

Primary necrotizing fasciitis of the chest wall

Abstract

Necrotizing fasciitis is a severe, necrotizing bacterial infection that affects the skin and soft tissues. We report a rare case of necrotizing fasciitis localized at the thoracic wall. Management requires antibiotic therapy followed by extensive necrosectomy taking all affected areas, thus preparing the bed for a possible skin graft. The prognosis is essentially based on the early diagnosis.

Keywords: necrotizing, infection, antibiotic therapy, prognosis, epidermis, evolution, latissimus dorsi

Volume 4 Issue 4 - 2018

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Received: April 24, 2018 | **Published:** September 14, 2018

Introduction

Necrotizing fasciitis (FN) or necrotizing bacterial dermohypodermatitis is the most severe stage of infections of the skin and soft tissues since it reaches all skin planes (epidermis, dermis, hypodermis), deep fasciae and sometimes muscles underlining unlike cellulite which is limited to the dermis.^{1,2}

It is mainly caused by group A streptococcus (*Streptococcus pyogenes*) but also by other bacteria such as *Vibrio vulnificus*, *Clostridium perfringens* or *Bacteroides fragilis*.

These bacteria are also called “flesh-eating bacteria”, but this name is false. In reality, these bacteria do not feed on the flesh, but release toxins, which are deadly for living cells, or which, by their effects on the immune system, lead to the production of free radicals. Necrotizing fasciitis is a therapeutic emergency whose rapid evolution is sometimes fatal.

We report here a case of FN primitive of the thoracic wall with myonecrosis of the latissimus dorsi.

Case report

This is a 65-year-old diabetic patient who has been poorly balanced for four years on oral antidiabetic drugs and who was admitted for right chest inflammatory tumefaction, very painful, whose installation was 10 days before admission by the onset an erythematopuraceous papule that had progressively spread. The clinical examination found an afebrile patient, polypneic, with the presence of an erythematous placard, surmounted by multiple pustules, in the right posterolateral thoracic wall, the palpation of which showed an impaction with infiltration of the wall. In addition, no infectious portal of entry has been found (Figure 1).

Ultrasonography of the soft tissues showed significant infiltration of the soft tissues and thoracic computed tomography showed infiltration of the chest wall reaching the muscular planes without an individualizable collection. The biological assessment showed predominantly neutrophil leukocytosis with a blood glucose level of 4g/l in ketoacidosis decompensation. The patient was admitted to the operating room after preparation. Surgical exploration revealed subcutaneous and subdermal cutaneous necrosis of the right posterolateral thoracic wall. A large necrosectomy exposing the large

serrated muscle and fascia was performed. The patient was transferred to intensive care under tri-antibiotic therapy and norepinephrine and died of septic shock.



Figure 1 It shows no infectious portal of entry.

Discussion

The FN is caused by contamination by so-called “flesh-eating” germs, but this name is false. In reality, these bacteria do not feed on the flesh, but release toxins, which are deadly for living cells, or which, by their effects on the immune system, lead to the production of free radicals.

The main germs found are group B streptococcus and *Clostridium Perfringens* however other germs may be involved, such as group G or S streptococci and *Staphylococcus aureus*. Multi-microbial associations have also been found in 40 to 90% of FN.³

Infection begins at the deep plane by necrosis of the hypodermis with vascular thrombosis, and then this necrosis extends to the underlying superficial aponeurosis, and secondarily to the dermis. Its evolution is fatal, generating a high mortality rate of 30% to 76%.³⁻⁵ The early diagnosis and initial management are the two main prognostic factors. The infection most often affects the lower limbs.^{3,4} The primary involvement of the chest wall remains exceptional. The few reported FN cases of the thoracic wall are secondary to thoracic drainage, pulmonary or oesophageal surgery, transparietal lung biopsy⁶ or thoracic empyema.^{4,5,7}

The risk factors complained of are age greater than 50 years, immunodepression in particular: diabetes, steroidal or nonsteroidal anti-inflammatory drugs, hematological diseases, cancers, immunosuppressive treatments, chemotherapy, alcoholism and drug addiction.³

The case here is distinguished by the fact that it is a primitive disease of the thoracic wall with myonecrosis of the latissimus dorsi. To our knowledge, it may be the 6th case of Idiopathic FN of the chest wall reported in the literature.^{8,9} The main risk factors were ill-balanced type II diabetes and peripheral microangiopathies of diabetic origin. Antibiotic treatment, undertaken early, must be made of a bi-antibiotic therapy, initially probabilistic, targeted on gram-negative bacilli and anaerobes and then will be adapted according to the results of bacteriological samples. It is complementary to the surgery because the local diffusion is weak because of the vascular thromboses responsible for the necrosis of the deep planes. Its purpose is to limit the progression of the infection. The treatment must be medical-surgical. There is no cure without complete excision of necrotic tissue, to limit the extension of the infectious process.

Conclusion

Our case is added to the few cases published in the literature, to alarm, against this entity, which is necrotizing fasciitis, which sits exceptionally at the level of the chest wall and usually occurs on debilitated sites. Only an early diagnosis and a rapid and adapted

management are able to transform its formidable prognosis.

Acknowledgements

None.

Conflict of interest

Author declares that there is no conflict of interest.

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