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Case Report
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Sweet Syndrome Variant Masquerading as a Necrotising Infection of the Breast

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Abstract

A 64-year-old female presented with significant superficial necrosis and cellulitis of the left breast following a fall injury. Initial examination revealed extensive skin necrosis, including the nipple-areola complex, leading to a diagnosis of a necrotising skin infection.

Despite intravenous antibiotics and surgical debridement, the necrosis progressed, necessitating multiple surgical interventions. Histopathological analysis suggested an acute necrotising neutrophilic dermatosis consistent with a rare necrotising variant of Sweet Syndrome. Eventually, complete recovery was achieved with steroids, regular wound care and ongoing dermatology review.

Keywords: Sweet syndrome; Fasciitis necrotising; Soft tissue infections

Introduction

A 64-year-old female presented with significant superficial necrosis and cellulitis of the left breast following a fall injury. Initial examination revealed extensive skin necrosis, including the nipple-areola complex, leading to a diagnosis of a necrotising skin infection. Despite intravenous antibiotics and surgical debridement, the necrosis progressed, necessitating multiple surgical interventions. Histopathological analysis suggested an acute necrotising neutrophilic dermatosis consistent with a rare necrotising variant of Sweet Syndrome. Eventually, complete recovery was achieved with steroids, regular wound care and ongoing dermatology review.

Case Presentation

A 64-year-old female was admitted to the breast unit after developing significant superficial necrosis and cellulitis of the left breast after a vague history of a trauma (falling onto furniture) 10-days prior. Clinical examination revealed extensive skin necrosis including the nipple-areola and surrounding cellulitis. A likely diagnosis of a necrotising skin infection related to trauma was made (Figure 1). Blood tests corroborated this impression with a raised white-cell count (WCC) of 20.2 × 10^9/L, neutrophilia of 19.6 × 10^9/L and an elevated C-reactive protein (CRP). Imaging studies were unremarkable.

The area of necrosis progressed despite receiving intravenous antibiotics targeting necrotising soft tissue infections. Concurrently, signs of systemic sepsis became more prominent, prompting urgent surgical intervention and extensive debridement. Intraoperatively, necrotic superficial fat and breast tissue was excised until viable bleeding tissue was encountered. Postoperatively, the patient was admitted to high dependency unit (HDU) for ongoing intravenous antibiotics and monitoring for systemic complications.

Five days after the initial debridement, the area of necrosis reappeared at the edges and continued to increase as did the patient's oxygen requirement, necessitating intubation and ventilation. Bloods showed deterioration with a WCC of 26.3 × 10^9/L, neutrophils of 23.3 × 10^9/L, rising inflammatory markers and a raised lactate. The deterioration of her symptoms was thought to be secondary to worsening sepsis. She underwent a further debridement of left breast tissue. The necrotic skin was excised until healthy tissue was visualised and primary closure of the wound was achieved with a drain in situ. Postoperatively she returned to the HDU. The infectious disease team suggested adding another antibiotic to the regime.

Five days later, she developed further skin edge necrosis with worsening of her systemic symptoms. She underwent a third and even more extensive debridement. On this occasion almost all

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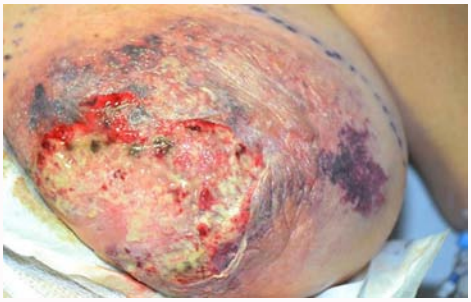

Figure 1: Clinical picture at presentation showing extensive skin necrosis and ulceration and intact underlying breast tissue.

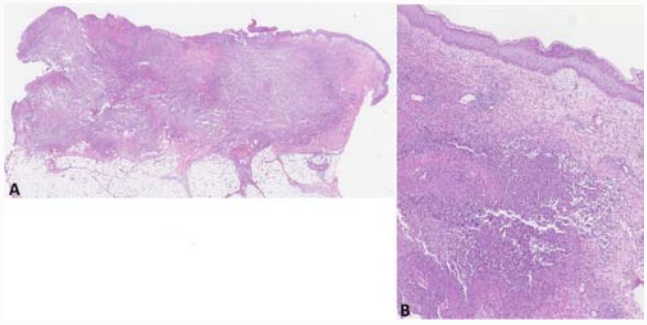

Figure 2: A) Skin biopsy showing surface ulceration, dense and diffuse neutrophilic infiltrate of the entire dermis and superficial fat associated with zones of necrosis.
B) Close up of the peripheral area with intact surface epidermis showing papillary dermal oedema and diffuse dermal neutrophilic infiltrate typically seen in Sweet Syndrome.
Haematoxylin and eosin: original magnification 20(A); × 200(B)

breast tissue was removed down to the pectoralis major muscle fascia. Dissection was performed under the viable skin and subcutaneous tissue flaps which were then secured to the pectoral fascia with a continuous quilting barbed suture. It was not possible to bring the flaps together and the centre of the wound was left open and dressed.

Tissue samples were sent for histopathology, culture and sensitivity during each operation. She remained culture negative throughout her admission.

Histopathological examination revealed focal surface ulceration and diffuse neutrophilic infiltration of dermis, subcutaneous fat and fascia. Focal areas of necrosis were seen involving the fat and fascia. In sections with intact surface epidermis, there was prominent papillary dermal oedema (Figure 2). No microorganisms were detected on Gram, periodic acid Schiff, Grocott methenamine silver, Ziehl-Neelsen and Wade Fite stains. A diagnosis of acute necrotising neutrophilic dermatosis was suggested. Given the papillary dermal oedema, monomorphic nature of the neutrophilic infiltrate with the absence of significant number of plasma cells, an unusual necrotising variant of sweet syndrome was favoured over other neutrophilic dermatoses e.g. pyoderma gangrenosum.

As per the dermatology team's recommendation she was commenced on IV methylprednisolone and a steroid cream with good effect. As the wound healed, she was stepped down to oral prednisolone with a slow taper to follow thereafter. She was discharged and assisted with regular dressing changes in the community.

At follow-up 6 weeks after discharge she had improved to the point of complete resolution.

Discussion

Sweet syndrome (SS) (acute febrile neutrophilic dermatosis) is a form of neutrophilic dermatosis characterized by the acute presentation of oedematous and painful erythematous papules, plaques, or nodules. Fever, leucocytosis, and multiple organ involvement can also co-exist [1,2]. Distinction of SS from other forms of neutrophilic dermatoses, especially pyoderma gangrenosum (PG) requires careful clinical and histopathological correlation. In this case, the clinicopathological features fulfilled the requisite major (abrupt onset of erythematous plaques/nodules with pseudovesiculation, dense diffuse neutrophilic infiltrate) and minor (response to steroids, abnormalities in laboratory tests including CRP, WCC and neutrophilia) criteria to justify a diagnosis a necrotising variant of sweet syndrome over PG [2].

Causes of SS are generally threefold: idiopathic, drug induced, or malignancy associated [1,2]. Occasionally pathergy following surgical or other forms of trauma is associated with the development of SS and other neutrophilic dermatoses [1-4]. Pathergy is a phenomenon which refers to the development of aberrant skin lesions or ulcers from minor cutaneous trauma such as a bump, bruise, incision or biopsy [4].

Only one other case of SS has been identified to affect primarily the breast and this was after a Deep Inferior Epigastric Perforator (DIEP) Flap Breast Reconstruction. This case was associated with malignancy and thyroid disease [5].

Necrotising Sweet syndrome (NSS) is a recently described variant of SS that poses significant challenges for both diagnosis and management. NSS was index-described in 2012, with only few cases reported in the literature to date [6].

Necrotising soft tissue infections are a clinically diagnosed surgical emergency, requiring serial debridement, broad-spectrum antibiotics, and intense supportive care. NSS may clinically mimic a necrotising mono or polymicrobial skin infections as both conditions can be rapidly progressive to demonstrate signs of septicaemia [7]. Aggressive surgical debridement of necrotising skin and soft tissue infections has been shown to improve the historically high mortality rate [7].

NSS is an acute necrotising neutrophilic dermatosis that clinically and histopathologically can masquerade as a necrotising soft tissue infection e.g. necrotising fasciitis [6-8].

Histopathologically, NSS shows dense neutrophilic infiltrate involving the dermis and deeper tissue planes including fat, fascia and skeletal muscle. In contrast, myonecrosis is rarely encountered in necrotising fasciitis, until late stages of the disease [9,10].

NSS may paradoxically be worsened by any surgical intervention via the mechanisms of pathergy as was evident in this case where serial debridement resulted in the exacerbation of the condition with more tissue loss. [4,7].

Conclusion

A diagnosis of NSS should be considered when a patient continues to deteriorate despite multiple debriding procedures, intravenous antibiotics, good supportive care while simultaneously remaining culture negative. A multidisciplinary approach, as was the case for this

patient, consisting of surgery, intensive care, dermatology, infectious disease, and dermatopathology input increases the likelihood of optimal outcomes. Early in the disease process, distinguishing a necrotising skin infection like necrotising fasciitis and NSS can be difficult clinically. Awareness of this entity and early recognition is critically important to avoid the vicious cycle of an erroneous diagnosis of a necrotising infection, repeated debridement and pathergy related tissue destruction [6].

This case describes an extremely rare differential for an emergent diagnosis that has thus far been poorly defined in the literature. It may constitute the second reported case of SS predominantly affecting the breast, and perhaps the first case of NSS affecting the breast without a drug or malignant trigger identified.

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