muscle-power grading was not documented. She reported no ptosis, diplopia, dysphagia, altered speech, or breathlessness. Duplex ultrasonography on April 4, 2026, excluded deep vein thrombosis.

Initial blood samples obtained before corticosteroid initiation demonstrated peripheral eosinophilia, with an absolute eosinophil count of 0.98×10^9/L (reference range: 0.01-0.5 $\times 10^9$/L). Serum creatine kinase (CK) was normal at 30 U/L (reference range: 0-144 U/L), and C-reactive protein was not elevated. Cardiac assessment before corticosteroid treatment showed a normal electrocardiogram (ECG), with high-sensitivity troponin I and N-terminal pro-B-type natriuretic peptide (NT-proBNP) within their respective laboratory reference ranges. An extended myositis antibody panel was negative. Tests for rheumatoid factor, anti-cyclic citrullinated peptide antibodies, and antineutrophil cytoplasmic antibodies directed against myeloperoxidase and proteinase 3 were also negative.

Given the initial concern for ICI-associated myositis, pembrolizumab remained withheld, and oral prednisolone was commenced at 60 mg daily on April 5, 2026. Pain, erythema, and swelling improved within days. The absolute eosinophil count decreased to 0.03×10^9/L during corticosteroid treatment, although woody skin induration persisted. Serial CK measurements remained within the normal range.

Rheumatology advice was obtained by telephone in approximately mid-April 2026, when EF was considered among the differential diagnoses. Non-contrast MRI of the lower legs was performed on April 24, 2026, 19 days after corticosteroid initiation and after symptomatic improvement. It demonstrated mild subcutaneous edema in the left lower calf on fat-saturated imaging (Figure *1*), with no evidence of myositis or organized fluid collection (Figure *2*). No characteristic fascial abnormalities or suspicious osseous or soft-tissue lesions were reported. A full-thickness biopsy was not pursued because symptoms were improving with corticosteroid treatment.

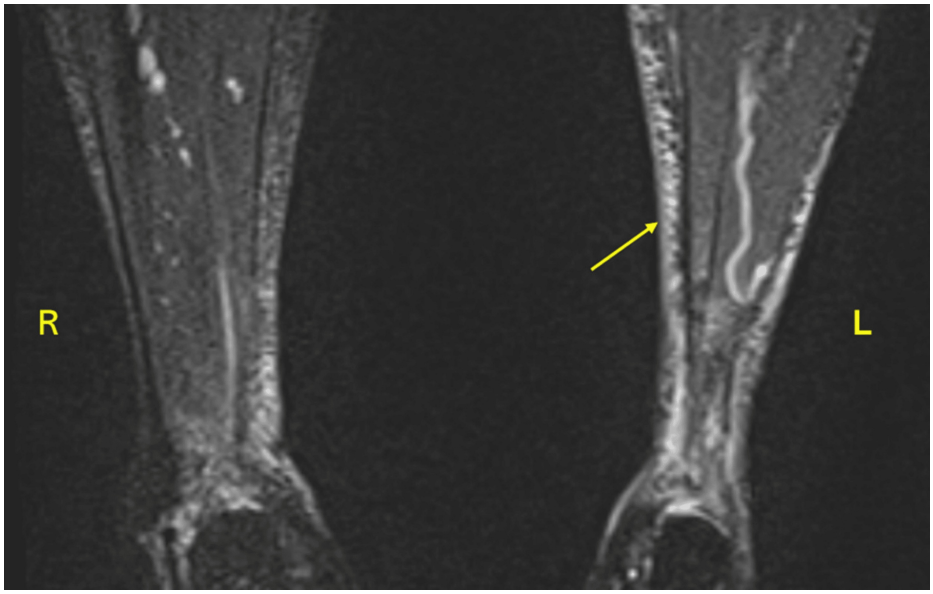


FIGURE 1: Fat-saturated MRI of both lower legs demonstrating mild left calf subcutaneous edema (yellow arrow)

MRI: Magnetic resonance imaging.

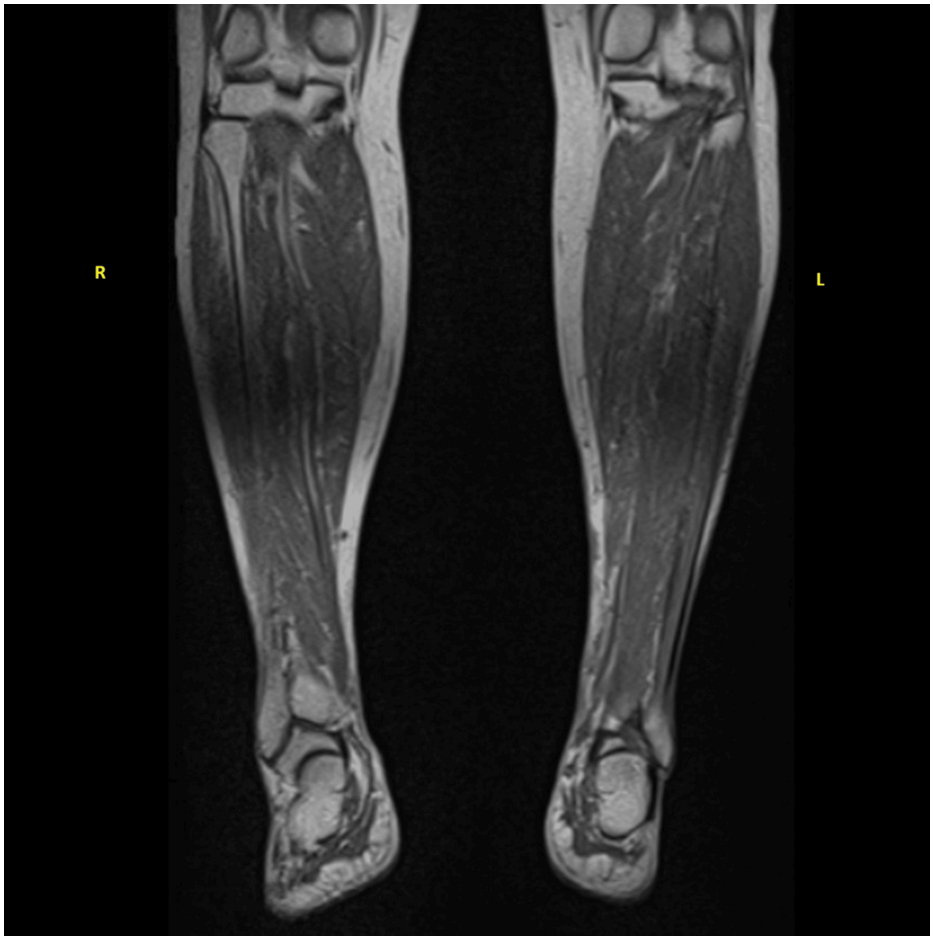


FIGURE 2: MRI of both lower legs showing no evidence of myositis, an organized fluid collection, or characteristic fascial abnormalities

MRI: Magnetic resonance imaging.

Breast MRI on April 27, 2026, demonstrated a partial radiological response in the primary breast tumor and axillary lymph nodes. These findings were reviewed at the multidisciplinary team meeting on April 30, 2026. Following multidisciplinary discussion before surgery, pembrolizumab was permanently discontinued because of a clinically significant suspected adverse reaction to pembrolizumab. She received no further neoadjuvant systemic therapy, and the remaining three planned epirubicin/cyclophosphamide-containing cycles were omitted.

She underwent mastectomy with axillary lymph node clearance on May 29, 2026. Histopathological examination demonstrated residual grade 3 invasive TNBC with lymphovascular invasion. Her first face-to-face rheumatology assessment took place on June 12, 2026. Specialist assessment supported a clinical working diagnosis of suspected EF based on the overall presentation, including woody skin induration and preceding peripheral eosinophilia.

Prednisolone was initially reduced in 10 mg weekly steps, followed by a slower taper, and was discontinued after approximately four months without additional immunosuppression. Adjuvant capecitabine commenced on July 16, 2026, while corticosteroid tapering was ongoing, and she had completed two cycles without significant toxicity at last review. At follow-up five months after calf symptom onset, she remained off corticosteroids, with complete resolution of calf pain, erythema, swelling, and residual skin induration. The clinical timeline is summarized in Table *1*.

Date or interval	Event
November 20, 2025	Neoadjuvant pembrolizumab, carboplatin, and weekly paclitaxel commenced. Baseline eosinophils: 0.24 × 10^9/L.
February 19, 2026	Final paclitaxel dose, 41 days before calf symptom onset.
February 26, 2026	First pembrolizumab/epirubicin/cyclophosphamide cycle, 34 days before symptom onset. Subsequent treatment was delayed because of fatigue and feeling unwell.
April 1, 2026	Acute left calf pain, erythema, and swelling began.
April 3, 2026	Clinical assessment demonstrated woody skin induration and ankle movement restricted predominantly by pain.
April 3, 2026	Eosinophils 0.98 × 10^9/L and CK 30 U/L. CRP was not elevated. ECG, high-sensitivity troponin I, and NT-proBNP were within normal limits.
April 4, 2026	Duplex ultrasonography excluded deep vein thrombosis.
April 5, 2026	Prednisolone 60 mg daily commenced; pembrolizumab remained withheld.
Within days of corticosteroid initiation	Pain, erythema, and swelling improved, although skin induration persisted. Eosinophil count decreased to 0.03 × 10^9/L.
Approximately mid-April 2026	Telephone rheumatology advice obtained; EF included in the differential diagnosis.
April 24, 2026	Non-contrast calf MRI, performed 19 days after corticosteroid initiation, demonstrated mild subcutaneous edema without myositis or characteristic fascial abnormalities.
April 27, 2026	Breast MRI demonstrated a partial response in the breast and axilla.
April 30, 2026	Breast MRI findings reviewed at the multidisciplinary team meeting.
Before May 29, 2026, exact date unavailable	Multidisciplinary decision to permanently discontinue pembrolizumab. Remaining three planned epirubicin/cyclophosphamide-containing cycles omitted.
May 29, 2026	Mastectomy and axillary lymph node clearance.
June 12, 2026	First face-to-face rheumatology assessment supported a working diagnosis of suspected EF.
July 16, 2026	Adjuvant capecitabine commenced during corticosteroid taper.
Approximately August 2026	Prednisolone discontinued after an approximately four-month course.
Approximately September 2026	Five-month follow-up after symptom onset. Off corticosteroids, with complete resolution of pain, erythema, swelling, and residual skin induration.

TABLE 1: Clinical timeline of treatment, symptom onset, investigations, and outcome

CK: Creatine kinase; CRP: C-reactive protein; ECG: Electrocardiogram; EF: Eosinophilic fasciitis; MRI: Magnetic resonance imaging; NT-proBNP: N-terminal pro-B-type natriuretic peptide.

Discussion

ICI-associated inflammatory sclerosing disorders present diagnostic challenges because their clinical features overlap and attribution is complicated by concomitant anticancer treatments. In this case, uncertainty concerned both the diagnosis of EF and the identification of the responsible drug.

Peripheral eosinophilia frequently occurs during the early inflammatory phase of EF but is neither universal nor required for diagnosis [4,5]. Beyond individual case reports, ICI-associated EF has been characterized in case series and more recent multicenter and pharmacovigilance studies, predominantly in association with PD-1 inhibitors [6-9].

The acute painful swelling, erythema, and marked woody induration supported consideration of EF, although these features overlap with other inflammatory sclerosing disorders [4,5]. Compared with morphoea profunda, the accompanying peripheral eosinophilia provided some additional support, as this finding was more frequent in EF in a comparative clinicopathological study [10]. However, the documented clinical findings offered limited discrimination between EF and a taxane-associated sclerodermoid reaction,

which can also present with edema progressing to sclerosis [11]. Symptom onset 41 days after the final paclitaxel dose established a relevant exposure interval but could not resolve this distinction. The unilateral distribution and absent groove sign limited support for classical EF, while corticosteroid responsiveness was nonspecific. These findings supported suspected EF as the specialist working diagnosis but did not reliably exclude morphoea profunda or a treatment-associated sclerodermoid reaction.

MRI can demonstrate fascial thickening, increased fascial signal on fluid-sensitive sequences, and enhancement following gadolinium administration. It can also help identify suitable biopsy sites and assess disease extent [4,12]. In this patient, non-contrast MRI demonstrated mild subcutaneous edema without characteristic fascial abnormalities or evidence of myositis. The scan was obtained 19 days after prednisolone initiation, when clinical improvement was already evident. Corticosteroid treatment may have attenuated inflammatory imaging findings, although no pretreatment scan was available to assess this possibility. Contrast enhancement could not be evaluated because gadolinium was not administered. Imaging therefore provided no confirmation of fascial involvement.

A full-thickness biopsy incorporating the fascia can provide histopathological support for EF and help distinguish competing sclerosing disorders [4,5]. Biopsy was not pursued because symptoms were improving with corticosteroid treatment. This left the anatomical distribution of the inflammatory and fibrosing process unconfirmed and limited distinction from morphoea profunda and a treatment-associated sclerodermoid reaction. The patient did not meet the criteria proposed by Jinnin et al. because the presentation was unilateral and lacked supportive histological or characteristic fascial imaging findings [4]. Failure to meet these criteria does not conclusively exclude atypical EF. Nevertheless, the absence of demonstrable fascial involvement substantially limits diagnostic confidence, and EF remained a suspected clinical diagnosis.

Morphea profunda remained an important alternative because it can overlap clinically and histologically with EF [10]. Morphea has also been reported following pembrolizumab treatment [13]. Without histological confirmation, this competing diagnosis could not be confidently excluded.

ICI-associated myositis was initially considered because of the acute painful presentation during pembrolizumab-containing treatment. Strength assessment was limited by pain, and formal muscle-power grading was not documented. The patient reported no ocular, bulbar, or respiratory symptoms. Persistently normal CK levels, negative myositis antibody testing, and the absence of myositis on MRI obtained after corticosteroid initiation did not definitively exclude ICI-associated myositis [14,15].

Cardiac assessment was relevant because ICI-associated myositis can overlap with myocarditis [14,16]. The normal ECG, high-sensitivity troponin I, and NT-proBNP obtained before corticosteroid treatment were reassuring, although they did not independently establish the absence of myocardial involvement.

Attribution to an individual drug was complicated by the combination treatment regimen. Symptoms began 132 days after treatment initiation, 41 days after the final paclitaxel dose, and 34 days after the first cycle of pembrolizumab, epirubicin, and cyclophosphamide. Paclitaxel-associated sclerodermoid reactions have been reported, making the preceding taxane exposure a plausible alternative explanation [11]. Epirubicin and cyclophosphamide were administered concurrently with the final pembrolizumab dose, further limiting attribution based on timing alone. The inflammatory presentation did not identify the responsible drug, and improvement following treatment withdrawal was confounded by corticosteroid administration.

Retrospective assessment using the World Health Organization-Uppsala Monitoring Centre causality assessment system supported a possible association between pembrolizumab and the inflammatory sclerosing presentation [17]. The temporal relationship was compatible with a drug-related reaction, but paclitaxel exposure provided a competing explanation. Corticosteroid treatment prevented assessment of the independent effect of drug withdrawal, and pembrolizumab was not reintroduced. This assessment did not establish the diagnosis of EF or exclude contributions from other treatments.

Systemic corticosteroids are the mainstay of EF treatment. Additional immunosuppression may be considered for an inadequate response or when prolonged corticosteroid exposure is undesirable [4,9]. In this patient, pain, erythema, and swelling improved within days of starting prednisolone 60 mg daily, while skin induration persisted. The eosinophil count also decreased, although glucocorticoids themselves rapidly lower circulating eosinophils, limiting the diagnostic significance of this response [18]. Early symptomatic improvement was compatible with suppression of inflammation and edema. Improvement of edema has been reported in EF, but the response did not distinguish EF from competing inflammatory sclerosing disorders [19].

Guidance from the American Society of Clinical Oncology (ASCO) and the European Society for Medical Oncology (ESMO) supports withholding ICIs and initiating corticosteroids according to the severity and organ involvement of suspected irAEs, with specialist input informing subsequent treatment decisions [14,16]. Pembrolizumab was permanently discontinued following multidisciplinary discussion in this case. This was an individual management decision made in the context of a clinically significant suspected

adverse reaction and diagnostic uncertainty. The remaining neoadjuvant chemotherapy cycles were also omitted, and residual disease at surgery must therefore be interpreted in the context of an incomplete neoadjuvant course. Adjuvant capecitabine was commenced for residual invasive TNBC. The Capecitabine for Residual Cancer as Adjuvant Therapy (CREATE-X) trial supports adjuvant capecitabine in patients with residual invasive disease after neoadjuvant chemotherapy but did not evaluate it as a substitute for pembrolizumab following an irAE [20].

This case illustrates the need to distinguish evidence supporting a suspected diagnosis from evidence implicating a particular treatment. Specialist multidisciplinary assessment helped guide management, but uncertainty about both fascial involvement and drug attribution persisted. Such uncertainty is particularly consequential when treatment decisions interrupt a potentially curative anticancer regimen.

Conclusions

Unexplained limb swelling and skin induration during combination anticancer therapy warrant specialist assessment. In this single case, EF remained a suspected clinical diagnosis without histological confirmation, and the responsible treatment could not be established. Further well-characterized reports, including histological and imaging confirmation, are needed to clarify the diagnosis and drug attribution of similar presentations.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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