

Empyema necessitans presenting as a gas-forming cellulitis in an HIV-positive man: Case report

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L Dvali, C Quan, R Pugash, E Poulin, W Peters. Empyema necessitans presenting as a gas-forming cellulitis in an HIV-positive man: Case report. Can J Plast Surg 1999;7(2):53-56.

A 45-year-old human immunodeficiency virus (HIV)-positive male presented with massive gas-forming cellulitis of the chest wall and left arm, which developed one month after the onset of a *Staphylococcus aureus* pneumonia that was untreated. This condition was due to an empyema necessitans – an empyema that had drained through the chest wall to the soft tissues of the chest and left arm. Surgical debridement and long term intravenous antibiotic therapy successfully treated the condition.

Key Words: Cellulitis; Empyema; Human immunodeficiency virus

Empyème de nécessité se présentant comme une cellulite gazogène chez un homme infecté par le VIH : rapport de cas

RÉSUMÉ : Un homme de 45 ans infecté par le VIH s'est présenté avec une cellulite gazogène massive de la paroi thoracique et du bras gauche, qui s'était développée un mois après le début d'une pneumonie à *Staphylococcus aureus* non traitée. Cette affection était due à un empyème de nécessité – empyème ayant drainé dans la paroi thoracique jusqu'aux tissus mous du thorax et du bras gauche. Une excision chirurgicale et une antibiothérapie au long cours par voie intraveineuse ont réussi à vaincre cette affection.

Cutaneous and soft tissue infections are common in human immunodeficiency virus (HIV)-positive patients (1). These can occur as typical infections, such as cellulitis or an abscess. Among HIV-positive patients, these infections can result from common bacteria such as staphylococci or streptococci. In other instances, they can have a very atypical presentation, which may result from organisms that are not generally considered to be pathogens in a nonimmunocompromised patient (2). The present case report describes a 48-year-old HIV-positive man who presented with an unusual form of a massive, gas-producing, soft tissue cellulitis of the chest and left upper arm.

CASE PRESENTATION

A 45-year-old HIV-positive male whose medications included stavudine, lamivudine, indinavir and glyburide had enjoyed stable health. His CD₄ count was 540 cells/mm³ and his viral load was undetectable at less than 500 copies/mL (Chiron 2.0 assay). He worked full-time, was physically active and had no history of acquired immunodeficiency syndrome (AIDS)-related illnesses.

One month before admission, he developed malaise, fatigue, cough, a pleuritic left chest and shoulder pain. His general practitioner prescribed a one-week course of ibuprofen. His symptoms resolved over about 10 days. Two weeks later (one month after his original symptoms) he presented to the emergency department with a 48 h history of progressive left anterior chest wall swelling, cellulitis (Figure 1) and floridly palpable crepitus that extended down the left arm to the el-

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Figure 1) Photograph showing cellulitis and swelling accompanied by crepitus of the left chest wall



Figure 2) The area of the left pectoralis major muscle is markedly swollen but not tender to palpation

bow. A tiny focus of redness was visible, approximately 2 cm in diameter, 3 cm lateral to the sternal border at the level of the second intercostal space. The left pectoralis major muscle was massively swollen (Figure 2), but was not tender to palpation.

The white blood cell count was 14,000 cells/mm³. A chest x-ray confirmed massive gas bubbles in the anterior chest wall (Figure 3). These were particularly noticeable in the region of the striations of the pectoralis major muscle. The lateral chest x-ray showed apical opacification, which was interpreted by the radiologist as 'soft tissue swelling' (Figure 3). An x-ray of the left upper limb showed gas bubbles, which were particularly noticeable against the striations of the pectoralis major muscle (Figure 4). These gas bubbles extended down the left arm to the elbow.

The patient was prepared directly for the operating room, with a presumed diagnosis of a gas-forming soft tissue infection. Under general anesthetic, a needle was first passed into

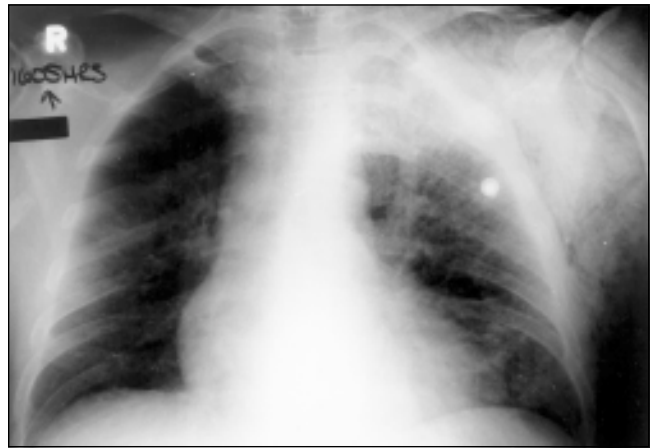


Figure 3) Posterior-anterior (top) and lateral (bottom) chest x-rays confirming massive air bubbles in the anterior chest wall (particularly in the pectoralis major muscle area) with apical opacification (lateral x-ray)

the apparent 'focus site' just above the left nipple. Five cubic centimetres of pus was extracted. A transverse incision was then made directly over the area just above the left nipple, and the pectoralis major muscle was split along its fibres. Under this muscle and superficial to the chest wall, there was a pocket containing additional pus (Figure 5). When the cavity was probed with a gloved finger, the chest wall was clinically intact and there was no undermining of the cavity in any direction. Intraoperative biopsies and cultures were taken. Postoperatively, the patient was placed on intravenous penicillin, clindamycin and ciprofloxacin while awaiting culture results.

Twenty-four hours later, the cellulitis of the chest wall had resolved. The patient spiked a fever to 39°C, and large amounts of pus began to drain from his left chest wound. The palpable crepitus was unchanged in the anterior chest and left arm. A computed tomographic (CT) scan of the chest demonstrated massive swelling of the pectoralis major muscle, a

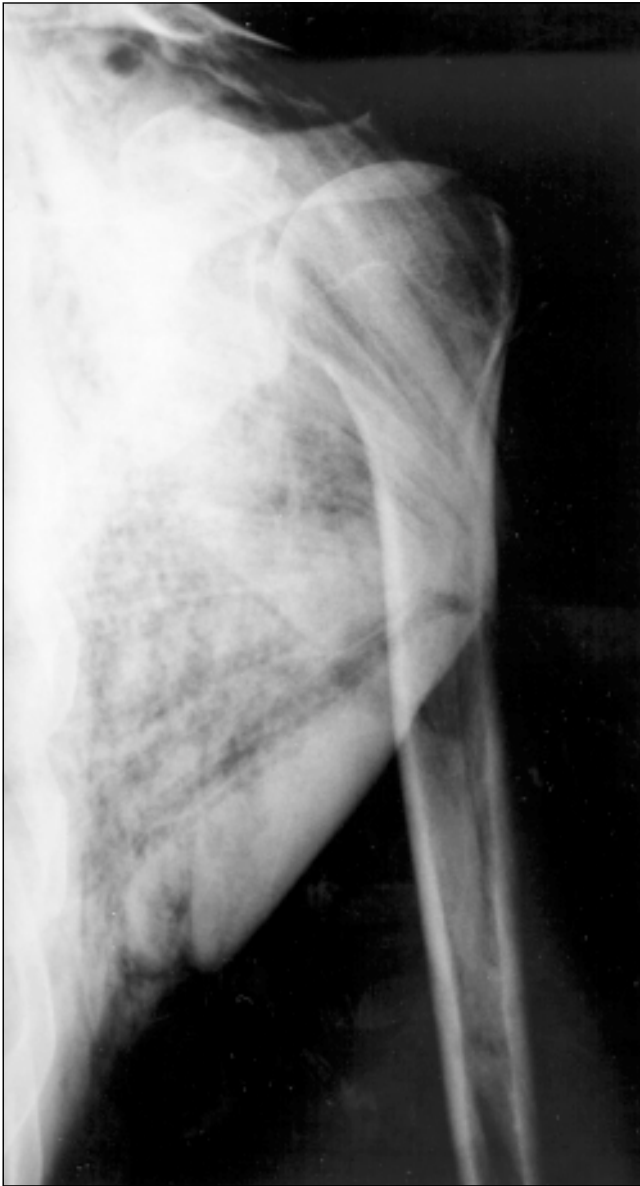


Figure 4) X-ray of the left upper limb showing gas bubbles in the pectoralis major muscle area extending down the arm to the elbow

collection of material on the anterior aspect of the left upper lung field, a sinus tract from the left upper lobe of the chest cavity extending through the rib cage to the left pectoralis major muscle and gas bubbles in the mediastinum (Figures 6,7). The sinus transversed the first anterior intercostal space 2 cm lateral to the manubrium (Figure 7). Intraoperative cultures grew only *Staphylococcus aureus*, which was sensitive to cloxacillin. Fungal cultures showed no growth. A skin test for tuberculosis was negative. This patient likely developed a bronchopectoral fistula with cellulitis of the chest wall secondary to an empyema.

He was placed on a six-week course of intravenous cloxacillin 2 g every 4 h, and was discharged home with a midline catheter. A nurse replaced the dressing of the wound to the



Figure 5) A pocket of infection deep to the pectoralis major muscle on the surface of the chest wall

left chest daily. Over the following six weeks the wound slowly healed by granulation and epithelialization. A repeat CT scan taken three months after the initial CT scans shown in Figures 6 and 7 demonstrated complete radiological resolution of any pathological process in the left lung.

DISCUSSION

In retrospect, the patient in this study appeared to have developed an *S aureus* pneumonia one month before presentation in the emergency department. This condition was not treated and appeared to have subsequently progressed to a chronic empyema in the pleural cavity (3). Pus from this empyema then appeared to have ruptured spontaneously through a weakness in the chest wall, such as an intercostal space, to erode into surrounding soft tissues including the pectoralis major muscle. This condition is termed empyema necessitans (EN) (4). In other cases of EN, rupture and drainage has also been reported into the breast, bronchus, mediastinum, esophagus, diaphragm, pericardium, retroperitoneum, flank, groin or thigh (3-5).

EN was first described in 1640 by Gullan De Baillon, when it developed after the spontaneous rupture of a syphilitic aneurysm (5). EN was much more common in the pre-antibiotic era than it is today. In 1940, Sindel (5) reviewed 115 cases of EN. Eighty-four of these cases were caused by tuberculosis and 31 by pyogenic infections, mainly *Streptococcus pneumoniae*. In 1941, Hochberg (6) reported four cases of EN in 300 cases of acute empyema. Causative organisms most commonly were included *Mycobacterium*

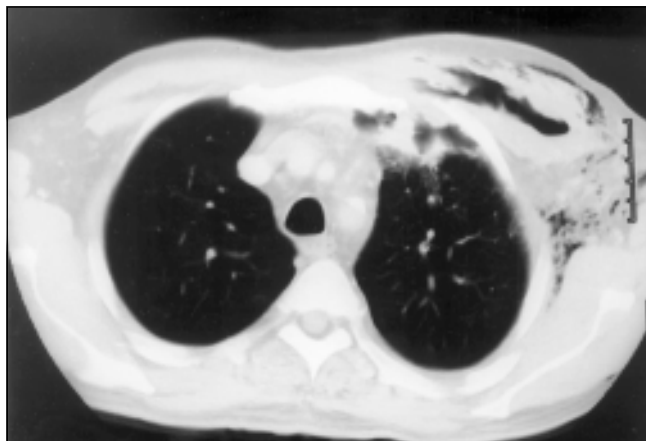


Figure 6) A computed tomographic scan of the chest demonstrating extensive subcutaneous emphysema of the left anterior superior chest wall with extensive inflammatory reaction within the pectoralis musculature and gas in the anterior mediastinum

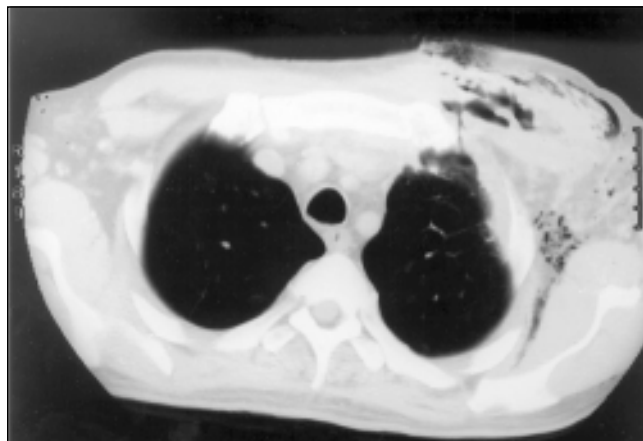


Figure 7) A computed tomographic scan showing a short linear tract extending into the chest cavity to the region of the left upper lobe that transversed the first anterior intercostal space approximately 2 cm lateral to the manubrium

tuberculosis, *S pneumoniae*, *Staphylococcus* species, polymicrobial infections, and Gram-negative bacilli (5).

After antibiotics were introduced in the 1940s, empyema became a much less common complication of tuberculosis and other infectious pneumonic diseases. Today, empyema occurs in fewer than 1% of cases of bacterial pneumonia. The number of cases of EN has also decreased dramatically. Only a small number of case reports have been published during the past 30 years (7,8). These cases have tended to develop in immunocompromised patients or in those with underlying tuberculosis. Causative organisms have included *Actinomyces* species, *S aureus*, and Gram-negative bacilli (7,8).

Although EN is now very rare, it is important to consider this process in immunocompromised patients with a sudden onset of a soft tissue mass of the chest with palpable crepitus.

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