Case Report

Necrotizing fasciitis complicated by multiple pneumoceles

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ABSTRACT

This paper reports a 14-year-old male patient who developed necrotizing fasciitis of the leg after a minor trauma. He was admitted to our intensive care unit with septic shock and acute lung injury. The clinical course was complicated by bilateral multiple pneumoceles resulting in bilateral pneumothoraces. The causative organism was found to be *Staphylococcus aureus*. This young patient survived and was discharged home in fair general condition.

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We present a case of necrotizing fasciitis after simple trauma to the leg. It was complicated by toxic shock syndrome, pneumoceles and a tension pneumothorax. The main infective organism was found to be *Staphylococcus aureus (S.aureus)*. *Staphylococcus aureus* species are significant human pathogens, these organisms are aerobic and facultative anaerobic gram-positive, coagulase-positive cocci. They are the most common cause of skin infection particularly impetigo in children.[1] The organism causes a wide spectrum of clinical diseases. However, necrotizing fasciitis caused by Staphylococcus alone is rare. Its evolution into necrotizing pneumonia with multiple pneumoceles with tension pneumothorax has not been, as far as we know, previously reported.

Case Report. A 14-year-old male patient sustained simple trauma to the left foot and ankle while playing football 3 days prior to presentation. Painful swelling of the left ankle and lower leg followed the trauma. He was initially treated with non-steroidal anti-inflammatory drugs and bandaging. The condition did not improve and swelling increased to involve mid leg. He was seen at the Accident and Emergency Department on the third day after trauma. He was then complaining of painful swelling of the leg, chest pain and shortness of breath. He was resuscitated and the initial working diagnosis was pulmonary embolism secondary to deep vein thrombosis (DVT). An urgent spiral computed tomography (CT) scan of the chest showed bilateral diffuse parenchymal infiltrates suggestive of acute lung injury (ALI). Doppler ultrasound of the left leg did not show any evidence of DVT, but only soft tissue edema. Hematological parameters revealed: white blood cells 21.10^3, hemoglobin 10.3 g/dL, platelets 481.10^3, while biochemical analysis showed BUN 13.7 mmol/L (3-9 mmol/L), S Creatinine 70 mmol/L (53-124 mmol/L). The patient was admitted to the medical intensive care unit (ICU) and received anticoagulants and antibiotics (cloxacillin and ceftriaxone).

At this stage, the patient's condition was deteriorating. He was tachycardic, tachypneic and distressed. He was consequently electively intubated and ventilated. Vital signs were as follows: pulse 150 beats/min, blood pressure 96/42 mm Hg and temperature 38°C. There were bilateral scattered crepitations at auscultation, while the abdomen showed no abnormality. The left lower limb was swollen with redness from 10 cm below the knee down to the

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dorsum of the foot raising the suspicion of necrotizing fasciitis. Surgical consultation was requested. An immediate exploration of the left leg was performed and a frozen section of the fascia and subcutaneous tissue confirmed the diagnosis of necrotizing fasciitis. An extensive debridement through 2 bilateral long incisions was carried out. All devitalized subcutaneous fat, fascia and muscles were excised and sent to the laboratory for gram stain, culture/sensitivity and histopathology. Post-operatively, the patient was admitted to the surgical intensive care unit (SICU) where ventilation was maintained. A Swan-Ganz catheter was also inserted. The following parameters were recorded: heart rate 160/min.,blood pressure 70/30 mm Hg, temperature 38.5°C, central venous pressure 9mm Hg, PAP 40/30 mm Hg, PAWP 18 mmHg. At that time, laboratory results showed an important leucocytosis ($34.10^3/mm^3$), severe anemia (hemoglobin 7.69 g/dL) and thrombocytopenia ($82.10^3/mm^3$). Arterial blood gasses, on FiO_2 1, showed mixed metabolic and respiratory acidosis. (pH 7.18, PaO_2 60 mm Hg, $PaO_2$189 mm Hg).

At this stage, a diagnosis of toxic shock syndrome (TSS) was made and aggressive resuscitation took place with fluids, packed red blood cells, fresh frozen plasma and platelets. Antibiotics were changed to tazocin and clindamycin. Noradrenaline, dopamine and frusemide infusions were used to support the cardiovascular system and maintain urine output. The initial gram stain showed gram-positive cocci in clusters. Blood and necrotic tissue cultures demonstrated *S.aureus*. Antibiotherapy was changed again to cloxacillin following culture and sensitivity results. Later on, sputum culture also showed growth of *S.aureus*.

To control the infection, 2 more debridements **(Figures 1 & 2)** were required after which the patient showed gradual improvement. On the forth SICU admission day, his oxygen saturation dropped suddenly and he had an episode of severe bradycardia. Examination of the chest revealed diminished air entry on both sides, more on the right. A tension pneumothorax was diagnosed requiring urgent needle pleural decompression. Bilateral chest tubes were then rapidly inserted and oxygen saturation improved. A repeat CT of the chest next morning showed multiple pneumoceles on both sides. Inotropes were gradually reduced from the sixth day but the patient remained intubated and ventilated as the chest condition was still critical. Weaning from the ventilator started on day 16th allowing extubation the next day. In ICU, feeding was achieved with total parenteral nutrition for 12 days, followed by enteral feeding and then normal food intake. The final histopathology report confirmed necrotizing fasciitis and necrotizing myositis. The left chest tube was removed and the right clamped but the clamp had to be released as of a significant pneumothorax. After 28 days of SICU management,

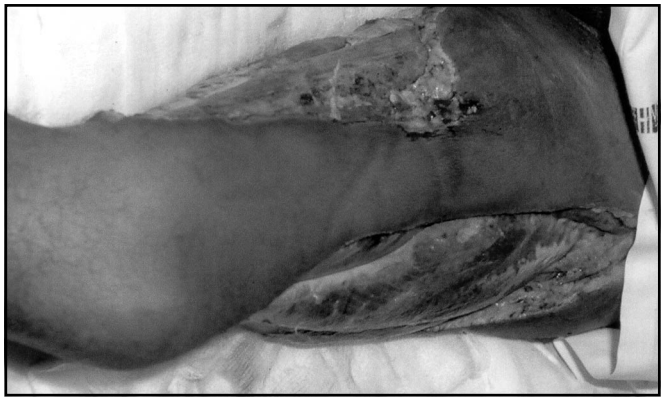

Figure 1 - Debridement of the thigh.

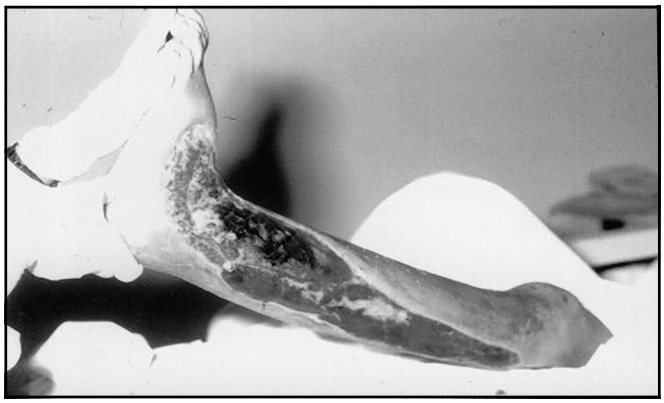

Figure 2 - Debridement of the leg.

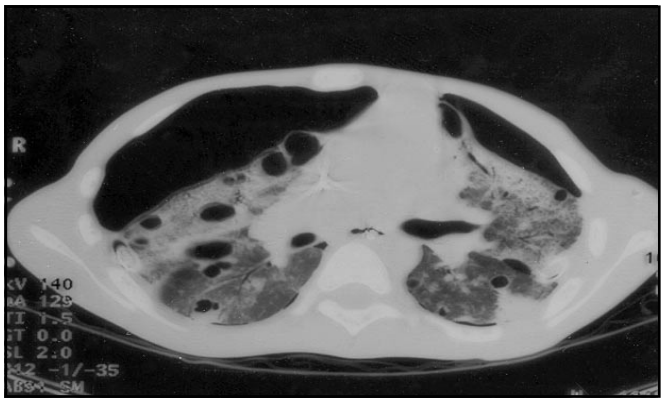

Figure 3 - Computed tomography scan of the chest showing multiple pneumoceles and bilateral pneumothoraces.

the patient was transferred to a general surgical ward and underwent skin grafting to cover the healthy looking defect in the leg. He was discharged home in fair general condition and followed by the chest physicians and the surgeons as an outpatient.

Discussion. Necrotizing soft tissue infection is rapidly progressive. Caused by toxin-producing virulent bacteria, it was first described by Melency[2] as hemolytic streptococcal gangrene in 1924, later it was coined necrotizing fasciitis by Wilson.[3] In necrotizing fasciitis, group A hemolytic streptococci and less often *S.aureus*, alone or in synergism, are frequently the initiating infecting bacteria. However, other aerobic and anaerobic pathogens may be present.[4] Edema extending beyond the area of erythema, skin vesicles, crepitus or air in the subcutaneous tissues and absence of lymphangitis and lymphadenitis are markers of necrotizing infection.[5] These are rapidly fatal unless promptly recognized and aggressively treated.[6] If caused by Staphylococcus, one should have a high index of suspicion for potential pulmonary complications otherwise it can be lethal in hours. Unfortunately, this patient received a NSAID believed to be one of the complicating factors in the development of necrotizing fasciitis.[7] These drugs adversely affect granulocyte function and curtail the disease clinical manifestations allowing more time to pass before intensive treatment is initiated. Early diagnosis with the help of frozen section biopsy,[8] followed by extensive debridement are important factors for successful outcome.[9] Aggressive fluid resuscitation in an intensive care setting is also required as sequestration of large volumes occur following soft tissue edema.[10] In this case, a Swan-Ganz catheter was used to guide fluids administration. Early enteral nutrition seems also to be an important adjunct in patients with necrotizing fasciitis.[11]

The efficacy of hyperbaric oxygen therapy (HBO) for necrotizing soft tissue infections remains unproven and only potentially useful. Hyperbaric oxygen therapy increases the normal oxygen saturation in the infected wounds by a thousand fold, leading to a bactericidal effect, improved polymorphonuclear function, and enhanced wound healing. Hyperbaric oxygen therapy has been confirmed to be of consistent value only in the treatment of clostridial myonecrosis.[12] Some authors feel anyhow that HBO should be used routinely in the treatment of necrotizing fasciitis, as it significantly reduces mortality and wound morbidity.[13]

In this case, the clinical course was complicated by a tension pneumothorax rapidly diagnosed and treated by needle thoracostomy followed by chest tubes. The chest x-ray and CT scans showed multiple pneumoceles in both lungs (**Figure 3**). In the literature, fulminating *staphylococcal pneumonias* occurring during influenza epidemics have been reported to cause death in a few hours.[14] To the authors' knowledge, this is the first case report of a young, otherwise healthy patient with staphylococcal necrotizing fasciitis complicated by pneumoceles and septic shock with good outcome.

In conclusion, necrotizing soft tissue infections caused by staphylococcal species are deadly especially if complicated by a pulmonary involvement. A good knowledge of patterns of disease can aid in adjusting treatment to decrease morbidity and mortality. Early accurate diagnosis and surgical intervention consisting of wide debridement are essential for the survival of these very sick patients.

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