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Necrotizing fasciitis after intramuscular self-injection: A case report

Necrotizing fasciitis (NF) is a rare, life-threatening soft tissue infection that affects the skin, subcutaneous tissue, fascia, and muscle, and is characterized by rapid spread and invasion of pathogens. Treatment is possible with early diagnosis, appropriate antibiotic therapy, and quick surgical intervention. Nevertheless, despite all interventions, it is sometimes difficult to manage NF.
The Authors present the case of a patient with extensive NF caused by multidrug-resistant Acinetobacter baumannii (A. baumannii) that developed following an intramuscular injection of diclofenac sodium administered at home by someone who was not a healthcare professional. Wound sample culture revealed multidrug-resistant A. baumannii. The patient was treated using negative-pressure wound therapy along with antibiotic treatment and extensive surgical debridement.

KEY WORDS: Multidrug-resistant acinetobacter baumannii, Necrotizing fasciitis, Negative-pressurelf-injection, Wound therapy

Introduction

Necrotizing fasciitis (NF) is a disease that rapidly progresses in the skin, subcutaneous tissue, fat tissue, and fascia, leaving large open wounds and resulting in destruction and sometimes death [1,2]. It is a rare and severe infection caused by pathogens known as "flesh-eating bacteria."
In general, the mortality rates are 3 . Some studies have reported that mortality rates of patients with NF are approximately 30%–60%. Mortality rates are reportedly higher in women aged >60 years and patients with chronic heart failure, liver cirrhosis, skin necrosis, pulse rate >130/min, systolic blood pressure >90 mmHg, and serum creatinine level ≥1.6 mg/dL [4-6].
Early diagnosis and surgical debridement are the main treatment options for NF. However, in some cases where-in the closure of large wounds is impossible following debridement, other surgical methods such as skin graft and local or free flap may also be considered. The rapid closure of a large wound is essential, otherwise it may result in general deterioration, sepsis and respiratory, kidney, and multiple organ failure in the patient, eventually leading to death [2,7].
On the other hand, in some cases, it is impossible to close the wound with a flap, and if the patient is unstable and the wound is contaminated, skin grafting may not be possible, which slows the healing. In such cases, negative-pressure wound therapy may be useful. It is reported that negative-pressure wound therapy is advantageous in increasing tissue perfusion, decreasing edema, and accelerating wound healing [2].
Here we present the case of a patient with extensive NF that progressed following an intramuscular injection of diclofenac sodium at home by someone who was not a healthcare professional. Multidrug-resistant *Acinetobacter baumannii* was found in the wound sample. Therefore, the patient was treated with negative-pressure wound therapy along with antibiotic treatment and extensive surgical debridement.

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Case Report

According to the anamnesis obtained from a 61-year-old female patient with diabetes mellitus 15 years ago, diclofenac sodium was intramuscularly injected by someone who was not a healthcare professional.

Approximately 1 week after the injection, the patient was admitted to an outpatient center because of severe pain in the gluteal area. Abscess was detected and drained, and ampicillin-sulbactam treatment was administered.

During the course of the antibiotic treatment, the patient was admitted to the emergency department because of agitation, deterioration of general condition, pain spreading from right side gluteal area to the thigh and leg, and discoloration of the skin. Evaluation of the patient revealed poor general condition; she was conscious and agitated and had a blood pressure of 90/50 mmHg, temperature of 35.5°C, leukocyte level of 22.8 × 10^3/μl, aspartate aminotransferase level of 104 U/L, alanine aminotransferase level of 69 U/L, creatinine level of 1.57 mg/dl, glucose level of 95 mg/dL, sodium level of 138 mmol/L, hemoglobin level of 10.2 g/dL, and C-reactive protein level of 345 mg/L. She had crepitation and discoloration spreading from the right gluteal area to the right thigh and top one-third of the right leg.

Following laboratory and post-examination evaluation, the patient underwent surgery and extensive surgical debridement (Figs. 1, 2). She was administered dopamine treatment for 2 days because of persistent hypotension despite intravenous hydration. During the postoperative period, she was followed up at the intensive care unit for 10 days and received negative-pressure wound therapy at 2 days postoperatively (Fig. 3).

Microbiological examination of the sample taken from the wound area revealed growth of *Acinetobacter baumannii*. On the seventh day of intensive care unit treatment, blood culture showed the growth of *Klebsiella pneumoniae*. It was found that *A. baumannii* was resistant to trimethoprim–sulfamethoxazole, netilmicin, gentamicin, ciprofloxacin–meropenem, imipenem, amikacin, piperacillin–tazobactam, and ceftazidime and *K. pneumoniae* was resistant to ampicillin, cefuroxime sodium, ertapenem, imipenem, and meropenem. Because *A. baumannii* was sensitive to colistin and *K. pneumoniae* to trimethoprim-sulfamethoxazole, the two antibiotics were included in the treatment. The patient was taken to a ward 11 days postoperatively and received negative-pressure wound therapy until 45 days postoperatively (15 courses), whereas appropriate antibiotic treatment was continued till 20 days postoperatively.

Debridement was performed twice again during her hospitalization period, and negative-pressure wound therapy dressing was replaced once every 3 days. Negative pressure was applied at 100 mmHg. At 58 days postoperatively, the defect after debridement in the gluteal area and thigh was completely filled with granulation tissue (Figs. 4, 5). At 70 days postoperatively, the defect area was almost completely closed (Figs. 6, 7). The patient

Fig. 1: First day after surgical debridement.

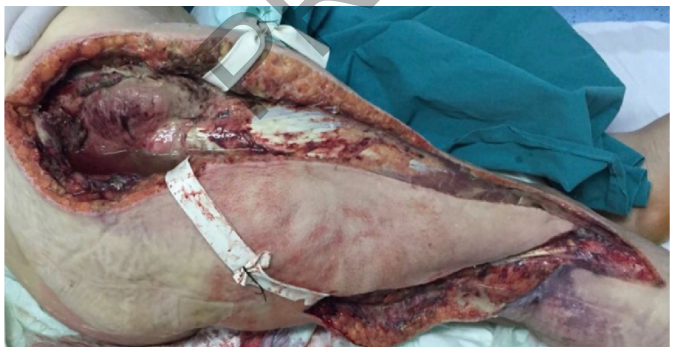

Fig. 2: First day after surgical debridement.

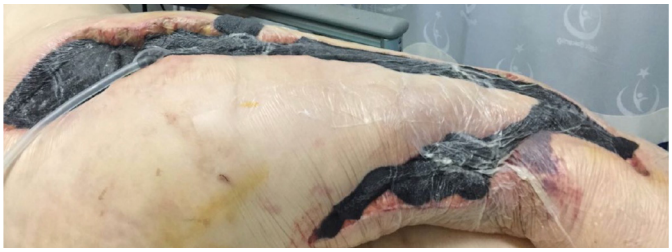

Fig. 3: At 2 days postoperatively, negative-pressure wound therapy was applied.

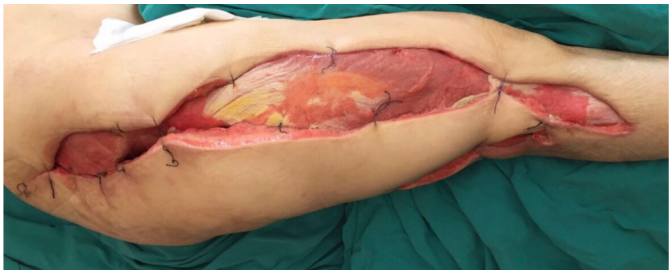

Fig. 4: Sutures for closure of the defected site after postoperative debridement.

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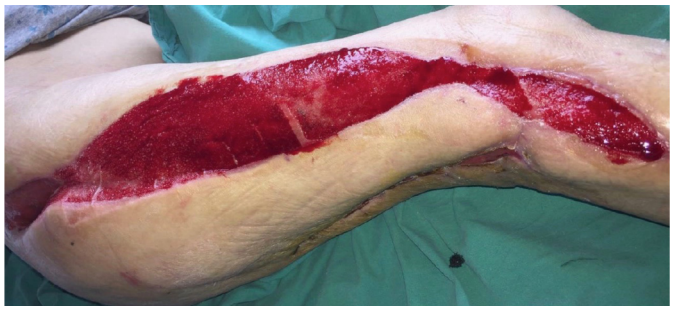

Fig. 5: Granulation tissue after debridement at 58 days postoperatively.

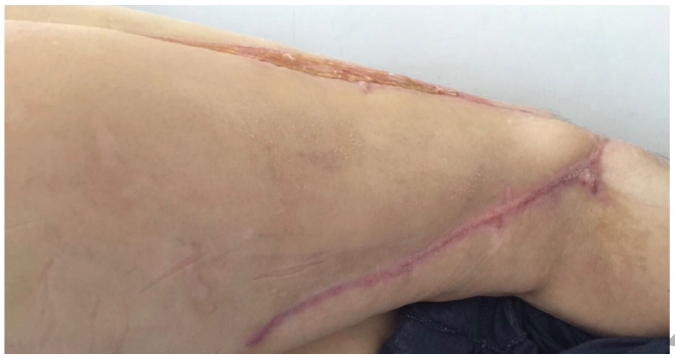

Fig. 6: Nearly full closure of the defect area at 70 days postoperatively.

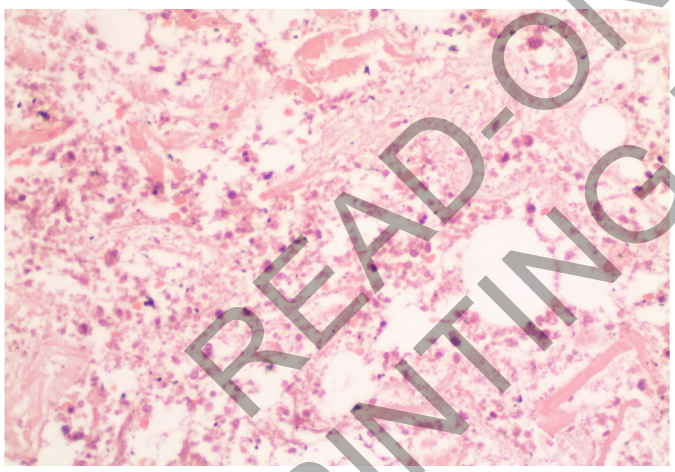

Fig. 7: Prevalent necrosis, sporadic bacterial colonization, and gas accumulation between the tissues (hematoxylin–eosin staining, ×400).

was recommended to have skin grafted from the other leg for the closure of the defected area.
However, she refused the graft because of the fear of developing another infection. The debridement material consisted of fragmented irregular tissues that embodied the skin, subcutaneous tissue, and deep fascia, with the largest being 12 × 11 × 2.5 cm and the smallest being 7 × 3 × 2.5 cm. Histopathology examination revealed prevalent necrosis, sporadic bacterial colonization, gas accumulation between tissues, and inflammation (Fig. 7).

Discussion

NF is a rare infection and has been reported to have an incidence of 4.3 out of 100,000 in the USA. It is characterized by rapid necrosis of tissue, which may result in multiple organ failure and septic shock [8-10].
Its etiopathogenesis is considered to be spontaneous or post-traumatic bacterial penetration into the fascia [11]. In addition, history of trauma, crush injuries, intravenous substance abuse, cancer, and autoimmune diseases are among the predisposing factors [12]. Microbial invasion contaminates the tissue either externally at the site of the trauma or by affecting the gastrointestinal and genitourinary systems. Tissue-invasive pathogens release exotoxins and endotoxins and increase the secretion of cytokines in the soft tissue, and the infection consequently spreads to deep fascial tissues. As a result, poor microcirculation, ischemia, cell death, and necrosis occur. Aerobic bacteria simultaneously cause coagulation by inducing platelet aggregation and complement fixation activation. Anaerobic bacteria cause formation of thrombosis by producing enzymes, such as collagenase and heparinase [2,9], resulting in bacterial increase in the fascia, infiltration of polymorphonuclear leucocytes, thrombosis in the veins, and finally necrosis.
Examination of a patient may reveal a complex clinical condition, such as erythema, hypothermia, pain, and agitation due to fever or septic shock [5,11]. NF is reportedly more common in people with diabetes mellitus and chronic alcohol addiction [7]. Our patient also had a history of diabetes mellitus for 15 years. Some cases reported that; NF can be seen in children [8].
NF most frequently occurs in lower extremities; serosanguineous blisters first occur, followed by necrosis. In addition, the trunk, abdomen, head, neck, or anus may also be infected. If the lesion is in the perineum or genital area, it is called "Fournier's gangrene" [9,12]. But interesting two cases have been reported that in the literature, cervicofacial necrotising fascitis decended from odontegenic origin [13].
NF is generally classified into two groups: polymicrobial and monomicrobial. Type I NF is called polymicrobial and is reported to include Bacteroides, *Peptostreptococcus* species, streptococci (non-group A β-hemolytic streptococcus), and Enterobacteriaceae family members (e.g., *Escherichia coli*, Enterobacter–Klebsiella, and *Proteus* species). Type II NF is called monomicrobial NF and includes invasive group A β-hemolytic streptococcus, *Vibrio vulnificus*, *Aeromonas hydrophila*, and various fungi (e.g., Mucor, Rhizopus, and Rhizomucor) [1,4,5]. A. baumannii is a gram-negative, aerobic bacillus and is isolated from the soil, water, and sewage. It is reported to cause sepsis, wound site infections, and pneumonia in immunocompromised people. A. baumannii is reported to be a hospital-derived infectious agent. Monomicrobial NF has only been reported in a few patients [1,14] Sinha et al. have reported a case of NF related to *A. baumannii*, which was fatal [15].

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In our patient, following a quick surgical intervention, negative-pressure wound therapy and appropriate antibiotic therapy based on the antibiotic sensitivity tests were administered. Thereafter, we achieved almost full recovery in this complicated case.

In conclusion, NF is a rapidly developing, life-threatening infection that affects the soft tissue and fascia. Without a correct diagnosis and appropriate treatment, high mortality rates can be observed. The most important factor in the diagnosis is suspicion of NF based on clinical findings. As soon as NF is suspected, infection control must be provided without delay through quick surgical debridement and appropriate antibiotic therapy. In addition to these treatments, use of negative-pressure wound therapy will heal the wound by providing drainage of secretions, safe protection of the wound, prevention of bacterial contamination, increase in microcirculation by angiogenesis, and formation of good-quality granulation tissue.

Riassunto

La fascite necrotizzante (NF) è una rara infezione dei tessuti molli che mette a rischio la vita, ed interessa la pelle, il sottocutaneo, la fascia ed I muscoli, caratterizzata da una rapida diffusione ed invasività di germi patogeni.

Il tratta,emto è possibile in caso di diagnosi precoce, trattamento antibiotico appropriato e rapido intervento chirurgico di sbrigliamento e necrosectomia.

Ciononostante nonostante tutti i provvedimenti il trattamento della NF è difficile.

Qui viene presentato il caso di una paziente con NF estesa causata da un Acinetobacter baumannii resistente a molti antibiotici, insorto a seguito di una iniezione intramuscolare di diclofenac sodio praticata a domicilio da un profano della professione sanitaria. Lo studio colturale della ferita ha dimostrato la presenza del A.baumannii antibiotico-multi-resistente. La paziente è stata trattata con terapia a pressione negativa insieme a trattamento antibiotico e sbrigliamento chirurgico esteso della ferita.

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