CASE REPORT – OPEN ACCESS

International Journal of Surgery Case Reports 5 (2014) 532–536

Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.casereports.com

Fatal skin and soft tissue infection of multidrug resistant *Acinetobacter baumannii*: A case report

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ARTICLE INFO

Article history:
Received 25 March 2013
Received in revised form 22 January 2014
Accepted 17 April 2014
Available online 19 June 2014

Keywords:
Acinetobacter baumannii
Soft tissue
Infection

ABSTRACT

INTRODUCTION: Acinetobacter baumannii is usually associated with respiratory tract, urinary tract and bloodstream infections. Recent reports suggest that it is increasingly causing skin and soft tissue infections. It is also evolving as a multidrug resistant organism that can be difficult to treat. We present a fatal case of multidrug resistant *A. baumannii* soft tissue infection and review of relevant literature.

PRESENTATION OF CASE: A 41 year old morbidly obese man, with history of alcoholic liver disease presented with left superficial pre-tibial abrasions and cellulitis caused by multidrug resistant (MDR) *A. baumannii*. In spite of early antibiotic administration he developed extensive myositis and fat necrosis requiring extensive and multiple surgical debridements. He deteriorated despite appropriate antibiotic therapy and multiple surgical interventions with development of multi-organ failure and died.

DISCUSSION: Managing Acinetobacter infections remains difficult due to the array of resistance and the pathogens ability to develop new and ongoing resistance. The early diagnosis of necrotizing soft tissue infection may be challenging, but the key to successful management of patients with necrotizing soft tissue infection are early recognition and complete surgical debridement.

CONCLUSION: A. baumannii is emerging as an important cause of severe, life-threatening soft tissue infections. Multidrug resistant *A. baumannii* soft tissue infections may carry a high mortality in spite of early and aggressive treatment. Clinicians need to consider appropriate early empirical antibiotic coverage or the use of combination therapy to include MDR *A. baumannii* as a cause of skin and soft tissue infections.

1. Introduction

Acinetobacter baumannii (*A. baumannii*) was first described as a distinct species within the genus *Acinetobacter* in 1986,[1] and is often associated with pneumonia, urinary tract and bloodstream infections.[2] There have also been previously reported cases of multi drug resistant *A. baumannii* infections in intensive care units.[3] Once considered a rare presentation, *A. baumannii* is now emerging as an important opportunistic multi drug-resistant (MDR) pathogen of skin and soft tissue infections (SSTI).[4]

The management of *A. baumannii* associated infections is increasingly difficult as a result of increasing rates of antimicrobial resistance.[3] Clinicians need to consider appropriate early empirical antibiotic coverage or the use of combination therapy to include MDR *A. baumannii* as a cause of SSTI. We describe a fatal case of a patient with MDR *A. baumannii* SSTI.

2. Case report

A 41 year old morbidly obese man was referred by his local medical officer to the emergency department with painful bilateral oedema of his lower legs and generalized abdominal pain. He had a past medical history of liver cirrhosis secondary to years of excessive alcohol abuse and hepatitis C infection with the complications of portal hypertension and oesophageal varices.

On examination he was febrile (37.6 °C) with significant bilateral pitting oedema and superficial pre-tibial abrasions were noted over his left leg. The capillary refill time was normal and the distal pulses were present in both lower limbs. His abdominal, respiratory, cardiovascular and neurological examinations were unremarkable. Baseline investigations revealed a low haemoglobin of 11.2 g/dl, platelet count of 44×10^9/l, albumin 19 g/l and an elevated bilirubin 36 μmol/l, CRP 37.mg/l, INR 2.1 and lactate of 8.6 mmol/l. Liver enzymes were at the upper limits of normal. On clinical and biochemical assessment of his liver cirrhosis using the Child's Classification of liver cirrhosis he was classified as a Child's B case of cirrhosis. Blood cultures and a swab of the left leg were performed and treatment was empirically started with intravenous flucloxacillin and benzylpenicillin for suspected cellulitis.

On the second day of admission his left leg demonstrated significantly more swelling than his right, with the overlying skin

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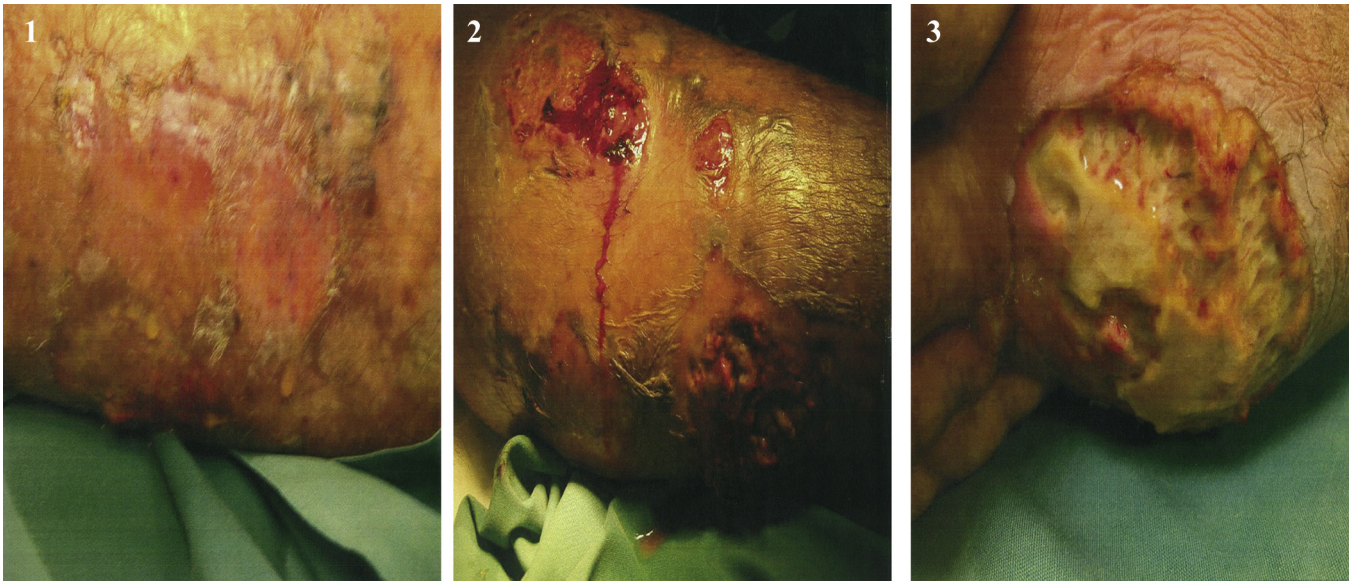
http://dx.doi.org/10.1016/j.ijscr.2014.04.019



Figs. 1–3. Cellulitis caused by *A. baumannii* on the left leg and forefoot of a 41 year old patient. This was initially presented as superficial pre-tibial abrasions and swelling of the lower limb, progressing to bacteraemia and extensive cellulitis and myositis.

erythematous and tender to touch with evidence of induration and blistering. The CRP increased to 101.7 mg/l, and intravenous ciprofloxacin and cephazolin was commenced for worsening cellulitis.

A left leg duplex ultrasound revealed severe oedema below the knee and a large knee joint effusion but no evidence of deep vein thrombosis within the femoral or popliteal veins. A CT scan of his legs confirmed bilateral knee joint effusions with non-specific synovitis. There was no evidence of necrotizing fasciitis.

Two consecutive blood cultures grew *A. baumannii*, with multi drug resistance to amoxicillin, amoxicillin/clavulanate, ceftriaxone and ceftazidime. It was sensitive to co-trimoxazole, gentamycin, meropenem, ciprofloxacin, and ticarcillin/clavulanate. His antibiotic regime was changed to intravenous meropenem and vancomycin to cover *A. baumannii* and also to cover the possibility of a potential co-pathogen Methicillin-Resistant *Staphylococcus aureus* (MRSA).

Conservative management of the lower limbs with leg elevation and compression bandages continued and he was referred to the reconstructive and orthopaedic surgeons for the evaluation of septic arthritis, a suspected compartment syndrome and exclusion of necrotizing fasciitis.

On the third day an MRI scan of his lower legs revealed extensive subcutaneous oedema of the left leg, mild to moderate subcutaneous oedema of the right leg, moderate bilateral joint effusions, cellulitic changes and extensive myositis of the left leg with possible fasciitis raising the suspicion of compartment syndrome. Despite the MRI changes the reconstructive surgeons did not believe that the clinical evidence for compartment syndrome was strong enough to require immediate surgical intervention. His left leg remained erythematous, bullous, and blistering secondary to the oedema and lincomycin was added as therapy on the recommendation of the reconstructive surgeons.

On the fourth day of admission he spiked a fever of 38.4 °C despite continuous broad spectrum antibiotic treatment and the left leg was surgically explored (Figs. 1–3). After general anaesthesia and application of a leg tourniquet a longitudinal incision was made in the mid lateral line. At surgery it was documented that there was no evidence of necrotizing fasciitis and no evidence of compartment syndrome in the lateral or anterior compartments. There was no evidence of an abscess or drainable collection but

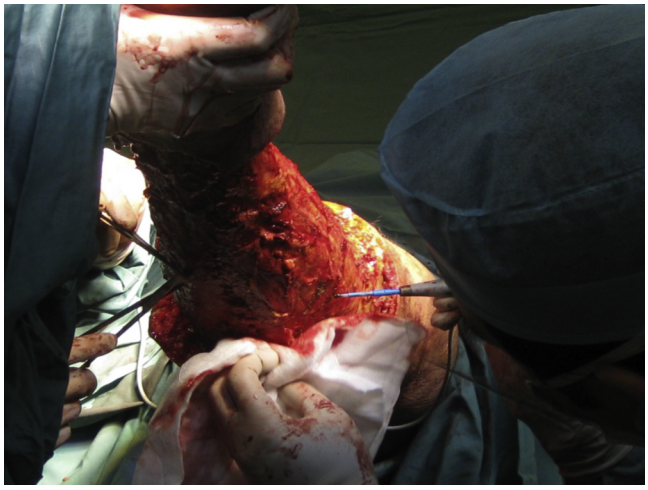


Fig. 4. Surgical debridement.

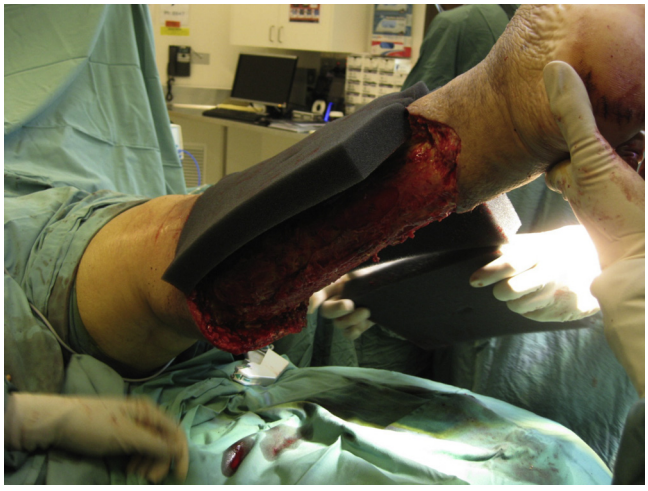


Fig. 5. The limb after extensive debridement.