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Case reports

Casi clinici

Type I necrotising fasciitis of heel in a diabetic patient

Fascite necrotizzante di tipo I del tallone in un paziente diabetico

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Necrotising fasciitis is a soft tissue infection that is characterized by widespread necrosis of fascia and subcutaneous tissue. Skin and muscle are usually spared from the necrotising process, even in the late stages. Since the first detailed description of necrotising fasciitis by Melleny in 1924, there have been several reports in the literature that have documented this disease process and its clinical presentation in detail [1].

Despite the recent developments of broader spectrum antibiotic agents and significantly improved methods of bacterial culture and sensitivity testing, the mortality and morbidity rates remain excessively high, ranging from 30 to 60 percent. Reviewing all cases from the past decades literature shows that the mortality rate from necrotising fasciitis has been 38 percent. [2, 3, 4]

CASE REPORT

A 54 year old woman was admitted to the hospital with a two day history of swelling and tenderness on her right foot following a puncture wound caused by a nail 15 prior. The patient was in good general condition, apyrexic and physical examination was normal except for the infectious view of the right foot. The dorsum of the foot bimalleolar regions were erythemateus and tender. Palmar surface of the heel was covered with necrotic skin and escar. There was prullant suppuration from the lateral malleolus (Figure 1).

The ray examination showed no signs of osteomyelitis.

Laboratory data showed a WBC count of 16,800/mmc and ESR of 124 mm/hour. Although the patient had no previous history of diabetes mellitus, her blood glucose level was 370 mg/dl.

After bedside debridment of the heel, insulin therapy and a broad spectrum antibiotic (Sulbactam/ampicillin 4 gr/day) were given to the patient for the rapid regulation of blood glucose levels and infection.

This treatment was given based upon the results of gram stained smears including Gram (+) Cocci. Aerobic and anaerobic cultures from pus were obtained prior to the administration of antibiotics. *Klebsiella spp.* was the only pathogen that could be isolated from aerobic cultures as nothing could be isolated from the anaerobic cultures.

The findings from these laboratory work-ups led to the addition of netilmicin at a dosage of 300 mg/day to the antimicrobial therapy.

After 32 hours, enlargement of dusky necroti-

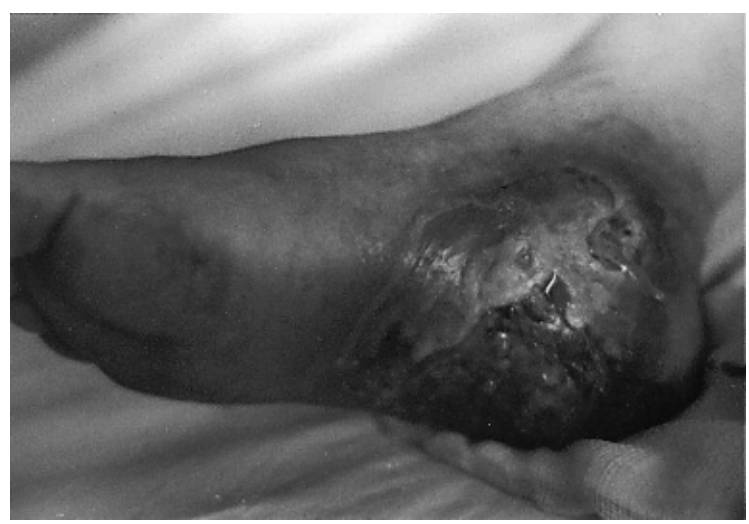

Figure 1 - The view of the foot with erithemateus look of the skin of the dorsum and necrotic escar on the heel.

sing areas towards the dorsum of the foot and the improving condition of the patient, surgery became a viable option. The patient was taken to the operating room where she underwent spinal anesthesia.

The area of necrotising fasciitis was debrided radically and extensively. All necrotic fascia, dead muscle and necrotic skin were removed during the initial operative procedure, regardless of the time required for this extensive debridment (Figure 2). Whenever possible, an effort was made to avoid amputation of the extremity and removal of viable tissue.

Meticulous care was always taken to preserve patent perforating blood vessels that supplied the overlying skin and subcutaneous tissue by taking probable reconstructive procedures into consideration.

The operative areas were packed with gauze's soaked with paniodine (Betadine).

Following this debridment, wound care involved changing of the dressings two times a day with paniodine.

This procedure in conjunction with antibiotic administration and regulated blood glucose levels resulted in successfully controlling the infection.

After 15 days of antibiotic administration, the wound began to granulate and signs of infection were no longer present. Antibacterial therapy was then stopped and the wound was allowed to granulate in order to be prepared for reconstruction (Figure 3).

On the 17th day of admission, the wound cultures were negative and the patency of the peripheric blood vessels of the foot which could be palpated as peripheric pulses were all checked by angiographic studies. Reconstruction of the weight bearing area (heel) with medial plantar artery pediculated neurovascular island and

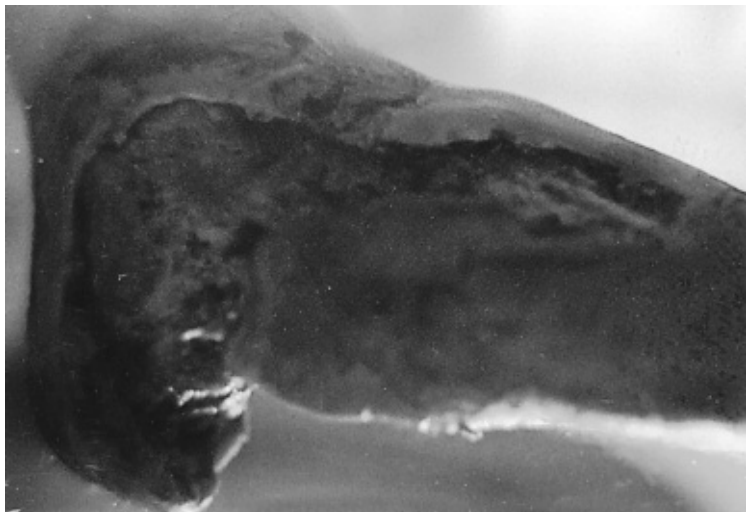

Figure 2 - The view of the foot after the first debridment.

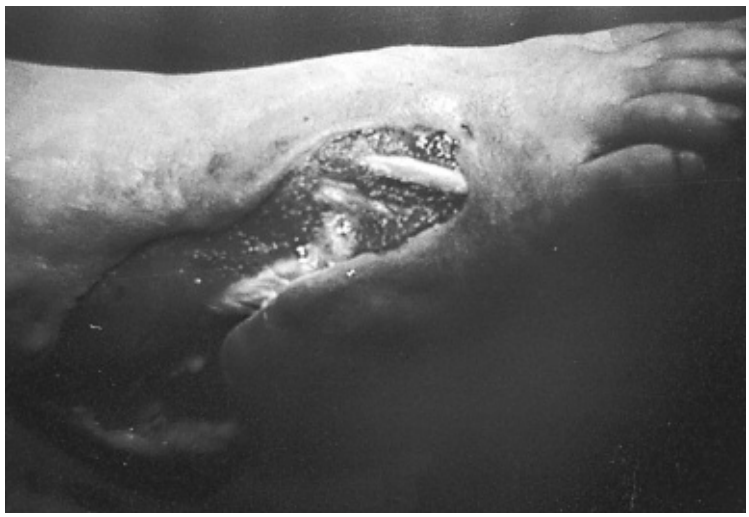

Figure 3 - The view of the foot after the defects were granulated.

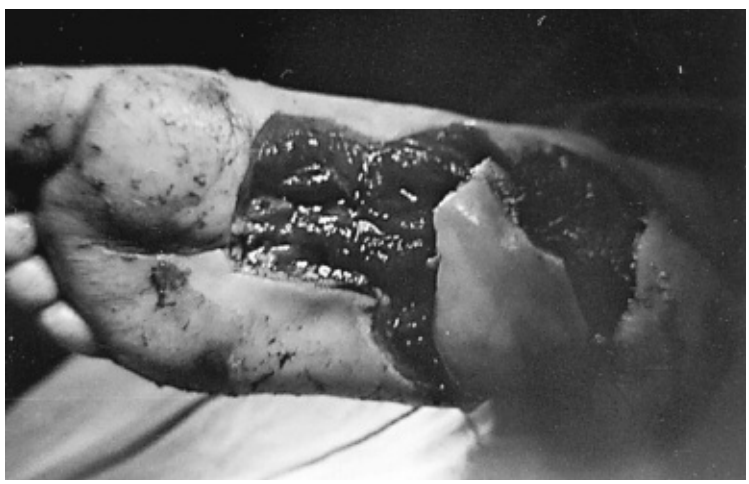

Figure 4 a The view of the foot during operation, the medial plantar flap was elevated.

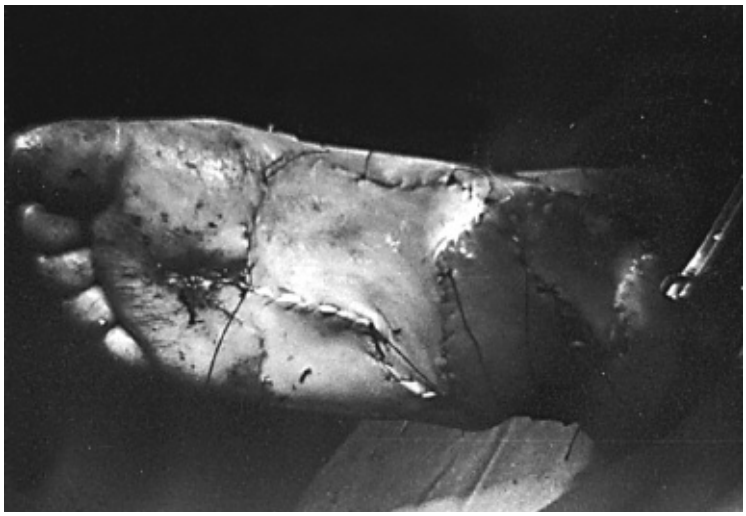

Figure 4 b Just after the operation, the heel was reconstructed by medial plantar flap, and the rest of the donor area was grafted.

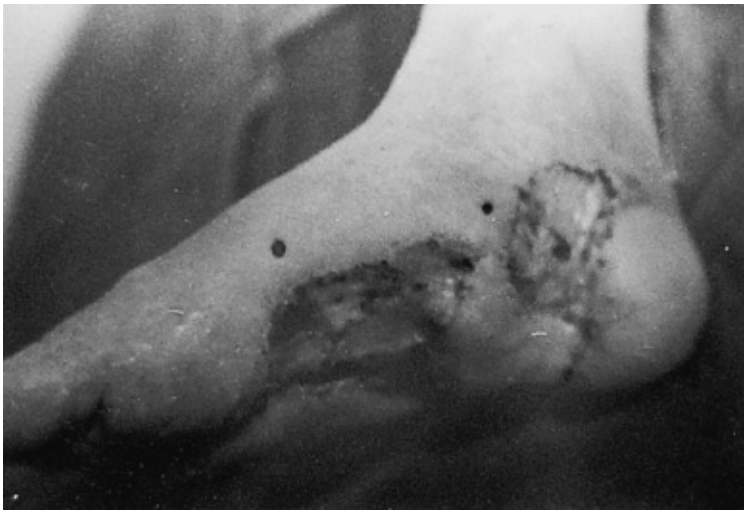

Figure 5 - The view of the foot showing the late result of the construction.

the flap donor area with full thickness skin grafts were performed (Figure 4).
Following the positive healing progress of her wounds and with the advice/instruction on insulin usage and foot care guidelines, she was discharged. This entire process was accomplished in only 29 days after admission. (Figure 5).

DISCUSSION

Necrotising fasciitis is an uncommon severe infection involving subcutaneous tissues and advancing along fascicle planes characterized by widespread necrosis of these tissues while skin and muscle are usually spared. [1, 2, 3, 4, 5].

Necrotising fasciitis is associated with a high mortality rate ranging from 30 to 60 percent. Factors associated with poor prognosis include: age over 50 years, extent of the disease when diagnosed, peripheral vascular disease, diabetes mellitus, obesity, malnutrition and involvement of the trunk or the head [3].
There is an increased incidence of necrotising fasciitis among patients with diabetes or peripheral vascular disease. Adult onset diabetes is present in 18 to 70 percent of patients with necrotising fasciitis and may increase the mortality rate to 68 percent.
Other conditions that are known to predispose to necrotising fasciitis include: malignancy, alcoholism, intravenous drug abuse, corticosteroid usage, the postpartum state [6].
Usually there is an initiating factor, a minor trauma, such as lacerations, cuts, abrasions, insect bites and inadequately treated cutaneous wounds.
More serious conditions such as perforated viscous, operative wounds (especially from abdominal and perineal operations), or infected prostheses may present the primary site of infection in some cases [3]. The lower abdomen, the perineum and the limbs are the most frequently affected sites [7].
Depending on the part of the body involved, different forms of necrotising fasciitis are described such as Fournier gangrene: necrotising fasciitis of scrotum and perineal region [8], necrotising fasciitis of neonates following onfalitis [9], and necrotising fasciitis of the head and neck [10, 11].
The clinical course of necrotising fasciitis has not changed since first described by Meleney in 1924 [1, 5]. Initially the skin is swollen, slightly red and tender. While the underlying subcutaneous tissue develops extensive and spreading necrosis. Pain may be a prominent symptom although it may be occasionally absent. From the second to the fourth day, the pathognomonic findings of necrotising fasciitis appear. The skin

turns dusky and becomes anesthetic due to the involvement of cutaneous nerves. Marked systemic toxicity is present in the majority of patients. By the eight to tenth day, the necrotic tissue suppurates.

The mechanism producing skin gangrene appears to be thrombosis of nutrient vessels passing through the involved deeper tissues. Bacteremia is common, especially with S. pyogenes and metastatic foci of infection may also develop [3] .

Meleney differs necrotising fasciitis into two forms: Streptococcal gangrene and gas gangrene. He describes gas gangrene as the deeper involvement of deeper structures while the skin is less affected and streptococcal gangrene as the involvement of superficial structures. In gas gangrene, systemic symptoms of toxicity are more apparent than the local symptoms. Streptococcal gangrene can be accompanied by a second infectious agent such as *Staphylococcus.* Associated *Staphylococcus* increases morbidity and mortality rates by causing bacteriemia and shock syndrome. Stained smears of the exudate in gas gangrene shows many gram positive rods while in streptococcal gangrene the exudate only contains gram positive diplococci [5].

The most complete documentation of bacteriology of necrotising fasciitis was reported by Gulliano et al in a study of 16 patients. Aerobic and anaerobic cultures and gram stained smears were obtained at the time of operative debridment. The cultures from three patients yielded *group A Streptococcus,* either alone or in combination with *Staphylococcus.*

The remaining 13 cultures yielded a mixture of facultative bacteria *(non-group A streptococcus* and *enterobacteriaceae)* and anaerobes. Gulliano and colleagues suggested that there are two types of infection: one caused by mixture of facultative and anaerobic bacteria and the second dueto *Streptococcus pyogenes* [2, 11, 12].

In the presented case study local symptoms with involvement of deep structures rather than involvement of skin and bacteriologic laboratory findings of gram positive cocci seen in smears and isolated *Klebsiella spp.* in aerobic cultures were all consistent with the type of a necrotising fasciitis caused by mixed infectious agents. Therefore, the clinical figure described here is accepted as Type-I necrotising fasciitis, as classified by Gulliano [12].

The cornerstone of the treatment of necrotising fasciitis is the early diagnosis and rapid treatment. The longer the time between the diagnosis and the treatment of this disease are directly associated with an increase morbidity and mortality rate.

Diagnosis of this infection is often difficult in the early stages where the normal appearance of skin may be deceptive, but the process develops rapidly, undermining large areas of normal appearing skin [4, 5, 13].

The most important measure in controlling necrotising fasciitis is the early and extensive debridment of the involved tissues. The pathognomonic changes of fascial necrosis and extensively undermining of skin are best appreciated in the operating room. Aggressive and adequate debridment at the time of surgical treatment is an important step, otherwise the patient may be more likely to have a lengthy hospital course that ends in progressive sepsis, progressive organ failure, and ultimately death [3, 4]. For faster provision of adequate debridment incision biopsies or frozen section examinations are advised [14].

During dissection and debridment all viable tissues must be preserved, including nerves, muscles, subcutaneous tissue skin, and blood vessels. Care must be exercised to preserve perforating vessels to the skin and subcutaneous skin, as these will aid in the reconstruction at a later date.

Frequently the patients were returned to the operating room for anesthesia because of severe pain associated with dressing changes and for additional debridment if necessary. The areas of necrotising fasciitis should be allowed to granulate and the wounds should be closed as soon as possible [4].

In addition to surgical debridment, prompt administration of antibiotics to possible causative organisms is essential. It should, however, be emphasized that antimicrobial agents alone have little effect on established infection in the absence of surgical intervention. The initial choice of antibiotics are guided by gram stained smears. Ampicillin + Gentamicin + Clindamicin or Ampicillin + Gentamicin and Metronidazole or Ampicillin/Sulbactam, Gentamicin may be used as first choices of therapy. This therapy can be modified when aerobic and anaerobic culture results become available [4, 13].

The other hallmark in the treatment of necrotising fasciitis is the intensive care on possible systemic disease of the patient including monitoring of the signs of septic shock, regulation of blood glucose levels in diabetics and aggressive nutritional support for the increased basal calo-

ric requirement [4].
For the reconstruction of plantar surface of the foot, split thickness skin grafts are not advised. Fifty percent of the patients reconstructed by skin grafts on weight-bearing surfaces of the foot required additional reconstructive procedures. Patients with accompanying peripheral vascular disease may not be suitable candidates for local flap reconstruction even though it would seem to be the most logical technique. Most local flaps described for the reconstruction of the plantar foot are based on antegrade blood flow through the posterior tibial artery and its media and lateral divisions.
Therefore, artherosklerotic or diabetic patients with occlusion or severe stenosis of these vessels may not be able to have a successful closure performed with these flaps. In these situations free tissue transfer techniques should be considered. In the case presented, the tissue defect as a process of necrotizing fasciitis was situating from dorsolateral of the foot to the medial malleol, including the heel.
All of the defect was well granulated. Peripheric pulses were palpable and the patency of peripheric circulation was checked by angiographic studies.
Covering with skin grafts was the first choice with minimal morbidity.
However, the defect on the heel, as a weight bearing part necessitated a flap coverage. A local neurovascular island flap based on medial plantar artery was used to cover heel and full thickness skin grafts were used for the remaining areas [15].
In conclusion, despite the known high rates of mortality and morbidity, a good prognosis may be obtained if necrotising fasciitis is diagnosed early and treated with a well managed therapeutic challenges.

Key words: Necrotising fasciitis, diabetesmellitus, heel, reconstruction

RIASSUNTO

La fascite necrotizzante è una malattia clinica potenzialmente fatale causata da infezione di diverse specie batteriche. Il diabete mellito, uno dei fattori predisponenti la fascite necrotizzante è anche un importante fattore nella progressione della malattia aumentando la percentuale di morbidità e mortalità. E' qui di seguito riportato il caso di una donna diabetica di 54 anni con fascite necrotizzante di tipo I del tallone la quale è stata trattata con sbrigliamento esteso, terapia antibiotica ad ampio spettro e regolazione dei liveli ematici di glucosio. Ne risultano nuovi dati riguardanti la morbidità e la mortalità di questa malattia suggerendo che il vecchio trattamento dovrebbe essere riesaminato.

SUMMARY

Necrotising fasciitis is a potentially fatal clinical disease caused by infection with various bacteria. Diabetes mellitus, one of the predisposing factors for necrotising fasciitis is also an important factor in the progression of the illness by increasing its morbidity and mortality rate. In this case report, a 54 year old diabetic woman with type-I necrotising fasciitis of the heel was treated with extensive debridments, broad spectrum antimicrobial therapy and intensive regulation of blood glucose levels. The resulting new positive data pertaining to the morbidity and mortality of this severe disease suggest that the old management of this disease should be re-examined.

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