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CASE REPORT

Necrotising Soft-Tissue Infection of the Upper Limb: A Case Report and Review of Current Management

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ABSTRACT

Necrotising soft-tissue infection is a life-threatening infection that requires a high index of suspicion for diagnosis. Treatment of this condition is an urgent and aggressive surgical debridement of all necrotic tissues within 12hours of presentation, high-dose broad-spectrum antimicrobial therapy and critical care support. It is a surgical emergency and requires a multidisciplinary team coordination between the surgeons, critical care and microbiology specialists. This approach has now been shown to reduce by 50% the high mortality of this condition. We present a rare case of upper limb Necrotising soft-tissue infection and review the current management of this surgical emergency.

Key words: Necrotising soft-tissue infection; Necrotising fasciitis; upper limb infections; Group A Streptococcus

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INTRODUCTION

Necrotising soft-tissue infection (NSTI) is uncommon but life-threatening, and rapidly spreading infection, involving various layers of the skin, subcutaneous tissue, deeper fascia and muscle. Delay in diagnosis and/or treatment increases the rates of morbidity and mortality. It is a surgical emergency and requires early surgical debridement within 12 hours of presentation.

It has been described as early as the 5th century BC by Hippocrates as a rapidly expanding infection with extensive necrosis and liquefaction of all soft tissues, associated with high mortality[1,2]. The modern term of "necrotising fasciitis" was first used by Wilson (1952) to describe this rapidly progressing fascial necrosis[3]. The current terminology of this condition is "Necrotising soft-tissue infection" (NSTI)[4]. We present a rare case of upper limb NSTI and review the current diagnosis and management of this surgical emergency.

CASE REPORT

A 57year-old man had noticed a small spot on the dorsal part of the right mid forearm, just below his tattoo, 48 hours prior to presentation and a picture was taken with his phone (Figure 1). The wound had grown rapidly in size and he presented with a very painful, swollen and infected wound on the posterolateral aspect of the right forearm. There was no history of insect bites or injury. He felt well in himself with no fever. Associated comorbidities included 27 years of intravenous heroin addiction which was terminated 2 years prior, and smoking with mild chronic obstructive pulmonary disease.

Examination showed slight tachycardia (pulse 107) and a respiratory rate of 16. Blood pressure was within normal limits (133/78). There was a 12 x 8 cm wound over the dorsal aspect of right mid-distal forearm, with necrotic centre, surrounded by erythema and swelling up to the elbow proximally and the wrist distally (area marked) (Figure 2). Radiographs were normal. Initial bloods tests showed a C reactive protein (CRP) of 113, white cell count (WCC) of 13. The clinical impression was that of a rapidly spreading NSTI and the patient was taken to theatre for emergency debridement within 3 hours from presentation. Wound swab and blood cultures were taken Intravenous fluids and high-dose broad spectrum antibiotics were given (clindamycin, piperacillin/tazobactam, gentamicin and teicoplanin) following discussion with the microbiologist.

Surgery involved debridement of all necrotic tissues down to the deep fascia and an area of 2 cm swollen tissue circumferentially up to healthy bleeding tissues (Figure 3). The wound was washed with 6L of saline solution and a vacuum dressing was applied. The wound was clean when re-explored 24h later and no further debridement was necessary. The patient was then referred to plastic surgeons for skin grafting. The patient made an uneventful recovery. His vitals improved within 12 hours with a heart rate of 79 and a respiratory rate of 11. Blood test results showed a CRP of 74, and a WCC of 11. Four days later the CRP decreased to 7 and the WCC to 6. Microbiology results showed heavy growth of β -haemolytic Group A Streptococcus (GAS).

DISCUSSION

NSTI is a rare surgical emergency with an incidence of 0.4 and 0.5 cases per 100,000 population and 500 cases per year in the UK[5-7]. It mainly affects the extremities but can occur in any part of the body. Risk factors include: intravenous drug use, alcohol dependence, diabetes mellitus, over 50 years of age, hypertension, and obesity[7,8].

Figure 1 Photograph taken by the patient 48 hours prior to admission.

Causal organisms of NSTI

(1) May include the following pathogens: Gram positive bacteria: (Mainly β haemolytic Group A streptococci (GAS), and less often Group B streptococci, Enterococci, Coagulase negative staphylococci, Staphylococcus aureus, Bacillus species).**Gram negative bacteria:** (Escherichia coli, Pseudomonas aeruginosa, Proteus species, Serratia species). **Anaerobic bacteria:** (Bacteroides species, Clostridium species, Pepto-streptococcus species). **Fungi:** (Zygomycetes, Aspergillus, Candida). **Other:** Aeromonas hydrophila, Vibrio species.

Figure 2 Photograph taken 48 hours later during hospital presentation.

Figure 3 Photograph following surgical debridement.

(2) Classification of NSTI.

Type 1: Is a polymicrobial infection mostly in immunocompromised individuals and includes anaerobes, Gram-negative and Gram-positive organisms. It is the most common type (70%) and occurs in the trunk and perineum. Affects patients with several comorbidities, (intravenous drug use, diabetes mellitus, cardiovascular)[7,9]. **Type 2:** Is mainly due to Group A streptococcus (GAS) with or without Staphylococcus aureus. Typically occurs in the limbs, with a history of usually minor injury. (20-30%)[7,9,10].

Less common types: Type 3: Is a monomicrobial necrotising fasciitis, which can be caused by water exposure with Aeromonas hydrophila, or Vibrio vulnificus, and is more common in Asia. **Type 4:** Caused by fungal pathogens particularly in immunocompromised patients[9].

The predominant organism in Type 2 necrotising fasciitis is GAS, which is common in UK based studies. The human pathogen Streptococcus pyogenes or group A Streptococcus (GAS), accounts for over 600 million infections globally per year with a high level of morbidity and mortality. New evidence suggests that invasive GAS (iGAS) is becoming more virulent. Recent epidemiological evidence points to a rapid emergence of the GAS genotype emm 89 as a leading cause of disease in the United Kingdom and other parts of the world. The new emergent clade variant was unexpectedly genetically acapsular, and exhibited enhanced production of the toxins[11].

Pathophysiology

Bacteria can invade soft tissues mainly through a break in the skin barrier or more rarely through haematogenous spread. Bacteria proliferate and release toxins, causing local tissue damage with subsequent inflammatory responses. Infection extends rapidly along fascial planes beyond the area of overlying cellulitis[12,13]. Some toxins produce vascular thrombosis and ischemic necrosis of all tissue layers. Toxins are responsible for systemic signs such as fever, hypotension, tachycardia, tachypnoea and altered mental status. Toxins may also activate T cells and macrophages resulting in a massive release of cytokines with an uncontrolled systemic inflammatory response that can lead to multi-organ failure and septic shock[13].

Diagnosis of NSTI.

It is a clinical diagnosis and NSTI requires a high index of suspicion. The early presentation is that of swelling, with severe pain and cellulitis. However, a rapidly progressive soft-tissue infection should always be suspected as a necrotising infection. There is severe pain disproportionate to the visible skin changes, and the oedema extends beyond the erythema area. Often there is an area of hypoesthesia over the site of infection. Necrotic skin, blisters, and systemic signs of infection (fever, tachycardia, hypotension and increased respiratory rate) may be late signs. Lymphangitis and lymphadenopathy are rare due to thrombosis of the vessels. The clinical picture may worsen very quickly within a few hours. Necrotising fasciitis is primarily a clinical diagnosis. However, investigations can support the diagnosis if this is unclear. Laboratory findings include rising inflammatory markers, WCC, and elevated serum creatinine kinase levels. Magnetic resonance imaging and Ultrasound scan have high accuracy rates but may be difficult to perform under emergency conditions[14,15].

The 'finger test' is a surgical method that can be performed under local anaesthesia at the bedside for the diagnosis of necrotising fasciitis. It involves making a 2 cm incision down to the deep fascia. Findings that suggest necrotising fasciitis following incision include: minimal resistance to finger dissection (a 'positive' finger test), absence of bleeding and pus, presence of necrotic tissue and murky or greyish 'dishwater' fluid[16,17].

Severity assessment of NSTI

(1) Laboratory Risk Indicator for Necrotising infection (LRINEC) score

Wong et al (2004) proposed the use of the Laboratory Risk Indicator for Necrotising infection (LRINEC) in order to predict the presence of NSTI[18]. It scores six independent variables: serum C-reactive protein level, white blood cell count, haemoglobin level, serum sodium level, serum creatinine level, and serum glucose level. A systematic review by Bechar et al (2017) concluded that the LRINEC score was useful in the diagnosis of necrotizing fasciitis[19]. However, a more recent meta-analysis of 5982 cases by Fernando et al (2018) showed that the LRINEC score had a low sensitivity of 68.2% and specificity of 84.8%, and should not be used to rule-out NSTI[20].

(2) Quick Sepsis-related Organ Failure Assessment score. (qSOFA score)

The third international consensus definitions for sepsis and septic shock (Sepsis-3) by Singer et al (2016) suggested that in hospital settings, adult patients with suspected infection can be rapidly identified as being more likely to have poor outcomes typical of sepsis if they have at least 2 of the following clinical criteria: respiratory rate of 22/min or greater, altered Glasgow coma scale, or systolic blood pressure of 100 mm Hg or less. These criteria constitute a new bedside clinical score termed the quick Sepsis-related Organ Failure Assessment (qSOFA) score. An increase in the of 2 points or more, is associated with an in-hospital mortality greater than 10%[21].

Mortality rate variation of NSTI

The progression of the infection is often rapid, and prognosis depends on quick diagnosis and appropriate treatment. The presenting signs of NSTI can be quite similar to those of other common soft tissue infections, such as cellulitis, making early diagnosis challenging. A high index of suspicion for NSTI is crucial to early recognition and intervention without delay. A recent review showed that 71.4% of the NSTIs are misdiagnosed on initial evaluation[22]. Delay in diagnosis and surgical debridement lead to an increased mortality rate.

Nawijn et al (2020) comparing mortality rates before and after 2000 in the Netherlands, reported a significant reduction in mortality from 28.3% to 20.6%. However, average mortality rates reported remained constant around 20% over the past 20 years[23].

Mortality rates for NSTI are considered high, and vary between 14% and 33% depending on the country of the study. Germany 32.8%[24], France 23.7%[25], Netherlands 21%[23], USA 14%[26]. Neilly et al (2019) reported the UK experience with an NSTI mortality of 17% and an amputation rate of 14%. The amputation cohort had a 50% mortality[7].

The mortality rates also vary according to surgical timing. Gelbard et al (2018) pooled the results from six studies and found that surgery within 12 h compared to surgery after 12 h from presentation had reduced mortality rates 13% vs 26% respectively[27].

A recent systematic review and meta-analysis of 109 studies by Nawijn et al (2020) showed a similar reduction in mortality if the initial surgery is performed within 12 h after presentation (19% vs. 34%) and demonstrated that early surgical debridement, lowers the mortality rate for NSTI by almost 50%[23].

Multidisciplinary team Managenent

A multidisciplinary team (MDT) is mandatory for the management of

this condition and requires coordination between the surgeons, critical care and microbiology specialists. The gold standard for treating NSTI is an early diagnosis, urgent surgical debridement of necrotic tissues, antimicrobial therapy, and haemodynamic support. The bacteria causing NSTI can spread rapidly along the fascial planes. The goal of the initial surgical procedure is therefore to prevent further spreading of the infection by aggressive debridement of all the infected and necrotic tissues until healthy viable tissue is seen. [17] In patients with a high suspicion for necrotizing infection, surgical debridement within the first 12 h after admission, is of paramount importance. Surgical debridement should be repeated as necessary every 12-24 h, until the patient has no necrotic tissue remaining[26].

Adjunctive antibiotic therapy and supportive care is crucial. Intravenous empirical antibiotics start after having obtained blood cultures. Until microbial identification and sensitivities are known, high-dose broad-spectrum antibiotics should be administered that cover the most common aetiologies for NSTI, according to local antimicrobial guidelines. In our hospital that includes: clindamycin, piperacillin/tazobactam, gentamicin and teicoplanin. Once culture results are available, therapy is tailored accordingly[17,28].

CONCLUSION

Necrotising fasciitis is a life-threatening soft-tissue infection that requires a high index of suspicion for diagnosis. The gold standard management of NSTI includes an early diagnosis, urgent and aggressive surgical debridement of necrotic tissues within 12hours after presentation, high-dose broad-spectrum antimicrobial therapy and critical care support. NSTI is a life-threatening surgical emergency and requires a multidisciplinary team coordination between the surgeons, critical care and microbiology specialists. This approach has now been shown to reduce by 50% the high mortality of this condition.

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