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Giant cellulitis-like Sweet syndrome mimicking cellulitis: a case report

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Abstract

Background Sweet syndrome (acute febrile neutrophilic dermatosis) is an uncommon inflammatory disorder characterized by the abrupt appearance of painful, edematous, and erythematous papules, plaques, or nodules on the skin. There are various subtypes, such as classical, drug-induced, malignancy-associated, and the less common variant giant cellulitis-like Sweet syndrome. This case is unique due to its presentation of the giant cellulitis-like variant of Sweet syndrome in a patient from Ethiopia. The unusual distribution of the skin lesions and the initial lack of response to antibiotics make this case particularly noteworthy. It underscores the importance of considering Sweet syndrome in differential diagnoses when faced with atypical skin manifestations and ineffective antibiotic treatment. This contribution adds valuable insights to the scientific literature by highlighting the need for heightened awareness of this rare variant and improving diagnostic accuracy in similar clinical scenarios.

Case presentation A 60-year-old Ethiopian male patient who presented to the accident and emergency department with a 5-day history of fever, chills, sweating, and rigor accompanied by a reddish skin color change around the anterolateral region of the right chest wall. On physical examination, there were erythematous, indurated tender plaques with ill-defined borders over the right antero- and posterolateral chest wall with extension to the lateral part of the right neck and medial aspect of the right arm. Subsequently, the patient was started on antibiotics, but there was a suboptimal response. Skin biopsy revealed features suggestive of giant cellulitis-like Sweet syndrome. He was then started on steroids, which significantly improved his symptoms.

Conclusion A cautious stance is essential when identifying Sweet syndrome in individuals displaying erythematous plaque-like skin lesions in atypical areas of the body with uneven distribution. Such presentation may signal Sweet syndrome rather than a common infection. If conventional treatments, such as antibiotics, fail to resolve symptoms, consider Sweet syndrome as a potential diagnosis. This approach ensures timely and appropriate treatment, preventing treatment delay and misdiagnosis.

Keywords Sweet syndrome, Cellulitis, Giant-cellulitis-like Sweet syndrome

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Background

Sweet syndrome (acute febrile neutrophilic dermatosis) is an uncommon inflammatory disorder characterized by the abrupt appearance of painful, edematous, and erythematous papules, plaques, or nodules on the skin. It is involved in a wide spectrum of diseases caused by neutrophilic dermatoses [1]. The etiology includes the following subtypes: classical (after upper respiratory tract infections), malignancy-associated and drug-associated Sweet syndrome. However, there are lesser common variants, such as giant cellulitis-like Sweet syndrome, which refers to the development of large infiltrated inflammatory plaques [2]. This case is unique for presenting the rare giant cellulitis-like variant of Sweet syndrome, characterized by large, infiltrated plaques in atypical areas such as the chest, neck, and arm. This clinical variant can masquerade as cellulitis because the patients present with an acute onset of large erythematous plaques, fever, and leukocytosis with neutrophil predominance [1]. This variant is often associated with systemic inflammatory and neoplastic disorders [2].

Case presentation

A 60-year-old Ethiopian male patient presented to the accident and emergency (A and E) department with a 5-day history of fever, chills, sweating and rigor accompanied by a reddish skin color change around the anterolateral region of the right chest wall that was small in size. In addition, he complained of right upper extremity and right-sided chest pain.

Since the onset of his symptoms, he has been fatigued and sleepy. Four days prior, he was injured in the proximal area of his right index finger by a metallic object while fixing a car. His medical history revealed essential hypertension with good control while on enalapril and dyslipidemia on atorvastatin. He had a personal history of alcohol intake of two to three beers per day, khat intake two times a week and cigarette smoking of eight pack years.

On presentation, he had a blood pressure of 90/60 mmHg, a pulse rate of 100, a respiratory rate of 26, an oxygen saturation of 75% with room air and a temperature of 35.8 °C. His random blood sugar was determined to be 45 mg/dL. On evaluation, there were erythematous, indurated tender plaques with ill-defined borders over the right antero- and posterolateral chest wall with extension to the lateral part of the right neck and medial aspect of the right arm.

The initial parameters were as follows: white blood cell count (WBC) 17.59×10^3/μL with neutrophilia of 88.4% and lymphopenia of 3.6%, a platelet count of 88×10^3/μL, a C-reactive protein concentration of 200 mg/L, a blood urea nitrogen concentration of 114.58 mg/dL, and a creatinine concentration of 1.59 mg/dL. On liver function tests, alanine aminotransferase was 3.8× elevated, and total and direct bilirubin were 1.75 mg/dL and 0.75 mg/dL, respectively. His serum sodium level was 115 mmol/L, hemoglobin A1C was 6%, and his human immunodeficiency virus (HIV) antibody was negative.

Soft tissue ultrasound revealed increased echogenicity and thickening/edema of the right lateral chest and abdominal wall subcutaneous tissue with a cobble stone appearance. There was slightly increased flow on color Doppler sonography with right lateral chest and abdominal wall soft tissue inflammatory changes suggestive of cellulitis. Chest X-ray was suggestive of right-sided pleural effusion.

As our initial impression was chest wall cellulitis, he was started on ceftriaxone 1 g intravenous twice daily and vancomycin 750 mg every 48 h (renal adjusted dose), but there was no improvement after 48 hours. Furthermore, the skin lesion extended from the chest wall to the abdominal wall and upper third of the thigh after admission (Fig. 1). The WBC count increased to 45.58×10^3/μL, with 96% neutrophilia. Therefore, ceftriaxone was changed to meropenem 1 g intravenous three times a day, and vancomycin was adjusted to a full dose of 1 g intravenous twice daily after the patient's renal condition improved. This management was continued for 14 days but there was no significant improvement. During this course of management the result of the histopathological examination for the skin biopsy showed parakeratotic and moderately acanthotic epidermis overlying significant edema of the papillary dermis associated with a pattern of nodular and diffuse dermatitis. The inflammatory cells were composed of numerous neutrophils and leukocytoclastic debris of neutrophils mixed with lymphocytes and few eosinophils as well as plasma cells in the absence of signs of vasculitis. The infiltrates also extended down to the subcutaneous fat, which was suggestive of Sweet syndrome as seen in Fig. 2.

The atypical distribution of the plaque, lack of early response, extensive subcutaneous edema and serous oozing were indicative of inadequate response to antibiotics alone. Therefore, a diagnosis of Sweet syndrome was made after the skin biopsy, and as a result, the patient was started on 1 mg/kg/day prednisone. One week after starting steroids, there was complete resolution of the subcutaneous edema and a significant decrease in oozing with general well-being and complete independence with activities of daily living. Moreover, there was a significant decrease in WBC count from 45.58×10^3/μL to 11.51×10^3/μL. The patient has been adherent to oral steroids and as a result he had 10 kg weight gain over a 4-month period. In addition, he had impaired glucose tolerance with hemoglobin A1C of 6.7%. Otherwise, he

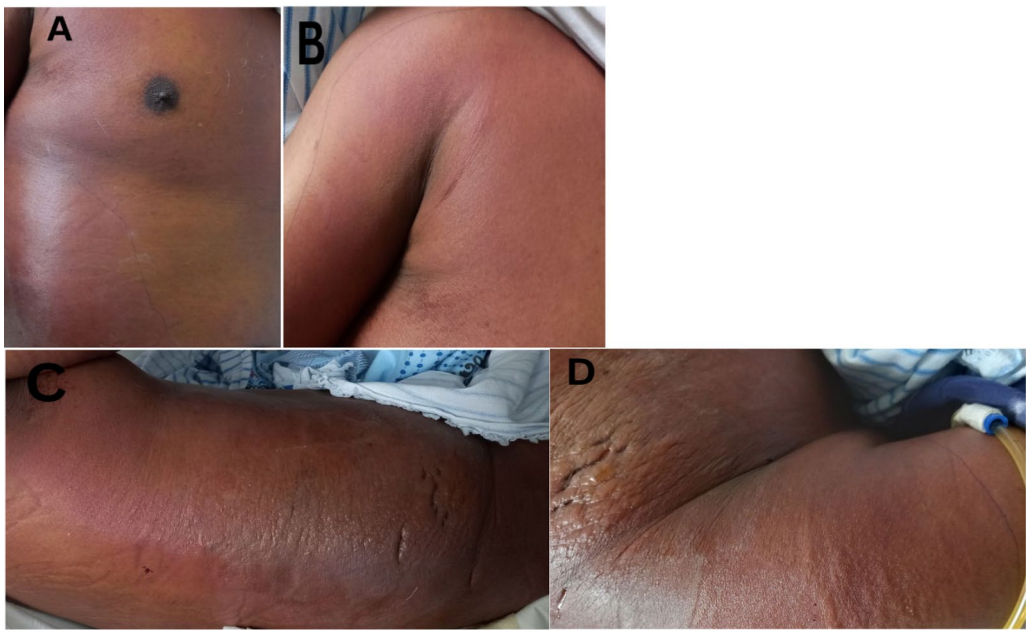


Fig. 1 Erythematous, tender plaque with ill-defined borders involving the **A** right side of the chest, **B** right medial aspect of arm, **C** right lateral trunk, **D** right flank and thigh

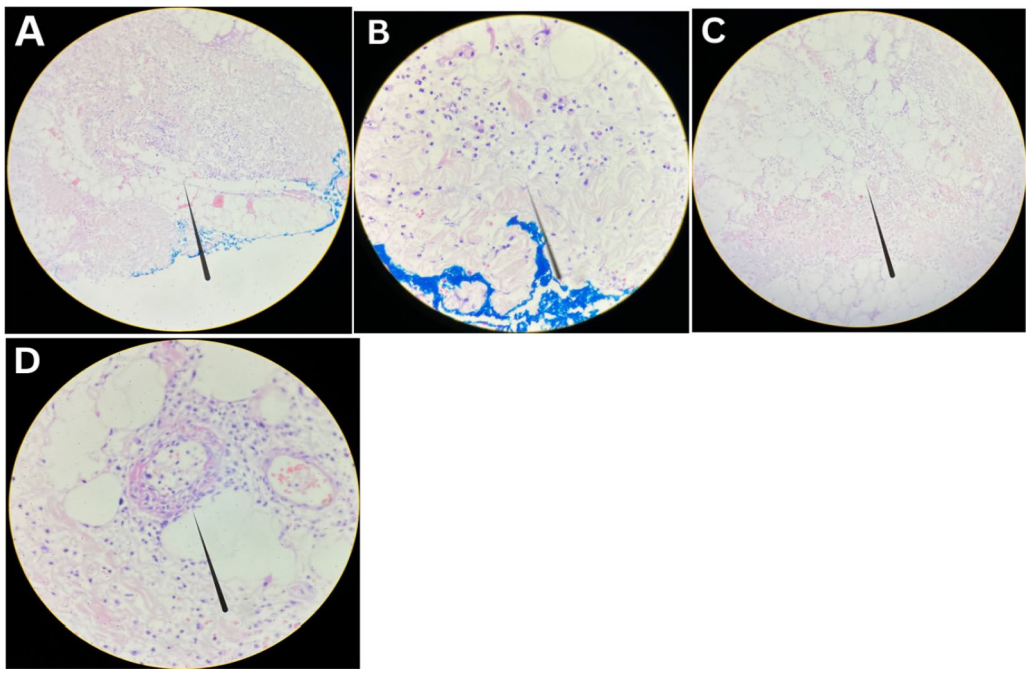


Fig. 2 **A**–**D** Neutrophils and leukocytoclastic debris of neutrophils mixed with lymphocytes and few eosinophils as well as plasma cells in the absence of signs of vasculitis, the infiltrates have also extended to the subcutaneous fat

did not have any epigastric discomfort, bloating or any sign of infection.

Discussion

Sweet syndrome is an inflammatory disorder characterized by erythematous plaques, nodules, or papules with prodromal symptoms of fever, malaise, arthralgia,

and diffuse infiltration of neutrophils in the papillary dermis. Skin findings are typically asymmetrical in distribution, with the upper extremities, trunk, head, and neck being the typical sites of involvement [3]. Although the size of the lesions ranges from millimeters to centimeters in diameter, there have been reported cases of large cellulitis-like plaques as a rare variant of Sweet syndrome, as observed in our patient [2]. Similar case reports have indicated history of malignancy, thus malignancy-associated Sweet syndrome [1, 2, 6]. While this was not observed in our patient, he may have had a preceding infection as seen in the 18 patients reported in 2013 [3].

Neutrophilic infiltration of other organs, such as the liver, spleen, heart, kidneys, central nervous system, and gastrointestinal system, may occur in Sweet syndrome. Although many extra-cutaneous manifestations of Sweet syndrome have been described in the literature, pulmonary manifestations are rare [4]. Sweet syndrome can present with uncommon features, such as pulmonary infiltrates and pleural effusion, as observed in our patient [5]. A case review of 34 cases on pulmonary involvement in Sweet's syndrome showed that skin involvement preceded pulmonary involvement, and bilateral or unilateral pulmonary infiltrates were the most common radiological feature. Pleural effusion accompanying lung infiltrates was uncommon and was present in only seven patients [6]. In patients with liver involvement, such as this patient, hepatic serum enzyme elevations in aspartate aminotransferase (AST) and alanine aminotransferase (ALT) with increased lactate dehydrogenase (LDH) might be observed [7]. However, hepatic involvement was not mentioned in other cases during our literature review [1–3, 6].

Skin biopsy is required to confirm the diagnosis, and it is demonstrated by detecting histopathological evidence of a dense neutrophilic infiltrate without leukocytoclastic vasculitis, which was similarly seen in this patient [2].

The gold standard of treatment is systemic corticosteroids, but topical and/or intralesional corticosteroids may also be effective as either monotherapy or adjuvant therapy. Prednisone is initially recommended at 0.5–1 mg/kg per day, followed by a tapering dosage to prevent recurrence. We expect clinical improvement after a few doses and complete resolution with 1–2 weeks of treatment [8]. This was evident in our patient as he had significant improvement of his symptoms within a week of starting prednisolone. In some cases, skin lesions may partially improve with antibiotics if there is a secondary bacterial infection [1].

Conclusion

We should be vigilant in diagnosing Sweet syndrome in patients who present with erythematous plaque-like skin lesions in atypical regions of the body with asymmetrical distribution. This case highlights the diagnostic challenges and effective treatment approach for a rare variant of Sweet syndrome. A 60-year-old Ethiopian male presented with skin plaques, fever, and systemic symptoms that initially mimicked cellulitis but did not respond to conventional antibiotics. The diagnosis was confirmed through a skin biopsy showing characteristic neutrophilic infiltration. The condition improved significantly with corticosteroid therapy, demonstrating the importance of considering Sweet syndrome in cases of atypical cellulitis-like presentations that do not improve with standard treatments. This underscores the need for a comprehensive diagnostic approach and highlights corticosteroids as an effective treatment for this rare dermatosis.

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SM, AK, and ED took responsibility in the construction of the body of the manuscript. KG, MN, and LT organized and supervised the course of the project. AA reviewed the article before submission for its intellectual content.

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Availability of data and materials
Data sharing is not applicable to this article as no datasets were generated or analysed during the current study.

Declarations

Ethical approval and consent to participate
Ethical approval is deemed unnecessary by Meqrez General Hospital Review Board as this is a single rare case faced during clinical practice.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing of interests
The authors declare that they have no competing interests.

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