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Bullous impetigo caused by *Streptococcus salivarius*: a case report

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SUMMARY A 19-month-old child presented with bullous impetigo around the perineal region, penis, and left foot. *Streptococcus salivarius* was the only isolate recovered from the lesions. The child was treated with parenteral penicillin, debridement of the bulli, and local application of silver sulphadiazine cream. This case of bullous impetigo illustrates another aspect of the pathogenicity of *Strep. salivarius*.

Streptococcal pyoderma or impetigo is a common problem in children. The infection usually begins with an outbreak of vesicular lesions on the arms and legs and on occasion they are seen around the nose, mouth, and scalp. The blisters are usually flat and contain clear fluid, which usually yields a pure culture of group A beta-haemolytic streptococci and, rarely, group C or G.[1]

This is a report of a 19-month-old child who presented with bullous impetigo. The organism recovered from the bullous fluid was *Streptococcus salivarius*, found in the normal mouth flora.[2 3] Although this organism has been isolated from patients with endocarditis, sinusitis, mastoiditis, cholangitis, peritonitis, pneumonia, and meningitis[4-6] it has never been recovered from bullous impetigo.

Case report

A 19-month-old black boy was admitted to Children's Hospital National Medical Center for the treatment of watery blisters around the penis, perineal area, and the dorsal area of the foot. These lesions appeared two days before admission and were accompanied by a temperature up to 38 °C. Past medical and family histories revealed no abnormalities. Physical examination showed a child in moderate distress, pulse 136/min, temperature 38·6 °C, and respiration 40/minute. Several blisters filled with light yellow liquid were observed around the perineal region, the glans and shaft of the penis, and the left heel and foot. The diameter of the blisters was 3-9 cm (Figs 1 and 2). Lymph nodes were enlarged in both inguinal areas. The rest of the physical and neurological examinations were normal. Admission laboratory data showed a haemoglobin of 12·9 g/dl, haematocrit 38%, white blood cells 11·2 × 10^9/l (11 200/mm^3) with 54% segmented neutrophils, 24% lymphocytes, 18% monocytes, 2% eosinophils, and 2% bands. Urinalysis was normal and blood cultures were negative.

The large bulli were aspirated and debrided, and their contents were sent for culture for aerobic and anaerobic bacteria and viruses. Cultures of vesicular fluid obtained from two vesicles by direct needle aspiration (the left foot and the perineal region) yielded a pure growth of an alpha-haemolytic streptococcus, which was identified by the Center for Disease Control as *Strep. salivarius*. Biopsy of the lesion demonstrated the presence of intraepidermal bulli containing Gram-positive cocci in chains and proteinaceous material, many neutrophils, and a few lymphocytes. Some of the bulli that were not aspirated persisted for 7 to 10 days. Treatment was started with the local application of silver sulphadiazine cream (Silvadene) and Phisohex soap baths and parenteral crystalline penicillin 100 000 units/kg per day for seven days. The patient responded well to therapy, and the denuded area showed slow epithelialisation and complete healing of the skin within two weeks.

Discussion

Current evidence suggests that impetigo can be divided on clinical and bacteriological grounds into two basic forms: a bullous type, which ultimately forms thin, varnish-like crusts and is primarily staphylococcal in origin, and a vesicular type, which

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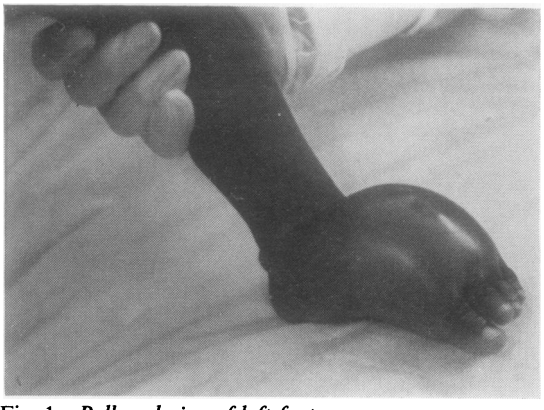

Fig. 1 *Bullous lesion of left foot.*

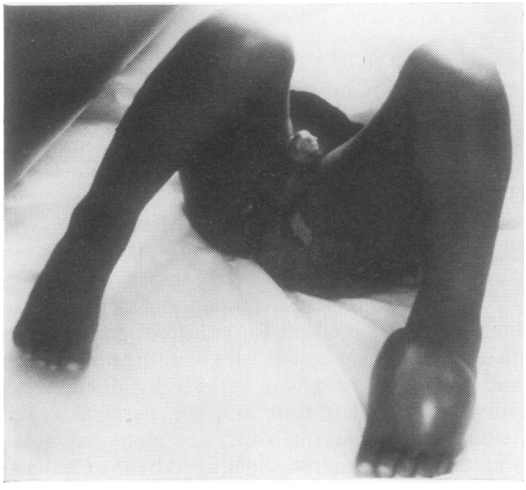

Fig. 2 *Bullous lesions in perineum and on left foot.*

develops thick 'stuck-on' crusts and is primarily streptococcal in aetiology.[7] The relative frequency of these two kinds of impetigo varies from place to place and from time to time.[7-9]

The initial lesion of streptococcal impetigo is a tiny papule. A vesicle filled with serous fluid and a few leucocytes rapidly forms and is surrounded by a narrow rim of erythema. The blisters are flat, with a diameter of 1 to 3 mm. The clear fluid of such a vesicle usually yields a pure culture of group A streptococci. The vesicles rapidly become pustular, and the fluid can infect by direct extension other areas, where new vesicles form. All these vesicles soon break, and the surface becomes covered by a thick, hard, brownish 'candy' crust, which is characteristic of streptococcal impetigo. Although the final lesions may be as large as 3 cm in diameter, they remain discrete. The lesions may later become secondarily infected by staphylococci which are present on normal skin.

Staphylococcal impetigo is characterised by persistent bullous lesions, which subsequently form thin, varnish-like crusts which may be white or grey in colour. The bullous form of impetigo usually yields pure cultures of phage type 71 staphylococci or closely related strains.[10 11]

It is of interest that our patient developed bullous lesions, which were microscopically and clinically similar to staphylococcal impetigo, whereas the lesions persisted for many days without spontaneous rupture. However, the only organism recovered was *Strep. salivarius*. *Strep. salivarius* is usually found in the mouth and throat. Several investigators have observed high levels of this organism on the dorsum of the tongue and in saliva but low levels in dental plaque.[2 3]

Strep. salivarius belongs to the group of poorly defined viridans streptococci. Differentiation of this organism may be best achieved by its physiological characteristics,[5] although a number of strains of *Strep. salivarius* show reactions with Lancefield group K antiserum.

As indicated by sensitivity tests and clinical response, penicillin G is the antibiotic of choice in treating infections due to *Strep. salivarius*.[4] These organisms are sensitive to the majority of antibiotics except aminoglycosides and tetracycline. Specific differentiation of viridans streptococci by biochemical and serological means should provide a better appreciation of the pathogenicity, antimicrobial susceptibility, and epidemiology of this heterologous group of microorganisms.

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