Anal Colonization of Group G β-Hemolytic Streptococci in Relapsing Erysipelas of the Lower Extremity

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Four patients who had frequent relapses of erysipelas but no obvious portal of entry and no β-hemolytic streptococci in specimens from conventional culture sites all had group G streptococci in cultures of specimens from the anal canal. It is suggested that anal colonization with group G streptococci, and possibly group A and other β-hemolytic streptococci, may constitute a reservoir for streptococci in such cases.

A puzzling feature of erysipelas and cellulitis is the high relapse rate, usually >20% during 1–3 years of follow-up, and the fact that cultures for β-hemolytic streptococci (BHS) are often negative [1]. For patients with a chronic leg ulcer or skin trauma, BHS can often be isolated from the site of entry. Usually, however, the skin is intact, and cultures of specimens from routine sites and of needle aspiration fluid are negative [1, 2]. Group A streptococci (GAS) may induce perianal cellulitis [3], and it is well known that anal carriage of GAS in hospital staff members is a source of postoperative wound infections [4].

Furthermore, transient colonization of the gut and perianal area by GAS may occur after throat infection [5], and group G streptococci (GGS) and group C streptococci (GCS) are regarded as frequent colonizers of the human gut [6]. Asymptomatic anal or perianal colonization with BHS may thus constitute a reservoir for streptococci in cases of erysipelas and cellulitis. To address this question, anal swab specimens from 4 patients with erysipelas were cultured. Informed consent was obtained from all patients.

Case Reports

Case 1. A man born in 1953 had a history of psoriasis, muscular dystrophy, obesity, and weakness on the left side after a cerebrovascular accident in 1994. On 5 occasions between April 1995 and November 1996 he was admitted with erysipelas involving the left thigh and buttock. In January 1996, swab specimens from the anal canal and a psoriatic lesion yielded rich growth of GGS. GGS were again recovered from a perianal psoriatic lesion during an erysipelas relapse in November 1996 and at follow-up in January 1997. In November 1996 long-term oral penicillin prophylaxis was started [7], and since follow-up in January 1997 he has not visited the department.

Case 2. A man born in 1944 had a history of hyperlipidemia type III, hypertension, angina pectoris, and adiposity. He underwent coronary bypass surgery in 1992, with a saphenous vein graft from the left leg. After the bypass he had numerous erysipelas attacks and started receiving long-term prophylaxis with oral penicillin in December 1995. Swabs specimens from the nose, nasopharynx, and throat obtained in December 1995 were negative for BHS, whereas a swab specimen from the anal canal yielded rich growth of GGS. At follow-up in March 1996 he had suffered no more relapses, but a swab specimen from the anal canal again yielded GGS, during ongoing penicillin treatment. He was later lost to follow-up.

Case 3. A woman born in 1928 had suspected deep vein thrombosis of the right leg during her pregnancy in 1959. She suffered at least 10 attacks of erysipelas during the 1980s, which always involved the right buttock and thigh. Routine cultures were negative except in July 1991, when blood cultures yielded GCS. From 1991 onward she was free of relapses until the first 6 months of 1995, when she suffered about 10 erysipelas episodes.

Routine cultures during a relapse in June 1995 were negative, whereas GGS were recovered from an anal swab specimen. Long-term prophylaxis with oral penicillin was started. During follow-up, until May 1997, she was taking penicillin prophylaxis and suffered no relapses. Swab specimens from the anal canal revealed no BHS.

Case 4. A severely obese man born in 1954 had ~15 attacks of erysipelas on the lower leg between January 1992 and December 1994, when long-term penicillin prophylaxis was initiated. During the following 2 years, he stopped taking penicillin on four occasions but each time relapsed immediately. A swab specimen from the anal canal taken in March 1996 yielded no BHS, whereas a swab specimen from the toe-web spaces of the left foot yielded rich growth of GGS. He was treated for tinea pedis.

At follow-up in December 1996, a swab specimen from the anal canal yielded abundant growth of GGS, whereas no BHS were recovered from toe-web samples. Topical mupirocin was applied perianally during a 2-month period. At follow-up in June 1997 he had suffered no further relapses. Cultures of specimens from the anal canal and from toe-web spaces, as well as a stool culture, were negative for BHS. At follow-up in March 1998, he reported that he had stopped taking penicillin around Christmas 1997 and had suffered no relapses.
Discussion

These 4 patients had frequent relapses of erysipelas but no obvious portal of entry and negative routine cultures for BHS. However, swab specimens from the distal part of the anal canal yielded GGS in all 4 patients. This finding seems not to have been reported previously. Although the causal relationship of anal GGS colonization and erysipelas is not proved by these findings, it is intriguing that all these patients were culture-positive. Patients 3 and 4 became culture-negative in terms of the anal canal specimens and stopped having relapses, despite stopping penicillin prophylaxis (patient 4). In patient 4, GGS colonization was also noted in the toe-web spaces of the left foot, which showed signs of athlete’s foot, a finding reported elsewhere, although not in conjunction with anal colonization [8].

One patient (case 3) had GCS isolated from blood on one occasion, but no culture of specimens from the anal canal was performed at that time. Similar to GGS, GCS may colonize the gut [6] and could thus be involved in anal colonization. In patient 1, GGS were once recovered from a psoriasis lesion but not from the anal canal. However, it is still possible that the original source of GGS colonization was the gut or the anal canal.

The hypothesis put forward is that asymptomatic anal colonization with BHS, primarily GGS, may constitute a reservoir of BHS in relapsing erysipelas/cellulitis. Although hypersensitivity against fungal antigens and effects of streptococcal toxins have been implicated in cellulitis [9], a recent study demonstrated the presence of BHS, primarily GAS and GGS, directly in the involved tissue [10]. Furthermore, streptococcal colonization of toe webs, primarily with GGS, was demonstrated in nearly all patients with athlete’s foot and cellulitis, whereas none in a control group without cellulitis were colonized [8].

It thus appears that BHS may be transferred from an asymptomatic colonization site to dermal tissue and induce cellulitis. Streptococci could be transferred by hands, or possibly via lymph vessels, to the skin of the legs and other parts of the body. Patients with predisposing factors might then have frequent relapses if anal colonization is not eradicated by standard penicillin treatment, as seems to be the case. This sequence of events bears some resemblance to recurrent furunculosis caused by *Staphylococcus aureus* being carried to the skin from the nostrils [11].

The identification of anal/perianal streptococcal colonization in cases of erysipelas and cellulitis makes eradication of this carrier state possible, which might prevent relapses. Controlled studies are now planned to further investigate this reservoir of GGS, and possibly GAS or other BHS, in these diseases.

References