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Staphylococcus aureus Sepsis as a Complication of Scabies

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To the Editor:

Scabies is a parasitosis considered to be a public health problem especially in developing regions of the world. Local and systemic secondary infections and other complications like acute poststreptococcal glomerulonephritis are major causes of morbility in this type of patient.[1]

A 2-month-old infant was admitted to hospital with a diagnosis of scabies and fever, and treatment with permethrin cream, 5%, was prescribed. The day before admission the infant developed a hot, erythematous, edematous plaque below the left knee (Figure 1). Two days later he was transferred to the pediatric intensive care unit with a high fever and respiratory distress. Physical examination revealed pale skin and mucous membranes, tachypnea, and subcostal and intercostal retractions. A hard, erythematous, and hot plaque had formed below the left knee with residual scabies lesions on the overlying skin. Vital signs included a heart rate of 108 beats per minute, respiratory rate of 55 breaths per minute, blood pressure of 51/21 mm Hg, oxygen saturation of 93% by pulse oximetry (oxygen mask with reservoir at 15 L/min), and a body temperature of 38ºC.

Laboratory analysis revealed a white cell count of 13 200 cells per mm^3 (16% band neutrophils and 20% segmented neutrophils) and an increase in acute phase reactants (C-reactive protein, 187 mg/L; procalcitonin, 50.5 ng/mL). Empiric treatment was started with intravenous cefotaxime (25 mg/kg/d) and teicoplanin (10 mg/kg/d).

The general condition of the patient deteriorated in the 24 hours following admission to the pediatric intensive care unit with increased signs of respiratory distress. A further chest x-ray revealed empyema and hemothorax in the left lung that required drainage of 35 cm^3 of yellowish fluid with a pH of 6.86. The abscess on the knee was also drained (Figure 2). Blood cultures and cultures from the skin lesion and the empyema were positive for *Staphylococcus aureus*. The prescribed antibiotics were replaced with intravenous cloxacillin (25 mg/kg/d) for 14 days, and a favorable response was seen in the patient.

Scabies is an infestation caused by the *hominis* variant of the *Sarcoptes scabiei* mite, a human parasite that tunnels under the epidermis. It affects both sexes and all age groups equally. There are more than 300 million new cases each year all over the world.[2] Scabies is a highly contagious disease that is generally transmitted by direct human contact, although cases have been described in which transmission occurred through contact with fomites and contaminated animals.

Secondary bacterial infections can sometimes occur in skin lesions and cause local and, less commonly, systemic complications.[3,4]

A study by Itzhak Brook[3] analyzed the bacterial flora found in lesions with secondary infections and found the most common aerobe was *S aureus*, while the most common anaerobes were *Peptostreptococcus* species.

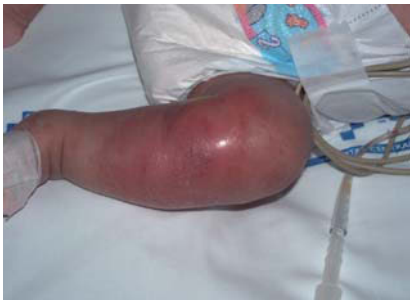

Figure 1. Hot erythematous and edematous plaque on the leg.

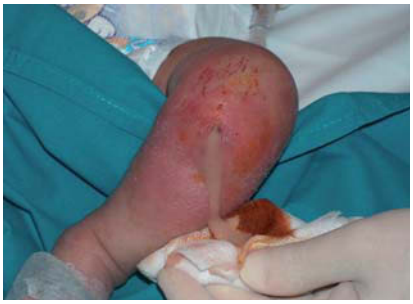

Figure 2. Drainage of purulent material from the abscess on the knee.

Secondary infection with *Streptococcus pyogenes* can trigger acute poststrepcococcal glomerulonephritis and rheumatic fever.[5,6]

The exact pathogenic mechanism associated with the organisms isolated in skin lesions with secondary infection has not yet been determined. In our case, the pathogenic role of *S aureus* was clear, as it was isolated in the blood, the pleural fluid, and the skin lesion. Thus the parasite entered the epidermis through a break in the skin leading to bacteremia and the consequent empyema.

Early diagnosis of scabies is essential in order to initiate appropriate treatment with a scabicide. Similarly, secondary bacterial infection must be managed through local or, occasionally, systemic antibiotics, and pus must be drained from abcesses[3] in order to avoid complications that could potentially endanger the life of the patient.

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Eruptive Xanthomas After Onset of Diabetes Mellitus

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To the Editor:

We recently treated a 33-year-old man who was admitted to our hospital with abdominal pain accompanied by nausea, vomiting, and hyperglycemia that had begun 4 days earlier and reflected the onset of diabetes mellitus. The patient had a history of hypertension diagnosed in the last 3 months, predominantly abdominal obesity, with a body mass index of 31.5 kg/m^2, severe alcoholism, and hypercholesterolemia diagnosed a year ago. He was receiving dietary treatment. The patient's father had type 2 diabetes mellitus that began in his thirties.

The patient reported polyuria, polydipsia, and polyphagia for the last 3 weeks, along with weight loss of 10 kg. Around that time, he began to develop erythematous papules of 1 to 4 mm in diameter on his back, and these turned yellow within a few days. Some of the lesions had a peripheral halo and were accompanied by mild pruritus. The lesions were initially distributed on the back but later spread to the arms and legs, buttocks, and in particular, the sacral region (Figure 1).

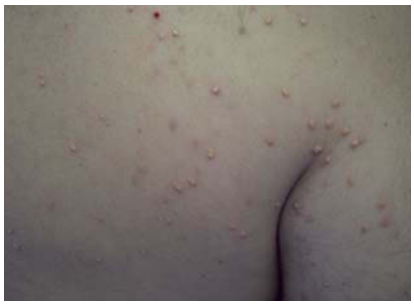

Figure 1. MMultiple yellow papules with a peripheral erythematous halo on the back of the arm and the back.

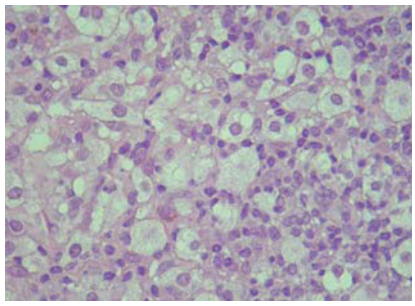

Figura 2. Macrophages loaded with intracellular lipids (foam cells) (hematoxylin-eosin, ×400).

Laboratory analysis during admission revealed the following: glucose, 257 mg/dL; total cholesterol, 418 mg/dL; triglycerides, 853 mg/dL; high-density lipoprotein cholesterol, 32 mg/dL; low-density lipoprotein cholesterol, 218 mg/dL; direct bilirubin, 0.1 mg/gL; indirect bilirubin, 6.1 mg/dL; aspartate aminotransferase, 18 mU/mL; alanine aminotransferase, 20 mU/mL; γ-glutamyltransferase, 66 mU/mL; lactate dehydrogenase, 398 mU/mL; and alkaline phosphatase, 230 mU/mL. Gasometric analysis of venous blood revealed slight metabolic acidosis. Thyroid function, insulinemia, and C-peptide concentrations were within normal ranges and analysis of anti-islet cell antibodies was negative.

Abdominal ultrasound revealed diffuse hepatic steatosis with hepatomegaly. Histology of the skin lesions (Figure 2) revealed infiltration of the superficial and middle dermis by uniform polygonal mononuclear macrophages with a foamy cytoplasm, with a tendency toward perivascular aggregation and without accompanying