Necrotizing fasciitis following drainage of Streptococcus milleri empyema

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Abstract

Streptococcus milleri (SM) is a heterogeneous group of Streptococci, which is a recognized cause of purulent infections of the mediastinal and pleural spaces. These bacteria are part of the normal oral flora and infections with SM are notoriously resistant to antibiotics and often require aggressive surgical management [1]. We present our experience with a 60-year-old patient, who developed necrotizing fasciitis of the chest wall after initial bedside drainage of a SM empyema. He required extensive debridement with significant soft tissue loss and subsequent latissimus dorsi flap reconstruction to cover the defect.

Keywords: Empyema; Necrotizing fasciitis; Streptococcus milleri

1. Introduction

Streptococcus milleri (SM) is a heterogeneous group of Streptococci which is a recognized cause of purulent infections of the mediastinal and pleural spaces. These bacteria are part of the normal oral flora and infections with SM are notoriously resistant to antibiotics and often require aggressive surgical management [1]. We present our experience of an empyema caused by SM which, despite surgical drainage, progressed into necrotizing fasciitis of the chest wall.

2. Case report

An otherwise fit and athletic 60-year-old male was initially seen by his GP and treated with antibiotics for community acquired pneumonia. He had no history of dental disease and co-morbidities. As the infection worsened, he was referred to his local hospital for treatment with intravenous antibiotics. Upon admission to the hospital, the patient was found to have a significantly deteriorated clinical state. Breathlessness, high fever, tachycardia, and mild hypotension along with physical findings of left-sided crackles and basilar dullness to percussion suggested possible lung consolidation and pleural effusion. Chest X-ray showed a large left basal opacification and computed tomogram (CT) revealed a loculated basal pleural fluid collection with gas bubbles within it. These finding prompted immediate insertion of a large bore chest drain at the bedside. Three litres of foul-smelling frank pus under pressure was evacuated. During the following few hours, despite decompression and drainage of the empyema, the patient continued to deteriorate clinically with ongoing fever, altered hemodynamics, and lethargy. Cultures of the sampled pleural fluid grew Streptococcus anginosus (‘milleri’) and antibiotic coverage was tailored to include high-dose penicillin, clindamycin, and 2nd-generation cephalosporin according to recommendations from our microbiologist. Despite this, the patient’s serum white blood cell count and C-reactive protein continued to rise, and it was soon noted that the skin surrounding the chest drain insertion site exhibited a progressive cellulitis with local crepitus. The patient was taken urgently to theatre for surgical exploration and debridement.

A standard left posterolateral thoracotomy revealed widespread acute inflammatory and necrotic changes of the chest wall involving skin and subcutaneous tissue with purulent extension deep to the latissimus dorsi, serratus anterior, and external oblique muscles. In addition, there were patches of myonecrosis extending from the subscapular region to the anterior superior iliac spine. Following chest cavity debridement, decortication, and copious washout of the pleural cavity, non-viable skin, subcutaneous tissues, and muscles were aggressively debrided until healthy back-bleeding tissues were demonstrated throughout. The entire wound was pressure irrigated with saline. Two large bore chest drains were placed into the pleural cavity and the ribs were re-approximated while the overlying wound was left open and packed with saline-impregnated dressings. Two large bore drains were placed in the wound itself and the entire surgical site was covered with an occlusive dressing. The pleural and wound drains were connected to wall suction at ~2 kPa pressure.

In the immediate postoperative period, the patient was clinically stabilized whilst being administered aggressive

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antibiotic therapy. Twelve hours later the patient was taken back to theatre for a second look inspection. Again, further skin, subcutaneous tissue and muscle (latissimus dorsi) was sharply debrided (Fig. 1). The wound was abundantly irrigated and a vacuum-assisted closure (VAC) dressing was fitted to cover the entire wound. Following this second look operation, there was no further need for chest wall debridement. Subsequently, the VAC dressing was maintained with twice weekly changes under general anaesthesia, whilst the underlying pleural cavity infection resolved. The two intra-pleural drains were removed during the third postoperative week.

The patient was taken to theatre for final soft tissue reconstruction by the plastic surgeons the following week. With a clean wound base lined by granulation tissue, the free contralateral latissimus dorsi muscle flap with split thickness skin grafting provided a satisfying result (Fig. 2).

3. Discussion

SM is a heterogeneous group of Streptococci, which is increasingly considered to be an important variety of pathogens causing serious purulent infections [2]. As such, it is a recognized cause of abscesses in various parts of the body, including the cerebrum, retropharynx, and pleura. SM is part of the usual flora of the mouth and the gastrointestinal tract. One-fifth of infections caused by SM are thoracic infections, and many of these evolve into pyogenic collections which are notoriously difficult to treat non-operatively [3]. SM is responsible for 24–51% of the total incidence of suppurative thoracic infections. Of these, empyema is the most common form (75%) [4], followed by mediastinitis and pulmonary abscess. SM reaches the thoracic cavity via several pathways: 1) aspiration of oral secretions, particularly in patients with periodontal abscess and defects in clearance of tracheobronchial secretions, 2) direct inoculation by penetrating trauma, instrumentation, such as bronchoscopy, or surgery, 3) direct extension of infection as in retropharyngeal abscess where the infection may descend along cervical planes into the mediastinum, and 4) haematogenous dissemination [4]. Underlying conditions that debilitate the host and hence facilitate progression of the infection often exist, such as diabetes, malignancy, immunosuppressant medications, and HIV. Patients with empyema caused by SM are often found to undergo stormy hospital stays which are prolonged due to significant morbidity [3]. At the forefront of care for these patients is aggressive surgical and antibiotic treatment with penicillin-based antibiotics [5]. An extreme example of the potential virulence of this infection is demonstrated in our case report where the empyema dissected into the overlying soft tissue planes, resulting in skin, fat, and muscle necrosis of the chest wall and flank. From this example, we emphasize prompt and serious attention to culture positive SM infection of the pleural space.

References