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Case report

A case report: Community-acquired *Pseudomonas aeruginosa* necrotizing fasciitis in a morbidly obese diabetic young man can be fatal

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ABSTRACT

We present a case study of a 26-year-old morbidly obese man with a three-day history of right leg pain and swelling. The swelling was associated with low grade fever. He was alert and conscious upon presentation to the hospital. His physical examination showed gross swelling of the entire right lower limb with no systemic manifestations. There was no discharge and bullae from the swelling area of the leg. He had high blood sugar and was newly diagnosed with type 2 diabetes mellitus. He was diagnosed with necrotizing fasciitis. An intravenous imipenem-cilastatin 500 mg every 6 h together with clindamycin 900 mg every 8 h was started empirically. Extensive wound debridement was performed. The swab culture obtained intraoperatively grew *Pseudomonas aeruginosa.* He required an above knee amputation due to worsening infection despite wound debridement. Post-operatively, he developed acute kidney injury with severe metabolic acidosis, which required daily hemodialysis. However, the patient deteriorated due to septic shock with multi-organ failure, resulting in his death.

Introduction

Necrotizing fasciitis is a rapidly progressive inflammatory infection of the fascia, with secondary necrosis of the subcutaneous tissues. The speed of spread is directly proportional to the thickness of the subcutaneous layer. Necrotizing fasciitis moves along the fascial plane [1], but it spares the muscle because of the abundant blood supply in the region [2,3]. Necrotizing fasciitis is part of complicated skin and soft tissue infections (cSSTIs). A three-year study from 2009 to 2011 by Ray et al. reported that diabetics were at a higher risk of SSTIs in comparison to non-diabetics [4].

Case report

A 26-year-old man presented with fever, right lower limb pain, and swelling for three days. He was morbidly obese with a body mass index (BMI) of 55 kg/m^2. Physical examination showed gross swelling of the entire right lower limb. He had heart rate of 111 beat per minutes, and temperature of 38 °C. Blood test results revealed leukocytosis and hyperglycemia. He was newly diagnosed with type 2 diabetes mellitus. The source of the infection was not adequately removed by extensive wound debridement. He underwent an above knee amputation on day three of Intensive Care Unit (ICU) admission. Intraoperatively, the procedure was uneventful. However, he required the use of a vasopressor. He developed acute renal failure with metabolic acidosis upon ICU admission. A swab culture obtained in the operation theatre (OT) grew *Pseudomonas aeruginosa* (*P. aeruginosa*), which was sensitive to amikacin, gentamicin, ciprofloxacin, imipenem, meropenem, piperacillin-tazobactam, and cefepime. Imipenem-cilastatin 500 mg every 6 h together with clindamycin 900 mg every 8 h was started. Post-operatively, the patient underwent daily hemodialysis due to acute renal failure. However, the patient deteriorated because of septic shock with multi-organ failure. He succumbed to death on day five of hospitalization despite the aggressive treatments he received in the ICU (Fig. 1).

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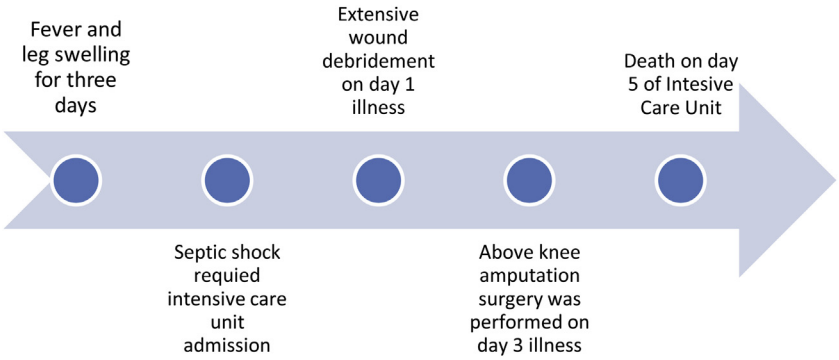

Fig. 1. Clinical course of disease.

Discussion

Necrotizing fasciitis is characterized by rapid progression of necrosis of the fascia, skin, soft tissue, and muscle. In type I necrotizing fasciitis, at least one anaerobic species, most commonly *Bacteroides* or *Pepto streptococcus*, is isolated together with ≥1 facultative anaerobic species, such as *Streptococci* (other than group A) and members of the *Enterobacteriaceae* family, such as *Escherichia coli*, *Klebsiella*, etc. Type II necrotizing fasciitis is usually caused by group A *Streptococci* alone or in combination with *Staphylococcus aureus*. A new category of necrotizing fasciitis (Type III) is caused by marine Gram-negative bacteria, such as *Vibrio vulnificus*. *P. aeruginosa,* even though rarely a component of a mixed infection, is a common cause of necrotizing fasciitis in immunocompromised patients [5,6]. Necrotizing fasciitis and sepsis caused by monomicrobial *P. aeruginosa* is a rare but life-threatening disease; it more frequently occurs in patients suffering from alcoholism, diabetes, or immunocompromised conditions [6]. Our patient had newly uncontrolled diabetes mellitus, which was a factor in the severity of the disease in our case. Intravenous imipenem 500 mg every 6 h based on the culture findings, in combination with clindamycin empirically targeted towards the toxin produced from Gram-positive coverage, was an acceptable antibiotic regime [6]. Clindamycin is used to inhibit the toxin production from *Clostridium* and *Streptococcus* species [6].

Other than pathogens, patient characteristics, infection sites, and the effectiveness of treatment are among the variables that affect survival. The treatment must be initiated without delay if the diagnosis of necrotizing fasciitis has been confirmed [2,7]. A multidisciplinary approach, including a surgeon, an infectious disease physician, and a microbiologist, is needed to manage the condition. Immediately, aggressive resuscitation must be initiated with careful monitoring of the patient's hemodynamic parameters. The key principles of managing necrotizing fasciitis include source control by doing immediate surgical debridement, administering antimicrobial therapy with good support, and intensive patient monitoring [8].

Immunocompromised patients, such as in our case, can pose a diagnostic challenge in this regard, as they may not manifest symptoms the same or as severely as immune-competent patients. Immunocompromised patients also have a 2-fold higher rate of necrotizing soft-tissue infection-related mortality [9]. Monomicrobial necrotizing fasciitis caused by *P. aeruginosa* is very rare with only a few cases reported in the literature [10,11].

Akamine et al. also reported one case of necrotizing fasciitis caused by *P. aeruginosa* with an atypical presentation that ended in mortality [10]. A review of the previous literature showed that most cases of *P. aeruginosa* necrotizing infection occur among immunocompromised individuals and can be acquired from the community or a hospital [12–14]. In our case, the patient probably acquired it from the community based on the short medical history that was given and the fact that he had no recent hospitalizations prior to admission. The microorganism grew from the necrotic tissue taken for the culture during extensive wound debridement, which was less than 48 h after hospitalization. It appears as a green-pigmented colony of *P. aeruginosa* MacConkey agar culture medium. (Fig. 2)

Anaya et al. identified six risk factors that predicted mortality: heart rate >110 bpm; temperature >36 °C; creatinine >1.5 mg/dL; age >50 years; white blood cell (WBC) count >40,000; and hematocrit >5 [6]. Our patient had three out of the six risk factors: tachycardia, fever, and abnormal creatinine values. A previous case report showed that infection with community-acquired *P. aeruginosa* necrotizing fasciitis was associated with multiorgan failure and death [15].

The main components of treatment are early goal-directed resuscitation, broad-spectrum antibiotic therapy, and surgical debridement. Although the antibiotics were started early in the case reported in this paper, the dosage and types of carbapenem that were chosen might have been inadequate in our patient, who was morbidly obese; moreover, the progression of the illness and the comorbidity, which was diabetes mellitus, impacted the outcome. At our institution, five antibiotics are commonly prescribed empirically for *P. aeruginosa*: carbapenem, tazobactam, ceftazidime, cefepime, and ciprofloxacin or levofloxacin. Combination therapy with gentamycin for five days is the common

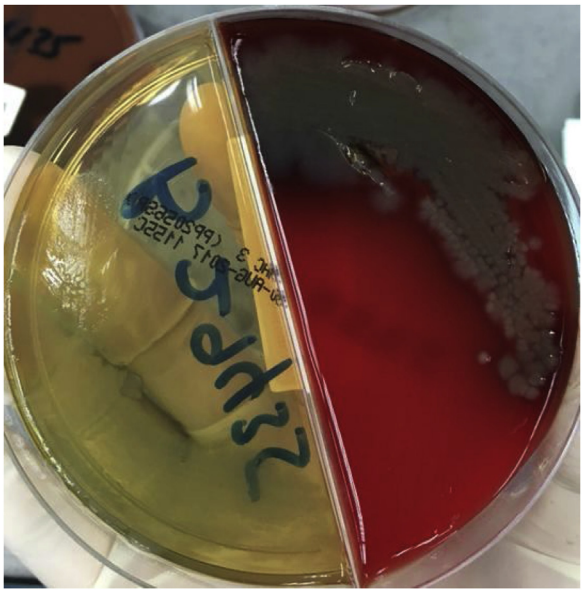

Fig. 2. Green-pigmented colony of *P. aeruginosa*.

aminoglycoside that is added as a synergistic effect for combination therapy, which is empirically started in selected cases. Our patient had acute renal failure; therefore, gentamycin was not added.

However, there is still no clinical data regarding the adequate dosage of an antibiotic in a morbidly obese patient. Moreover, no data were available to prove which of the previously mentioned antibiotics are superior. They all need to be considered based on the severity of the patient's illness during presentation. Unfortunately, trials focusing on optimal dosing in obese patients are scarce, and underdosing of the antibiotics used may increase the risk of treatment failure, unnecessary escalation to broader-spectrum antibiotics, resistance, and possibly death. Inconsistent and limited results in therapeutic outcomes suggest that clinicians should consider dosing beta-lactams within the upper limit of normal for obese patients to obtain the correct therapeutic concentration [16–21]. The literature has reported that an obese patient needs a 300 % increment of the antibiotic dosage [19].

Conclusion

In conclusion, the community-acquired *P. aeruginosa* and the aggressive nature of the necrotizing infection in diabetes mellitus with the sub-optimal dosing of antibiotic worsened the patient's condition and led to mortality.

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Consent

No consent required as there are no studies done on patients.

Author contribution

Mohd Zulfakar Mazlan –writing.
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Nik Abdullah Nik Mohamad- writing.
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Mohd Hafiz Abdul-Aziz – writing.
Kamaruddin Ibrahim – editing.
Mahamarowi Omar-editing.

Declaration of Competing Interest

The authors report no declarations of interest.

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