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Periorbitary Necrotizing Fascitis by Acinetobacter, Proteus and Escherichia Coli Differential Diagnosis

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ABSTRACT

Necrotizing fascitis which has been the object of public attention due to the apparentent emergence and the growing virulence of the causative agent is not a novel disease. From 1960 only has been reported 38 cases from the scientific literature and no one was in Spain. Of the 38 cases-12 has been described on forehead and periorbita. In the present article, necrotizing fascitis is reviewed and a comment is made on its classification and causative agents. Finally, the importance of performing a wide surgical desbridation of the necrotic tissue is underlined , for delimitation of this desbridation image diagnostic techniques mainly, Nuclear Resonance

Keywords: Necrotizing fascitis, Antibiotic treatment, Surgical desbridation, IRM

INTRODUCTION

Fascia superficialis [1] is crossed by facial expression muscles. The temporal region presents a peculiar superficial fascia: the zygomatic-temporo frontal [1] or pretemoral fascia [1], a set of several thin conjunctive laminae [2] and a continuation of the aponeurosisepicranea [3] which together with the superficial temporal aponeurosis, and both being inserted into the upper border of the zygomatic arch they leave between them a cell that acts as a container for bruises or infections [3]. Both are on the structures of the temporal region and have the same limits as this [4]. This region is where the fascitis referred to in this article began, which later spread to the ipsilateral (left) palpebral region, an area easily colonized by the laxity of the subcutaneous cellular tissue [5] and easy affectation of the orbicularis muscle due to lack of aponeurosis [1]. Necrotizing fasciitis is an inflammatory pathology of the subcutaneous soft tissues, especially the fascia superficialis and sometimes the deep fascia [6].

It is of infectious cause and rapid evolution, which can go unnoticed and can cause death by the activity of bacterial exotoxins. According to the cases published to date, the mean age of presentation necrotizing facial fasciitis is 48.7 years [7] and affects 22 women and 16 men. In the 38 cases reviewed by Shindo and Cols [7], they are found as pathological antecedents: Diabetes in 8 cases, Alcoholism in 8 cases, Arteriosclerosis in 2 cases and Premature delivery in 1 case. As in the general population the prevalence of diabetes is between 2 and 4% [12] and alcoholism presents a prevalence of 6% of the population. As there were 8 cases in each of these groups there is a relative risk of suffering from this pathology being diabetic of 8.08 with respect to the healthy population, and of 3.90 being alcoholic. Other predisposing factors are malnutrition, hypoalbuminemia, obesity, peripheral vascular disease, addition to intravenous drugs, leukemia, lymphomas, renal failure, use of corticosteroids, cirrhosis and AIDS [8]. Although there have been cases of spontaneous onset, necrotizing fasciitis usually arises after some type of skin lesion of infectious type (Varicella or superficial bacterial infection), after some type of solution of continuity of the skin although this is minimal, after puncture, after surgery, decubitus ulcers, etc.

Such lesions allow colonization of anaerobic bacteria, enterobacteriaceae, streptococci, or even fungi such as cryptococcalneoformans. According to Fitzpatrick [6] "is a mixed infection in which one or more anaerobes are involved along with at least one facultative species (non group A streptococci; member of the Enterobacteriaceae such as Enterobacter, proteus, etc.) [6, 9]. Aerobic cocci or

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bacilli sometimes coexist with some species of clostridium.

From the surface or directly in depth the superficialis fascia is colonized. The time it takes to colonize and the inflammatory reaction is 1 to 4 days in rapid forms and 3 to 15 days in Necrotizing Synergistic Cellulitis [6] The mechanism has not been clarified to date [10]. After colonization of the fascia they occur3893/5000 microthromboses of the perforating vessels that cause posterior ischemic necrosis of the supraadyacent skin.

It is a more frequent pathology in lower extremities, periné (Fournier gangrene) and abdominal wall. The face is a rare localization, although within it the most frequent is the periorbital location [11].

Histopathologically there is fibrinoid necrosis in the middle layer of some arteries and veins that cross the infected fascia, as well as microthrombos [12].

The first symptoms that usually occur within 4 days after the aggression of the skin [6] are: Blush that looks a little more violaceous than cellulite and diffuse margins as opposed to simple cellulite with marked margins (sharp margins), edematous swelling soft and depressible, heat, not as evident as in erysipelas or cellulitis (Felix patiño considers it non-existent in true fasciitis) [13], and pain. Later on, after 3 to 5 days, symptoms appear as: purple coloration of the skin, bullae with thick pink or purple purulent content [14], crepitation in 25% of cases fitzpatrick pag2317 por presence of gas, especially in polymicrobial fasciitis and in diabetics [14] numbness, anesthesia due to thrombosis of small perforating vessels that destroy the sensory nerves of the area [14] this sign is an indication that we are faced with a fascitis and not before a simple cellulite [14] and frank necrosis of the skin that characteristically begins in blue-gray patches that converge to form a zone black like a black scab of a burn [14]. Unfortunately the necrosis of the skin is the sign that most often leads to the diagnosis. The disease is more extensive than it appears. Secondary thrombophlebitis is frequent in the case of streptococcal disease, but lymphangitis and lymphadenitis are rare. The body temperature is high (more than 39°C or 102-105°F) and systemic toxicity is usually patented by the patient's severity status

Streptococcal Gangrene is now considered the same entity, produced by the hemolytic streptococcus [6].

Fournier gangrene (strep throat gangrene, perineal phlegm) Fitzpatrick et al. [6] consider the same pathology but localized in the perineum

Necrotizing synergistic cellulitis (necrotizing cutaneous myositis, non-clostridial synergic anaerobic synechia, anaerobic cutaneous gangrene by gram negative) [6,14] a form of a slower incubation period and it also affects the underlying muscles.

The first diagnostic method is clinical examination. If necrotizing fasciitis is suspected in its initial stages, in which it does not present necrosis but presents an inflammatory zone a little more violaceous than the cellulite and of not very well defined margins, a TAC is performed - preferably , MRI or ultrasound that will determine the depth of the presence of the gas if any and of the inflammatory exudate [14]. Subsequently, the area is incised and a hemostatic clamp is inserted over the deep fascia: if the fascia is still incipient, the fascia dissects with extreme ease [6], which would not occur in ordinary cellulite [14]. Gas and pus are often found between fascia muscularis and fascia superficialis. Doing a Gram staining of the exudate smear, one can rule out the presence of sporulated gram-positive bacilli that would overshadow the diagnosis".

Subsequently, a bacterial culture of exudate should be performed for aerobes, anaerobes and fungi. In addition to a blood culture that is frequently positive [6].

The hemogram shows leukocytosis and asymptomatic hypocalcemia can be associated if necrosis of the subcutaneous fat is extensive [14]. Serum CPK levels that are elevated in muscular involvement of the clinical form called necrotizing synergistic cellulitis

The definitive diagnosis is by a freezing biopsy [15]. The etiological diagnosis is by cultures of the purulent fluid that infects the fascia duperficialis. The differential diagnosis is made with the inflammatory and / or necrotic pathologies of the skin and adjacent tissues that are reflected in the **Table 1.**

Differential Diagnosis of Necrotizing Fascitis

Erysipelas: It is a superficial type of simple cellulitis that presents heat, marked redness and some vesicula or bulla. The edges of the lesion are well delimited as corresponds to a superficial infection in which the relief produced by the infection of the dermis is not attenuated by supraadyacentes gross tissues. It produces a large lifangitis and the typical orange peel. Sometimes it can progress in depth to cellulitis and more deeply to fascitis.

Cellulite simple by aerobic germs

After an incubation period that may be as short as gaseous gangrene, the symptomatology appears with its characteristic orange peel, flushing, heat, pain, more marked blush of a frank red color. The edges of the lesion are diffuse as corresponds to an inflammation of the subcutaneous tissues without gas, presents /displays lymphangitis, when it does not separate the muscular fascia of the fascia superficialis with ease [14].

Clostridial anaerobic cellulitis: The incubation period is similar to fasciitis and its onset is less acute but may be rapid as that of fasciitis

Presenting less swelling but more gas than necrotizing fasciitis. The pain in the early stages is mild The secretion is dark and diluted unlike fasciitis that is seropurulent Toxicity toxemic is less striking than that of necrotizing fasciitis [14] (box) and not there is muscle involvement.

Table 1. Differential Diagnosis of Necrotizing Fasciitis

Inflammatory pathology	Differential diagnosis	Comments
Erysipelas	lymphatic involvement and "peau d'orange	It can extend more deeply, producing cellulitis, abscesses or even necrotizing fasciitis. This is treated using antibiotics.
Anaerobic cellulitis	lymphatic involvement and "peau d'orange"	It can extend more deeply, producing necrotizing fasciitis. This is treated using antibiotics.
Non-clostridial anaerobic cellulitis	More superficial lesion determined by biopsy	The treatment is the same except for the smaller margin of debridement.
Clostridial anaerobic cellulitis	More gas and more superficial lesion than Necrotizing Fasciitis	More serious than the one above but with the same treatment.
Pyomyositis (tropical pyomiositis)	Intramuscular abscess observed by CT, MRI or ultrasound.	It is uncommon in developed countries. It never results in skin necrosis.
Group A streptococcal necrotizing myositis **(peracute streptococcal pyomyositis, spontaneous streptococcal gangrenous myositis)**	Intramuscular swelling observed by CT, MRI or ultrasound. The affected muscle can be observed during debridement.	It never gets to produce skin necrosis.
Gas gangrene (clostridial myonecrosis)	With less erythema than NF. Intramuscular swelling observed by CT, MRI or ultrasound. The affected muscle can be observed during debridement.	

NF= Necrotizing Fasciitis, CT=Computed Tomography, MRI= Magnetic Resonance Imaging.

Clostridia anaerobic cellulitis

The onset is gradual, there is more gas than necrotizing fasciitis

The murky, serous oozing odor characteristic of the lesion is characteristic [14], crepitation may extend beyond the limits of infection by the dissector effect of gas [14]. Swelling is more moderate than in necrotizing fasciitis because it does not affect a plane of easy dissection as in the case of this [14] although the amount of gas is higher.

"Tropical" pyomyositis: According to Morton [14] in the United States is very rare whereas in some tropical areas the frequency reaches from 1 to 4 percent of the admissions are abscesses produced in muscles damaged by various causes and simultaneously or later colonized by Staphylococci (usually Streptococcus or other bacteria from adjacent or distant areas after hematogenous dissemination. In stage II or suppurative it presents a swelling in the superficial tissues to the affected muscle that can resemble to the necrotizing fascitis but whose differential diagnosis is easily realized by CAT, ultrasound or MRI in which the intramuscular

collection is observed. In addition it never produces necrosis of supraadyacente skin.

Anaerobic streptococcal myositis, periaphatic (hyperacute or subacute) pyomyositis streptococcal or streptococcal spontaneous gangrenous myositis:

At a more gradual beginning than gas gangrene without posterior necrosis of the skin, it appears erythematous or with petechiae and vesicles, with a moderate presence of gas, these clinical data allow to distinguish this pathology from gas gangrene and necrotizing fasciitis. In addition there is little superficial swelling and inflammation of the underlying muscle that is objectified with the accomplishment of an ultrasound, MRI or CT scan in which the swollen and exuded muscle is seen in the muscle compartment, which allows differential diagnosis with necrotizing fasciitis in that the swelling is between the muscular fascia and the fascia superficialis. There is a large increase in

Serum levels of CPK (Creatine Phosphokinase) [Morton W Swartz [14] Mortality is 80- 100%

Glaucoma or Clostridialmyonecrosis: Affects 1 to 2% of major open wounds [14] In which the abundant muscle affection and accompanied by patches of dermal blackish green necrosis [14] the symptomatology is more serious than in necrotizing fasciitis, the first symptom, which appears after 1 or 2 days of incubation, is pain, greater than that of fascitis (sometimes even atrocious) and which is accompanied by a tense edema of the area that is very sensitive but without an appearance as inflammatory as fascitis and much less than simple cellulite or erysipelas. The appearance of supra-adjacent skin rapidly passes from the initial pallor to a yellowish or tan tone on which appear dark bullae with serosanguinolent exudate and patches of greenish-blackish necrosis [14]. The patient in a few hours becomes pale, sweaty, restless and even stuporous and delirious sometimes reaching shock [14] in the images of the CT or MRI we see that the inflamed area is the muscle. In a fasciotomy that includes the muscular fascia, one would see the muscle hernia through the incision, releasing gas in an amount similar to that of the necrotizing fasciitis and a serosangunolent exudate with a peculiar slightly fetid odor and Mandell-924 dulzon. If we stimulate the affected muscle with electrodes, it does not contract [14]. The hemogram shows hematocrit decrease due to hemolysis that does not appear in necrotizing fasciitis. Total hyperbilirubinemia appears in Biochemistry. In the pathological anatomy there is necrosis of fibersmuscle, destruction of connective tissue, gas, large numbers of bacilli and very few inflammatory cells.

Infected vascular gangrene: It happens on an area affected by peripheral arterial insufficiency usually known on which after a period of incubation from the contamination longer than the fascitis and with a gradual course appears a blackish, swollen but dry lesion although maloliente and with presence of gases. The underlying muscle is affected by vascular insufficiency and infection. The systemic involvement is minimal.

Synergistic Progressive Bacterial Gangrene

It is a necrotic ulcer surrounded by a dark margin and with an erythematous periphery. It grows slowly.

Necrosis Cutanea due to mucormycosis

Necrotic zone with high purple margin

It is important to distinguish between erysipelas and simple cellulitis, which require only medical treatment on the one hand and other soft tissue infections requiring early surgical treatment for:

The treatment, in the case of suspected necrotizing fasciitis in the initial stages in which it does not yet present necrosis, begins with the incision of the inflamed area until it reaches fascia superficialis, detachment of it, cleaning of pus, cure with daily antiseptics (H2O2 and povidone iodine which has the advantage of being active in the presence of blood and pus), oral antibiotics (Amoxicillin plus clavulanic or cloxacillin). If the cause has been a contaminated wound, an early debridement of all devitalized tissues should be carried out, which has demonstrated its ability to reduce the incidence of necrotizing infections in war wounds [14].

In the case of presenting necrosis, it should be treated more aggressively with debridement of the entire fascia superficialis that peels off easily on examination, even with a margin of 2 cm and all the necrotic tissue. The surgical wound should be left open.

Antibiotherapy should include clindamycin, intravenous gentamicin, and penicillin. The antibiotic regimen should be changed according to the result of the antibiogram.

Later, once the infection is considered cured, the wound is closed by cooling the edges and taking off the two margins of the wound or the defect is covered with a flap or graft.

Mortality is approximately 35%. The main cause of complications is due to the confusion of this pathology with a cellulitis, which delays the diagnosis, which in pathologies of rapid evolution like these can be fatal.

Acinetobacteranitratus, proteus mirabilis and Escherichia coli

Acinetobacteranitratus is a gram-negative non-enteric (non-digestive tract) bacillus that forms part of the normal flora of the skin and mucous membranes. It is a known producer of severe respiratory infections in patients with mechanical ventilation or automatic inhalers, has been isolated in the cefaloraquide fluid in cases of meningitis and has been isolated in blood cultures of septic patients of diverse origins.

Proteus mirabilis is a gram negative bacillus belonging to the group of enterobacteria (facultative anaerobes) and has many flagella which makes it very mobile. Proteus mirabilis is the only species of proteus that are indole negative although some proteus mirabilis indole positive strains have been found. It produces the majority of infections that are out-of-hospital infections - urinary tract infections that produce struvite stones.

It is usually quite sensitive to commonly used antibiotics. Amoxicillin plus clavulanic - unlike the rest of proteus that usually produces hospital infections and are usually more resistant to antibiotics.

DISCUSSION

Necrotizing fasciitis, although well known in other locations such as the lower extremities, abdomen and perineum - where it is called gangrene of fournier - is rare in other locations such as the face and neck. This is the first time necrotizing fasciitis of the head and neck has been reported in Spain. The importance of knowing the existence of this location goes beyond the pure anecdote.

The severity of the disease from which 35% of those affected [7] die; and the serious aesthetic sequels that it produces, make this knowledge a necessity for the early diagnosis and treatment that would avoid these complications.

There is no single nomenclature for necrotizing soft tissue infections. Necrotizing fasciitis has been called in many ways - acute streptococcal gangrene, hemorrhagic streptococcal gangrene, necrotizing erysipelas, erysipelas gangrenous, suppurative fasciitis, hospital gangrene, etc.

There have been many classifications of necrotizing soft tissue infections that are reflected as:

Clostridial Infections

Non-Clostridial Infections

The real problem is the early diagnosis in the initial stage where it is confused with bacterial cellulitis by aerobic germs [6] and that without surgery, since the treatment of cellulite is antibiotic therapy, the picture can quickly become complicated seriously. In most of the published cases the diagnosis happened when the necrosis had already appeared with which the defect of skin and soft tissues required the placement of grafts or flaps with the subsequent esthetic deterioration in a pathology that of suspecting precociously and knowing the protocol diagnosis would avoid the sequelae and of course the cases of death. In cases where necrosis appears, it is a frequent mistake to wait until the area of necrosis is delimited, as in third-degree burns and ischemic necrosis.

Diabetes mellitus is a recognized risk factor for the production of necrotizing fasciitis [8] as well as the use of steroids. The patient we are referring to is a known diabetic.

The greatest error that usually occurs is the delay in debridement, perhaps due to the constancy of waiting for the necrosis to be delimited in cases where it is produced by a non-septic cause (burns, frostbite, ischemic necrosis, etc.) , or to confuse it, in the initial stages with a cellulite by aerobic germs.

CONCLUSIONS

The knowledge of this pathology is a necessity - given the high mortality and the serious avoidable aesthetic sequels -, for an early diagnosis and treatment. Being a rare pathology, we must insist on disclosure because we do not usually think about it, and we begin to consider the differential diagnosis when it may already be too late because of the rapid onset of the disease.

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