Recurrent Erysipelas Secondary to a Late Prosthetic Femoropopliteal Bypass Infection

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Erysipelas is a common acute, infectious skin disease, which is often recurrent. A 67-year-old man presented with 4 attacks of erysipelas within 6 months, after having received a prosthetic femoropopliteal bypass 6 years previously. An extended focus search, including anti-granulocyte scintigraphy with ^{99m}Tc monoclonal antibodies, indicated a prosthesis infection. After the synthetic vessel was replaced, the patient experienced no further episodes of erysipelas over a period of 30 months.

CASE REPORT

A 67-year-old man presented with recurrent erythema, accompanied by local warmth, oedema and tenderness on his right leg (Fig. 1). The patient reported generally being unwell and chills. An increase in body temperature was not observed. He had been treated for erysipelas with i.v. penicillin and clindamycin 3 times in the past 6 months. The last i.v. antibiotic treatment with clindamycin (600 mg tid, for 12 days) had finished 13 days prior to presentation. The portal of entry for the recurrent erysipelas was assumed to be mycologically-proven tinea pedis, which was treated with topical ciclopirox olamine. Furthermore, the patient reported having received a femoropopliteal bypass with a vessel prosthesis in his right leg because of peripheral vascular disease 6 years previously.

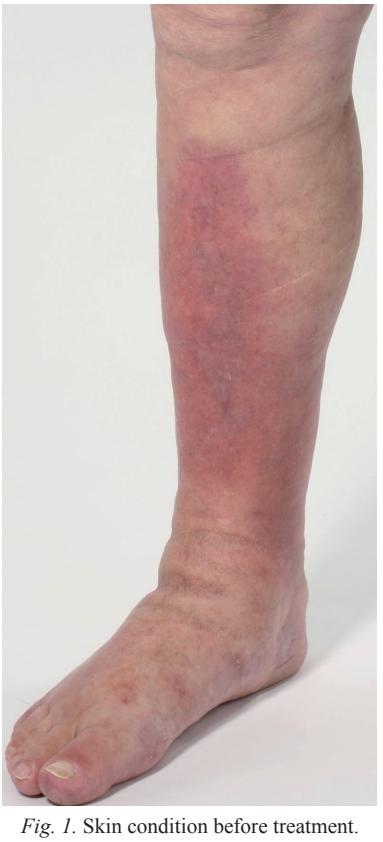

Fig. 1. Skin condition before treatment.

His erysipelas was treated with a combination of clindamycin (600 mg tid) and penicillin (10 million IU tid).

An extended search for a focus of underlying infection was undertaken. Arterial duplex sonography showed a patent bypass with a hypoechoic structure around the graft. Computed tomography (CT) examination suggested fasciitis and myositis, as well as inflammation of the femoropopliteal bypass. To explore this issue further, anti-granulocyte scintigraphy was performed. After 24 h, an intensive, long and inhomogeneous enrichment was seen medial to the right femur (Fig. 2). Furthermore enrichment was also detected on the lower limb. In addition, bone marrow expansion was seen, supporting the likelihood of a chronic infection. Thus, the suspicion of an infected femoropopliteal prosthesis emerged. After 15 days of antibiotic treatment, the patient's old prosthesis was replaced by a 6-mm silver-coated graft by vascular surgery. At discharge the wound was dry with no sign of infection. The old graft removed at operation was cultured. An infection with *Staphylococcus aureus* was proven. The antibiotic treatment was continued with penicillin G benzathine (1 g daily) for 4 weeks, followed by moxifloxacin (400 mg daily) for 10 weeks. The patient has been asymptomatic for more than 30 months.

DISCUSSION

Erysipelas is characterized by an asymmetrical, locally limited, oedematous, tender warm erythema, with flame-like offshoots. The erythrocyte sedimentation rate (ESR) may be drastically increased. The blood count shows a neutrophil leukocytosis. Erysipelas is frequently recurrent. The pathogenesis of the original infection and recurrence are similar. The usual causative agents are β-haemolytic streptococci (group A). Streptococci of groups B, C or G, as well as *S. aureus* and other bacteria, are found rarely (1–3). The pathogen usually enters through a site where the skin barrier function is not intact. Thus, tinea pedis, ulcus cruris, minor injuries and surgical procedures often correlate with an infection (2–4). Additional predisposing factors include any dermatitis of the distal legs, limb oedema, chronic venous insufficiency and obesity. Furthermore, reports of erysipelas following coronary artery bypass surgery are described in the literature (4–6).

In our case a femoropopliteal bypass infection was identified as the main cause of recurring erysipelas. To the best of our knowledge, this association has not been described previously. The bypass infection was proven by anti-granulocyte scintigraphy, using monoclonal ^{99m}Tc-marked anti-granulocyte-antibodies. For detection of the inflammation, antibiotic therapy should be interrupted for several days if medically feasible (7–9).

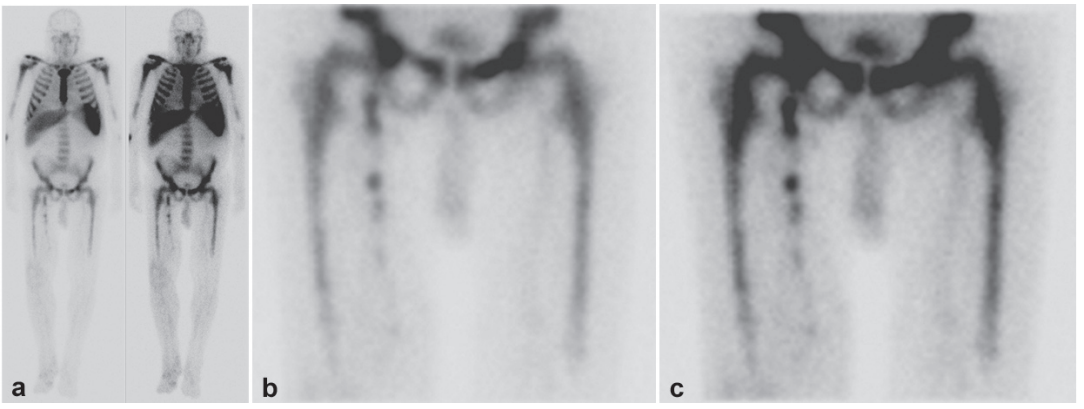

Fig. 2. Anti-granulocyte scintigraphy with ^{99m}Tc monoclonal antibodies after: (a) 3.5 h and 24 h: enrichment medial to the right femur and on the lower limb as well as bone marrow expansion (b) 4 h and (c) 24 h: intensive, long and inhomogeneous enrichment medial to the right femur.

Because of the severity of the infection, stopping antibiotics was impossible in our case. Nevertheless enrichment in the vicinity of the bypass was detectable. The sensitivity of granulocyte scintigraphy in the detection of vascular prosthetic graft infections is described as more than 90% (10).

The patient had a late bypass infection. Such infections may be a result of inadequate postoperative antibiotic prophylaxis, and are normally observed 25–41 months following the primary procedure, rarely later (11). In our case, it is not sure whether the bypass infection was caused by the implant itself or by a secondary infection as a result of tinea pedis. The risk of a late infection is increased when a synthetic graft is employed. Bacteria may accumulate around the graft where they cannot be completely destroyed by the immune system and thus cause a chronic infection, which can consequently exacerbate in an acute event (12).

In our case the revision of the bypass was inevitable. Thirty months after the procedure the patient is still asymptomatic. Since his first infection developed 6 years after his primary bypass, we must be cautious in making a final evaluation.

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