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Review Article

Necrotizing Fasciitis

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ABSTRACT

Necrotizing fasciitis (NF) is among the most challenging surgical infections faced by a surgeon. The difficulty in managing this entity is due to a combination of difficulty in diagnosis, and also of early as well as late management. For the patient, such a diagnosis means prolonged hospital stay, painful dressings, an extended recovery, and in some unfortunate cases even loss of limb or life. Necrotizing fasciitis is a fairly common condition in surgical practice in the Indian context resulting in a fairly large body of clinical experience. This article reviews literature on MEDLINE with the key words ''necrotizing,'' ''fasciitis,'' and ''necrotizing infections'' from 1970, as well as from articles cross referenced therein. The authors attempt to draw comparisons to their own experience in managing this condition to give an Indian perspective to the condition.

KEY WORDS

Fasciitis; necrotizing necrotising infections; synergistic infections

INTRODUCTION

Necrotizing fasciitis is any necrotizing soft tissue infection spreading along fascial planes with or without overlying cellulitis.[1] It has also been described as a rapidly progressing necrotizing process accompanied by severe systemic toxicity.[2] Necrotizing fasciitis has been historically reported from almost all parts of the world and is now understood to be caused by either a single organism or more frequently by a variety of microbes — both aerobic and anaerobic.[3,4] Necrotizing infections of the soft tissue have been known since ancient times. They were described by Hippocrates and Galen, also by Avicenna and the great renaissance surgeon Pare.[5-8] Suppuration in the tissue beneath the skin (sopha), with danger of pus spreading to surrounding healthy tissues has been mentioned in Susrutha Samhita.[7] The first clear description was reported by Joseph Jones, a surgeon in the Confederate Army of United States in 1871.[9] This condition occurred in 2642 soldiers during the civil war with a mortality of 46 percent. He termed it "Hospital Gangrene," a rapidly progressing fascial necrosis of bacterial causes.[2] Since his description, it has been variously labeled as "necrotizing erysipelas," "hemolytic streptococcal gangrene," "suppurative fasciitis" and "acute dermal gangrene."[10-15] In 1884, a classic description of gangrenous infection of the perineum and male genitalia by Fournier appeared.[16] In 1924, Meleney published a detailed description of progressive necrotizing ''acute hemolytic streptococcus gangrene'' which he, with limited microbiologic techniques of his time, considered to be exclusively due to beta hemolytic streptococcus.[5] In a later publication he described the synergistic association of anaerobic streptococci and staphylococcus and proposed the differential diagnosis between certain types of infectious gangrene of the skin with particular reference to hemolytic streptococcus

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gangrene and bacterial synergistic gangrene.[17] The present preferred term "Necrotizing fascits" was coined by Wilson in 1952, for he observed that cutaneous gangrene is not invariably present, but fascial necrosis is a constant feature of the syndrome.[9]

Although Pasteur was first to report on anaerobic bacteria in 1861, the "anaerobic renaissance" started in late 1960s.[18] Gorbach and Bartlett attribute this to the technical advances in the isolation and identification of non-sporing anaerobic bacteria.[19] It was later in 1977 Giuliano determined that necrotizing fasciitis was due to polymicrobial infections by a variety of micro-organisms including both aerobic and anaerobic gram positive cocci and gram negative bacilli.[3]

CLINICAL PROFILE

Necrotizing fasciitis seems to have preponderance in males, perhaps due to increased incidence of trauma in males. [20,21] In our own series of 54 cases there was a distinct male preponderance. However, larger series show no significant sex predilection. Necrotizing fasciitis is relatively uncommon in childhood. Goldberg and associates found only 14 reported cases in infancy, several resulting from circumcision or fetal scalp monitoring.[22] Fustes Morales have reported a series of 39 pediatric cases.[23] Other causes include scalp vein infusions and newborn omphalitis.[24,25] Association with necrotizing enterocolitis and urachal anomalies too have been noted.[26] Collette has reported necrotizing fasciitis associated with Hemophilus influenza type-b, and with chickenpox.[27,28] Adeyokunnu reports six children aged between 3 and 12 weeks who were treated for Fournier's syndrome.[29] In these cases, circumcision, diaper rash, and perianal skin abscesses were suspected as portals of entry. Fournier's gangrene in children has been well reviewed by Adams along with a case report of a 4-year-old boy.[30] Rea, in his study of 44 cases found necrotizing fasciitis affected people of all age groups from 9 months to 81 years, mostly in patients over 40 years of age.[21]

The source of infection is variable but usually occurs after trauma or postoperatively.[3,9,20,21] Frequently no history of trauma can be elicited.[2] Seemingly insignificant and easily forgotten trauma, including minor lacerations, abrasions, or insect bites can initiate the process.[21] Insignificant trauma in a marine environment leading to necrotizing fasciitis has been reported by Pessa.[8] It is reported in a significant number of survivors injured following a volcanic cataclysm in Colombia.[31] Necrotizing fasciitis in contused areas has been noted by Svensson.[32] Drug abusers, mentally disturbed and malingering prison inmates form occasional patients of necrotizing fasciitis.[33-35]

The most common surgical wound infection is a simple abscess, but any patient who develops redness and induration of an incision is at risk for necrotizing fasciitis. It has been reported from laparoscopy, endoscopic gastrostomy, tube thoracostomy, and thoracotomy sites as well as after endoscopic sphincterotomy.[36-41] Abdominal incisions are most commonly involved, especially if a viscus had been opened.[42] The risk according to Ahrenholz is so great in patients with frank peritonitis that he advises delayed primary or secondary closure in all such cases.[4] Spontaneous infections may herald perforated diverticulitis or occult malignancy.[43] Conn describes a case of necrotizing fasciitis secondary to a strangulated femoral hernia containing a perforated sigmoid diverticulum.[44] Feeding jejunostomy and Caesarian section associated with non-steroidal anti-inflammatory drug intake have also been cited as initiating factors.[45,46]

Though the above instances and Fallahzadeh describe a predilection for the abdomen and groin, most investigators have noted that the infection is most common on the extremities.[3,9,30] It can however, occur in any part of the body including back, neck and orbit.[2,47] Cervicofacial necrotizing fasciitis have followed mandibular fractures, dental infections and have even been fatal following peritonsillar abscess and tooth extraction.[48-50]

FOURNIER'S GANGRENE

Necrotizing fasciitis of scrotum readily causes dermal gangrene (Fournier's gangrene) due to lack of subcutaneous fat.[1] It is usually the first sign of necrotizing infection arising from the perineal area, especially a perirectal abscess.[1,51] Spinark and co-workers have identified urologic and colorectal sources of contamination.[52] Chatterjee and Dandapat have reported association with filarial scrotum.[53,54] Urinary extravasation as a predisposing factor was noted by Bhandari.[55] Increased risk in patients with granulocytopenia following

chemotherapy for solid or hematologic malignancy has been reported recently.[56,57]

Equivalent of Fournier's gangrene in women is a necrotizing infection of the vulva or perineum.[1] Bartholin's cyst abscess may occasionally lead to severe necrotizing infection.[58] Episiotomy, hysterectomy, septic abortion, and cervical or pudendal blocks have lead to infections of necrotizing nature.[59,60]

Diabetes and advanced age have been identified as pre-disposing factors.[61] The disease is more prevalent in developing countries and is attributed to their low standards of hygiene and poor socioeconomic status.[62] Reduction in the overall resistance of the individual following general debility and delay in treatment play a vital role in severity of the disease.[55]

The *sine qua non* of necrotizing fasciitis is fascial necrosis and widespread undermining of skin with sparing of muscles by the initial necrotizing process.[2] Little has been added to Meleney's original description of clinical features of the disease.[5] Whatever be the initiating cause, necrotizing fasciitis begins as an exquisitely tender, erythematous, hot and swollen area of extensive cellulitis, atypical in that it does not respond to the standard regimen of antibiotics, heat application and raising of the affected part.[56] Lymphadenitis and lymphangitis are rare, as lymphatic obstruction occurs early in the necrotic process and lymphatic channels are rapidly destroyed.[2,57] Stone noted small skin ulcers that drained thin, reddish-brown, foul swelling fluid (usually referred as dish-water pus) as the cause for referral to surgical service. Surrounding these draining wounds were variable amounts of skin necrosis, though rarely was superficial gangrene very extensive.[58]

Classically, progression of disease is said to be rapid with skin changing from red and purple to pathognomic blue-grey ill-defined patches as early as 36 h after onset, but occasionally after 3 to 5 days.[2,3] By fourth or fifth day, frank cutaneous gangrene develops in patients with skin sloughing resulting from thrombosis of nutrient vessels. Bullae filled with clear, thick, pink, and deep purple fluid may develop, often giving the appearance of a partial or full thickness burn.[56,57] Although at first excruciatingly painful, affected areas become anesthetic secondary to cutaneous nerve destruction.[10,57,59] This may occur before the onset of gangrene, providing a clue that the process is indeed necrotizing fasciitis.[56] Subcutaneous fat and fascia become edematous, dull gray and necrotic with serosanguinous exudations and extensive underming of skin.[3,21] Skin death is subsequent to subcutaneous necrosis. Far advanced instances reveal muscles and tendons "standing out almost as an anatomic dissection."[2] Pink, viable muscle can be seen beneath grossly gangrenous fascia, fat and necrotic skin.[3] [Figures 1 and 2]

The reason for muscle sparing with primary fascial involvement is unknown.[2] However, muscle or bone can be involved if the original penetrating injury exposes these structures, or if the fascial envelope surrounding a muscle is entirely involved by the necrotizing process.[3,13] Diagnosis is confirmed in some patients when a probe can be passed laterally along the fascial cleft in an open wound.[1]

Local tissue involvement is accompanied by severe systemic manifestations. Patients are extremely toxic, and have an indifference to surroundings.[2] Severe

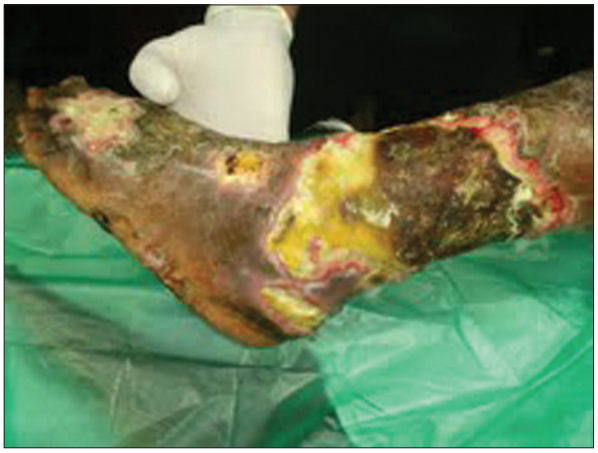

Figure 1: Necrotising fasciitis involving the leg

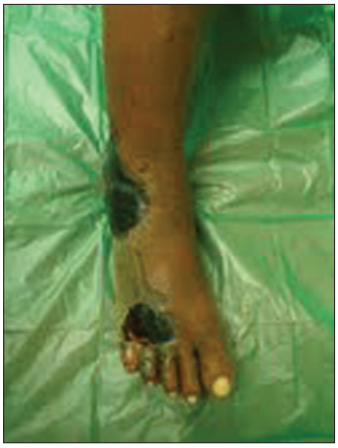

Figure 2: Necrotising fasciitis appearing as patchy necrosis

dehydration and hypoalbuminemia occur secondary to massive edema. Associated septic shock, respiratory failure have been described.[60] Protean manifestations are not invariably present and may be masked by steroid intake.[2] Associations with either systemic disease, such as diabetes mellitus or by a local condition such as peripheral vascular disease have been noted.[8]

MICROBIOLOGY

In the first major series of patients with necrotizing fasciities in 1924, Meleney with careful bacteriological studies disclosed a beta hemolytic streptococcus cultured from each of his patients and named the disease "acute hemolytic streptococcal gangrene."[5] It is said Meleney's studies were limited by the bacteriologic techniques of his time. Some groups of bacteria may not have been cultured and were incompletely identified.[3]

Accurate isolation and culture techniques for the most fastidious anaerobes have been developed only recently.[2] Few investigators have been able to present complete bacteriologic data on necrotizing fasciitis until the study by Giuliano.[3] Aerobic and anaerobic cultures and gram stained smears were made immediately of the fluid aspirated and the tissue debrided from the center of the lesion. No single microbe pathognomic for necrotizing fasciitis was found in his series.

Giuliano proposed two clinically indistinguishable but bacteriologically distinct groups.[3] Type 1 — comprising a majority of the patients, isolate anaerobic bacteria including *Bacterioides* with facultative anaerobic bacteria, *Enterobacteriaceae* and non-group A streptococci. Anaerobes were never cultured exclusively. Type 2 — is less common and isolate Group A *Streptococcus* alone or in combination with bacteria other than anaerobes or *Enterobacteriaceae*. In his series, *Streptococci* were the single most predominant bacteria, and non-group A streptococci with *B. fragilis* was the most common combination isolated. Recently, an association with MRSA organisms have been reviewed and found to have a worse prognosis.[4]

Single organism reports include certain patients exposed even briefly to marine environment. These patients developed fulminating infections caused by halophilic bacteria of the genus *Vibrio*, especially *V. vulnificus*.[61] Cases of fulminating mucormycotic necrotizing infection were identified in the survivors of the Colombian volcanic cataclysm.[31] The fungus isolated here was *Rhizopus arrhizus*.

MANAGEMENT

Meleney recognized the importance of early surgical intervention. He said "Surgery should not be delayed an hour after the diagnosis has been made."[17] He had also written that "complications are minimized by the wide exposure of the originally infected tissues and the outpouring of the organisms..."[5] This is true even today when the cornerstone of treatment is considered to be early diagnosis and aggressive treatment. Delay in diagnosis and treatment is associated with a greater morbidity and mortality.[62] If necrotizing fasciitis is suspected, an incision should be made to the level of the fascia and a probe passed along the fascial plane. If undermining of the skin occurs, immediate operation is indicated.[57,59] However, we believe that it is better to concentrate on resuscitation and control of infection and take up desloughing after clear cut demarcation of the dead tissue is recognizable, which is generally in the form of a blackish discoloration associated with lack of sensation. This often results in less tissue loss as well as less bleeding [Figures 3 and 4].

PRE-OPERATIVE PREPARATION

Surgical exploration is undertaken after adequate resuscitation. Penicillin along with, an aminoglycoside and clindamycin or metronidazole is needed to be administered preoperatively in moribund patients. Cefoxitin or imipenem is used in more limited infections.[4]

SURGERY

It is the author's experience that a fair amount of desloughing can be done without anesthesia as the tissues are devoid of sensation. However, extensive desloughing involving healthy margins must be done under adequate anesthesia, regional, or general. Blood loss must be anticipated and provision made for the same.

Incisions are made through the discolored skin down to the fascia, parallel to the cutaneous nerves and blood vessels. A thin, foul, murky fluid with blackened necrotic fascia

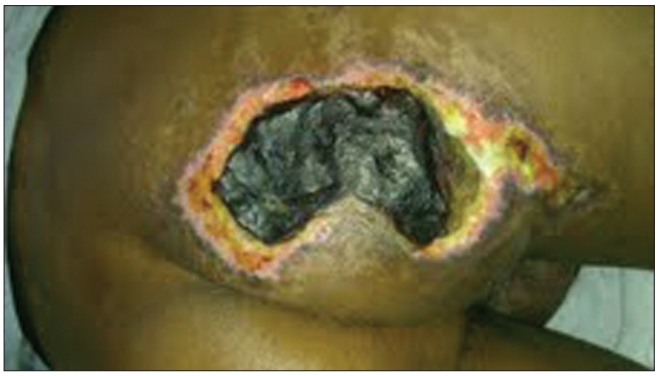

Figure 3: Necrotising fasciitis gluteal region

is found in patients with a mixed facultative anaerobic infection.[1] The process extends into the overlying fat as patchy greenish-black liquefaction necrosis. All such tissues are excised primarily, and specimen sent for culture studies immediately after the procedure and not later. Total debridement of all necrotic material must be performed until the skin and subcutaneous tissue can no longer be separated from deep fascia.[9,60,63] If there is circumferential necrosis in an extremity, the fascial planes should be bluntly dissected with hand.[64] Patino states that in such cases aggressive debridement may signify leaving the extremity totally nude of subcutaneous fat and skin.[16] Majeski emphasizes the need to preserve all viable tissues including nerves, muscles, subcutaneous tissue, skin, and blood vessels.[62]

Debridement into the tissues below the deep fascia is not indicated, except when external injury has penetrated this layer. Amputation is rarely indicated because of the superficial involvement.[64] However, if the causative operation is from the bone amputation is unavoidable. When burrowing areas of fat necrosis are absent (*i.e.* streptococcal gangrene), every attempt is made to preserve skin flaps since tissue cellulitis resolves rapidly after surgery, leaving viable skin.[65]

A diverting colostomy reduces wound soiling in patients with open perineal wounds.[8,51] Ahrenholz advocates a lower quadrant abdominal counter incision with extraperitoneal dissection into pelvis permitting through and through drainage for infection spreading upward into the periectal space.[1]

POSTOPERATIVE WOUND CARE

Various topical agents have been used. Chlorinated soda dressing was being used in 1984.[57] Silver nitrate

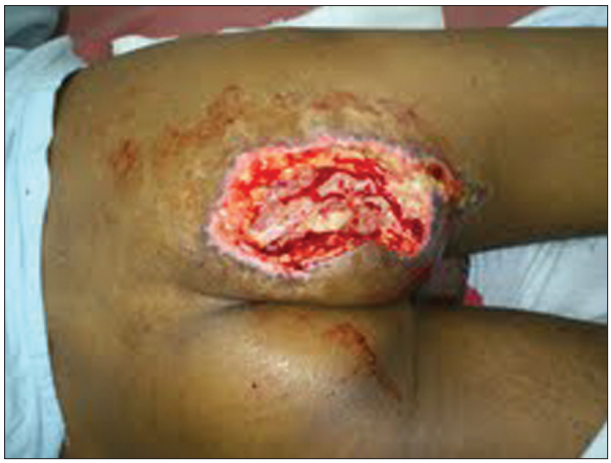

Figure 4: Necrotising fasciitis gluteal region after desloughing

dressings were noted to arrest the disease process in one study.[66] Other agents include: Topical antibiotics, Dakin's solution and hydrogen peroxide.[3] Studies using mainly dressings of saline solution with frequent wound inspection have also been beneficial.[8,63] Recent studies report use of providone-iodine solution dipped gauze dressings.[1] Kaiser and Cerra report unsatisfactory results with either early application of porcine xenografts or burn wound topical antimicrobials, unlike Baxter.[63,65] Frequent dressing changes are performed until granulation tissue forms. Toxic patients and those truncal and perirectal infections require daily exploration under general anesthesia.[1] Pooling of secretions in the wound predisposes to the growth of Pseudomonas and other opportunistic organisms. Eventually, remaining skin flaps adhere to underlying fascia.

ADJUNCTS TO THERAPY

All patients require intensive enteral or parenteral nutritional support.[62,63] Pessa and Howard used adjunctive hyperbaric oxygen in selected patients. However, they felt there was no evidence of an improved clinical course. Janeviscus in his collective review has noted other adjuncts to therapy in literature — like the use of heparin, high dose steroids, topical honey, and regional antibiotic perfusion.[2]

COVER OF RAW AREA

Patients with extensive skin loss require meshed split thickness skin grafts [Figure 5]. These contract and draw the remaining undamaged skin together, leaving a reduced soft tissue defect.[1] Marlex mesh has been used as a temporary cover of abdominal wall lesions.[67] Temporary biologic

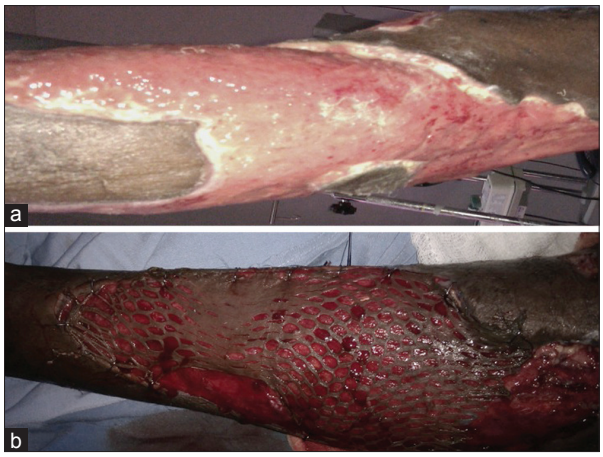

Figure 5: (a) Necrotising fasciitis leg, after initial debridement. (b) after application of meshed split skin graft

wound closure in debilitated patients may be provided with cadaveric grafts. If bone, tendons, nerves or blood vessels are exposed, even rotation or microvascular flaps may attempt after infection is checked.[1] Primary closure with simple suturing can be done when raw area is small or when skin around can be mobilized, as in scrotum.[68]

Incorporating all the above steps, Sudarsky has advocated an unified plan consisting of vigorous preoperative resuscitation, broad spectrum antibiotics including penicillin, aggressive surgical debridement with mandatory re-exploration at 24 h and thereafter as needed, parenteral or enteral nutritional support and early soft tissue coverage.[67]

MORTALITY

High mortality rates have changed little since the 1920s.[69] Young non-diabetic patients with extremity infections have the best prognosis. [3] In one series, patients over 50 years had a mortality of 57 percent while it was 4 percent for under 50 years.[21] Truncal and perineal involvement, diabetes and delay in diagnosis have been known to decrease the survival rate.[21] Failure to control the infection at the first operation can be highly lethal.[70] Overall mortality ranges from 9 to 64 percent in Jeneviscus's collective review.[2] In our own series we noticed that the mortality was related more to the associated medical illness than the wound itself.

Death is usually due to sepsis, respiratory failure, renal failure, or multi-organ failure.[60] Lesions of extremities have better prognosis than trunk and head.[57] Obesity, malnutrition and systemic illness have also been high risk factors.[2,66]

REFERENCES

1. Ahrenholz DH, Necrotising soft-tissue infections. Surg Clin North Am 1988;68:199-214.
2. Janeviscus RV, Hann S, Butt MD. Necrotising fasciitis. Surg Gynecol Obstet 1982;154:97-102.
3. Giuliano A, Lewis F Jr, Hadley K, Blaisdell FW. Bacteriology of necrotising faciitis. Am J Surg 1977;134:52-7.
4. Changchien CH, Chen YY, Chen SW, Chen WL, Tsay JG, Chu C. Retrospective study of necrotizing fasciitis and characterization of its associated Methicillin- resistant Staphylococcus aureus in Taiwan: BMC Infect Dis 2011;11:297.
5. Meleney FL. Hemolytic streptococcus gangrene, Arch Surg 1924;9:317-64.
6. Berg A, Armitage JO, Burns CP. Fournier's gangrene complicating aggressive therapy for hematologic malignancy. Cancer 1986;57:2291-4.
7. Ray P, Gupta HN, Roy M. In Susrutha Samhita (A scientific synopsis), New Delhi: Indian National Science Academy; 1980. p. 99.
8. Pessa ME, Howard RJ. Necrotising fasciitis. Surg Gynecol Obstet 1985;161:357-61.
9. Wilson B. Necrotising fasciitis. Am Surg 1952;18:416-31.
10. Hammar H, Wanger L. Erysipelas and necrotising fasciitis. Br J Dermatol 1977;96:409-19.
11. Ledingham IM, Tehrani MA. Diagnosis, clinical course and treatment of acute dermal gangrene. Br J Surg 1975;62:364-72.
12. Collins RN, Nadel MS. Gangrene due to the hemolytic streptococcus: A rare but treatable disease. N Engl J Med 1965;272:578-80.
13. Meleney FL. Hemolytic streptococcus gangrene: Importance of early diagnosis and early operation. JAMA 1929;92:2009-12.
14. Webb HE, Hoover NW, Nichols DR, Weed LA. Streptococcal gangrene. Arch Surg 1962;85:969-73.
15. McCafferty EL, Lyons C, Suppurative fasciitis as the essential featureof hemolytic streptococcus gangrene. Surgery 1948;24:438-42.
16. Patino JF, Castro D. Necrotising lesions of soft tissues A review. World J Surg 1991;15:235-9.
17. Meleney FL. A differential diagnosis between certain types of infectious gangrene of the skin — with particular reference to hemolytic streptococcus gangrene and bacterial synergistic gangrene. Surg Gynecol Obstet 1933;56:847-67.
18. Pardanani DS, Punekar SV, Usgaokar RP, Galwankar DN, Pardiwala BB. A study of non-clostridial anaerobic infection in surgical sepsis. Indian J Surg 1977;39:666-9.
19. Gorbach SL, Bartlett JG. Anaerobic infections. N Engl J Med 1974;290:1237-45, 1289-94.
20. Lancerotto L, Tocco I, Salmaso R, Vindigni V, Bassetto F. Necrotising fasciitis: Classification, diagnosis, and management. J Trauma Acute Care Surg 2012;72:560-6.
21. Rea WJ, Wyrick WJ. Necrotising fasciitis. Ann Surg 1970;172:957-64.
22. Goldberg GN, Hansen RC, Lynch DJ. Necrotising fasciitis in infancy: Report of three cases and review of the literature. Pediatr Dermatol 1984;2:55-63.
23. Jamal N, Teach SJ. Necrotising fasciitis. Pediatr Emerg Care 2011;27:1195-9.
24. Gibboney W, Lemons JA. Necrotising fasciitis of the scalp in neonates. Am J Perinatal 1986;3:58-60.

25. Lally KP, Atkinson JB, Woolley MM, Mahour GH. Necrotising fasciitis. A serious sequela of omphalitis in the newborn. Ann Surg 1984;199:101-3.
26. Kosloske AM, Cushing AH, Borden TA, Woodside JR, Klein MD, Kulasinghe HP, *et al*. Cellulitis and necrotising fasciitis of the abdominal wall in pediatric patients. J Pediatr Surg 1981;16:246-51.
27. Collette CJ, Southerland D, Corrall CJ. Necrotising fasciitis associated with Haemophilus influenzae type b. Am J Dis Child 1987;141:1146-8.
28. Falcone PA, Pricolo VE, Edstorm LE. Necrotising fasciitis as a complication of chicken pox. Clin Pediatr 1988;27:339-43.
29. Adeyokunnu AA. Fournier's syndrome in infants. A review of cases from Ibadan, Nigeria. Clin Pediatr 1983;22:101-3.
30. Rajan S. Skin and soft-tissue infections: Classifying and treating a spectrum. Cleve Clin J Med 2012;79:57-66.
31. Patino JF, C astro D, Valencia A, Morales P. Necrotising soft tissue lesions after a volcanic cataclysm. World J Surg 1991;15:240-7.
32. Svensson LG, Brookstone AJ, Wellsted M. Necrotising fasciitis in contused areas. J Trauma 1985;25:260-2.
33. Jacobson JM, Hirschmann SZ. Necrotising fasciitis complicating intravenous drug abuse. Arch Intern Med 1982;142:634-5.
34. Orangio GR, Pitlick SD, Della Latta P, Mandel LJ, Marino C, Guarneri JJ, *et al*. Soft tissue infections in parenteral drug abusers. Ann Surg 1984;199:97-100.
35. Oh C, Ginsterg-Fellner F, Dolger H. Factitial panniculitis and necrotising fasciitis in juvenile diabetes. Diabetes 1975;24:856-8.
36. Sotrel G, Hirsch E, Edelin KC. Necrotising fasciitis following diagnostic laparoscopy. Obstet Gynecol 1983;62(Suppl):67-69s.
37. Sultan HY, Boyle AA, Sheppard N. Necrotizing fasciitis. BMJ 2012;345:e4274.
38. Korula J, Rice HE. Necrotising fasciitis and percutaneous endoscopic gastrostomy (letter). Gastrointest Endosc 1987;33:335-6.
39. Welch NT, E llis DJ, Bradby GV. Necrotising fasciitis — a complication of " successful" endoscopic sphincterotomy (letter). Gastrointest Endosc 1990;36:425-6.
40. Pingleton SK, Jeter J. Necrotising fasciitis as a complication of tube thoracostomy. Chest 1983;83:925-6.
41. Van de Sadt J, Roemans P, Thys JP, Kahn RJ. Synergistic necrotising cellulitis after pneumonectomy. Intensive Care Med 1985;11:158-9.
42. Casali RE, Tucker WE, Petrino RA, Westbrook KC, Read RC. Postoperative necrotising fasciitis of the abdominal wall. Am J Surg 1980;140:787-90.
43. Galbut DL, Gerber DL, Belgraier AH. Spontaneous necrotizing fasciitis: Occurrence secondary to occult diverticulitis. JAMA 1977;238:2302.
44. Conn IG, Martin DL, La Ferla G. Necrotising fasciitis secondary to a strangulated femoral hernia containing a perforated sigmoid diverticulum. J R Coll Surg Edinb 1964;31:191-2.
45. Sriram K. Necrotising fasciitis following feeding jejunostomy. JPEN J Parenter Enteral Nutr 1988;12:322.
46. Van Ammers PM, Moore PJ, Sacho H. Necrothising fasciitis after caesarean section — association with non-steroidal anti-inflammatory drugs. South Afr Med J 1991;80:203-4.
47. Lloyd RE. Necrotising fasciitis of the orbit. Br J Oral Maxillofac Surg 1987;25:314-32.
48. McAndrew PG, Davies SJ, Griffiths RW. Necrotising fasciitis caused by dental infection. Br J Oral Maxillofac Surg 1987;25:314-22.
49. Wenig BL, Shikowitz MJ, Abramson AL. Necrotising fasciitis as a lethal complication of peritionsillar abscess. Laryngosc 1984;94:1576-9.
50. Crowson WN. Fatal necrotising fasciitis developing after tooth extraction. Am Surg 1973;39:525-7.
51. Adinolfi MF, Voros DC, Moustoukas NM, Hardin WD, Nichols RL. Severe systemic sepsis resulting from neglected perineal infections. South Med J 1983;76:746-9.
52. Spinark JP, Resnick MI, Hampel N. Fournier's gangrene; A report of 20 patents. J Urol 1984;131:289-91.
53. Chatterjee P: Filariasis. In: Basu AK, editor. Rob C, Smith R's Clinical Surgery (8), Tropical Surgery. London: Butterworths; 1965. p. 56-7.
54. Dandapat DC, Patra SK, Rao JM. Fournier's gangrene of the scrotum. Indian J Surg 1985;47:283-5.
55. Bhandari M, Acharekar KL, Kalla VK. Urinary extravasation and gangrene of genitalia. Indian J Surg 1983;45:129-34.
56. Vishwanath G, Basarkod SI, Katageri GM, Mirji P, Mallapur AS. Necrotising fasciitis of the breast with shock and postpartum psychosis. J Clin Diagn Res 2011;5:1117-9.
57. Buchanan CS, Haserick JR. Necrotising fasciitis due to group Ab-hemolytic streptococci. Arch Derm 1970;101:664-8.
58. Yamasaki O, Nagao Y, Sugiyama N, Otsuka M, Iwatsuki K. Surgical management of axillary necrotizing fasciitis: A case report. J Dematol 2012;39:309-11.
59. Wilson HD, Haltalin KC. Acute necrotising fasciitis in childhood. Am J Dis Child 1973;125:591-5.
60. Tehrani MA, Ledingham IM. Necrotising fasciiitis. Postgrad Med J 1977;53:237-42.
61. Howard RJ, Pessa ME, Brennaman BH, Ramphal R. Necrotising soft-tissue infections caused by marine vibrios. Surgery 1985;98:126-30.
62. Majeski JA, Alexander JW. Early diagnosis, nutritional support and immediate extensive debridement improve survival in necrotising fasciitis. Am J Surg 1983;145:784-7.
63. Kaiser RE, Cerra FB. Progressive necrotising surgical infections – a unified approach. J Trauma 1981;21:349-55.
64. Miller JD. The importance of early diagnosis and surgical treatment of necrotising fasciitis. Surg Gynecol Obstet 1983;157:197-200.
65. Baxter CR. Surgical management of soft tissue infections. Surg Clin North Am 1972;52:1483-98.
66. Hirsch JH. The value of silver nitrate in controlling necrotising fasciitis. Arch Surg 1974;109:708-11.
67. Sudarsky LA, Laschinger JC, Coppa GF, Spencer FC. Improved results from a standardized approach in treating patients with necrotizing fasciitis. Ann Surg 1987;206:661-5.
68. Ciftci H, Verit A, Oncel H, Altunkol A, Savas M, Yeni E, *et al*. Amputation of the penis and bilateral orchiectomy due to extensive debridement for fournier's gangrene: Case report and review of the literature. J Pak Med Assoc 2012;62:280-2.
69. Freeman HP, Oluwole SF, Ganepola GA, Dy E. Necrotising fasciitis. Am J Surg 1981;142:377-83.
70. Hussein QA, Anaya DA. Necrotizing Soft Tissue Infections. Crit Care Clin. 2013 Oct;29(4):795-806.

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