

CASE REPORT

Atypical localized neutrophilic dermatosis. Localized neutrophilic dermatosis in post-mastectomy lymphedema.

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Key words: neutrophilic dermatosis, Sweet
syndrome, lymphedema, neutrophilia

How to cite this article:
Ortiz L., Úraga V., Garcés JC. Dermatitis
neutrofílica localizada atípica. Dermatitis
neutrofílica localizada en linfedema post-
mastectomía. – Reporte de un caso clínico.
Rev Dermatol Cent Úraga. 2026;8(2).

Date of receipt: 28/5/2026
Date of acceptance: 7/7/2026

ABSTRACT

Localized neutrophilic dermatosis arising in areas of post-mastectomy lymphedema represents a rare variant of Sweet syndrome, characterized by inflammatory lesions confined to regions with impaired lymphatic drainage and regional immune dysfunction. We present the case of a 76-year-old woman with a history of right ductal breast carcinoma treated with mastectomy, lymphadenectomy, chemotherapy, radiotherapy, and hormonal therapy, who subsequently developed chronic ipsilateral lymphedema. She presented with erythematous maculopapular lesions on the right arm associated with episodes of fever, tenderness, and localized warmth. Laboratory studies revealed leukocytosis, neutrophilia, hyperfibrinogenemia, and elevated C-reactive protein levels. Skin biopsy demonstrated superficial and deep perivascular inflammatory infiltrates with scattered neutrophils, swollen endothelial cells, and focal erythrocyte extravasation. A diagnosis of localized neutrophilic dermatosis arising in post-mastectomy lymphedema was established, likely favored by the presence of an immunologically vulnerable district related to the patient's previous oncological treatments.

INTRODUCTION

Sweet syndrome, also known as acute febrile neutrophilic dermatosis, was originally described by Robert Douglas Sweet in 1964 as an entity characterized by fever, neutrophilic leukocytosis, and painful erythematous plaques histologically associated with a dermal neutrophilic infiltrate in the absence of infection. Since then, multiple clinical variants associated with inflammatory, infectious, neoplastic, and drug-related conditions have been described. Among these, localized forms represent an uncommon presentation, particularly those confined to areas of impaired lymphatic drainage secondary to mastectomy and axillary lymphadenectomy.^{1,2}

Localized neutrophilic dermatosis occurring in post-mastectomy lymphedema has been considered by some authors as a localized and less severe variant of classic Sweet syndrome. Its pathophysiology remains poorly understood; however, lymphatic stasis has been proposed to promote a local inflammatory microenvironment characterized by impaired immune surveillance, cytokine accumulation, and disruption of cellular trafficking, transforming these regions into immunologically vulnerable districts or loci minoris resistentiae. We present the case of a patient with a history of breast cancer treated with mastectomy, radiotherapy, and lymphadenectomy who

developed localized neutrophilic dermatosis involving the arm affected by chronic lymphedema.^{3,4}

CASE PRESENTATION

A 76-year-old woman with a history of right ductal breast carcinoma diagnosed in November 2021 was treated with right mastectomy, axillary lymphadenectomy, chemotherapy, radiotherapy, and hormonal therapy with anastrozole. As a sequela of oncologic treatment, she developed chronic lymphedema of the right upper limb. Additional medical history included recurrent herpes simplex infection in the anal region, for which she used valacyclovir during episodes of reactivation.

She presented with a three-day history of unilateral erythematous maculopapular lesions involving the right forearm with progressive extension to the ipsilateral arm, associated with documented febrile episodes, local tenderness, and a sensation of warmth in the affected limb. The patient reported the application of topical ketoprofen to both upper limbs prior to consultation; however, lesions developed exclusively in the arm affected by lymphedema. Four days before symptom onset, she experienced reactivation of anal herpes simplex infection and initiated treatment with valacyclovir.

Physical examination revealed evident lymphedema of the right upper limb compared with the contralateral

arm, together with multiple ill-defined erythematous maculopapular lesions scattered over an erythematous background involving the right arm and forearm, without vesicles, pustules, or ulceration (Figure 1A, Figure 1B). The lesions were asymptomatic to palpation, although the patient reported tenderness and increased local temperature coinciding with febrile episodes.

Laboratory studies showed a hemoglobin level of 13.3 g/dL, hematocrit of 40.8%, leukocytosis of 12,300/mm³ with absolute neutrophilia of 8,140/mm³, elevated plasma fibrinogen of 521 mg/dL, and C-reactive protein of 28.01 mg/L. Initial differential diagnoses included allergic contact dermatitis, cellulitis, and erysipelas.

A skin biopsy was obtained from a maculopapular lesion located on the affected right arm. Histopathological examination revealed skin with preserved architecture, an orthokeratotic epidermis, and a superficial and deep perivascular inflammatory infiltrate predominantly composed of lymphocytes with an interstitial component, associated with histiocytes, occasional eosinophils, and scattered neutrophils. In addition, swollen endothelial cells and focal erythrocyte extravasation were observed, without evidence of frank vasculitis (Figure 2).

Based on the clinical findings, laboratory results, and histopathological features, a diagnosis of localized

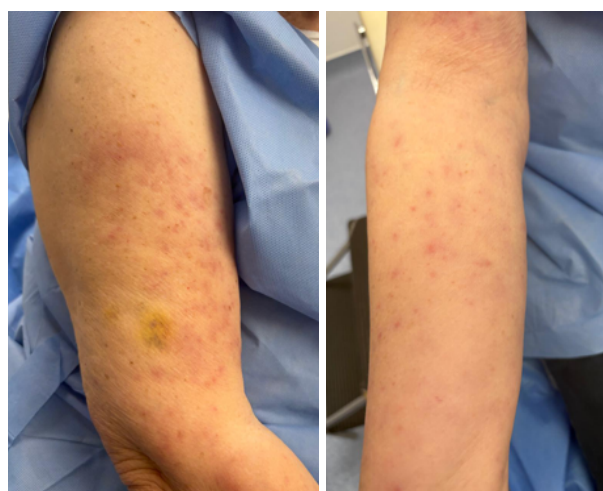


Figure 1. (a) Scattered erythematous maculopapular lesions distributed over an erythematous background on the arm, and (b) the right forearm affected by post-mastectomy lymphedema, observed at the time of the initial consultation.

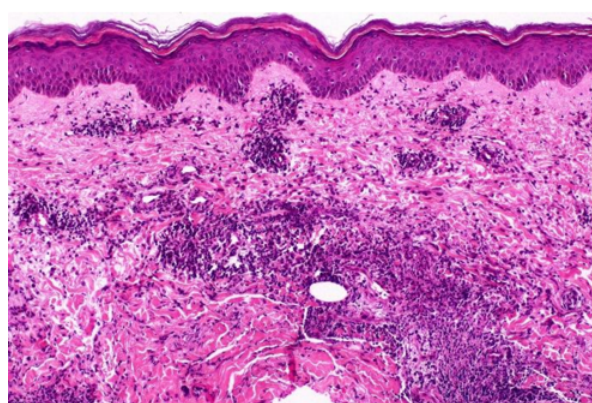


Figure 2. Histopathological examination of a cutaneous lesion from the right arm. A superficial and deep perivascular inflammatory infiltrate predominantly composed of lymphocytes with an interstitial component is observed, associated with histiocytes, occasional eosinophils, and scattered neutrophils, as well as swollen endothelial cells and focal erythrocyte extravasation, without evidence of frank vasculitis.



Figure 3. (a) Resolution of the cutaneous lesions on the right arm and (b) forearm after topical treatment with fluticasone, with mild residual erythema.

neutrophilic dermatosis arising in post-mastectomy lymphedema was established. The patient was treated with paracetamol every 8 hours and topical fluticasone emulsion every 12 hours for seven days, with progressive improvement observed from the fourth day onward, leading to near-complete resolution of the lesions without residual pigmentation or subsequent recurrence (Figure 3A, Figure 3B).

DISCUSSION

Sweet syndrome, also known as acute febrile neutrophilic dermatosis, was originally described by Robert Douglas Sweet in 1964 as an entity characterized by fever, neutrophilic leukocytosis, and painful erythematous plaques histologically associated with a dense dermal neutrophilic infiltrate without evidence of infection. Subsequently, multiple clinical variants were recognized, including classic forms, malignancy-associated forms, drug-induced forms, and localized variants. Clinically, lesions usually present as painful erythematous plaques or papules of sudden onset, predominantly affecting the face, neck, and extremities, and are frequently accompanied by systemic symptoms and elevated inflammatory markers.^{1,2}

Neutrophilic dermatoses confined to areas of post-mastectomy lymphedema represent an uncommon and probably underdiagnosed variant. Since the first case

described by Demitsu et al., several reports have documented patients with a history of breast cancer treated with mastectomy, lymphadenectomy, and radiotherapy who develop inflammatory lesions exclusively on the limb prone to lymphedema. Compared with classic Sweet syndrome, these variants tend to show lower clinical severity, fewer recurrences, and a lower frequency of marked neutrophilia or systemic involvement. Some authors have proposed the term “neutrophilic dermatosis on postmastectomy lymphedema” (NDPL) to differentiate this localized and less aggressive presentation from conventional Sweet syndrome.^{3,5,6}

The pathophysiology of these localized neutrophilic dermatoses remains a subject of debate. It has been proposed that chronic lymphedema generates a persistent inflammatory microenvironment characterized by the accumulation of protein-rich interstitial fluid, impaired lymphatic drainage, and disruption of normal immune cell trafficking. This may promote local production of cytokines and chemokines capable of attracting neutrophils and perpetuating dermal inflammation. In addition, altered regional immunosurveillance transforms these areas into “immunologically vulnerable districts” or loci minoris resistentiae, predisposing them to the localized development of infections, neoplasms, and immune-mediated dermatoses. The strict unilateral distribution observed in many reported cases supports this pathophysiological hypothesis.^{4,7}

In the present case, the patient had multiple factors potentially related to the development of localized neutrophilic dermatosis, including chronic lymphedema secondary to lymphadenectomy and radiotherapy, a history of breast malignancy, and a recent episode of herpes simplex reactivation. Although the histopathological infiltrate did not fully correspond to the classic dense neutrophilic pattern described in Sweet syndrome, the clinical, laboratory, and histological findings were compatible with localized neutrophilic dermatosis arising in an area of post-mastectomy lymphedema. Likewise, the absence of lesions on the contralateral limb despite bilateral application of topical ketoprofen supports the hypothesis of regional immunological susceptibility. Finally, the rapid improvement with topical treatment and conservative management is consistent with the less aggressive clinical behavior described in localized lymphedema-associated variants.^{3,5}

CONCLUSION

Localized neutrophilic dermatosis arising in areas of post-mastectomy lymphedema represents an uncommon variant within the spectrum of neutrophilic dermatoses and is likely underdiagnosed. Its recognition is important because of its clinical similarity to infectious processes such as cellulitis or erysipelas. The present case supports the hypothesis of lymphedema as an immunologically vulnerable district capable of promoting localized inflammatory responses, possibly triggered by concomitant systemic or infectious stimuli. Clinicopathological correlation and awareness of this variant allow timely diagnosis and help avoid unnecessary treatments.

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