# An unusual case of *Escherichia coli* cellulitis and bacteremia in an immunocompetent patient

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## Abstract

Cellulitis secondary to *Escherichia coli (E. coli)* is a rare phenomenon, particularly in an immunocompetent patient. We report an unusual case of an immunocompetent 84-year-old female presenting with *E.coli* bacteremia and *E. coli* cellulitis in the right lower leg. We postulate that bacterial translocation from the gastrointestinal tract to the bloodstream is the most likely source of *E. coli* infection. Whilst a common condition, cellulitis can pose a diagnostic and therapeutic challenge when a causative organism is not identified. Thorough investigation and consideration of atypical organisms such as *E.coli* are essential to permit targeted antimicrobial therapy and prevent patient deterioration.

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## Introduction

*Escherichia coli (E. coli)* is a gram-negative bacillus infrequently involved in skin and soft tissue infections.[1,2] The few reported cases of *E. coli* cellulitis are described in immunocompromised patients.[1] Early identification is essential as *E. coli* cellulitis can be severe and require prolonged antimicrobial therapy and hospitalization.[3] Here, we report a case of *E. coli* cellulitis in an immunocompetent patient.

## Case Report

An 84-year-old female presented to the emergency department of a major tertiary hospital with a 10-day history of a painful and progressive rash on her right lower limb. This was associated with bilateral lower limb edema and dyspnea. Her medical history was significant for atrial flutter, heart failure with reduced ejection fraction, hypertension, hypercholesterolemia, and previous breast cancer completely treated with mastectomy.

On examination, she was hypotensive (93/67 mmHg), tachycardic (120 bpm) but afebrile (36.8°C). There was an erythematous to violaceous patch extending from her right calf to her thigh that was tender and non-pruritic. Laboratory investigations revealed a white cell count of $3.0\times10^9$/L, C-reactive protein of 227 mg/L, lactate of 2.7 mmol/L, and creatinine kinase level of 53 U/L (normal <145). Sensitive *E. coli* was isolated from a single set of blood cultures on admission, with time-to-positivity under 24 hours. A chest x-ray demonstrated evidence of pulmonary edema. A computed tomography (CT) scan of the abdomen and pelvis was performed to investigate a possible source of *E. coli* bacteremia, which was unremarkable. Culture of the urine and stools were also unremarkable. A venous ultrasound scan showed no evidence of deep venous thrombosis. She was then commenced on intravenous piperacillin-tazobactam for the management of cellulitis and bacteremia. She was also found to be in decompensated heart failure, accounting for her symptoms of dyspnea and bilateral lower limb edema. This was managed with intravenous frusemide.

There was concern for necrotizing fasciitis given the systemic instability and persistent pain. A CT scan of the right lower limb showed subcutaneous soft tissue thickening with inflammatory stranding; however, there were no discrete collections or any evidence of subcutaneous emphysema (Figure 1). The Plastics and Reconstructive Surgery Unit performed a fascial biopsy, and at this time, the muscle and fascia appeared viable with normal compartment pressures. Skin biopsies were also taken. Histopathological examination of the skin demonstrated a patchy perivascular and focally interstitial inflammatory infiltrate in the dermis comprising lymphocytes and neutrophils, consistent with cellulitis. Fascial biopsy did not show evidence of necrotizing fasciitis. A gram stain and culture of the skin subsequently yielded

sensitive *E. coli* growth. A diagnosis of *E. coli* bacteremia and cellulitis without necrotizing fasciitis was made.

The patient improved and her antibiotic regime was downgraded to oral clindamycin and ciprofloxacin after 11 days of intravenous antibiotic therapy. Due to the patient's severe lower limb edema, her cellulitis and site of surgical wound exploration was complicated by wound breakdown with small areas of full-thickness necrosis (Figure 2). This was managed with wound care, dressings, and a six-week oral tail of amoxicillin-clavulanic acid. At the three-month follow-up, repeat imaging demonstrated the resolution of previous radiologic findings, her cellulitis had resolved, and surgical wounds had healed.

## Discussion

*E. coli* is a gram-negative bacillus known to colonize the gastrointestinal tract as a commensal bacterium. Pathogenic strains typically cause enteric disease, urinary tract infections, or meningitis.[2] *E. coli* is rarely involved in skin and soft tissue infections.[4]

More notable causative agents of cellulitis include *Streptococcus pyogenes* and *Staphylococcus aureus*, the key targets of empirical anti-microbial therapy.[5] Patients with impaired immunity can be more susceptible to infection with atypical gram-negative organisms such as *E. coli*.[3] The few reported cases of *E. coli* cellulitis have been described in immunocompromised patients with liver cirrhosis, chronic renal failure, post-organ transplantation, and with hematological malignancies.[1,6,7] Immunosuppression results in the functional deficiency of polymorphonuclear cells, impairing the inflammatory response which can then predispose immunocompromised patients to *E. coli* cellulitis from gastrointestinal or urinary translocation.[6]

We present an unusual case of *E. coli* cellulitis and bacteremia in an immunocompetent patient with no evidence of *E. coli* in stool or urine samples. Nevertheless, given that commensal *E. coli* constitutes part of the endemic gut flora, the gastrointestinal tract is still a potential source for *E. coli* bacteremia and subsequently cellulitis.[2] It is possible that our patient's decompensated heart failure further predisposed to bacterial translocation. It has previously been described that venous congestion of the gastrointesti-

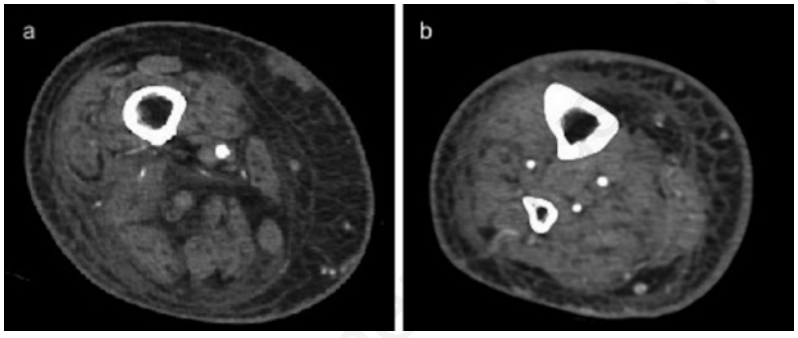


**Figure 1.** Axial computed tomography imaging of the right leg demonstrating diffuse subcutaneous stranding and edema: a) at the level of the thigh; b) at the level of the calf.

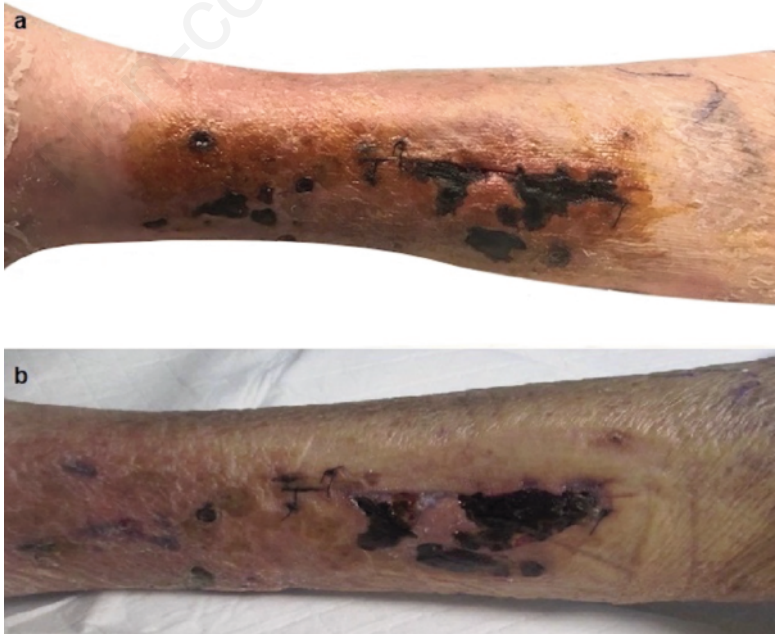


**Figure 2.** a) Right lower leg wound with areas of black necrotic eschar secondary to cellulitis and soft tissue biopsies; b) healing of the inferior aspect of the wound 15 days later.

nal tract from congestive heart failure can result in higher levels of lipopolysaccharide, a toxic component of pathogenic bacterial walls, which can lead to increased intestinal permeability and bacterial overgrowth.[8] Furthermore, our patient also had significant peripheral lower limb edema, a well-recognized risk factor for cellulitis.[9]

*E. coli* cellulitis, particularly with bacteremia or sepsis, can result in significant clinical deterioration and systemic upset. Our patient required prolonged hospitalization and extended course of anti-microbial therapy. Buchanan *et al*. also reported on a case of *E. coli* soft tissue infection with a complicated hospitalization requiring surgical debridement on multiple occasions.[3] They attributed this to a delay in effective treatment, as initial empirical therapy only covered gram-positive organisms. Furthermore, a microbial diagnosis of cellulitis can often be difficult to make.[10] Since the organisms are present in the dermis rather than on the skin surface, non-invasive investigations such as skin swabs have low diagnostic yield.[11] There should be a low threshold to perform blood cultures, cross-sectional imaging, as well as invasive investigations such as skin and soft tissue biopsy when the diagnosis is equivocal.

## Conclusions

Our case highlights the need to consider *E. coli* as a potential causative agent for skin and soft tissue infections in both immunocompetent and immunocompromised patients. While rare, thorough investigation and consideration of atypical causes of cellulitis are essential to allow targeted anti-microbial therapy and prevent patient deterioration. Acute or chronic states such as congestive heart failure, which increase intestinal permeability or promote bacterial overgrowth, should prompt consideration of gastrointestinal enteric flora as a possible source for bacterial translocation.

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