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CASE REPORT / OLGU SUNUMU

Sweet Syndrome Presenting to the Emergency Department as Febrile Neutropenia

Acil Servise Nötropenik Ateş Olarak Başvuran Sweet Sendromu

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ABSTRACT

Aim: Sweet syndrome is an acute febrile neutrophilic dermatosis classically characterized by fever, painful erythematous skin lesions, and neutrophilia. However, its presentation in the setting of neutropenia is rare and may mimic infectious conditions such as febrile neutropenia in the emergency department. This case aims to highlight the importance of considering Sweet syndrome in febrile neutropenic patients with atypical skin lesions and to emphasize the diagnostic role of early dermatologic evaluation and skin biopsy.

Case Presentation: A 64-year-old woman presented to the emergency department with fever, dyspnea, generalized weakness, and painful skin lesions. Physical examination revealed erythematous bullae on the right wrist, an ulcerated and crusted erythematous plaque on the right breast, and an erythematous nodule with a black center on the right finger. Laboratory evaluation demonstrated pancytopenia, including leukocytes of 0.65 × 10^3/μL and neutrophils of 0.01 × 10^3/μL. The patient was initially evaluated as having febrile neutropenia, and broad-spectrum antibiotic therapy was initiated. Because of the atypical and persistent skin lesions, dermatology consultation was obtained, and a punch biopsy was performed. Histopathological examination showed dense dermal neutrophilic infiltration without vasculitis, confirming the diagnosis of Sweet syndrome. Further hematological evaluation revealed no evidence of an underlying malignancy. Her clinical findings regressed during follow-up, and she was discharged with oral and topical treatment.

Conclusion: This case demonstrates that the absence of neutrophilia does not exclude Sweet syndrome. In febrile neutropenic patients presenting with painful, atypical, ulcerated, or bullous skin lesions, Sweet syndrome should be included in the differential diagnosis. Early dermatologic consultation and skin biopsy are essential to distinguish this condition from infectious mimics and to guide appropriate management in the emergency setting.

Keywords: Acute neutrophilic dermatosis, emergency department, neutropenia, pancytopenia, Sweet syndrome

ÖZ

Amaç: Sweet sendromu, klasik olarak ateş, ağrılı eritemli deri lezyonları ve nötrofili ile karakterize akut febril nötrofilik dermatozdur. Ancak nötropeni varlığında ortaya çıkması nadirdir ve acil serviste febril nötropeni gibi enfeksiyöz durumları taklit edebilir. Bu olgu, atipik deri lezyonları olan febril nötropenik hastalarda Sweet sendromunun göz önünde bulundurulmasının önemini vurgulamayı ve erken dermatolojik değerlendirmenin ve deri biyopsisinin tanısal rolünü öne çıkarmayı amaçlamaktadır.

Olgu Sunumu: Altmış dört yaşında kadın hasta, ateş, dispne, genel halsizlik ve ağrılı deri lezyonları ile acil servise başvurdu. Fizik muayenede sağ el bileğinde eritemli büller, sağ memede ülserleşmiş ve krutlu eritemli plak ve sağ parmakta siyah merkezli eritemli nodül saptandı. Laboratuvar değerlendirmesinde lökosit sayısı 0,65 × 10^3/μL ve nötrofil sayısı 0,01 × 10^3/μL olmak üzere pansitopeni izlendi. Hasta başlangıçta febril nötropeni olarak değerlendirildi ve geniş spektrumlu antibiyotik tedavisi başlandı. Atipik ve persistan deri lezyonları nedeniyle dermatoloji konsültasyonu istendi ve punch biyopsi yapıldı. Histopatolojik incelemede vaskülit olmaksızın yoğun dermal nötrofilik infiltrasyon saptanarak Sweet sendromu tanısı doğrulandı. İleri hematolojik değerlendirmede altta yatan maligniteye ilişkin kanıt saptanmadı. Takipte hastanın klinik bulguları geriledi ve hasta oral ve topikal tedavi ile taburcu edildi.

Sonuç: Bu olgu, nötrofili yokluğunun Sweet sendromunu dışlamadığını göstermektedir. Ağrılı, atipik, ülseratif veya büllöz deri lezyonları ile başvuran febril nötropenik hastalarda Sweet sendromu ayırıcı tanıda düşünülmelidir. Erken dermatoloji konsültasyonu ve deri biyopsisi, bu durumun enfeksiyöz taklitçilerinden ayırt edilmesi ve acil servis yönetiminin doğru şekilde yönlendirilmesi açısından önemlidir.

Anahtar Kelimeler: Akut nötrofilik dermatoz, acil servis, nötropeni, pansitopeni, Sweet sendromu

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Introduction

Sweet syndrome, also known as acute febrile neutrophilic dermatosis, is a rare inflammatory disorder characterized by fever, painful erythematous plaques or nodules, and dermal neutrophilic infiltration. It can present as idiopathic (classical), malignancy-associated, or drug-induced (1). Due to its acute onset and fever, Sweet syndrome is often misdiagnosed as an infectious process in the emergency department. Diagnostic criteria for Sweet syndrome include laboratory findings such as elevated C-reactive protein and erythrocyte sedimentation rate, leukocytosis, and neutrophilia (2). However, there are a few reports in the literature of Sweet syndrome in neutropenic patients (3, 4).

Clinically, Sweet syndrome typically presents with high fever, severe joint pain, and the sudden appearance of painful, red, rapidly enlarging plaques, most commonly on the arms, face, or neck (5). The skin findings are generally erythematous nodules or plaques, while vesiculation or bullae are considered rare manifestations (5). Histopathological examination usually reveals a dense neutrophilic infiltration in the mid to upper dermis without evidence of vasculitis.

The aim of presenting this case is to emphasize the importance of considering Sweet syndrome in the differential diagnosis of febrile neutropenic patients with atypical skin lesions, to highlight the diagnostic challenges, and to discuss management strategies in the emergency setting.

Case presentation

A 64-year-old female presented to the emergency department with fever, dyspnea, and generalized weakness. Her past medical history was significant for hypertension, for which she was taking nebivolol, 5 mg daily. Five days prior to admission, she had been evaluated at another hospital because of generalized body pain, chills, and a high fever of 40°C. She received empirical antibiotic therapy and was discharged with recommendations for further evaluation. After discharge, her symptoms persisted, and she developed new painful skin lesions. These lesions initially appeared as small papules, subsequently became increasingly painful, and later developed discharge.

On admission, her vital signs were as follows: heart rate 105 bpm, blood pressure 96/63 mmHg, respiratory rate 20/min, oxygen saturation 93%, body temperature 38°C, and blood glucose 121 mg/dL. She was alert and oriented, with a Glasgow Coma Scale score of 15. Physical examination revealed bibasilar rales on lung auscultation, abdominal distension, and bilateral pretibial edema. Neurological examination was normal. Dermatologic examination revealed erythematous bullae on the right wrist, an ulcerated and crusted erythematous plaque on the right breast, and an erythematous nodule with a black center on the right finger (Figure 1).

Laboratory investigations showed pancytopenia with hemoglobin 8.9 g/dL, platelets 108 × 10^3/µL, leukocytes 0.65 × 10^3/µL, neutrophils 0.01 × 10^3/µL, and monocytes 0.17 × 10^3/µL. The peripheral smear showed no blasts but revealed atypical lymphocytes. The sedimentation rate was 69 mm/h and C-reactive protein was 24.6 mg/L. She was evaluated by internal medicine, infectious diseases, and dermatology specialists. Empirical broad-spectrum antibiotic therapy with piperacillin-tazobactam (4.5 g IV every 6 hours) was initiated for neutropenic fever, and topical antibiotic therapy with fusidic acid or mupirocin was recommended. During the evaluation, blood cultures were obtained as part of the febrile neutropenia workup. A swab culture from the ulcerated lesion site was obtained and revealed Staphylococcus aureus. Dermatology performed a punch biopsy with a preliminary diagnosis of Sweet syndrome; histopathology later demonstrated a mixed cellular infiltrate with a predominance of polymorphonuclear neutrophils without vasculitis, consistent with Sweet syndrome. During follow-up, her complaints regressed, and she was discharged with oral amoxicillin-clavulanate (1 g twice daily) and fusidic acid (500 mg three times daily) for one week, with outpatient follow-up arranged. At dermatology follow-up, the lesions had regressed significantly by the time the biopsy results were reviewed; therefore, systemic corticosteroids were not started. Subsequent outpatient evaluations, including hematological investigations, revealed no evidence of an underlying malignancy, and her complete blood count gradually returned to normal ranges during follow-up.

Written informed consent was obtained from the patient.

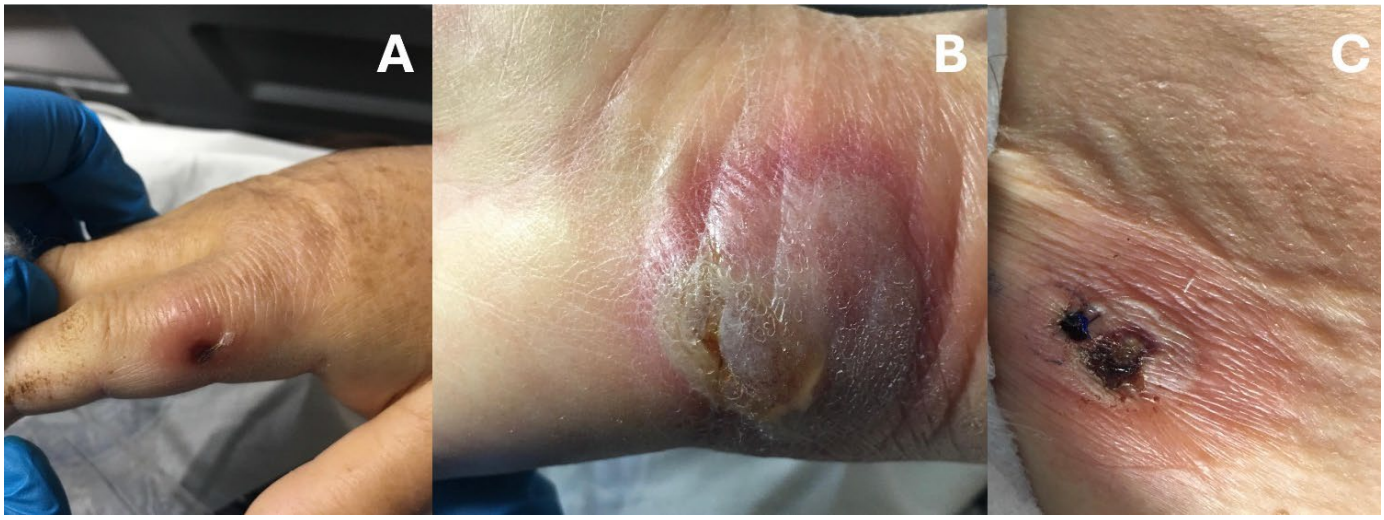

Figure 1. A: A plaque with a crusted infiltrate and more erythematous edges on the right wrist, B: An erythematous papule on the right finger, C: An erythematous plaque with a central ulcer and crust on the right breast.

Discussion

Sweet syndrome is an uncommon but clinically significant disorder that often mimics bacterial infections in the acute setting. It is typically defined by the triad of fever, neutrophilia, and painful erythematous skin lesions (1). Sweet syndrome may arise in various clinical contexts, including idiopathic forms, malignancy-associated cases, drug-induced reactions, autoimmune or inflammatory diseases, infections acting as triggering factors, and states of immune dysregulation or immunodeficiency (6). The presence of pancytopenia and profound neutropenia complicated the diagnostic process in our patient, masking the usual laboratory finding of neutrophilia.

From an emergency medicine perspective, several factors made the diagnosis challenging. Although Sweet syndrome is rarely encountered in the emergency department, this patient also presented with an atypical clinical picture compared with the classic presentation. The initial clinical impression was neutropenic fever with cutaneous infection, a common and potentially life-threatening condition in immunocompromised patients. In this setting, Sweet syndrome can closely mimic severe cutaneous infections (including cellulitis and ecthyma gangrenosum), and diagnostic suspicion may be further reduced because the classic laboratory clue of neutrophilia can be absent in the presence of neutropenia/pancytopenia. The lack of response to broad-spectrum antibiotics prior to admission highlighted the need to consider non-infectious etiologies in patients with persistent fever and atypical skin lesions. Histopathological evaluation of the punch biopsy revealed dermal neutrophilic infiltration without evidence of vasculitis, which was consistent with Sweet syndrome and critical for the definitive diagnosis.

Consistent with our findings, a few previous reports have described Sweet syndrome presenting in the setting of neutropenia, which can obscure the characteristic laboratory finding of neutrophilia and lead to an initial misdiagnosis of infection. Liu et al. reported a rare case of Sweet syndrome with histiocytic infiltration accompanied by neutropenia and emphasized that the absence of neutrophilia does not rule out the diagnosis (4). Isaac et al. described acute neutrophilic dermatitis in a patient with acute myeloid leukemia who developed severe neutropenia after chemotherapy and emphasized that Sweet syndrome should be considered in the differential diagnosis even in patients with febrile neutropenia and skin rashes (7). Additionally, a recently reported pediatric case demonstrated histiocytic Sweet syndrome with neutropenia and was diagnosed with autoimmune neutropenia (3). Together, these reports support our findings and underscore the importance of considering Sweet syndrome in neutropenic patients with persistent fever and atypical skin lesions.

Sweet syndrome is frequently associated with hematological malignancies, most notably acute myeloid leukemia and myelodysplastic syndrome (8). The pancytopenia and atypical lymphocytes observed in our patient raise concern for an underlying hematological disorder. This emphasizes the importance of close follow-up and hematology consultation to exclude malignancy, particularly in secondary Sweet syndrome. However, despite these concerning findings, no evidence of an underlying hematological malignancy was identified during her subsequent evaluations, demonstrating that Sweet syndrome can also occur in the absence of malignancy even when initial laboratory results suggest otherwise. Furthermore, our patient had no prior condition that would cause chronic immunosuppression, suggesting that the transient immunosuppressed state related to her infectious process may have predisposed her to the development of Sweet syndrome.

Although systemic corticosteroids are considered the gold standard treatment for Sweet's syndrome and often result in dramatic improvements, improvements following antibiotic treatment have also been reported (1, 9). In our case, systemic corticosteroids were not administered during the acute emergency department visit and hospitalization because the patient was evaluated as having severe neutropenia with fever and was treated with broad-spectrum antimicrobial therapy and close monitoring. When the biopsy results confirmed Sweet syndrome during the outpatient follow-up, steroid therapy was not added because the lesions had regressed. Her improvement with supportive therapy and antibiotics suggests that the clinical course may also have been influenced by the natural disease process and adjunctive topical treatment. Additionally, prior literature has reported variable improvement of Sweet syndrome lesions following appropriate antibiotic therapy in cases where lesions are secondarily infected/impetiginized (9). It could not be determined whether the improvement observed in our case was due to antibiotic therapy or the natural course of Sweet syndrome, but the growth of Staphylococcus aureus in the swab culture suggests that antibiotics contributed to the improvement.

The overall prognosis of Sweet syndrome is favorable in idiopathic cases but largely depends on the presence of associated underlying disease in secondary forms. Recurrence has been reported in up to 30–69% of cases, and malignancy-associated Sweet syndrome generally carries a poorer prognosis (10, 11). Therefore, patients should be monitored carefully not only for recurrence but also for the potential underlying hematological malignancies.

This case demonstrates that Sweet syndrome can develop in patients presenting with profound cytopenia and clinical features initially suggestive of neutropenic fever. In our patient, pancytopenia and fever increased suspicion of underlying hematologic malignancy; however, subsequent evaluations revealed no evidence of malignancy, demonstrating that Sweet syndrome can occur in the setting of immunosuppression without an underlying neoplastic disorder. Our case highlights the importance of considering Sweet syndrome in the differential diagnosis of febrile neutropenic patients with atypical skin lesions and underscores the value of dermatologic evaluation and skin biopsy in clarifying the diagnosis.

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Informed consent: Written informed consent was obtained from the patient.

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