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Descending necrotizing mediastinitis: report of a case following steroid neck injection

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Abstract

Cervical necrotizing fasciitis is a rare, rapidly progressive, severe bacterial infection of the soft tissues of the neck. Uncommonly, it may descend into the mediastinum. We describe a case of descending necrotizing mediastinitis in a young man, where there was diagnostic confusion and delay, with an eventual fatal outcome. A steroid injection for neck pain is thought to be the source of infection. In this case, the signs of mediastinitis were initially masked, and the diagnosis delayed until cardiopulmonary arrest occurred. Early recognition with a low threshold for computed tomography (CT) scanning is essential. Aggressive multidisciplinary therapy with mediastinal drainage is mandatory.

Key words: Necrosis; Neck; Mediastinitis; Steroids

Introduction

Necrotizing fasciitis (NF) is a rare but well-known, rapidly fulminant polymicrobial infection of subcutaneous tissues. It is characterized by progressive destruction of fascia and adipose tissue, with sparing of the overlying skin and muscle in the initial stages. It most commonly occurs in the trunk, perineum and limbs after mild trauma, insect bites, falls, furuncles and surgery. Mortality ranges from 8.7 to 73 per cent.[1] NF is seen less often in the head and neck where an odontogenic infection is the most common aetiology.[1] Cervical necrotizing fasciitis (CNF) may rapidly spread into the thorax along fascial planes, and the associated diagnostic delay makes this descending necrotizing mediastinitis (DNM) the most lethal form of mediastinitis, with a mortality of approximately 40 per cent.[2]

NF was first described during the American Civil War by Joseph Jones in 1871, who reported a 46 per cent mortality in a series of 2642 cases of 'hospital gangrene'.[3] In 1918, Pfanner reported a case of streptococcal necrotizing erysipelas.[3] In 1924, Meleney reported 20 cases from Peking of streptococcal gangrene with subcutaneous necrosis.[3] Recent interest has been generated in the public domain with the lay press reporting deaths caused by the 'flesh eating bug'.[4] There are a variety of synonyms: hospital gangrene, streptococcal gangrene, gangrenous erysipelas, necrotizing erysipelas and Meleney's gangrene.

In the pre-antibiotic era, Pearse reported a mortality of greater than 50 per cent in 44 DNM patients in a series of 110 cases of mediastinitis.[5] Even in the antibiotic age the mortality remains relatively high, Corsten *et al.*'s, meta-analysis yielding a fatality rate of 31 per cent in 69 cases between 1970 and 1997.[5] NF may develop in patients in all ages and has no sex or race predilection.[4] CNF is relatively uncommon with Banerjee *et al.*'s review finding only 59 cases of CNF between 1945 and 1994 in the medical literature.[6] The peak age of CNF is in the fifth decade (mean 44.3, range 1–81), which is comparable to NF in other sites.[6] There is a male bias with 68 per cent of cases occurring in men.[6] Dental infection is the commonest origin (30–53 per cent) followed by trauma (28 per cent).[4,6] DNM is predominantly a disease of young men (average 38 years and 86 per cent male).[5] Dental infection (57 per cent) is the most common origin of infection in DNM followed by in order: retropharyngeal abscesses (14 per cent), peritonsillar abscesses (11 per cent), traumatic endotracheal intubation (seven per cent), trauma (five per cent), cervical lymphadenitis and clavicular osteomyelitis (seven per cent).[2] NF as a complication of steroid injection is an extreme rarity, with only a case following treatment for a painful shoulder reported in the literature.[7] We report a case of DNM, possibly secondary to a steroid injection for neck pain, in a young fit, but obese man, where there was diagnostic confusion and delay, with a fatal outcome.

Case report

A 32-year-old obese man was referred by his General Practitioner with a 10-day history of a sore throat and neck pain. He had previously been prescribed amoxycillin, which had been ineffectual. Two weeks prior to admission he had had a left-sided steroid injection to the C2/3 intervertebral disc as treatment for chronic neck pain. Otherwise he was fit and well. On examination, he looked comfortable, but his temperature was 39.5°C. He had marked trismus, with an area of left peritonsillar swelling associated with a slight palatal shift to the right. Examination of the neck demonstrated several tender, discrete masses in the left upper deep cervical chain of nodes. Aspiration of the peritonsillar swelling failed to yield any pus, and a diagnosis of left peritonsillar cellulitis with reactive lymphadenopathy was made. Initial investigations revealed a raised white cell count of 21.6×10^9/l. A lateral neck radiograph was normal. Intravenous benzylpenicillin was commenced, in association with dexamethasone and intravenous fluids.

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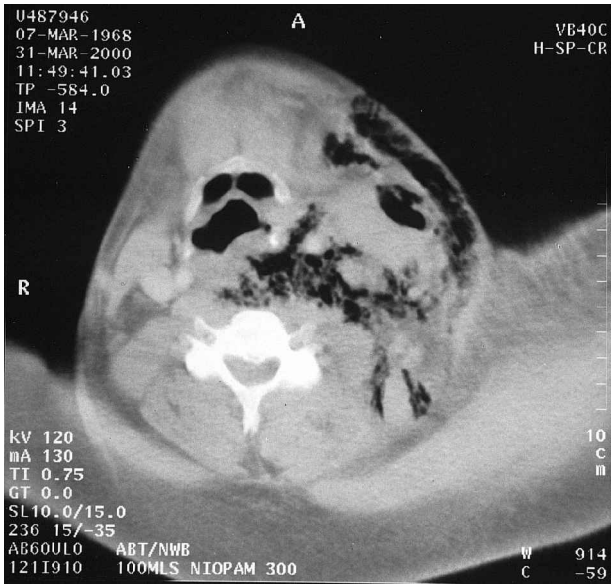

FIG. 1

An axial CT scan of neck demonstrating extensive surgical emphysema on the left side of the neck.

On review the following morning, his condition had improved. His temperature had settled and he was able to tolerate a soft diet. However, by that evening his neck swelling had increased in size. On examination the swellings were tender and fluctuant. Intravenous metronidazole was added. The following day, the neck swelling was unchanged, but he was apyrexial and relatively well. Fibre-optic laryngoscopy revealed a mild degree of medial displacement of the pharyngeal and hypopharyngeal wall on the left side but the airway was well preserved. He underwent a CT scan of his neck and chest. This was reported as follows: Neck: 'There is extensive what amounts to surgical emphysema in the deep and superficial soft tissues on the left side.

Some of the air is tracking up across the midline behind the pharynx. There is no soft tissue mass or collection identified, and I imagine there must be a perforation in the pharynx somewhere to have caused this. There is some shift of the airway to the right side' (Figure 1). Chest: 'Heart size normal. Lung clear.' In the absence of any identifiable pus on CT and with a background of an apyrexial patient it was concluded by two Consultant Otolaryngologists that the surgical emphysema was simply upper aerodigestive tract air, which did not warrant surgical intervention.

The following day, in the early hours of the fourth day, the patient developed dyspnoea and a feeling of tightness in his neck. His respiratory rate increased to approximately 25 breaths per minute and his oxygen saturation dropped to 87 per cent. Flexible nasendoscopy was unremarkable. He was given oxygen via facemask and his condition improved. A portable chest X-ray was unremarkable. There were no acute changes seen on electrocardiography. He was reviewed a few hours later, at which time his symptoms remained settled but he had by now developed back pain. His neck swelling was noted to have diminished in size. Orthopaedic review was sought regarding his back pain, but no acute orthopaedic problem was detected.

For most of the day he remained well, with stable clinical parameters until the early afternoon of the same day when he again complained of shortness of breath. On examination, he appeared sweaty and pale, with obvious stridor. His respiratory rate was again raised, and his oxygen saturation low. His chest was clear to auscultation. Nasendoscopy again demonstrated a well preserved airway. However, removal of the fibre-optic endoscope precipitated a cardiac arrest. Resuscitation was performed as per European Resuscitation Council guidelines, and cardiac output was restored.

He was intubated and transferred to Intensive Care. Despite aggressive management and increasing doses of inotropic support, his condition deteriorated. He suffered several episodes of fitting, suggestive of hypoxic brain injury. Shortly following this he underwent an asystolic arrest, and further attempts at resuscitation were unsuccessful.

A post-mortem examination was performed which demonstrated extensive green discolouration and putrefaction throughout the retropharyngeal space, extending inferiorly to the posterior mediastinum. The pericardial cavity contained a trace of yellow fluid, while the right pleural cavity contained 600 ml and left pleural cavity 1000 ml of turbid green fluid. Ante-mortem blood culture was positive for *Prevotella* and *Eubacterium lentum*.

Discussion

NF may initially be mistaken for a superficial soft tissue infection such as cellulitus and thus undertreated.[5] Characteristically the clinical course is dramatic with severe systemic toxicity and alarmingly rapid spread. Infection spreads in the subcutaneous tissues and along fascial planes initially sparing muscle and skin, with focal tenderness out of proportion to the local clinical findings. Later the skin may become tense and shiny, eventually ending in blistering and necrosis of the skin. Crepitus may be absent clinically due to gas being deep in the tissues. Death may occur as a result of overwhelming sepsis, respiratory failure, multisystem organ failure and major arterial erosion. Alcoholism and diabetes are considered to be risk factors and poor prognostic indicators.[6] NF following steroid injection is an extreme rarity, although not unheard of.[7] Whether our patient had peritonsillar cellulitis is a moot point, as parapharyngeal pathology may have resulted in tonsillar proptosis which in a patient with trismus may have led to a mistaken impression.

As in our case, plain radiography may fail to detect gas in the tissues or the early stages of mediastinitis. CT scan will allow earlier diagnosis of mediastinitis and assess the extent of abscess formation in the neck and the extent of thoracic spread. CT scanning may reveal mediastinal fluid collections, pericardial effusions, pneumonia and emphysema. A CT scan may reveal the presence of gas in the tisssues not detectable by plain radiographs.[8] It is unfortunate that the surgical emphysema in our case was interpreted as an air leak from pharyngeal penetration by the steroid injection canula, rather than bacterial as formation. This is against a background of a patient who was apyrexial and systemically well. His neck swelling had diminished in size and thus his infection seemed to be resolving. The decision not to explore the neck was made two days prior to his death, by two consultant head and neck surgeons, who concurred that in the absence of any pus requiring drainage, his surgical emphysema should be managed conservatively. His general obesity and medical treatment masked the seriousness of his deep neck infection. His initial dyspnoea and later backache on the fourth day failed to alert the attending physician to the possibility of developing mediastinitis. At this stage his chest X-ray was falsely reassuring, showing no abnormality. A CT scan may have been informative at this time, but one performed only a day earlier was completely normal. His swift deterioration demonstrates the rapidity of progression of this life-threatening condition. We

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recommend a low threshold for CT scanning, should the suspicion of DNM arise. If CT scanning is equivocal, a labelled white blood cell scan may be helpful at demonstrating mediastinal involvement.[3]

The pathogenesis of the rapidity of spread of this gangrenous infection is incompletely understood, but it is postulated that the spread of infection is attributable to bacterial production of endotoxins, protease, lipase, collagenase, elastase, chondroitin sulphatase and hyaluronidase. Aerobic and anaerobic bacteria act synergistically in most cases, with isolates including b-haemolytic streptococci, *Strephylococcus aureus*, *Bacteroids* sp, *Peptostreptococcus* sp, *Enterobacter* sp, *Pseundomonas* sp and *Bifidiobacterium*.[1–3]

Antibiotic and medical management alone are insufficient treatment for DNM. Prompt mediastinal drainage and debridement of the necrotic skin, fascia and muscle is necessary. Corsten *et al*.'s meta-analysis demonstrated a significant survival advantage, in those patients who were treated by a combined drainage of the neck and mediastinum versus cervical drainage alone (19 *vs* 47 per cent mortality respectively). The authors recommend a formal thoracotomy to drain the chest.[5] Brunelli and colleagues, however, recommend a cervicomediastinal drainage (cervical incision to enter the thorax) of the mediastinum for cases of superior mediastinitis, with a formal thoracotomy reserved for those cases where the DNM involvement extends beyond the carina. In their series of five cases all patient survived.[2] Hyperbaric oxygen therapy may have a role to play as an adjunct to the above therapies. Shupak successfully treated five patients with CNF with hyperbaric oxygen therapy.[9] Hyperbaric oxygen therapy may reduce mortality in general cases of necrotizing fasciitis.[10]

In summary CNF is a rare but serious infection, which is easily over looked but requires prompt diagnosis. Otolaryngologists need to be increasingly vigilant and aware of this potentially fatal condition. Even in the absence of clinical signs, should the possibility of DNM exist an urgent CT scan may help to rapidly diagnose this devastating condition. Disease progression is alarmingly rapid. DNM requires aggressive multidisciplinary treatment, including mediastinal drainage.

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