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Original Research Paper

Epidemology

NECROTIZING FASCIITIS: NARRATIVE REVIEW

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ABSTRACT Necrotizing fasciitis (NF) is an entity that is part of the spectrum of necrotizing skin and soft tissue infections (NSTI), characterized by extensive and rapidly progressive tissue destruction, accompanied by signs of sepsis. It is classified as polymicrobial (type I) and monomicrobial (type II). The incidence is estimated at 0.3-15 cases per 100,000 inhabitants. Early signs of the disease include swelling, pain, and erythema. Early diagnosis is imperative, which is why prediction scales have been created, with LRINEC being the most widely used. Treatment has two fundamental pillars: early surgical debridement to control the septic focus and broad-spectrum antibiotic therapy. However, despite advances in the diagnosis and treatment of FN, its mortality continues to be around 25-35%.

KEYWORDS :

INTRODUCTION

Necrotizing skin and soft tissue infections (NSTI) are a spectrum of pathologies, within which we can find cellulitis, myositis and fasciitis (1). These infections are characterized by extensive and rapidly progressive tissue destruction, accompanied by signs of multi-organ dysfunction.

The high mortality implies that early diagnosis and treatment are imperative to achieve the best possible outcome. The therapeutic pillars should include early surgical intervention and antibiotic therapy (2-3).

Necrotizing fasciitis is a deep tissue infection that causes progressive destruction of muscle fascia and subcutaneous fat. The muscle tends to be saved frequently given its generous blood supply. In contrast, the fascia, having little blood supply, is more susceptible to extensive destruction (4).

The development of anesthesia may precede the appearance of skin necrosis, which gives a clue to the presence of necrotizing fasciitis (5).

Diagnosing it and differentiating it from myositis is complicated, since direct visualization of the fascia is required to make the diagnosis.

METHODS

This narrative review was based on a search strategy that was carried out in databases such as PubMed/Medline, Lilacs and Redalyc, EBSCO. The MeSH and DeCS thesauri were used. Articles such as clinical trials, systematic reviews, topic reviews between the years of 1999 and 2022 were included (**Figure 1**).

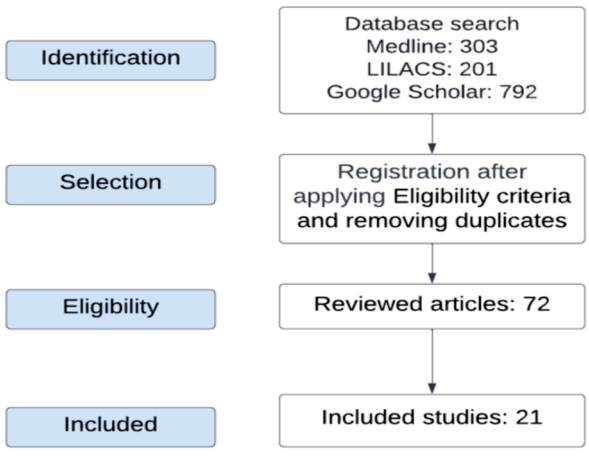

Figure 1: PRISMA

Classification

Necrotizing fasciitis can be divided into 2 microbiological categories: polymicrobial infection (type I) and monomicrobial infection (type II).

Polymicrobial infection (type I)

It is caused by aerobic and anaerobic bacteria, usually an anaerobic species (Bacteroides, Clostridium, Peptostreptococcus) is isolated in combination with Enterobacteriaceae (E. coli, Proteus, Klebsiella) and one or more facultative anaerobic streptococci. Obligate aerobes such as P. aeruginosa and fungi such as Candida are rarely isolated from polymicrobial infections (6).

Monomicrobial infection (type II)

It is usually caused by group A Streptococcus, hemolytic group B Streptococcus and also Staphylococcus aureus (7) (8). Infection without a clear portal of entry occurs in approximately half of the cases and hematogenous bacterial translocation from other sites, for example throat (asymptomatic pharyngitis) should be suspected (9). Other pathogens can also cause necrotizing fasciitis. Vibrio vulnificus and Aeromonas hydrophila should be suspected in the context of a traumatic injury that is associated with seawater or freshwater, respectively (10).

Epidemiology

The incidence of necrotizing fasciitis is estimated at 0.3-15 cases per 100,000 inhabitants (1,10).

Polymicrobial necrotizing fasciitis (type I) occurs more frequently in older adults with associated comorbidities, the most important being diabetes and its associated peripheral vascular disease.

Monomicrobial necrotizing fasciitis (type II) can occur in any age group and without associated comorbidities (11). Despite advances in treatment, mortality from necrotizing fasciitis remains quite high and it is estimated that around 25-35% of patients diagnosed with necrotizing fasciitis die (12).

Pathophysiology

Diffuse damage to superficial tissue extends to the deep muscle plane and fascia, with certain unique characteristics that depend on the offending microorganism, which causes the infection. In type II necrotizing fasciitis, tissue injury is more associated with the release of exotoxins, which generate an inflammatory response that includes cytotoxic T cells, the release of cytokines, and toxic shock syndrome. Microvascular damage or thrombosis underlying uncontrolled inflammation can lead to tissue ischemia with subsequent necrosis (13).

Risk factor's

The most common risk factors for NSTI are major penetrating trauma, skin injuries (stings, injections), recent surgery, immunosuppression, malignancy, obesity, alcoholism, and most important of all diabetes, these patients have a predominant presentation. of necrotizing fasciitis in lower limbs, perineum, head and neck (14).

Clinical manifestations

Necrotizing fasciitis should be suspected when any of the following signs or symptoms are present (1) significant pain inconsistent with physical examination findings, (2) rapidly progressive clinical deterioration, (3) SIRS, (4) blisters, (5) tense edema (6) ecchymosis or necrotic skin, (7) palpable crepitus, (8) hypoaesthesia localized to the skin. In cases of doubt, the modified LRINEC scale is recommended to guide the decision on surgical management. In a systematic review by Goh et al, the three earliest signs of necrotizing fasciitis were found to be swelling (80.8%), pain (79%), and erythema (70.7%) (15). Ecchymotic rash, epidermolysis, tissue necrosis, and septic shock, findings that have been described as "hard signs" of fasciitis by Wang et al., although they are more specific for necrotizing disease, correspond to an advanced, late stage, with few possibilities. therapeutic and prognostic (16).

Spawn Sites

Perineum:

Necrotizing fasciitis of the perineum, known as Fournier's gangrene, can occur because of a breach in the integrity of the gastrointestinal or urethral mucosa. Fournier's gangrene is a form of polymicrobial infection (type I) and typically begins with extensive pain which can rapidly spread to the muscles of the anterior abdominal wall, buttocks, and genitalia (17).

Head and neck:

Necrotizing fasciitis of the head and neck can be due to a rupture of the mucous membrane of the oropharynx after surgery or instrumentation or in the context of an odontogenic infection (18).

Extremities:

it may be due to the presence of skin lesions which favor infection by pathogenic microorganisms, it is more common to find it in patients with diabetes and peripheral arterial disease, as explained in the pathophysiology section (18).

Diagnosis

As mentioned many times, the diagnosis of necrotizing fasciitis can become a challenge, since the initial manifestations are non-specific and rapidly progressive, and when characteristic findings of the disease are seen, we are already in a potentially fatal advanced stage. Wong and collaborators constructed a predictive scale based on laboratory results (19), called "Laboratory Indicators of Risk for Necrotizing Fasciitis (LRINEC for its acronym in English). The scale takes into account variables such as blood glucose, creatinine, sodium, hemoglobin, CRP and leukocytes. A score from 0 to 13 is given and classifies the risk of necrotizing fasciitis as mild, moderate and severe. A score greater than 6 has a sensitivity of 68.2% and a specificity of 84.8% to diagnose necrotizing fasciitis. The main utility of this scale lies in early recognition of cases of necrotizing fasciitis in which the clinic is not conclusive (20).

Treatment and prognosis

The mainstay of treatment in NSTIs is early surgery to control the infectious focus (debride) and broad-spectrum antibiotics. The initial debridement should be before 24 hours of admission, which is associated with lower mortality, compared to those in which surgical management was delayed for more than 24 hours (20). Since the etiology can be polymicrobial, including MRSA, a combination of vancomycin and cefepime or piperacillin/tazobactam and clindamycin is recommended; in case of kidney failure, vancomycin is replaced by linezolid. If there is suspicion of vibrio vulnificus as the etiology (history of stay or contact with salt water or maritime zone), it is recommended to start treatment with 3 or 4 generation cephalosporin and doxycycline. In patients with suspected *S. pyogenes* infection, the use of penicillin and clindamycin is recommended. Remember that once the causal germ is isolated, antimicrobial therapy must be adjusted (21). **Table 1** contains the doses and adjustment for renal function of the most used antimicrobials.

Table 1. Antibiotic therapy for necrotizing fasciitis

Type of therapy	Antibiotic	Dose	Dosage Interval	Renal failure adjustment
Oral	Amoxicillin/clavulanate	875/125 mg – 500/125 mg	12 horas 8 horas	GFR 10-50: 250 mg/12 h GFR <10: 250-500 mg/24 h
	Cephalexin	500 mg – 1 gr	6 h	GFR 10-50: 500 mg/12 h. GFR< 10: 150 mg /12 h
	Clindamycin	300 mg	8 h	Not require adjustment
	Dicloxacillin	500 mg	6 h	Not require adjustment
	Doxycycline	100 mg	12 h	Not require adjustment

	Linezolid	600 mg	12 h	Not require adjustment
	Trimethoprim /Sulfamethoxazole	160/800 mg (tab)	1-2 tab/ 12 h	GFR: 30-90: 5-20 mg/kg/day GFR: 10-29: 5-10 mg/kg/day GFR <10 : not recommended
Intravenosa	Cefazolin	1 – 2 gr	8 h	GFR 10-50: 1 -2 gr /12 h GFR <10: 1-2 gr/24-48 h
	Clindamycin	600-900 mg	8 h	Not require adjustment
	Daptomycin	6-10 mg/kg/day	24 h	GFR < 30: 6-10 mg/kg/day every 48 h
	Linezolid	600 mg	12 h	Not require adjustment
	Oxacillin	2 gr	4 h	Not require adjustment
	Trimethoprim /Sulfamethoxazole	8-10 mg/kg/day	6 – 12 h	GFR 30-90: 5-20 mg/kg/day GFR: 10-29: 5-10 mg/kg/day GFR <10 : not recommended
	Vancomycin	15-20 mg/kg/dose	12 h	GFR: 10-50: every 25-96 h Hemodialysis 7,5 mg/48-96 h

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