

Necrotizing Fasciitis

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Summary

A 31-year-old Pakistani man was admitted to hospital after sustaining a Grade I compound fracture of the mid-shaft of the left tibia and fibula following a motor vehicle accident. He developed septicaemic shock, acute renal failure and Group A streptococcal necrotizing fasciitis of the left leg. The patient underwent an above knee amputation followed by disarticulation of the left hip with extensive debridement. He was treated with benzylpenicillin, vancomycin, inotropes and continuous haemodialysis and survived without further sequelae. Subsequently, skin grafting was done over the wound site. This case highlights the role of Group A streptococcus as a cause of this rare and life-threatening infection.

Key Words: Necrotizing fasciitis, Group A Streptococcus

Introduction

Group A streptococcal necrotizing fasciitis is a rapidly progressing condition involving subcutaneous tissue and fascia. It is characterized by vascular thrombosis and cutaneous gangrene. Often, it is accompanied by severe systemic toxicity, seen as haemorrhagic bullae as part of toxic-epidermal necrolysis, septic toxic shock and progressive multiple, organ failure. It is therefore important that medical and laboratory staff be aware of this infection and its life-threatening consequences.

Case Report

A 31-year-old man was admitted to the Kuala Lumpur Hospital after he sustained a Grade I compound fracture of the mid-shaft of the left tibia and fibula following a motor-vehicle accident. The initial operative finding was a laceration measuring 0.5cm in diameter with a piece of bone fragment measuring 1.5 x 1.5 x 0.5cm underneath the wound. Debridement was done and a full length plaster of Paris (POP) applied post-operatively. He was not given any antibiotic at that time. On the 5th day of admission, the patient suddenly developed septicaemic shock with symptoms of shortness of breath, cyanosis, hypotension and tachycardia. His condition worsened and he was

transferred to the Intensive Care Unit (ICU) where he was paralysed and mechanically ventilated. Upon removal of the POP, the left lower limb was found to have haemorrhagic blisters with cutaneous gangrene and cellulitis extending up to the inguinal region. On the same day, fasciotomy, debridement and an above knee amputation (AKA) was done. There was extensive necrosis mainly involving the subcutaneous tissue, fascia, overlying skin, and some of the extensor and flexor muscles of both upper and lower left leg. The post-operative diagnosis was necrotizing fasciitis of the left lower leg. The patient was given intravenous cefoperazone 1gm 12-hourly and metronidazole 400mg 8-hourly before the operation and post-operatively. While still under intensive care, patient was noted to have oliguria and persistent hypotension despite the administration of fresh frozen plasma and crystalloids.

Inotropes were started with adrenaline 5ug/min and dopamine 3ug/kg/min. His condition worsened and both adrenaline and dopamine dosages were increased. He was noted to have acute renal failure and disseminated intravascular coagulopathy (DIVC). Continuous haemodialysis on alternate days and treatment for DIVC was commenced immediately.

Group A beta-haemolytic *Streptococcus* was isolated from the culture of tissue collected from the amputation site. This organism produced large, mucoid, round and dome-shaped colonies, surrounded by a thin zone of beta-haemolysis. The strain was sensitive to the bacitracin identification disc. Grouping by the coagglutination test (Streptex) confirmed it as group A *Streptococcus*. The organism was sensitive to penicillin, cefuroxime, tetracycline and erythromycin. Blood cultures were negative. Anti-streptolysin 'O' titre was more than 200 IU/ml initially and a repeat sample a month later was less than 200 IU/ml.

On the third post-operative day, the patient was commenced on benzylpenicillin 2 mega units 4-hourly based on the microbiology report of the tissue culture. However, the patient continued to have spiking temperature and unstable blood pressure. Intravenous vancomycin 500mg 6-hourly was added and benzylpenicillin increased to 4 mega unit 4-hourly. Disarticulation of the left hip joint with tissue debridement was done on the sixth day of the above knee amputation.

The patient was managed in the intensive care unit for 16 days. Following that, he was stable and remained afebrile. Split skin grafting was carried out with 95% uptake. This was to be followed by a skin flap later. He was referred to physiotherapy for rehabilitation.

Discussion

Necrotizing fasciitis is a relatively rare, severe infection characterized by necrosis of the fascia and subcutaneous tissue. It usually follows vascular thrombosis and cutaneous gangrene and is accompanied by severe systemic toxicity and progressive and multiple organ failure. Necrotizing fasciitis has to be differentiated from clostridial fasciitis, haemolytic streptococcal gangrene and anaerobic non-clostridium fasciitis. The usual cause of necrotizing fasciitis is a mixture of aerobic and anaerobic organisms but group A *Streptococcus* alone can be responsible for it. The organism reaches the subcutaneous tissue by extension from a contiguous infection or through trauma to the area, including surgery.

Early diagnosis of septic shock and necrotizing fasciitis

is important for management and better outcome of the illness. However, necrotizing fasciitis is a rare condition and therefore diagnosis may be delayed, as happened in this case. The opening of a window in the full length plaster of Paris for wound inspection may have facilitated an early diagnosis.

Lack of awareness of this condition amongst medical and laboratory staff may have also resulted in delay in starting the treatment. Benzylpenicillin was commenced only after five days of diagnosis of necrotizing fasciitis.

Necrotizing fasciitis can appear in individuals without obvious risk factors. However, a study done in 1993 identified various risk factors such as diabetes mellitus, intravenous drug abuse, age greater than 50, hypertension, malnutrition or obesity¹.

Group A streptococcal necrotizing fasciitis was first described in Beijing by Meleney, F.C. in 1924². The incidence and severity of streptococcal infections decreased in the later half of the 20th century but over the past decade, a resurgence of severe group A streptococcal infection has been reported from Europe and the USA. This includes increased identification of the organisms in blood cultures and cases of necrotizing fasciitis.

In the past the mortality rate in cases of necrotizing fasciitis was up to 40%. However, it has been reduced with surgical debridement or amputation to remove the necrotic tissue, combined with early use of benzylpenicillin.

Mucoid strains of *S. pyogenes* belonging to M-types 1 and 3 are commonly associated with severe and invasive infections. These strains also produce streptococcal pyogenic exotoxin (SPE-A, B and C) which are now known to act as superantigens. These toxins can induce T cells to synthesize tumour necrosis factor (TNF-alpha) and interleukin 1 and 6, resulting in excessive activation of cytokines, complement and clotting cascades together with the production of oxygen-free radicals and nitric oxide which cause shock and multi-organ failure.

Benzylpenicillin is the drug of choice for group A

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streptococcal necrotizing fasciitis. Aggressive and extensive surgical debridement or amputation to remove the necrotic tissue are also indicated. Vancomycin has been successfully used in some cases of severe and life-threatening streptococcal infection in which penicillin treatment had failed³. In this particular case, no antibiotics was given even though he sustained a grade I compound fracture. Whether this would have prevented the necrotizing fasciitis is debatable. Nevertheless, the antibiotic regimens currently recommended by Ministry of Health for this purpose would have covered for group A Streptococcus.

Intravenous immunoglobulin may also have a role in the management of such cases. It can reverse the hyperproliferation of T cells, neutralise superantigens and regulate the production of tumour necrosis factor³.

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A Report of the First Three Cases of Diffuse Panbronchiolitis in Malaysia

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Summary

Three cases of diffuse panbronchiolitis (DPB) occurring in two Malaysian Chinese patients and one Malay patient are reported. They had similar clinical, radiological and physiological features which are characteristic of DPB. The diagnosis in one of the cases was confirmed histologically by transbronchial lung biopsy. These could be the first three cases identified in Malaysia.

Key Words: Diffuse panbronchiolitis, Malaysians