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**Case Report****Necrotizing fasciitis after internal fixation of fracture of femoral trochanteric^{☆,☆☆}**

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ABSTRACT

Necrotizing fasciitis is a rare and potentially lethal soft tissue infection. We report a case of trochanteric femur fracture in a patient who underwent fracture fixation and developed necrotizing fasciitis. A literature review on the topic will be addressed.

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Fasciíte necrosante pós-osteossíntese de fratura transtrocantérica do fêmur**RESUMO**

A fasciíte necrosante é uma rara e potencialmente letal infecção de partes moles. A seguir, descreveremos o caso de uma paciente portadora de fratura transtrocantérica do fêmur que evoluiu com fasciíte necrosante após a osteossíntese da fratura. Uma revisão da literatura acerca do tema será abordada.

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Introduction

Necrotizing fasciitis is a rare infection, misdiagnosed as a benign infection. The physician must have a high degree

of clinical suspicion for the establishment of an immediate diagnosis/treatment. The most important variable influencing mortality is the time of surgical debridement.

Orthopedic surgeons are often the first to evaluate patients with necrotizing fasciitis and therefore must be

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knowledgeable of the clinical presentation and treatment. Timely diagnosis, surgical debridement and broad-spectrum parenteral antibiotic therapy are the keys to proper treatment.

The objective of this study was to describe the fatal course of a patient who developed necrotizing fasciitis after osteosynthesis of transtrochanteric fracture and to review the literature of this serious infection.

Case report

Seventy-one year old female with a history of falling in the bathroom. The patient was in pain, unable to walk, with shortening, external rotation and limited range of motion of the left leg. Presented with arterial hypertension, diabetes, congestive heart failure, liver cirrhosis, schistosomiasis with portal hypertension and thrombocytopenia. Radiographs of the pelvis and left hip showed an unstable transtrochanteric fracture (31-A2.2/AO-ASIF).

The patient was operated on the fourth day of hospitalization after her release of the medical clinic. Relative stability of the fracture (osteosynthesis with DHS screw) and postoperative control in the ICU were obtained.

On the second day postoperatively, the patient was discharged from the ICU and started motor physical therapy with progressive partial load with walker. She was discharged on the eighth hospital day.

Six days after discharge, the patient returned to the hospital complaining of pain in her left leg, with edema, ecchymosis and swelling of the left calf. The duplex scan of the lower limbs depicted deep vein thrombosis (DVT) in the left lower limb.

Anticoagulation with enoxaparin SC, followed by warfarin PO, was initiated.

After two days, the patient reported persistent pain in the medial aspect of her left thigh, with fever (38°C). Absence of skin changes in the wound.

Laboratory tests were requested: hemoglobin: 7.5 g/dL, total leukocyte: 2400 mm^{-3} (bands: 7%, segmented: 82%); platelets: $46,000 \text{ mm}^{-3}$, RNI 1.94; APTT: 41/26, PCR: 28.7 mg/dL.

Progression the next day with hypotension (BP: $80 \times 40 \text{ mmHg}$), prostration and appearance of blisters on the medial aspect of the left thigh. Absence of pain relief with the use of opioids. Started empirical antibiotic therapy (meropenem), with no improvement.

Suspicion of necrotizing fasciitis because of severe pain, rapid appearance of bullous lesions in her left thigh and refractoriness to analgesia.

Indication of debridement of the left leg, but the patient had blood dyscrasias (INR 7.36 and PTTA 73/26).

Changing antibiotics to intravenous tigecycline. The patient was referred on an emergency basis to the operating room for wide left leg fasciotomy, surgical debridement and collection of material for analysis (Figs. 1-3).

Occurrence of severe septic shock refractory to the use of amines. The patient eventually died.

The results of blood cultures and samples collected during surgery identified multiresistant *A. baumannii/haemolyticus*. The germ was only sensitive to trimethoprim-sulfamethoxazole, tetracycline and tigecycline.



Fig. 1 – Necrotizing fasciitis in left leg.



Fig. 2 – Anterior aspect of the left leg. Extensive necrosis and presence of blisters.



Fig. 3 – Extensive necrosis in the anteromedial aspect of left thigh.

Discussion

Necrotizing fasciitis was described in 1871 by the U.S. Army surgeon Joseph Jones. In 1883, Fournier identified necrotizing fasciitis that affects the perineum and external genitalia. But it was Ben Wilson, in 1952, who described the superficial fascia and subcutaneous necrosis.

Necrotizing fasciitis follows an injury to the epidermis. In 45% of cases, it is not possible identify the site of the initial injury.¹⁻³ The extremities are the most commonly affected local, but the involvement of the trunk and perineum is related to the high mortality rate.^{4,5}

Patients over 65 years old have the highest incidence of the disease.¹

Initially, the disease presents with a local edema. However, with the involvement of the surrounding tissues, local toxicity is triggered and simulates a cellulitis. But the patient presents with severe pain, disproportionate to the skin lesion. The progression of the margins of erythema at greater than 1 cm/h speed is an important signal to the diagnosis in the early stages of necrotizing fasciitis.⁶ With the evolution of the underlying necrotic process, serous blisters possibly becoming hemorrhagic can be observed.

Usually, fever, chills, hypotension, tachycardia, and altered level of consciousness are present.^{2,6}

Acute renal failure is present in 35% of patients, coagulopathy in 29%, acute respiratory failure in 14%, and bacteremia in 46%.⁷

The burning pain is the most prominent symptom, that can be observed in almost 100% of patients with necrotizing fasciitis.⁸

The diagnosis is mainly clinical; it is essential that the physician has a high degree of suspicion. The average time between onset of symptoms and diagnosis is 2-4 days.⁹

WBC count > 15,400 cells/mm³ and serum level of sodium < 135 mmol/L have a sensitivity of 90% for necrotizing fasciitis. The specificity is 76% and the positive predictive value is 26%, and that only serves to exclude the disease.¹⁰

Phosphocreatin levels > 600 IU/L have 58% sensitivity and 95% specificity.¹¹

MRI has high sensitivity (93–100%) for diagnosis. Liquefied tissue inflammation and edema around the fascia are detected by an increased signal on T2-weighted images and absence of attenuation of gadolinium in T1.

"The finger test" is a simple procedure, done under local anesthesia. The surgeon makes an incision of 2 cm to the deep fascia, and his/her gloved finger is inserted. The presence of liquefied subcutaneous tissue, absence of bleeding and poor adherence of subcutaneous tissue during blunt dissection define a positive test. A sample of tissue must be resected and sent to bacterioscopy, culture and anatomopathological examination.

Histologically, necrotizing fasciitis is characterized by suppurative focal necrosis of fascia, fat and nerves, edema of the fibrous septa and infiltration by polymorphonuclear cells.

The parenteral empirical antibiotic therapy can be initiated with imipenem, meropenem, ampicillin/sulbactam or piperacillin/tazobactam associated with clindamycin. The antibiotic is complementary to debridement.

In patients allergic to penicillin, ceftazidime, associated with clindamycin, is an option.¹²

The mortality of necrotizing fasciitis ranges from 6% to 76%.¹³ Patients over 60 years old have a high mortality rate. Thrombocytopenia, abnormal liver function, hypoalbuminemia, acute renal failure, and increased serum lactate are associated with mortality.¹⁴

The mortality can reach 100% in non-operated cases and in the case of myonecrosis.⁷ However, the mortality rate drops to 12% if the diagnosis and treatment are made in the first four days after the onset of symptoms.¹⁵

Conclusion

Necrotizing fasciitis is a severe infectious disease. It requires a high index of suspicion to initiate antibiotic therapy and debridement.

Despite the diagnosis of necrotizing fasciitis and the indication of debridement, blood dyscrasias precluded surgery in a timely manner, so there was no reversal of the clinical picture.

Conflicts of interest

The authors declare no conflicts of interest.

REFERENCES

- Dufel S, Martino M. Simple cellulitis or a more serious infection? *J Fam Pract.* 2006;55(5):396-400.
- Bisno AL, Stevens DL. Streptococcal infections of skin and soft tissues. *N Engl J Med.* 1996;334(4):240-5.
- Mulla ZD. Treatment options in the management of necrotising fasciitis caused by *Gro^{*}up A streptococcus*. *Expert Opin Pharmacother.* 2004;5(8):1695-700.
- Levine EG, Manders SM. Life-threatening necrotizing fasciitis. *Clin Dermatol.* 2005;23(2):144-7.
- Carter PS, Banwell PE. Necrotising fasciitis: a new management algorithm based on clinical classification. *Int Wound J.* 2004;1(3):189-98.
- Wong CH, Chang HC, Pasupathy S, Khin LW, Tan JL, Low CO. Necrotizing fasciitis: clinical presentation, microbiology, and determinants of mortality. *J Bone Joint Surg Am.* 2003;85(8):1454-60.
- Kaul R, McGeer A, Low DE, Green K, Schwartz B. Population-based surveillance for Group A streptococcal necrotizing fasciitis: clinical features, prognostic indicators, and microbiologic analysis of seventy-seven cases. Ontario Group A Streptococcal Study. *Am J Med.* 1997;103(1):18-24.
- Young MH, Aronoff DM, Engleberg NC. Necrotizing fasciitis: pathogenesis and treatment. *Expert Rev Anti Infect Ther.* 2005;3(2):279-94.
- Wysoki MG, Santora TA, Shah RM, Friedman AC. Necrotizing fasciitis: CT characteristics. *Radiology.* 1997;203(3):859-63.
- Wall DB, Klein SR, Black S, de Virgilio C. A simple model to help distinguish necrotizing fasciitis from nonnecrotizing soft tissue infection. *J Am Coll Surg.* 2000;191(3):227-31.
- Simonart T. Group a beta-hemolytic streptococcal necrotising fasciitis: early diagnosis and clinical features. *Dermatology.* 2004;208(1):5-9.
- Smith RJ, Berk SL. Necrotizing fasciitis and nonsteroidal anti-inflammatory drugs. *South Med J.* 1991;84(6):785-7.

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13. Wong CH, Khin LW, Heng KS, Tan KC, Low CO. The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections. *Crit Care Med.* 2004;32(7):1535-41.
 14. Golger A, Ching S, Goldsmith CH, Pennie RA, Bain JR. Mortality in patients with necrotizing fasciitis. *Plast Reconstr Surg.* 2007;119(6):1803-7.
 15. Morantes MC, Lipsky BA. "Flesh-eating bacteria": return of an old nemesis. *Int J Dermatol.* 1995;34(7):461-3.