

BRIEF ARTICLES

Sweet Syndrome Following Gynecologic Surgery: Pelvic Abscess as a Cause

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ABSTRACT

Sweet syndrome (acute febrile neutrophilic dermatosis) is characterized by painful, erythematous skin lesions clinically, and a dense infiltrate of neutrophils in the upper and mid-dermis on histopathology. It may be associated with infection, particularly of the respiratory or gastrointestinal tract. We describe a case of Sweet syndrome in a 54-year-old woman provoked by a post-surgical pelvic infection.

INTRODUCTION

Sweet syndrome (SS), also known as acute febrile neutrophilic dermatosis, is characterized by the sudden onset of tender, erythematous cutaneous lesions (papules, nodules and plaques) accompanied by fever and leukocytosis. Extracutaneous manifestations may involve the eyes, internal organs, or the musculoskeletal system. SS may be associated with infection, malignancy or autoimmune disorders. Various drugs have also been implicated in this disorder.¹ A dense neutrophilic infiltrate in the absence of vasculitis is characteristically present in the upper dermis.² We describe a unique case of a patient who developed SS following pelvic abscess formation subsequent to gynecologic surgery.

CASE REPORT

A 54-year-old woman presented to her dermatologist six-weeks post-operatively after undergoing robotic hysterectomy, bilateral salpingo-oophorectomy and pelvic lymph node dissection for endometrial cancer. She had a three-day history of a burning, papulovesicular rash on her extremities. She also mentioned vague lower abdominal discomfort and right hip pain that developed two-weeks following her hysterectomy. She was afebrile upon presentation. Erythematous papules were noted on her extremities and trunk, with the largest lesions present on her knuckles and heels (Figure 1). Mucous membrane involvement was limited to one erosion on her soft palate with no conjunctival or genital lesions noted.

Figure 1: Erythematous papulovesicular lesions of Sweet syndrome on the patient's lower extremities.

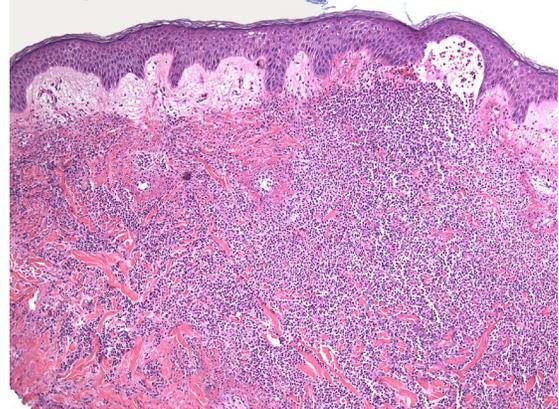


Punch biopsies taken for hematoxylin-eosin staining and direct immunofluorescence were taken from her right arm and revealed a dense neutrophilic infiltrate in the dermis with focal papillary dermal edema, consistent with SS (Figure 2). Laboratory analysis was significant for an elevated alkaline phosphatase level of 179 U/L (reference range, 33-130 U/L), alanine transaminase level of a 65 U/L (reference range, 6-29 U/L), aspartate transaminase level of 60 U/L (reference range, 10-35 U/L), a white blood cell (WBC) count of 6300 μ /L (78.4% neutrophils), and a platelet count of 363 $\times 10^3/\mu$ L. Additional laboratory findings included an erythrocyte sedimentation rate of 113 mm/h (reference range, <30 mm/h), C-reactive protein of 26.62 mg/dL (reference range, <0.80 mg/dL), and elevated anti-streptolysin O titer of 299 IU/mL. Anaplasma and Ehrlichia titers were negative.

Three days later the patient returned with a low-grade fever and worsening of her cutaneous symptoms. She noted arthralgias in her fingers and no myalgias. Her skin lesions were now painful and spreading more diffusely, involving her tongue, nose, buttock and feet. She was started on prednisone 40 mg/day, tapered every 5 days by 10 mg to which her skin lesions responded well. However, her abdominal pain progressively worsened and a CT of the abdomen and

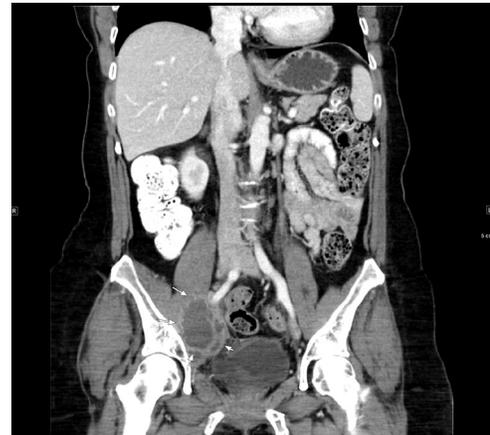
pelvis with IV contrast demonstrated a large 7.6 x 4.5 x 8 cm complex multiloculated, rim-enhancing fluid collection along the right pelvic sidewall, highly suggestive of a post-surgical abscess (Figure 3).

Figure 2: Dense neutrophilic infiltrate in the dermis with focal papillary dermal edema, consistent with Sweet's syndrome.



Under CT guidance, a drainage catheter was placed and 20 cc of purulent material was aspirated. Gram stain of the pelvic abscess drainage showed Gram positive cocci in chains. Culture of the same fluid grew out abundant Group C beta hemolytic streptococci, and no anaerobes. She was admitted to the hospital for treatment with intravenous ceftriaxone and metronidazole.

Figure 3: CT of the Abdomen & Pelvis with Intravenous Contrast, coronal view, showing a right iliopsoas abscess with displacement of the external iliac vasculature.



The patient was discharged five days later with a peripherally inserted central catheter for continued administration of antibiotics for an additional four weeks. She was also instructed to continue her prednisone taper for which she had eight days remaining. The patient's cutaneous lesions and discomfort improved dramatically and she attained complete resolution over the course of two weeks.

DISCUSSION

Diagnostic criteria for SS were last revised in 1994 by von den Driesch.³ To accurately diagnose SS, the patient must satisfy two major criteria: abrupt onset of tender erythematous nodules or plaques and histopathological evidence of a neutrophilic infiltrate without evidence of vasculitis. Additionally, the presence of two of four minor criteria is required. The minor criteria includes: 1) pyrexia over 38°C; 2) association with malignancy, inflammatory disease or preceded by an upper respiratory or gastrointestinal infection or vaccination; 3) excellent response to systemic corticosteroids or potassium iodide; and 4) laboratory values demonstrating erythrocyte sedimentation rate >20 mm/hr, positive C-reactive protein, >8,000 leukocytes or >70% neutrophils.³ Our patient fulfilled both major criteria and the latter 2 minor criteria for a diagnosis of SS.

Literature suggests that hypersensitivity reactions may be responsible for SS, given its association with malignancy, drugs, autoimmune diseases and infections.² Our patient was not on any SS-inducing medications.¹ Furthermore, despite our patient's history of endometrial cancer, her dermatosis developed 6 weeks after cancer resection, mitigating against malignancy-associated SS. Blood work also ruled out

hematologic malignancy, the most common cause of malignancy-associated SS.² The most common infectious associations include those of the upper respiratory tract (*Streptococcus*) and gastrointestinal tract (*Salmonella* and *Yersinia*). It is postulated that streptococcal infection induces SS from a hypersensitivity reaction to bacterial antigens.⁴

The gold standard of therapy for SS is administration of systemic corticosteroids, often beginning with 1 mg/kg/day of prednisone and tapering down to 10 mg/day over 4 to 6 weeks. Some patients however, may require treatment for 2 to 3 months.⁵ In a report by Panagiotakis et al, complete remission was achieved after 10 days of antibiotic therapy without the use of systemic corticosteroids.⁴ Similarly, although our patient's symptoms improved on prednisone, complete resolution of her symptoms occurred after drainage of her pelvic abscess, enabling prednisone to be discontinued.

The chronology in this case suggests that Group C beta hemolytic *Streptococcus* abscess formation following gynecologic surgery triggered the development of SS. Elevated anti-streptolysin O titers can indicate recent or current group A, C and G streptococcal infection. The elevated anti-streptolysin O titer was a clue to the underlying infection. Cutaneous findings compatible with SS may hint at an underlying infectious complication.

Conflict of Interest Disclosures: None.

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