NECROTIZING FASCIITIS (N.F.)

A REMINDER

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ABSTRACT :

Necrotizing fasciitis (NF) is one of the necrotizing soft tissue infections which are marked by the absence of clear local boundaries or palpable limits. This lack of clear boundaries accounts for both the severity of the infection and for the frequent delay in recognizing its surgical nature.

The overlying skin has a relatively normal appearance in the early stages of infection and the visible degree of involvement is substantially less than that of the underlying tissue(1).

N.F is a serious mixed infection due to a beta hemolytic streptococcus pyogenes or staphylococci and peptostreptococci. The infection is associated with excessive collagenase production which leads to dissolution of connective tissue. The infection complicates wounds, lacerations, abrasions or punctures and involves the superficial fascia and subcutaneous fat. It may be immediately fulminant and spreads with dramatic speed or may remain dormant for 6 or more days before beginning to spread rapidly resulting in extensive necrosis of soft tissue accompanied by extensive undermining of the skin and resulting in gangrene(1 , 2).

N.F has a high mortality rate and its rapid destructive nature necessitate early recognition and aggressive surgical debridement combined with high dose broad spectrum antibiotic and appropriate systemic support to overcome the disease (3 , 4 , 5 , 6)

INTRODUCTION :

Most publications indicate that necrotizing fasciitis is of rare incidence(7). N.F has been reported to be predisposed to by diabetes mellitus (D.M), lower extremity ischemia and alcoholism.

In D.M the premature atherosclerosis, small blood vessel affection, poor leukocyte function related to hypoxia, acidosis, hyperglycemia and other tissue abnormalities may be among the factors that predispose to N.F (8). Gas may also be produced in diabetic tissue by gas producing organisms

or by facultative anaerobic bacteria. The rapid growth of these bacteria and the rapid gas production is favored by the presence of abundant substrate for sugar fermentation (8).

The mean age of incidence of NF was reported to be 59.7 years (7).

N.F was reported to affect children who did not suffer from any chronic disease or immunosuppression and the infection was attributable to varicella in 20% of cases (5).

Some authors (5 , 9) emphasized that children who present two to three days after the onset of varicella with symptom complex of fever, tachycardia, elevated white cell count in association with an erythematous indurated painful lesions with marked tissue oedema and peau d'orange appearance of the skin, such children should be diagnosed to have N.F until proven otherwise and should be treated as emergency with I.V. fluid, high doses of antibiotics, and close observation. If the symptoms persist for several hours despite treatment the patient must be shifted for surgical exploration of the cellulitis despite the risk of a negative operative findings (5 , 9).

CLINICAL DESCRIPTION OF N.F :

N.F was reported to occur after minor trauma, injection abuse, minor surgery, ischemia, burns, malignancy, foreign body introduction, leukopenia and immunodeficiency (4 , 6 , 8).

Group A streptococci (streptococcus pyogenes) were isolated in 25% of cases of N.F and synergistic mixed infection with aerobic and anaerobic bacteria was found in 75% of cases (10). The predominant isolates were peptostreptococcus, streptococcus pyogenes, bacteroides fragilis group, clostridium perfringens, escheridia coli and prevotella spp (10).

In a study of necrotizing infection of soft tissue it was concluded that the condition is polymicrobial and no single bacterium is associated with a specific clinical entity (11).

Group A streptococci are known to cause a variety of life threatening infectious complications including N.F, purpura fulminans and streptococcal toxic shock syndrome in which bacteremia is associated with shock, organ failure or limb amputations (12 , 13).

Strains of group A streptococci isolated from patients with invasive disease have been predominantly M types 1 and 3 which produce pyrogenic exotoxin A or B or both (14). The standard rapid streptococcal diagnostic kit detects the presence of streptococcal antigen and has an overall accuracy of 95% (15) .

Necrosis in infections may occur by several mechanisms(8) :

* pressure necrosis from extensive inflamma tory swelling which may be augmented by tissue gas especially in closed spaces such as digits or male genitalia.
* Vascular necrosis produced by direct acceleration of coagulation or production of heparinase by anaerobic organisms.
* Bacterial extracellular toxins or enzymes such as lecithinase produced by clostridium perfringens.
* Certain organisms in combination cause gan grene whereas individual organisms do not.
* Direct invasion of blood vessels causing vas cular occlusion as in phycomycetes.

The necrotizing cellulitis progresses rapidly after a minor trauma and involve the superficial fascia and subcutaneous tissue. The necrotic fascia is undermined easily surgically and characteristically shows no resistance to the passage of a blunt instrument along the fascial plane.

The cellulitis is painful and is associated with marked systemic toxemia.

There is no gas production or odor to the exudate if the infection is caused by Group A streptococci (streptococcus pyogenes).

If the necrotizing fascitis is caused by mixed infection gas may be present in the tissue and the odor is foul.

The overlying skin may appear normal but as the disease affects blood vessels the skin becomes eodematous, anaesthetic and progresses to cyanosis blister formation and gangrene.

The clue for surgical intervention in N.F is an acutely ill patient with mental confusion and spreading cellulitis despite antibiotic therapy.

Any delay in diagnosis and management carries the danger of increased mortality.

Differential diagnosis of N.F(1 , 2 , 8 ,16 , 17) :

Necrotizing soft tissue infection is characterized by massive tissue destruction and may be caused by any of the following :

1 - Clostridial cellulitis which is a serosanguinous crepetant cellulitis caused by clostridium species of which C. perfringens , C. Novyi and C. septicum are the most common(17). It differs from gas gangrene in that the infection does not involve muscles(2).

2 - Non clostridial cellulitis caused by facultative anaerobic bacteria. Infections caused by obligate or facultative anaerobes produce insoluble gases in tissues such as hydrogen, nitrogen and methane(17).

Both clostridial and nonclostridial cellulitis are characterized by mild pain, minimal skin changes, slight or moderate systemic toxicity and extensive tissue gas which smells foul in clostridial and putrid in nonclostridial infection

3 - Progressive bacterial synergistic gangrene (Post operative progressive gangrene) : The infection almost always occurs at an abdominal or thoracic operative wound at sites where wire retention sutures are employed.

Few days to few week after operation (usually 7 - 14 days) tender red swollen indurated area develops near the wound and slowly evolves into gangrenous ulcer which is progressive and has a gangrenous purple margin and oedematous erythematous periphery.

If untreated it spreads to enormous size with severe pain and little accompanying toxicity. It closely resembles pyoderma gangrenosum.

4 - Clostridial myonecrosis (Gas gangrene) is a fulminant anaerobic infection of muscle characterized by profound toxemia related to break down products of muscle. C. perfringens is the most common causative organism and produces a variety of toxins including hyaluronidase, collagenase, four different hemolysins, five necrotizing lecithinases and six other necrotizing lethal toxins. With progression of infection clostridial bacteremia occurs, hemolytic anemia ensues, hypotension and kidney failure. In treatment, mutilating surgery and amputation may be required. High doses of intravenous penicillin (15-30 million units per day), broad spectrum antibiotic and blood transfusion are given pre

and post operative. Hyperbaric oxygen gives good results but it has no effect in other anaerobic infections.

5 - Non clostridial myositis (non clostridial gas gangrene, peptostreptococcal myositis).

Early in the disease there is no pain or local gas. The disease starts several days after trauma by erythema, oedema, purulent foul exudate and no systemic toxicity. The foul smell is characteristic of anaerobic streptococcal infection.

6 - Synergistic necrotizing cellulitis (gram negative anaerobic cutaneous gangrene, Fournier gangrene syndrome).

It is an infection usually of diabetics. It often begins in the perineum and results in acute painful toxemic infection. There is foul smelling pus with patchy skin necrosis and prominant muscle necrosis in absence of major vascular occlusion. Bacterial synergism is responsible for muscle necrosis. The bacteria are facultative gram negative enteric bacilli or bacteriodes species. The majority of patients die(16).

7 - Phycomycotic gangrenous cellulitis. There is vascular invasion by hyphae of the fungi (Rhizopus, Mucor, Absidia). There is thrombosis and necrosis. The characteristic lesion is a central black anaesthetic ulcer surrounded by purple oedematous margin. Spread may be rapid and toxicity may be severe. Diagnosis depends on finding the hyphae either by crushed tissue treated with 20% KOH or by frozen section or by histopathology of biopsy specimen.

8 - Infected vascular gangrene : Peripheral vascular insufficiency and distal gangrene may be complicated by infection marked by development of putrid odor and accumulation of gas in gangrenous tissue.

9 - Gangrenous cellulitis in the compromised host.

Some opportunistic organisms may present in this group of patients with gangrenous cellulitis with or without gas. Pseudomonas infection and rarely other facultative gram negative bacilli may produce such infection.

Aspergillosis, phycomycosis, cryptococcosis and hydrophilia sepsis can cause dermal necrosis.

Many drugs given in combination to treat HIV infections and tumors in AIDS are rapidly changing resulting in improved palliation but no cures are available at present.

Principles of therapy(1 , 2 , 17) :

1 - Cardiopulmonary resuscitation

2 - Surgical treatment is the most essential and includes longitudinal incisions and debridement of all necrotic tissue. The debridement may involve 35% of the body surface. In clostridial infection necrotic muscle tissue must be removed and amputation of an extremity may be required (16). Smears and swabs are taken from exudate for examination, culture and sensitivity.

3 - Extensive skin grafting

4 - High doses of antibiotic with subsequent change based on culture result.

5 - The antibiotics generally used are :

a - Penicillins : (Penicillin G, Methicillin, Oxacillin, Nafcillin, Ampicillin, Amoxicillin, Carbenicillin, Ticarcillin, Mezlocillin, Pipercillin).

b - Beta lactamase inhibitor combination :
- Clavulonic acid + Ticarcillin
- Clavulonic acid + Amoxicillin
- Sulbactam + Ampicillin
- Tazobactam + Pipercillin

c - Cephalosporins :
- First generation (Cephalothin, Cephapirin, Cefazolin)
- Second generation (Cefamandole, Cefuroxim, Ceforanide, Cefonicid, Cefoxitin, Cefmetazole, Cefotetan)
- Third generation : (Cefotaxim, Ceftriaxone, Cefoperazone, Ceftazidim)

d - Monobactams : (Aztreonam)

e - Carbapenems : (Imipenem)

f - Quinolones : (Norfloxacin, Ciprofloxacin and ofloxacin)

g - Aminoglycosides : (Gentamycin, Tobramycin, Netilmycin, Amikacin)

h - Other antianaerobes : (Chloramphenicol, Clindamycin(there is evidence that early use of Clindamycin for infection caused by group A streptococci may be beneficial) (15), Metronidazole)

i - Glycopeptides : (Vancomycin)

j - Macrolides : (Erythromycin, Azithromycin, Clarithromycin)

k - Tetracyclines : (Tetracycline, doxycycyline)

l - Antifungals : (Amphotericin, Ketoconazole, Itraconazole, Fluconazole)

The antibiotics with predominantly aerobic cover age are : Gentamycin, Tobramycin, Amikacin, Netilmicin, Cefotaxime, Cefuroxime, Ceftriaxone, Ceftazidime, Aztreonam, Ciprofloxacin.

The drugs with predominantly anaerobic coverage are : Clindamycin, metronidazole and chloramphenicol, imipenem, and a combination of a penicillin with a betalactamase inhibitor (Ticarcillin/Clavulonate ; Ampicillin/Sulbactam ; Pipercillin/Tazobactam).

j - Hyperbaric oxygen (17) : is used to treat necrotizing soft tissue infections. Patients are placed in 100% oxygen chamber at 3 times atmospheric pressure. Hyperbaric oxygen inhibits production of alpha toxin by clostridium. It makes the patient less toxic and diminishes the amount of tissue requiring excision. Hyperbaric oxygen has no effect on other anaerobic infection caused by nonclostridial organisms. Hyperbaric oxygen should not be used before surgical debridement. Hyperbaric oxygen has possible complications (Injury to middle ear if eustachian tube is blocked, trauma to a sinus, pnemothorax and air embolism). Oxygen toxicity can cause neurotoxicity resulting in reduced seizure threshold and pulmonary toxicity if treatment is prolonged. Other potential problems include a feeling of claustrophobia and reversible visual changes.

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