鏈球菌性肛門周圍皮膚炎

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Perianal Streptococcal Dermatitis

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Perianal streptococcal dermatitis (PSD) is a superficial bacterial infection of the parianal area. PSD is usually caused by *group A β-hemolytic streptococci* and rarely, the *group G β-hemolytic streptococci* or *staphylococcus aureus*. Symptoms include perianal rash, itching and rectal pain; blood-streaked stools may be also seen in one third of patients. It primarily occurs in children between 7 months and 11 years of age. We report a 6-year-old boy with PSD who developed desquamation of his fingers and perioral skin. The desquamation developed concomitant with resolution of perianal dermatitis. We propose that desquamation in this patient was due to PSD and its release of streptococcal exotoxin. Therapy by oral penicillin and topical mupirocin is effective. (Dermatol Sinica 21 : 142-145, 2003)

Key words: Perianal streptococcal dermatitis, PSD, *Group A β-hemolytic streptococci*, Desquamation, Exotoxin

鏈球菌性肛門周圍皮膚炎(Perianal streptococcal dermatitis)是因感染A群貝它融血性鏈球菌(*group A β-hemolytic streptococci*)導致肛門周圍皮膚之表淺性皮膚炎。臨床上的表現有紅斑、癢感、肛痛；三分之一的病人大便帶有血絲。此病好發於7月大到11歲大的小孩。在此我們報告一位6歲的男孩罹患鏈球菌性肛門周圍皮膚炎。此病患經給予口服penicillin和局部塗抹mupirocin軟膏後痊癒，然而此病患在病灶緩解時伴隨出現手指末端和唇部周圍皮膚脫皮之現象。我們認爲這脫皮之現象是鏈球菌外毒素所引起的。(中華皮誌21 : 142-145, 2003)

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INTRODUTION

Perianal streptococcal dermatitis (PSD) was first described by Amren et al in 1966.[1] It was initially described as a perianal cellulitis. More recent reports have suggested using the term perianal streptococcal dermatitis since that its skin inflammation tends to be superficial.2 PSD is caused by *group A β-hemolytic streptococci* (GABHS). PSD in children is much more prevalent than was previous thought. The disease is frequently misdiagnosed and treated inappropriately. The typical clinical presentation includes well defined erythema arising in the perianal area, perioral and acral scarletiniform desquamation. The clinical features are being continually expanded as this disorder becomes more widely recognized. We describe a 6-year-old boy with PSD who developed desquamation of his fingers and perioral skin.

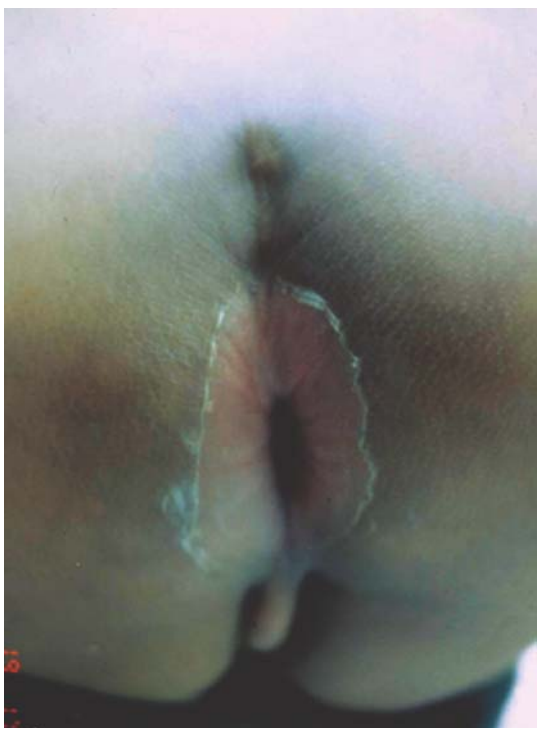

Fig. 1
The perianal area and gluteal cleft were edematous and erythematous, with well demarcated margins and minimal desquamation.

CASE REPORT

A 6-year-old boy presented with a 3-day history of perianal rash with itching and painful defecation. He denied fever, sore throat or other skin rash. On physical examination his oropharynx and tongue was clear of erythema and exudate. The perianal area and gluteal cleft were edematous and erythematous, with well demarcated margins and minimal desquamation (Fig. 1). A scant mucoid discharge was presented. Digital examination of the rectum revealed soft blood-streaked stools. Results of laboratory examinations, including red and white blood

Fig. 2
Desquamation of the boy's fingertips was noted after 4 days of therapy.

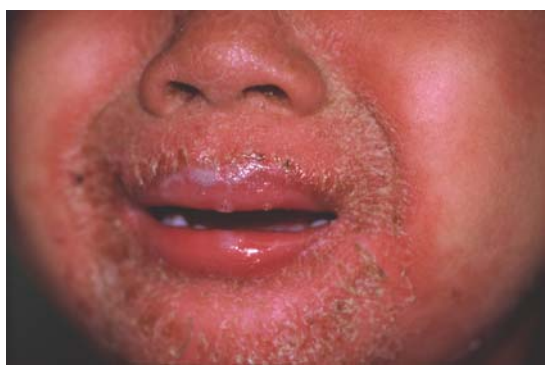

Fig. 3
Desquamation of the perioral skin was also noted.

cell counts, erythrocyte sedimentation rate, urine microscopy and sedimentation as well as stool examination were within normal limits. Cultures were obtained both from the perianal skin and the oropharynx. The perianal swab culture grew for GABHS. The throat swab culture was negative for GABHS. The antistreptolysin-O (ASLO) test was positive at 1:320 (normal < 1:160). Oral penicillin and topical mupirocin ointment (Bactroban) were prescribed. Four days following the initiation of antibiotic therapy, his perianal erythema and painful defecation had resolved. Then, the boy began to have desquamation of his fingertips and perioral area (Fig. 2 and Fig. 3).

After a 14-days course of treatment, further bacteria culture by perianal and throat swabs were negative growth of GABHS. Regular urine analysis to follow up poststreptococcal glomerulonephritis was suggested.

DISCUSSION

A distinctive perianal eruption caused by GABHS was initially described by Amren *et al*.[1] However, little attention was paid to it until 1985 when Spear *et al*.[2] described 14 children with the condition and designated it cellulitis. Increased awareness of this entity has fostered better understanding of the clinical features. More recently, reports have suggested using the term perianal streptococcal dermatitis, since that its inflammation tends to be superficial.[3]

In one study, the incidence was reported to range from one in 218 to one in 2000 pediatric outpatient visits.[4] Probably PSD is more frequent than previously thought, possibly because it is often misdiagnosed. It is usually seen in children aged 7 months to 11 years. However, it has been described in adults.[5] For unknown reasons, this condition is more commonly found in males than in females.[1, 3, 4] Familial cases of PSD have been reported but the disease is rarely contagious.[6]

The causative agent of PSD is the GABHS and rarely, the *group G β-hemolytic streptococci* (GGBHS) or *staphylococcus aureus*.[7, 8] The GABHS is a frequently identified bacteria in the pharynx and tonsils of children, especially as a consequence of streptococcal throat infection or scarlet fever. It is likely that the GABHS descends from the throat into the stomach, where it survives the acid habitat, reaches the lower intestinal tract and colonizes the perianal area.[3] Infection is also possible through digital contamination from an infected oropharyx or other site infected by GABHS.

The clinical presentation is quite constant with well-defined erythema which always arising in the perianal area. It can be inflammatory and edematous with induration and tenderness. The most common features include dermatitis (90%), perianal itching (78%), rectal pain (52%), and blood-streaked stools (35%).[3] It is often associated with anal fissure, painful defecation, constipation and even protocolitis. Rarely, febrile cases of PSD had been reported.[9] Its course can be chronic and relapsing. Although most skin lesions of PSD do not spread, sometimes involvement of the perianal region as well as the genitalia resulting in vulvovaginitis or balanitis can be observed.[2, 6] Symptoms and signs can persist for weeks or months, since the diagnosis is generally late and the condition is frequently misdiagnosed.[10]

Digital desquamation was first reported by Michael J. *et al*. in 2000.[11] The patient that we described eventually developed desquamation not only of his fingers but also perioral skin, which to our knowledge, has not been heretofore described with PSD. Digital desquamation is well known following scarlet fever and streptococcal pharyngitis, resulting from the production of exotoxins. The timing of the desquamation in our case was in keeping with that seen following other streptococcal infections. It was developed concomitant with treatment and resolution of perianal dermatitis. We propose that desquamations in this patient of his fingers and perioral skin were due to PSD and its release of exotoxin since no other source of streptococcal infection was found.

Although the clinical picture of a sharply demarcated erythema is very characteristic, PSD is often misdiagnosed for long periods and

patients are subjected to treatments for a variety of diagnoses. The differential diagnosis includes cellulitis, erysipelas, intertrigo, candidiasis, contact dermatitis, seborrheic dermatitis, psoriasis, sexual abuse and pinworm infection. Kawasaki disease shares some clinical features with the PSD, including perineal erythema and periungual desquamation. Nevertheless, the absence of conjunctival injection, strawberry tongue, erythematous induration of limbs, and lymphadenopathy argues against a diagnosis of Kawasaki disease.

For many years, the preferred treatment has been a 14-day course of oral penicillin. Medina *et al*[12] suggested topical 2 % mupirocin alone or in addition to oral penicillin for PSD. Other treatment options include oral erythromycin and topical erythromycin could be considered.[13] Carrier status of streptococcus or post-streptococcal glomerulonephritis should be checked at follow-up visits.

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