



# Cutaneous eruption associated with *Streptococcus dysgalactiae* (group C *Streptococcus*) bacteremia

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*Streptococcus dysgalactiae*, a group C *Streptococcus* (GCS), is a Gram-positive bacterium that is a commensal colonizer of the upper respiratory tract and skin. Initially thought to be nonpathogenic, increasing evidence suggests it is an important bacterial pathogen in humans.<sup>1</sup> It has been implicated in a variety of infections including pneumonia, endocarditis, and cellulitis.<sup>2,3</sup> Recognition of this pathogen is critical, given the 25% mortality rate associated with serious GCS infection.<sup>4</sup>

Although there is increasing evidence that GCS is an important pathogen, little exists in the literature about dermatologic manifestations associated with GCS infection. Here we report a case of cutaneous lesions associated with GCS bacteremia.<sup>5</sup>

## CASE REPORT

A 48-year-old male with a history of Down syndrome and gout presented to a dermatology clinic with a 2-day history of a new rash. The rash first appeared on his buttocks and spread to his thighs. It was pruritic and nontender. The only new medication exposure was a short course of indomethacin 3 weeks before presentation. The patient denied new exposures, travel, or new contactants. A review of his medical records indicated he had a productive cough for a week that prompted his primary care physician to obtain a chest x-ray, the findings from which were normal. Physical examination showed symmetric, well-demarcated, and erythematous plaques on his buttocks and thighs bilaterally. Drug reaction was less likely given the lack of truncal

### Abbreviations used:

GCS: group C *Streptococcus*  
URI: upper respiratory tract infection

involvement. There was no pain or rubor suggestive of cellulitis. A diagnosis of contact dermatitis was favored given the bilateral and symmetric distribution and pruritus. He was instructed to use emollients and triamcinolone 0.1% ointment, as well as hydroxyzine for the itch.

A punch biopsy revealed dermal edema and a mixed inflammatory infiltrate extending to the mid-dermis comprised of lymphocytes, neutrophils, and rare eosinophils (Fig 1). Overall, the findings of the biopsy were interpreted as most consistent with a hypersensitivity reaction.

Two days later, the patient presented to the emergency department with a worsening rash, fever, and chills. Clinical exam showed tachycardia, a temperature of 101°F, and a rash, which was now diffuse on his trunk and legs (Fig 2). Labs were positive for leukocytosis of  $17.3 \times 10^3$  cells/uL with a neutrophilic predominance of 91%, transaminemia with an aspartate transaminase of 58 U/L and alanine transaminase of 75 U/L, as well as an elevated alkaline phosphatase of 310 U/L. He was admitted for presumed sepsis and treated with 1.25 g vancomycin intravenously twice daily. For the rash, diagnostic consideration was given to cellulitis, Sweet syndrome, and acute generalized exanthematous pustulosis. Subsequently, blood

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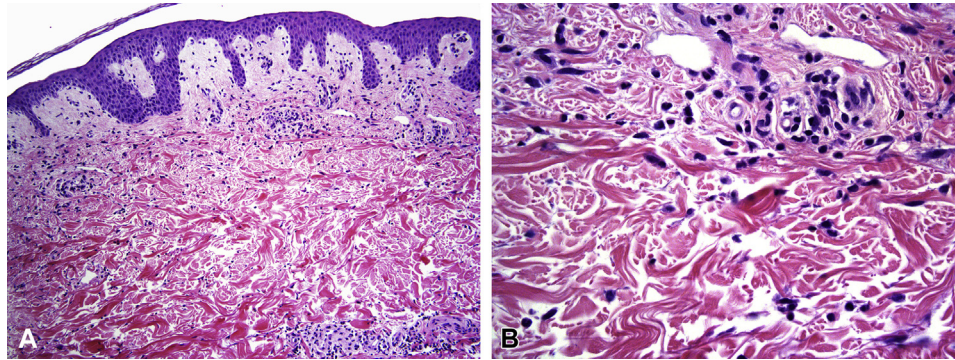
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**Fig 1.** **A**, Punch biopsy from the right lateral thigh showing superficial dermal edema and a mixed inflammatory infiltrate. **B**, The infiltrate is composed primarily of lymphocytes and neutrophils with rare eosinophils. (**A** and **B**, Hematoxylin-eosin stain; original magnifications: **A**,  $\times 10$ ; **B**,  $\times 40$ .)



**Fig 2.** Cutaneous eruption associated with *Streptococcus dysgalactiae* bacteremia. **A**, Erythematous plaques on the lateral right thigh and right lower leg with associated scattered petechiae at the periphery. **B**, Diffuse brightly erythematous and edematous plaques on the back with islands of sparing. **C**, Erythematous and edematous plaque on the right abdomen.

cultures returned positive for *Streptococcus dysgalactiae* in 3 out of 4 vials; however, given the pruritus, diffuse and bilateral nature of the eruption, a reactive process to the patient's infection was favored.

On day 4 of hospitalization, his blood cultures had been clear for >72 hours, his rash was markedly improved, and his liver enzymes had normalized, which was most consistent with cholestasis secondary to sepsis. He was discharged with a 2-week course of cefadroxil 1 g twice daily by mouth. The patient's rash resolved within 2 weeks of discharge.

## DISCUSSION

GCS only constitutes 0.28% of blood stream infections.<sup>6</sup> Most GCS bacteremia is seen in patients with preexisting medical conditions such as malignancy or immunosuppression.<sup>4</sup> Given that our patient was young and healthy, his GCS bacteremia and associated skin changes make it an unusual case.

This case is the second reported in the literature of cutaneous lesions associated with GCS bacteremia.<sup>5</sup> Our case shares many similarities with the previously reported case; both patients developed pruritic,

erythematous cutaneous eruptions in the setting of GCS bacteremia, both rapidly cleared with antibiotics, and both had skin biopsies showing sparse inflammation. In contrast with the previous case, our patient had a more widespread rash and also primarily had indurated plaques rather than figurate cutaneous lesions. This demonstrates that the dermatologic manifestations of GCS infection can be variable. His rapid recovery with antibiotics further supported that the rash was infection-related and suggests that other etiologies on the initial differential diagnosis, such as drug reaction, Sweet syndrome, and acute generalized exanthematous pustulosis, were unlikely.

It remains unclear how the patient acquired GCS bacteremia. Two of the most common GCS primary infection sites are the upper respiratory tract and skin.<sup>4</sup> An upper respiratory tract infection (URI) is the more likely route in this case given the productive cough preceding his rash. A URI was also the primary infection in the previously reported case. Down syndrome is a risk factor for frequent and severe URIs, which might have predisposed the otherwise healthy patient to the bacteremia.<sup>7</sup> Although GCS is a

common culprit in cellulitis, this was not favored by 2 different dermatologists.<sup>3</sup>

One possibility for the cutaneous lesions is a toxin-mediated process. GCS shares many virulent factors with group A *Streptococcus* including superantigens implicated in toxic shock syndrome, such as streptococcal exotoxin type G.<sup>8</sup> Thus, it is possible that dissemination of superantigens or toxins led to a reactive cutaneous process. Although, it is worth noting our patient's rash morphology was distinct from the classic scarlatiniform rash seen with group A *Streptococcus* toxic shock syndrome.

GCS is increasingly becoming recognized as an important human pathogen. It should be considered in cases of URI or cellulitis with an associated cutaneous eruption. Fortunately, GCS is universally susceptible to  $\beta$ -lactam antibiotics and is easily treated with first-generation cephalosporins.

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