Clinical study of necrotizing fasciitis and its management

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ABSTRACT

Background: Necrotizing fasciitis is a severe flesh-eating disease of soft tissue infection. It is characterized by widespread infection of the fascia with necrosis. It may lead to MODS and shock in a very short period of time. In this study we have evaluated the predisposing factors, clinical manifestation and different modalities of investigation which helps in management of this near fatal disease.

Methods: This study was conducted in Department of Surgery, Gandhi Medical College, Bhopal, Madhya Pradesh, India for 3 years (October 2013-September 2016). A total of 107 patients having necrotising fasciitis were admitted in this period. The diagnosis was confirmed by detailed history and physical examination followed by haematological, microbiological and radiological investigations priority wise. After initial resuscitation patients were taken to OT where debridement of dead necrotic tissue was done, laying open of deeper planes with proper and effective drainage of pus. Most of the dressings used were silver sulfadiazine or iodine depending upon the sensitivity to sulpha group.

Results: Diabetes mellitus was the most common predisposing factor (65.4%) followed by trauma (28%). Perineum and thigh were the most commonly affected site (70%). Most of the patients (84%) presented with skin erythema and blistering. Fever and tachycardia were seen in almost all the patients (95%). Leukocytosis and neutrophilia were seen in 91.5%. Most common organisms isolated were E. coli and proteus. Mortality rate was 24.29%.

Conclusions: Necrotizing fasciitis is a flesh eating, highly lethal disease. Early diagnosis, early and radical surgical debridement supported by appropriate antibiotic and correction of metabolic disorder are the cornerstones of successful management.

Keywords: Debridement, Gangrene, Necrotizing fasciitis

INTRODUCTION

It is a rapidly progressive inflammation and near fatal infection of the fascia and subcutaneous tissue with secondary necrosis of involved tissues. Spread is directly proportional to the thickness of the layers involved. It mostly spreads by lymphatic and blood route. It involves skin, subcutaneous tissue, adipose layer, fascia and muscles. When muscles are also involved then it may be termed as necrotizing myositis. It may start as lesser aggressive infection and inflammation of the skin and subcutaneous tissues named cellulitis. Some diseases described as meleneys ulcer or synergistic gangrene are forms of necrotizing fasciitis only. Fournier’s gangrene is also a kind of necrotizing fasciitis involving the area of perineal and scrotum.

The causative bacteria may be aerobic anaerobic or mixed. They get access in from traumatic skin and or mucus membrane. The predisposing factors are diabetes mellitus, immune compromised state, other metabolic disorders and vascular insufficiency.
There is increased incidence of this disease recently as it is directly proportional to the immune deficiency in the patient and which is in rise due to HIV infection. It may also be associated with cancer, alcoholism, chronic liver, chronic renal and peripheral vascular disease. It may occur as a complication of cardiac catheterization, sclerotherapy, diagnostic laparoscopy or may be idiopathic. A minor insect bite may start such disease in immuno compromised patients.

It is more common in adult age than paediatric, more seen in men than women. It is more common in underdeveloped countries than developing and developed countries.

**Etiopathogenesis**

It is bacterial proliferation causing local ischemia which leads to hypoxia of the affected area and if the host defence is compromised the proliferation increases considerably and ischemia finally leads to gangrenous changes. Carbon di oxide and water are the end product of aerobic metabolism whereas hydrogen, nitrogen, hydrogen sulphide and methane are end products of anaerobic and aerobic metabolism. These products except for carbon di oxide are not absorbed in tissues and accumulate there and cause cell damage.

It can be divided into 3 types

- **Type I - Polymicrobial**
- **Type II - Group A beta haemolyticus streptococcal**
- **Type III - Clostridial or gas gangrene.**

Out of this polymicrobial is most common of those are *E. coli, Klebsiella, Pseudomonas, Bacteroides,* and *proteus.* Earlier GABS i.e. Group A beta haemolytic streptococcus and staphylococci aureus alone or in synergy has been identified as major cause but now considered as initiating bacteria, other bacteria often follow.

**Presentation**

Skin erythema, skin vesicles, rashes, high grade fever, delirium, finding of crepitus is the most graded clinical feature. The complications of the disease are very fast to occur. The complications are renal failure, septic shock, cardiovascular collapse and toxic shock syndrome all of which have a very high mortality rate. If the patient survives, scarring and limb loss or loss of muscle function is inevitable.

Treatment and management is a surgical emergency. Once the diagnosis is made, immediate optimising and resuscitation, use of proper antibiotics and surgical debridement which is of wide and extensive area is done. Use of hyperbaric oxygen is an integral part in life saving therapy in some institutes. Still there is high mortality rate from 20-80% because of pre-existing diabetes, chronic liver and renal conditions and immune compromised states.

**METHODS**

This study was conducted in unit III Department of Surgery, Hamidia Hospital, Gandhi Medical College, Bhopal, Madhya Pradesh, India for 3 years (October 2013-September 2016). The patient presenting with gross skin lesions and sign and symptoms of lesion requiring surgical debridement were included in the studies. A total of 107 patients were admitted in this period. The diagnosis was confirmed by detailed History and physical examination followed by haematological, microbiological and radiological investigations priority wise.

**Pathology**

Complete blood picture, fasting and postprandial blood sugar, LFT including SGOT, SGPT, Serum bilirubin and RFTs including Serum creatinine blood urea and arterial blood gas analysis were done.

**Microbiology**

Culture and sensitivity of pus, blood and urine were done.

**Radiological investigations**

X-ray CT scan were done as and when required.

As soon as the patient was admitted and diagnosed patient was optimized and resuscitated when required and was taken to OT as early as possible where debridement of dead necrotic tissue was done, laying open of deeper planes with proper and effective drainage of pus followed by haemostasis and copious irrigation. Full precaution of the team by double gloving was done. Most of the dressings used were silver sulfadiazine or iodine depending upon the sensitivity to sulpha group. The metabolic state was corrected by use of proper intravenous fluids, insulin in cases of diabetes mellitus. Bolus dose of antibiotics were administered in one to all cases. When patients survived the initial catastrophe, they were reassessed and antibiotic and supportive treatment was given as per sensitivity of cultured organism. Frequent dressings where required, few patients required serial debridement also. When the granulation tissue started appearing skin coverage and soft tissue reconstruction was done.

**RESULTS**

**Predisposing factors**

- Diabetes mellitus in 70 patients
- Post trauma in 30 patients
- AIDS in 4 patients
- Urethral fistula in 3 patients (Table 1).
Table 1: Predisposing factors.

<table>
<thead>
<tr>
<th>Predisposing factors</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes mellitus</td>
<td>70</td>
</tr>
<tr>
<td>Post trauma</td>
<td>30</td>
</tr>
<tr>
<td>AIDS</td>
<td>4</td>
</tr>
<tr>
<td>Urethral stricture and fistula</td>
<td>3</td>
</tr>
</tbody>
</table>

Area involved
- Perineal and thigh in 75 patients
- Groin and thighs in 25 patients
- Abdomen in 4 patients
- Chest in 3 patients.

Presentation
- Skin erythema, edema, blistering in 90 patients
- Ulceration and discharge in 10 patients
- Gangrene and discharge in 45 patients
- Loss of sensorial in 6 patients.

On examination
- Fever in 102 patients
- Tachycardia in 101 patients
- Crepitation in 2 patients
- Altered sensorium in 6 patients
- Oligouria in 15 patients.

Investigations
- CBP- leukocytosis and neutrophilia in 98 patients
- deranged RFT in 27 patients
- Increased blood sugar level 69 patients
- Culture and sensitivity- most common organism were E. coli and proteus thus Type I polymicrobial as the most common cause of necrotizing fasciitis
- USG- chest and abdomen showed hypo echoic areas in region and area involved. It differentiated the involvement of peritoneum and peritoneal cavity we did not find such involvement in any case
- CT scan revealed the depth of involvement of muscles and fascia.

Table 2: Operations performed.

<table>
<thead>
<tr>
<th>Operation performed</th>
<th>Number of patients</th>
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<tbody>
<tr>
<td>Debridement</td>
<td>101</td>
</tr>
<tr>
<td>Suprapubic cystostomy</td>
<td>3</td>
</tr>
<tr>
<td>Skin grafting</td>
<td>10</td>
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</table>

Operative intervention
- Surgical debridement done in 101 cases
- Suprapubic cystotomy in 3 patients with urinary fistula (Table 2).

Out of 107, 26 patients died hence mortality rate was 24.29% (Figure 1).

DISCUSSION

Necrotizing fasciitis is also known by other names but is the most apt one as per clinical presentation is flesh eating disease. In present study we found that necrotizing fasciitis is rapidly spreading, progressive infection of skin and subcutaneous tissue fascia and muscle involving adult male patents much more than females and paediatric age. Average age for men was 44 and females 35. The most common predisposing cause was diabetes mellitus followed by trauma. The most common area was perineal followed by thigh and lower limb. Patient commonly presents with skin erythema and blisters foul smelling discharge and colour changes of skin. Investigations were mandatory to know the liver, renal and immunological functions of the body. In present study we found that 65.42% of patients had diabetes. Others were post trauma-27.90%. Other causes were HIV 3.75%, urethral fistula2.75%. Associated factors were chronic liver diseases, obesity and drug use. Special investigations were colored Doppler to know the vascular status. Radiological examination CT scan and MRI where deep tissues were much more involved than skin and subcutaneous tissue and it was difficult to assess the depth. This was essential to make the diagnosis and to rule out other concomitant pathologies like leomyosarcoma of underlying structures.

Treatment was aggressive and consisted of optimizing and resuscitation, early surgical intervention and diversion of urine wherever necessary. As we did aggressive treatment our mortality rate was 24.29% whereas it is mentioned in literature that it may be as high as 80%. 101 of the patients underwent surgical intervention, 6 of the patients survived for very short time after reaching hospital inspite of all resuscitative measures could not survive and hence could not be taken to the operation theatre.
Using wide spectrum antibiotic was the essential part depending upon the sensitivity of the bacteria. In our institute, E. coli was the most common organism and sensitivity to cephalosporin (3rd generation- ceftixime, ceftaperazone) was reported the most hence was the commonly used in bolus form. The other antibiotics were aminoglycosides and metronidazole. Imipenem and meropenem were used in few cases which were showing resistance to all antibiotics. \(^7\)\(^,\)\(^22\)

The survivors underwent serial debridement followed by multiple dressings and skin coverage by skin grafting in 10 patients. They also required prolonged physiotherapy and nutritional support was mandatory.

We lost 26 patients out of which 6 patients presented very late and could not survive more than 20 minutes therefore could not be taken to OT whereas 20 died on 2nd and 3rd day of admission inspite of ventilator support due to respiratory failure following ARDS secondary to septicemia. If awareness is created in public regarding this fulminate disease the mortality can still be brought down further.\(^23\)

Necrotizing fasciitis is a flesh-eating disease is highly lethal, early diagnosis, early and aggressive surgical debridement supported by appropriate antibiotic and correction of metabolic disorder were key to success.\(^7\)\(^,\)\(^22\)

The susceptible patients and public can be made aware about such complications and to report to hospital as early as possible and by doing so the dreaded disease can be fought successfully.

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**Conflict of interest:** None declared

**Ethical approval:** The study was approved by the institutional ethics committee

**REFERENCES**
