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Escherichia coli Cellulitis: Two Cases

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We report two cases of cellulitis of the legs occurring in adults where *Escherichia coli* (*E. coli*) was, or probably was, the causative bacterial agent. *E. coli* and other gram-negative bacilli cellulitis are rarely reported. However, in cellulitis, the causative microorganism is rarely identified, and some cases of *E. coli* cellulitis could be unrecognized. Furthermore, classical risk factors for gram-negative sepsis are characterized by a state of leucocyte dysfunction which could explain the possibility of a severe, even lethal, course of gram-negative cellulitis. Therefore, the occurrence of cellulitis in patients with risk factors should prompt attempts at isolating the pathogenic microorganism, and a broad spectrum of antibiotic therapy should be initiated. ***Key words: Necrotizing cellulitis; Gram-negative cellulitis; Soft-tissue infection; Erysipela; Fasciitis.***

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Cellulitis is an acute spreading infection of the skin extending deeper than erysipelas to involve the subcutaneous tissues (1). In adult cellulitis, the causative bacterial agent is rarely identified even when fine needle aspiration and culture of cutaneous biopsies are performed. However, β-hemolytic *Streptococcus* group A is thought to be the responsible bacteria in the majority of cases, especially because cellulitis is usually cured with penicillin G (1). Furthermore, an immunohistological study of skin biopsies from cellulitis has shown that streptococcal antigens are present, even when *Streptococcus* itself cannot be isolated (2). Other bacterial agents are sometimes encountered, such as other streptococcal groups or *Staphylococcus aureus*, but *Escherichia coli* (*E. coli*) cellulitis seems to be exceptional.

We report two cases of *E. coli* cellulitis occurring in adults. Presence of risk factors for gram-negative bacteria (GNB) sepsis, leading to a state of immunodeficiency, could account for the severity of the cellulitis, more than the bacteria.

CASE REPORT

Case 1. A 77-year-old woman was seen with a 72-h history of fever (38°C) and bilateral inflammatory edema of the lower legs. She had a past medical history of leg ulcer secondary to a superficial venous insufficiency with varicosae and statis pigmented dermatitis. She had also recently had a hepatitis C post-transfusional liver cirrhosis complicated with hematemesis due to esophagal varices.

At examination she was confused and her temperature was 39°C. She had a necrotizing cellulitis of the lower legs: both limbs were swollen, erythematous, with bullae, pustules, ulcerations and necrosis. There were a hepatomegaly, a collateral abdominal wall circulation and a splenomegaly. The white cell count was 20,000 with 90% neutrophils. Hemostasis tests showed a hepatocellular insufficiency and a disseminated intravascular coagulation: the fibrinogen was 1.7 g/l, the platelet count was 60,000, the thromboplastin time ratio was 2, the coagulation factor V was decreased at 56% of normal. A test for fibrin-split products was negative, but soluble complexes were present. Serum electrolytes and renal function were normal. *E. coli* was isolated from 3 blood cultures, blister fluid, fine needle aspiration from cellulitis and skin biopsy. No associated bacterial agent was found. Skin biopsy showed a massive polymorphonuclear infiltrate in the dermis and subcutaneous tissue, with presence of fibrinous thrombi in the vascular lumen. A treatment was administered intravenously with pefloxacine (400 mg b.i.d.) and amoxicillin-clavulinic acid (1 g × 4 times a day). The patient was less ill and afebrile after 24 h. A surgical debridement was performed, but after an initial clinical and biological improvement, local signs worsened despite antibiotic treatment, requiring a second surgical debridement. Subsequently, the hepatocellular insufficiency worsened and the patient died.

Case 2. A 72-year-old man presented with an erysipelas-like cellulitis of the left lower leg which had started 3 days earlier. On examination, he had a swollen leg with diffuse erythema, tenderness and warmth. There was a left inguinal inflammatory lymph node. The patient's temperature was 39°C, with shivers and sweatings, and he appeared acutely ill. There was a small traumatic skin erosin on the internal aspect of the left ankle that had not healed for 1 month. There was a superficial venous insufficiency. He was an alcoholic and a smoker. The physical examination was otherwise normal. Specimens of blood, fine needle aspiration, and a swab from the ankle erosin were obtained for culture. The white cell count was 11,000 with 63% neutrophils. The hepatic tests were normal. The glucose was 7.6 mmol/l at admission but became spontaneously normal. The antibodies to streptococcal exoenzymes were negative at admission and 2 weeks later. Intravenous oxacillin (6 g per day) was started. Forty-eight hours later, local and general signs worsened and the temperature remained high. Three blood cultures were positive for *E. coli* and the antibiotic regimen was modified for amoxicillin (9 g per day) and dibekacine (225 mg per day). The cultures of swab, fine needle aspiration and skin biopsy specimens were sterile. The search for another site of infection than skin remained negative: a urine culture performed before antibiotics was sterile, a stool culture was normal, a colic roentgenogram and ultrasonographic scan of the abdomen were normal.

Seventy-two hours later the patient felt better, but the temperature was still 38°C for 7 days and the leg remained erythematous for 10 days. Six months later the patient was seen in good health, with total healing of his lower leg.

DISCUSSION

We have reported two cases of cellulitis of the lower legs where *E. coli* was or was suspected to be the causative bacterial agent. In the first case, *E. coli* was cultivated from the blood, the cutaneous blisters, skin aspiration, and cutaneous biopsy. In the second case, despite the lack of positive culture from the skin, the role of *E. coli* is more than putative since it could be isolated from 3 blood cultures and because the cellulitis was not improved with oxacillin therapy but was secondarily cured with amoxicillin treatment.

E. coli cellulitis seems to be rare. In adults, only one case of

perianal cellulitis due to both *E. coli* and *Morganella morganii* has been reported (2). In children five cases of *E. coli* cellulitis have been reported (3, 4). All had a corticodependent nephrotic syndrome. Other GNB have been occasionally reported as causative for cellulitis in adulthood (5, 6, 7, 8). In necrotizing cellulitis, GNB are more frequently isolated, but almost exclusively in association with gram-negative or anaerobic bacteria (9, 10). In these cases, whether GNB are secondarily present or causative agents is unclear.

Nevertheless, the rarity of *E. coli* cellulitis must be cautiously interpreted for several reasons. First, in the majority of cases of cellulitis, no causative infectious agent can be found (1). Second, in the published cases of GNB cellulitis, as in our cases, the clinical features are unremarkable and impossible to distinguish from streptococcal cellulitis. Third, some of the GNB cellulitis might be cured with an empiric antibiotic treatment. Thus, it is possible that some of the *E. coli* or other GNB cellulitis remain unrecognized.

Although empiric treatment could cure some of the GNB cellulitis, *E. coli* and other GNB cellulitis seem to have a high risk of severe evolution, as our first case. It is uncertain whether such an evolution is due to the infectious agent itself: GNB cellulitis can have a favorable course despite the absence of surgical treatment, whereas *Streptococcus* remains the main etiologic agent of necrotizing cellulitis and fasciitis (9, 10). Presence of risk factors for GNB sepsis, neutropenia, diabetes mellitus, renal insufficiency, hepatocellular insufficiency, corticosteroid treatment, and chronic alcohol consumption (5) are characterized by a state of functional deficiency of polymorphonuclear cells, leading to a defect in the non-specific inflammatory reaction which seems to have a major role in the healing process of cellulitis (11). Presence of risk factor for GNB sepsis could explain the high risk of severe evolution of GNB cellulitis. Thus, when one of these risk factors is present, it is important to try to isolate the causative agent(s) by fine needle aspiration and culture of cutaneous biopsy sample, even if its yield is usually low, in order to avoid delay in effective treatment. Indeed, it has been claimed that in patients with an underlying disease associated with immunologic dysfunction, microbiologic evaluations of cutaneous cellulitis yield pathogenic organisms at a low but tangible rate (11). Moreover, in these cases, the antibiotic regimen should not be penicillin G or oxacillin: the chosen antibiotic should have a wider spectrum as for example the combination amoxicillin-clavulinic acid. An early well-adaptated antibiotic treatment is probably the best way to prevent necrotizing evolution and fascial spreading of an infectious cellulitis.

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