Microbiological Aspect of Necrotizing Fasciitis

An Essay
Submitted for partial fulfillment of
master degree of clinical Pathology

By
Gamila Fouad Ali
M.B.B. Ch.

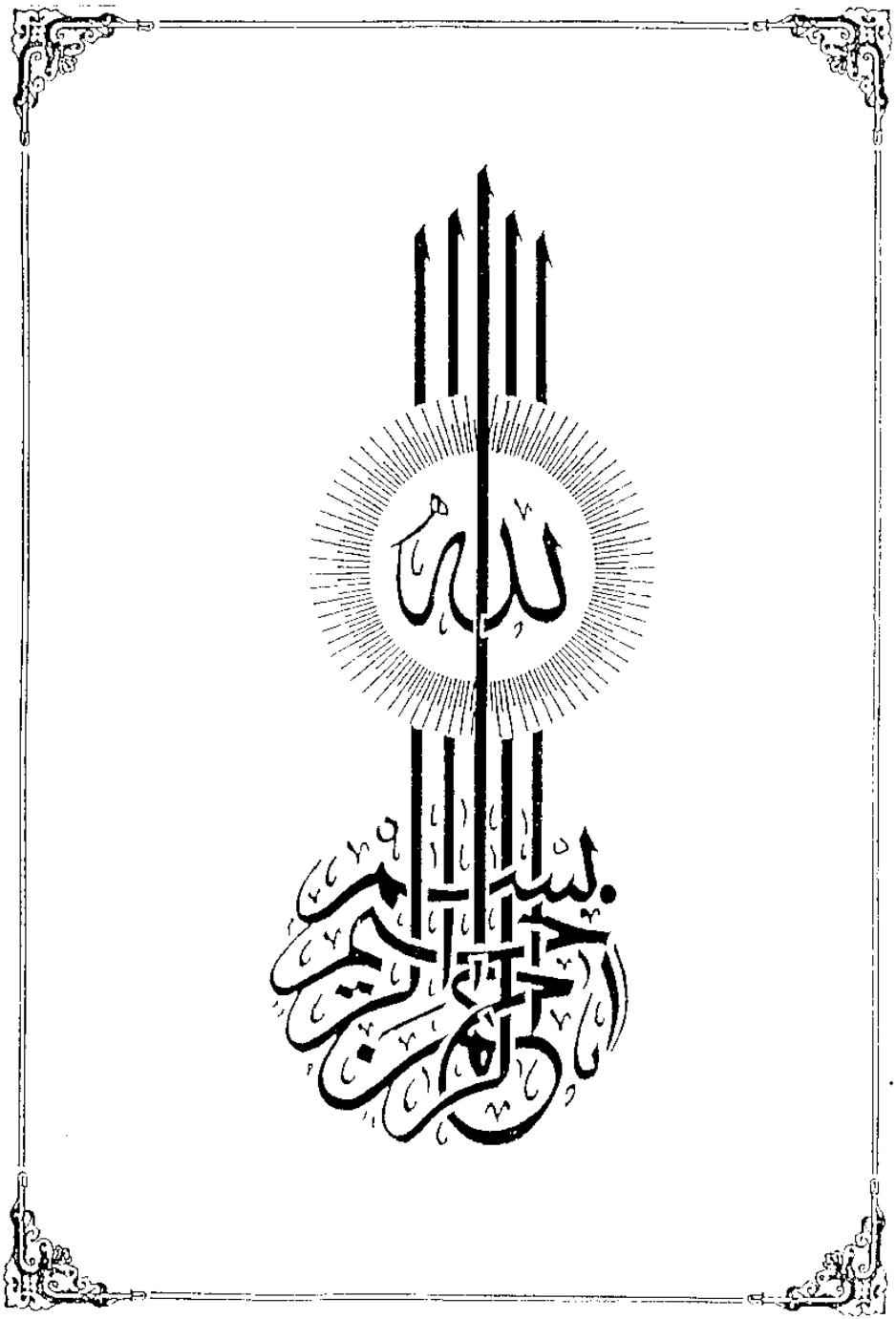
Supervised by

Dr. Ibrahim Khalil Ali
Professor of Clinical Pathology
Faculty of Medicine
Ain Shams University

Dr. Eman Mohamed Kamel Abu El-Fotouh
Lecturer of Clinical Pathology
Faculty of Medicine
Ain Shams University

Dr. Omnia Abu El-Makarem Shaker
Lecturer of Clinical Pathology
Faculty of Medicine
Ain Shams University

Faculty of Medicine
Ain Shams University
1995



ACKNOWLEDGMENT

I would like to express my deepest gratitude to Dr. Ibrahem Khalil Ali Professor of clinical pathology Faculty of Medicine - Ain Shames University for his fatherly encouragement, valuable guidance and sincere help.

I wish to express my sincere gratitude to Dr. Eman Mohamed Kamel Abu-El Fotouh. Lecturer of clinical pathology. Faculty of Medicine. Ain Shams University for her kind support, Supervision and Suggestion.

I also express my sincerest thanks to Dr. Omnia Abu El-Makarem Shaker. Lecturer of clinical pathology. Faculty of Medicine. Ain Shams University. Who offered this study much of here time and smart concepts.

Contents

Introduction
Historical Aspect

Introduction

Necrotizing fasciitis is a relatively rare but potentially fatal disease **[Freeman. et al., 1981]**. It is a rapidly progressing necrotizing process which affects subcutaneous tissue and fascia and is accompanied by severe systemic toxicity. Initially the skin is spared from involvement, but as the infection progresses, thrombosis of nutrient blood vessels in the subcutaneous tissue eventually leads to cutaneous gangrene. Muscular necrosis is less common, probably because of the better vascular supply of striated muscles **[Janevicus. et al., 1982]**.

Necrotizing fasciitis frequently results from neglected acute or chronic infections and rarely there is no apparent cause **[Rouse et al., 1982]**. Clinical pattern of the disease can be either distinct or non specific, hence the prognosis is influenced mainly by early diagnosis and surgical treatment.

Unfortunately, because of the rarity of this disease, recognition, radical surgical excision of the necrotic tissue and proper antimicrobical therapy are frequently delayed resulting in a significant mortality rate **[Pauzner et al., 1994]**.

Aim of the study:

The aim of this study is to review the subject of necrotizing fasciitis with special emphasis on the bacteriological etiology.

Historical Aspect

Necrotizing fasciitis infection of soft tissue have been known since ancient time and was described in the writting of hippocrates, Galen and Avicenna. The first description was reported by **Jones 1871**; a surgeon in the confederate army of the United States. He was the first to describe what he termed "hospital gangrene" which is a rapidly progressing fascial necrosis of bacterial cause.

In 1918 **Pfanner. et al.,** named it necrotizing erysipelas The syndrome was first described in details by **Meleney, 1924**; He reviewed twenty patients from China and gave an accurate description of this infection which he called it "haemolytic streptococcal gangrene" because Beta hemolytic streptococci were isolated from all his original cases. Meleney's studies were limited by the bacteriologic technique of his time.

Later reports indicate that this entity can be caused by a variety of bacteria **[Collins and Nadel; 1965]**. The disease was named suppurative fasciitis by **McCafferty and Lyons, 1948** and also, named acute dermal gangrene by **Ledingham and Tehrani; 1975**.

Wilson, 1952 referred to it as necrotizing fasciitis which is the preferabl name, regardless of the specific bacterial infection and because the most consistant manifestation is fascial necrosis, no involvement of underlying muscle and normal appearance of skin overlying.

Classification

Classification

In the last years, there has been a trend in the literature to include the entity of necrotizing fasciitis in a category termed progressive necrotizing infections **[Dellinger et al., 1981]**. These different necrotizing infections of fascia and subcutaneous tissue should not be lumped together as a single clinical entity to aid in both recognition and treatment as they may be presented differently and may require different approaches especially in relation to the extent of debridement **[Dellinger et al., 1981]**.

The first example is acute streptococcal gangrene, where the diagnosis can be easily made by gram stain and the therapy is quite different from that required for necrotizing infections in that extensive fascial debridement is unnecessary. The second example is acute clostridial cellulitis which occurs after intra-abdominal operations and there are disastrous complications if it is not treated aggressively by early extensive debridement. Occasionally hyperbaric oxygen therapy is beneficial where as it is not clearly indicated in the treatment of streptococcal gangrene or necrotizing fasciitis **[Ledingham and Tehrani;1975].**

Necrotizing soft tissue infections can be classified into the

1. Anaerobic cellulitis. [Clostridial Cellulitis].
2. Clostridial myonecrosis (gas gangrene).

3. Synergistic necrotizing cellulitis.
4. Necrotizing fasciitis.

1. Anaerobic cellulitis (Clostridial cellulitis)

It usually occurs following trauma, especially in military wounds. Clinically, it appears following 3-5 days incubation period and is characterized by producing a foul smelling sero purulent fluid in small blebs which may or may not be followed by frank necrosis of the skin **[Baxter, 1972]**. There is little or no oedema and extreme wound pain, clostridial cellulitis either co exist with myonecrosis or healthy muscle is not involved **[Alterneier and Fullen, 1971]**. Systemic effects and toxicity are variable and the mortality rate is usually low **[Kaiser and Cerra, 1981]**.

2. Clostridial myonecrosis (gas gangrene)

It most commonly follows trauma to large muscles but it can occurs after elective surgical procedures in areas that contain anaerobic flora such as the colon, the vagina, the perineum and the mouth. The onset of this infection may be insidious or rapid with a variable incubation period.

Crepitus may or may not be present, the skin may assume a characteristic discoloration which has been labelled "bronze erysipelas". There may be a serosanguineous perfuse wound discharge with a sweat odour and wound pain is usually present. Systemic manifestation may be minimal to severe and massive haemolysis may occur. Clostridial species may occure alone, but the infection is usually mixed and the outcome is frequently fatal **[Kaiser and Cerra, 1981]**.

3. Synergistic necrotizing Cellulitis

Progressive synergistic infection is usually found around the perineum and in the lower extremities but may occurs anywhere. The majority of these infections occur in patients with diabetes mellitus and or peripheral vascular diseases. Clinically, although the onset of infection may be days to weeks, once the process sets in it assumes a rapid progressive toxic course **[Kaiser and Cerra, 1981).**

Wound pain is usually present, the systemic manifestation vary from minimai to severe toxicity. The muscle may be involved. Infection is usually mixed, consisting mainly of the anaerobic streptococci, bacteroids and the gram negative aerobes as klebsiella, proteus and Ecoli. The outcome is frequently fatal **[Kaiser and Cerra, 1981].**

4. Necrotizing Fasciitis

The infection most often occurs following minor trauma but may also occurs following operative procedures diabetes mellitus and peripheral vascular diseases are common predisposing factors. It is usually caused by mixed infections the mortality is high **[Kaiser and Cerra, 1981]**. There is local pain and areas of hypoesthesia around the wound edges, the infection involves the fascia **[Guiliano et al., 1977]**.

Differentiation of the common necrotizing bacterial soft tissue infection (Barond and Jenning, 1991)

	Anaerobic cellulitis (clostridial cellulitis)	Clostridial myonecrosis (gas gangrene)	Synergistic necrotizing cellulity	Necrotizing Fasciitis
incubation peroid	More than 3 days.	Variable (less than 3 days).	Variable (3-14 days).	1-4 days.
Onset	Gradual	Insidious or rapid	From days to weeks	Acute.
Toxaemia	Non or slight	Minimal to severe	Minimal to severe	Moderate to marked
Pain	Extreme wound pain	Severe	Severe	From moderate to severe (local pain with areas of hypothesia around edge of wound).
Exudate	Seropurulent fluid	Serosanguinous profuse.	Dish water pus	Serosangiunous.
Odour of exudate	Possibly foul	Sweet odour	Possibly foul	foul
Gas	Abundant	Not pronounced	Not pronounced	Usually not precent
Muscle	No change	Marked change	Marked change	Variable
Skin	Little change	Bronze erysipelas	Minimal change	Pale red cellulitis
Mortality rate	5-10 %	15-30 %	75%	30%

Heparin-induced cutaneous necrosis :

Mini-dose heparinization has been widely accepted for patients at risk for postoperative deep venous thrombosis. Those patients are usually obese, diabetic or have underlying vascular diseases. They are also at risk for necrotizing fasciitis. Heparin induced necrosis is a rare complication associated with subcutaneous heparin injection and should be included in the differential diagnosis for patients presenting with necrotic skin lesions. **[Cohen et al., 1988]**.

Heparin induced necrosis does not lead to the same sequelae as necrotizing fasciitis, and does not warrant immediate surgical intervention. These patients do not appear septic despite the large areas of necrosis. This lack of septic signs and symptoms prompt a biopsy of the area rather than a radical debridement. **[Cohen et al., 1988]**.

The biopsy specimen shows vasculitis, extravasation of red blood cells and intravascular thrombi with a minimal amount of inflammation. These findings are characteristic of heparin necrosis **[Levine et al., 1983]**. Necrotizing fasciitis is characterized by intact superficial dermal necrosis with polymorphonuclear infiltration, vasculitis and the presence of micro organism **[Stamenkovic and Lew.,1984]**, also Roentgeno graphic studies for soft tissue gas can differentiat the condition **[Fisher et al., 1979]**.

Pseudonecrotizing fasciitis

It is reported due to dialysate and air leak in a peritoneal dialysis patient. While free intraperitoneal air is known to occur in peritoneal dialysis patients, dissection of this air into the abdominal wall mimicks the subcutaneous emphysema of necrotizing fasciitis.

So to exclude this possibility, peritoneal dialysis patients should have an initial small incision in the area of emphysema followed by intraperitoneal instillation of dialysate containing Evan's blue dye. The lack of foul odour or necrotic tissue and the presence of Evan's blue in the wound should suggest the diagnosis of pseudonecrotizing fasciitis and avoid unneccessary extensive abdominal wall debridement with its accompanied morbidity **[Graves et al., 1987]**.