

Necrotising Fasciitis-An Old Enemy or a New Foe

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Abstract

Necrotising fasciitis when described by Meleney was caused predominantly by Streptococcus pyogenes. Since then, there were several reports which confirmed streptococcus as the main organism identified in this disease entity. However, recently there were reports of necrotising fasciitis caused by organisms other than streptococcus.

We analysed 19 cases of necrotising fasciitis seen in the Department of Orthopaedic Surgery over a period of 24 months to see if this disease entity has changed significantly. The patients in our series were between the ages of 19 to 85 years with an average age of 57.2 years. Males were more often affected (16 out of 19). The majority of the patients have some form of underlying disease (16 out of 19 cases). There was a trend towards polymicrobial infection and many were resistant to standard antimicrobial therapy. The mortality rate was 21.1%. Our results are comparable to many earlier series.

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Introduction

Necrotising fasciitis is a soft tissue infection characterised by rapidly spreading inflammation and subsequent necrosis of the muscle fascia, subcutaneous fat, and in some cases, the epidermis. Necrosis is limited, at least initially, by the muscle fascia with sparing of the muscle. The disease is usually accompanied by systemic toxicity and may be fatal.

In 1883, this condition was first described by Alfred J Fournier in a specific body part as idiopathic scrotal gangrene, better known as Fournier's gangrene.¹ It was only in 1924 that Meleney first described the clinical picture of this potentially deadly disease, which he called "acute hemolytic streptococcal gangrene".² The name "necrotising fasciitis" that is widely used currently was introduced by Wilson in 1952.³

Over the years, numerous studies and reports on this condition were published which shed more light on its aetiology, microbiology, pathology and management. However, despite our better understanding of this condition, it still carries a high mortality rate (27% to 43%).⁴⁻⁷ In 1994, a cluster of cases of necrotising fasciitis in the United Kingdom caught the attention of the media and terms like "flesh-eating disease" and "killer bug" were coined.

Apart from the report by the local media and some scanty publications, the extent of the problem of necrotising fasciitis and the disease pattern in our population is under reported. Hence, in this paper, we set out to review the disease pattern in our hospital and attempt to compare with other reported series.

Patients and Methods

A total of nineteen patients who were treated in the Department of Orthopaedic Surgery, Tan Tock Seng Hospital for necrotising fasciitis from December 1993 to November 1995 were retrospectively reviewed. Twelve patients were admitted directly through the Emergency Department while the remaining seven were referred from other departments of the hospital.

All patients underwent aggressive surgical debridement except one lady who refused surgery. The patient's age and sex, pre-existing medical conditions and presenting symptoms were analysed. We also paid special attention in detecting the aetiological cause in each case and any delay in surgical treatment was recorded and the reasons identified. Our data also included the laboratory findings, bacteriology, histology, treatment method and outcome of treatment.

The patients were followed up regularly in our outpa-

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tient clinic after discharge from the ward. The duration of follow-up ranged from 3 months to 18 months. The assessment of the result of treatment was based on the outcome of wound healing.

Results

There was a male preponderance in our series with 16 male and 3 female patients. Their average age was 57.2 years (range 19 to 85 years). Sixteen patients were found to have underlying medical conditions, of which, 8 were diabetics (42.1%). Other common medical conditions were hypertension (4/19), ischaemic heart disease (3/19) and chronic obstructive lung disease (3/19).

Like many other series, most of our patients did not have any definite aetiological cause for the necrotising fasciitis. Only 5 patients (26.3%) gave history of some trivial injuries leading to the infection, and interestingly, two patients had injury from a crab bite.

As our study only included patients treated by the Orthopaedic Department, most patients had infection involving the extremities. The lower limbs were affected in 16 patients (84.2%) compared to two in the upper limbs (10.5%). Only one female patient had necrotising fasciitis of the perineum.

The commonest presenting symptoms were severe pain (15/19), swelling (17/19) and fever (12/19) (Table I). The duration of symptoms ranged from 1 to 12 days (average of 4.3 days). The time from presentation to surgery ranged from few hours to 6 days, with an average of 2.5 days. The reasons for the delay in surgery included delay in diagnosis (2/19); delay in referral by other departments (4/19); and time required to optimise the patient for surgery (2/19).

Blood glucose level was found to be raised in all 8 diabetic patients, but only mildly elevated or normal in the 11 non-diabetic patients. White cell count was raised in all patients ranging from 16 900 to 33 200 cells per ml (average 23 800). Ten patients had coagulation studies (prothrombin time and partial thromboplastin time) done preoperatively with 5 cases (50%) showing derangement.

Bacteriological studies were carried out with aerobic and anaerobic culture of the tissue taken during surgery. In the case of the lady who refused surgery, blood culture was taken. Most patients had polymicrobial infection (63%), i.e. more than one organism were isolated from the tissue or blood taken for culture. The common organisms were β -haemolytic streptococcus, *Staphylococcus aureus* and *Pseudomonas* (Table II).

Only 10 patients were diagnosed with certainty to have necrotising fasciitis preoperatively. These patients had florid signs of skin necrosis, extensive swelling and blistering. The remaining 9 patients were diagnosed at the time of surgical debridement based on findings of

TABLE I: CLINICAL PRESENTATION

Swelling	17	(89.5%)
Pain	15	(78.9%)
Fever	12	(63.2%)
Ulcer	3	(15.8%)
Discharge	3	(15.8%)
Septic shock	3	(15.8%)

TABLE II: BACTERIOLOGY

Organism isolated	Number of patients
B-haemolytic streptococcus	7 (3gpA, 3gpB, 1gpG)
<i>Staphylococcus aureus</i>	6
<i>Pseudomonas</i>	4
<i>Proteus mirabilis</i>	3
<i>Klebsiella</i>	3
<i>Enterococcus</i>	3
<i>Acinetobacter</i>	2
<i>Escherichia coli</i>	1
<i>Corynebacterium</i>	1

necrosis of the subcutaneous tissue and underlying fascia and muscle. Eight patients had tissue sent for histology which confirmed the positive diagnosis in all 8 cases. The diagnostic features included inflammation and necrosis of the epidermis, dermis, subcutaneous tissue and collagen bundle of the fascia.

All except one patient underwent one or more debridements. Subsequent treatment included split skin graft in 6 patients, above-knee amputations in 4 patients, below-knee amputation in 1 patient and free flap in 1 patient.

There were 4 deaths in our series (21.1%) of which 2 resulted from septicaemia and 2 from acute myocardial infarction. All these 4 patients had underlying medical disease including ischaemic heart disease and diabetes mellitus. The first patient was a lady with necrotising fasciitis of the perineum presenting with septicaemic, shock. Blood culture taken showed group B streptococcal infection. She refused all forms of surgical treatment and succumbed to sepsis after spending six days in the intensive care unit. The second patient was a man with liver cirrhosis and presented with necrotising fasciitis of the left leg and had above-knee amputation done. However, postoperatively, he continued to have swinging fever with necrosis of the stump and died 4 days after the surgery from sepsis and liver failure. The third patient was an elderly man admitted to the Cardiology Department for angina. He was noted to have necrotising fasciitis of his right leg and underwent one surgical debridement. Postoperatively, he had myocardial infarction and died three days after surgery. The last patient was another elderly man with history of myocardial infarct and diabetes mellitus who underwent surgical debridement for necrotising fasciitis of his

left leg. He passed away on the first postoperative day following another myocardial infarction.

Diabetic patients were found to have higher amputation rate (4 out of 8) compared with non-diabetic patients (1 out of 11). This is probably due to the fact that the existence of diabetic peripheral vascular disease impaired wound healing. Excluding those with limb amputation, 9 out of the remaining 10 patients had good wound healing. Only one patient required re-admission for further debridement. The average hospital stay of the patients was 27.6 days (range 6 to 86 days).

Discussion

Since the original description of necrotising fasciitis by Meleney in 1924, many changes have taken place, including the discovery of penicillin. Despite better understanding of this condition and more aggressive surgical treatment, the fatality rate of necrotising fasciitis in most series was no better than that reported by Meleney in his series of 20 patients from Peking with only 4 deaths.²

Following the series presented by Meleney, there were many subsequent reported series of necrotising fasciitis citing streptococcus as the causative organism.⁸⁻¹² Many of the patients presented with fulminant infection and systemic toxicity and they had a high mortality rate despite aggressive therapy. Chelsom et al⁹ reported 13 cases of necrotising fasciitis due to group A β -haemolytic streptococci in Norway. In their series, 8 patients had signs of shock, 8 had impaired renal function and there were 3 deaths among the 13 patients. Many other series reported that a variety of different organisms were isolated in necrotising fasciitis including *staphylococcus*, *proteus*, *enterococcus* and *acinetobacter*.^{4,5,13,14} In the series reported by Ward and Walsh,⁵ haemolytic streptococcus was only isolated in one out of thirteen patients. Our series is similar to the latter group.

In our study, β -haemolytic streptococcus was only isolated in 7 out of 19 patients (37%). The majority of our patients had polymicrobial infection (12 out of 19), of which, many were resistant to the standard antibiotic therapy (eg. penicillin and cloxacillin). Similarly, Wang and Shih¹⁴ reported polymicrobial infections in 16 out of the 18 patients in their series. In another study by Chin et al,⁴ β -haemolytic streptococcus was implicated only in 6 out of 15 cases. There are a few reasons that could account for the differing bacteriology pattern of our series as compared with that presented by Meleney in 1924. Firstly, some of our patients were initially treated with penicillin group of antibiotics before culture were taken. This might have eradicated or suppressed the penicillin-sensitive streptococcus leaving the resistant organisms to emerge. Secondly, 4 of our patients developed necrotising fasciitis during their stay in the hospital for other medical conditions. The causative organ-

isms isolated were those commonly implicated in hospital-acquired infection such as methicillin-resistant *staphylococcus aureus*, *pseudomonas* and *acinetobacter*. Thirdly, the inclusion criteria of the cases in our study were based on clinical and histological findings as opposed to bacteriology in some series.^{8,10} Finally, the difference may be attributed to the unique population studied, clinical settings and practices in each reported series.

Besides 4 patients who presented with septic shock, the remaining patients had subacute type of presentation with duration of symptoms up to 12 days. In the early stages, clinical signs of necrotising fasciitis were minimal and the skin may look deceptively normal. Many were diagnosed to have cellulitis and some of our cases were admitted to other departments and treated initially with antibiotics. Hence, diagnosis at this stage requires a high index of suspicion, in particular when patient complains of pain disproportionate to the clinical signs of cellulitis, which may be labelled as "atypical cellulitis". We found that a high total white cell count and deranged coagulation results may help in alerting us to the possibility of underlying necrotising fasciitis. With progression of disease, the signs are more characteristic with the presence of increasing swelling, duskiness, skin blistering, necrosis and subcutaneous crepitus with systemic toxicity. With our observation in this series, we concurred with the point made by Burge and Watson¹⁵ that necrotising fasciitis may simply lie towards one end of a spectrum of soft tissue infections ranging from simple cellulitis to myositis requiring amputation.

The key to the success in managing necrotising fasciitis is early diagnosis and aggressive surgical intervention. Most patients require repeated surgery where the necrotic tissues, including fascia and underlying muscle in some cases, are extensively debrided. Ward and Walsh⁵ showed that delayed diagnosis and inadequate surgery are the most important factors contributing to the mortality of necrotising fasciitis. Subsequent management of the resultant defect created can include skin grafting, secondary suture or some form of myocutaneous flap. Small defect can be left to granulate and heal by second intention. Appropriate antibiotics based on sensitivity study as well as metabolic support are equally important in the total management of the patient.

Conclusion

Necrotising fasciitis that we know for the past century has changed little in its clinical entity, although the bacteriology has changed. There were many reports of polymicrobial infection and non-streptococcal infection as the causative organisms. Our report has similar findings. We are also more aware of its varied presentations, bacteriology and clinical course. We realised that it is not

that uncommon as we used to think and can mimic simple cellulitis with atypical symptoms as shown in our series. To achieve favourable outcome, a high index of suspicion is required to diagnose the condition early. Radical debridement remains the key to success in treatment.

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