Contents lists available at ScienceDirect

Asian Pacific Journal of Tropical Biomedicine

journal homepage:www.elsevier.com/locate/apjtb

Document heading doi:10.1016/S2221-1691(12)60052-5

Severe monobacterial necrotizing soft tissue infection by group A *Streptococcus*: A surgical emergency

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ARTICLE INFO

Article history:
Received 6 August 2011
Received in revised form 5 September 2011
Accepted 28 September 2011
Available online 28 March 2012

Keywords:
Necrotizing soft tissue infection
Group A *Streptococci*
Fulminant skin necrosis

ABSTRACT

Eight percent of necrotizing soft tissue infections (NSTI) are attributable to group A *Streptococci* (GAS), and among these, 50% develop streptococcal toxic shock syndrome. The reported mortality associated with NSTI reaches 32%. We present cases of two healthy individuals with minor GAS skin infection which developed to a rapidly progressed NSTI and sepsis despite of the antibiotic treatment, aiming to discuss the lessons learned from the course and management of these patients.

1. Introduction

Necrotizing soft tissue infection (NSTI) is a broad diagnostic term used to describe a spectrum of extremely aggressive and potentially life and limb threatening bacterial soft tissue infections, characterized by extensive, rapidly progressive soft-tissue necrosis[1-4]. The disease is classified on the basis of the principal soft-tissue layer involved with necrosis *i.e.* cellulitis, fasciitis or myositis[5].

Streptococcus pyogenes, one kind of group A *Streptococci* (GAS), is a gram-positive, very common human pathogen which may cause severe invasive disease in approximately 2.79 per 100 000 population. Eight percent of these patients are diagnosed with GAS-NSTI, and among these, 50% develop streptococcal toxic shock syndrome (STSS)[6]. The reported mortality associated with NSTI reaches as high as 25%-32%[6,7].

We present two recent cases of fulminant GAS-NSTI aiming to discuss the lessons learned from the course and management of these patients.

2. Case report

Both cases presented within a month and had no

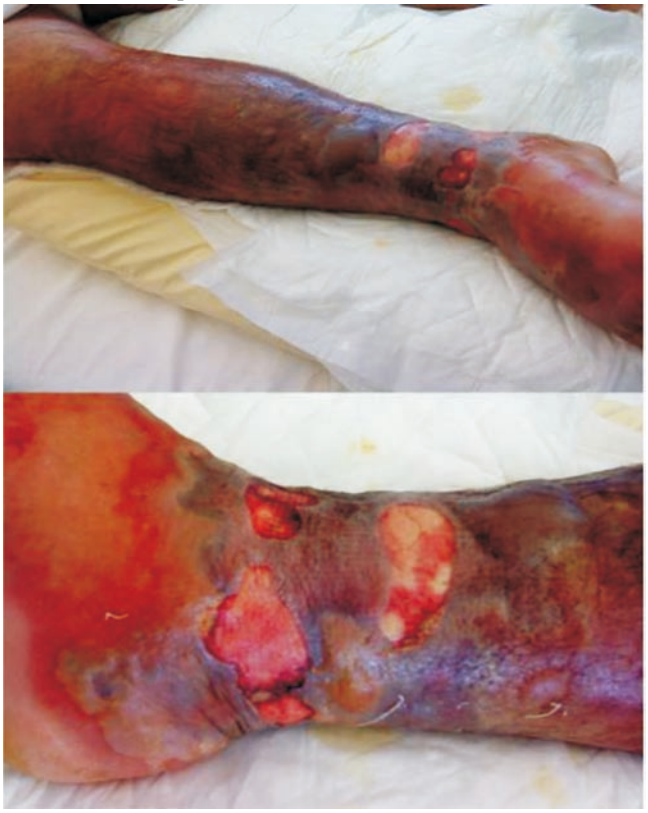

Figure 1. Intraoperative picture of case 1 showing areas of skin necrosis, purulent discharge and cellulitis.

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significant past medical history. On presentation they were febrile (-39.5 ℃), tachycardic and normotensive. Both patients had raised inflammatory markers (WBC, CRP) and ESR but otherwise normal biochemistry. Doppler studies demonstrated normal blood flow on the involved limb while there was no evidence of subcutaneous gas on imaging.

Details about the presentation and management of the patients were included in Table 1.

Table 1
Presentation, management and outcomes of the two cases.

Parameters	Case 1 (Figure 1)	Case 2 (Figure 2)
Sex	Male	Male
Age	23 years old	56 years old
Original side	Small skin lesion on left arm	Right foot
Presentation	24 h history of high T° and arm inflammation	48 h history of an exquisitely painful and swollen foot
Initial management	Fluid resuscitation, tetanus anti-toxin, wound swabs for culture	
Antibiotics	Amoxicillin/clavulanic acid: 1 g/12 h; clindamycin: 300 mg/8 h	Clindamycin: 600 mg/8 h; linezolide: 600 mg/12 h; piperacillin/ tazobactam: 4.45 mg/6 h
Outcome with medical treatment during the first 48 h	Progression of the local infection and deterioration of the general condition with spiking temperature and sepsis, hemorrhagic and non hemorrhagic bullae, progression to severe necrotizing fasciitis	Grossly oedematous and inflamed right limb extending from the foot to the mid anterior thigh, large infected bullous lesion on ankle, rapid progression of the infection, deterioration of general condition and renal function, signs of sepsis
Surgery	Extensive debridement of the necrotic skin and fascial plane through irrigation and draining of a deep abscess extending from the arm dorsally to the mid-forearm	Aggressive debridement of the full thick skin necrosis involving most of the leg, drainage of the abscess and irrigation of the wound
Postoperative antibiotics	Clindamycin: 900 mg/8 h; linezolide: 600 mg/12 h; piperacillin/tazobactam: 4.5 mg/6 h	Same treatment
Outcome	Wound with healthy red granular base, arm fully functional, discharged on postoperative day 12	Complete resolution of the inflammation on postoperative day 20 when wound demonstrated a healthy red granular base. The leg function completely recovered.

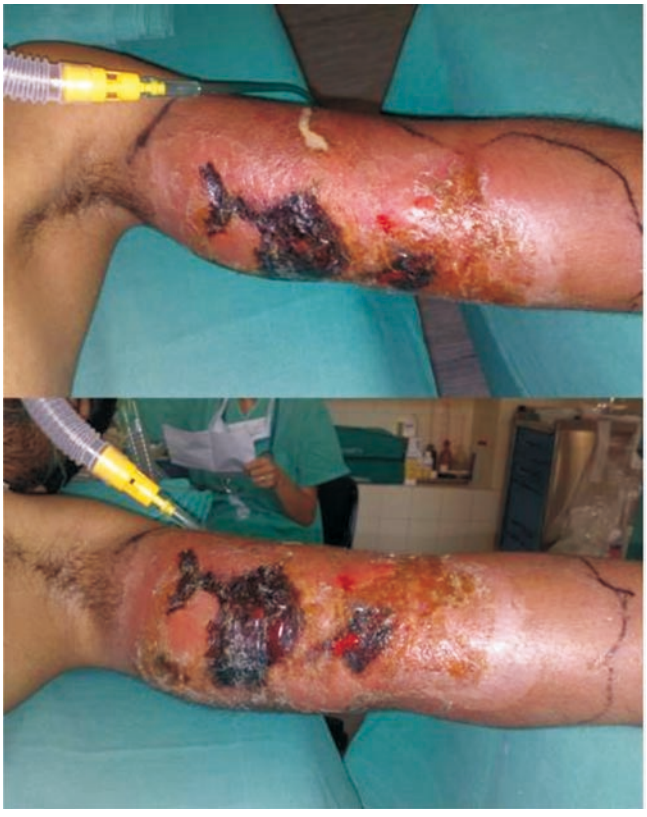

Figure 2. Intraoperative picture of case 2 showing inflammation of the grossly oedematous right limb extended from the foot to the mid anterior thigh and a large infected bullous lesion on the ipsilateral ankle.

3. Results

In both patients the early surgical intervention and the extensive debridement of all necrotic tissue along with the abscess drainage controlled the fulminant course of the disease and in combination with the broad spectrum antibiotics reversed the systemic signs. The motility of the extremities was preserved and there was a minimum need for reconstruction.

4. Discussion

NSTI is usually polymicrobial (type I) in 55% to 75% of the cases[8]. On the other hand, type II NSTI is usually monobacterial, caused by GAS and may lead to STSS[8,9]. NSTI occurs predominantly in immunocompromised patients[10-12]; however, both of our cases were otherwise fit and healthy individuals suffered type II NSTI caused by GAS which progressed rapidly from a superficial minor skin infection to NSTI and sepsis refractory to antibiotic therapy.

The recommended medical treatment for severe GAS disease includes a combination of a β-lactam and clindamycin since no resistance has yet been reported

against these drugs[13], although local conditions and tissue ischemia, prevent adequate delivery of antibiotics in the area, and prevent bacterial destruction *via* aerobic mechanisms[9], thus surgical debridement is imperative[14].

Both of our patients received early in their course an appropriate combination of antibiotics which nevertheless, did not delay or confine the extent of the infection. The fast deterioration of our patients' general condition was a major determining factor for a prompt surgical debridement.

Today, initial empiric therapy is recommended to include vancomycin, linezolid, daptomycin, or quinupristin/dalfopristin and it should continue until no further surgical debridement is required and the patient is free from systemic symptoms[8].

Waiting for the classic crepitus or skin necrosis for the establishment of the diagnosis of NSTI may delay patient's management since the skin might have a normal appearance until advanced stages of the disease[15], despite the extensive subcuticular course. A large review of 198 patients found the nonspecific signs of inflammation such as pain, swelling, and erythema were the most common presenting symptoms, while the specific signs of NSTI such as crepitus and blistering, were found in less than 40% of patients at the time of admission[7].

In our cases the progression from stage 1 to stage 3 based on Wang *et al* description was extremely fast indicating an aggressive form of the disease[16]. Surgical intervention and exploration remains the most sensitive diagnostic tool and the cornerstone of therapy[2,3,8], especially in highly aggressive forms of the disease. It is recommended to proceed with extensive resections of all necrotic skin up to a point where healthy, bleeding tissue is present regardless of the extent of debridement required[8].

In conclusion, monobacterial (type II) NSTI may present in healthy individuals, progress rapidly and have a highly aggressive course thus rendering imaging modalities, broad spectrum antibiotics and other conservative measures useless for both the diagnosis and the treatment.

A high index of suspicion along with a low threshold for surgical intervention and early debridement is necessary in order to prevent compromise of the general condition and minimize the tissue and function loss. The appropriate timing of surgical intervention is not yet determined but ideally surgical debridement should be performed as early as possible if there are signs of local progression or systemic deterioration of the patient despite the appropriate empiric antibiotic therapy.

Conflict of interest statement

We declare that we have no conflict of interest.

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