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

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## 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.

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### 1. Introduction

*Acinetobacter baumannii* (*A. baumannii*) was first described as a distinct species within the genus *Acinetobacter* in 1986,<sup>1</sup> and is often associated with pneumonia, urinary tract and bloodstream infections.<sup>2</sup> There have also been previously reported cases of multi drug resistant *A. baumannii* infections in intensive care units.<sup>3</sup> 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).<sup>4</sup>

The management of *A. baumannii* associated infections is increasingly difficult as a result of increasing rates of antimicrobial resistance.<sup>5</sup> 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

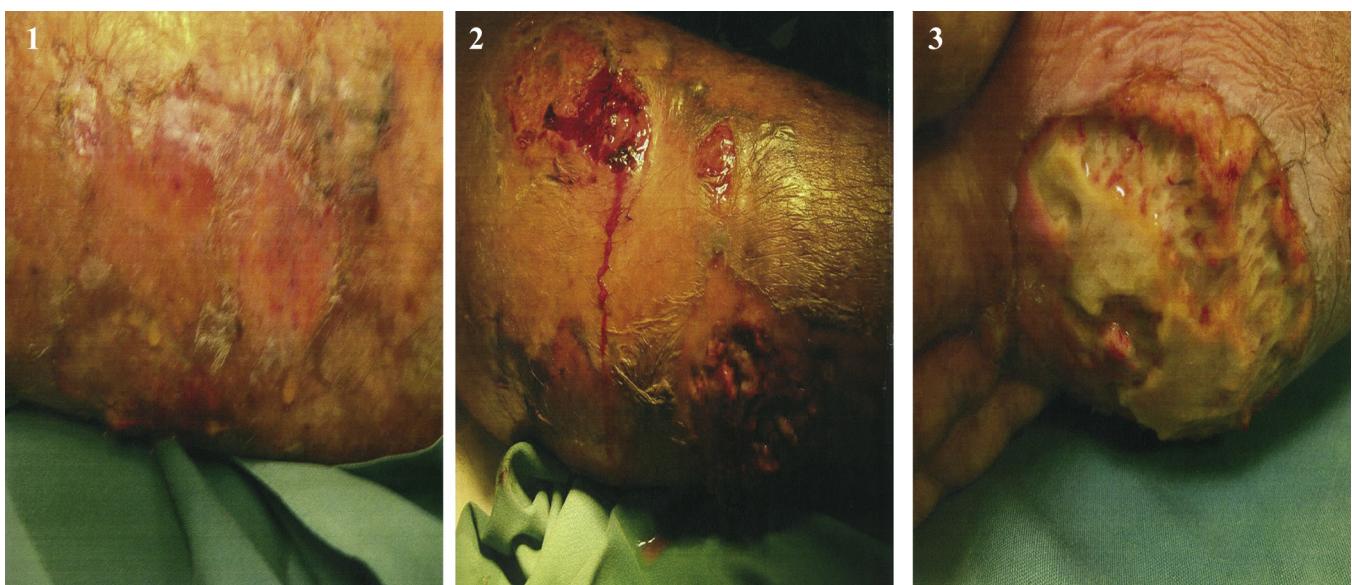
A 41 year old morbidly obese man was referred by his local medical officer to the emergency department with painful bilateral oedema of his lower legs and generalized abdominal pain. He had a past medical history of liver cirrhosis secondary to years of excessive alcohol abuse and hepatitis C infection 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  $44 \times 10^9/l$ , 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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**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.



**Fig. 6.** The limb after placement of the VAC dressing.

there was extensive subcutaneous oedema noted. The surgery involved circumferential debridement of fat necrosis and muscle and washout of his left leg wound (Figs. 4 and 5). The explored leg was managed with a vacuum assisted closure (VAC) dressing (Fig. 6) and antibiotics were continued. The subcutaneous fat and fascia was sent for microscopy, culture and sensitivity and histopathological review. The cultures from the wound confirmed *A. baumannii*.

He remained ventilated post operatively and was commenced on a noradrenaline infusion due to hypotension despite adequate fluid resuscitation. Cultures from the left leg subcutaneous tissue confirmed a light growth of *A. baumannii* resistant to amoxicillin, augmentin, ceftriaxone and ceftazidime and sensitive to co-trimoxazole and gentamicin. After an infectious disease physician consult, his antibiotic regime was changed to intravenous ciprofloxacin, meropenem and lincomycin. Vancomycin was ceased as blood cultures for MRSA and Vancomycin-Resistant Enterococci (VRE) were negative.

Further surgical debridement was performed 6 days later and the surgical findings revealed necrotic circumferential subcutaneous fat. At surgery circumferential debridement of all the subcutaneous tissue in the left lower leg was performed. There was extensive washout of this area with hydrogen peroxide and a new VAC dressing was applied. Histopathology of the left leg subcutaneous fascia revealed epidermal and dermal infarction with focal fat necrosis and acute inflammation. There was no histological evidence of fasciitis and his antibiotic management was tailored to intravenous meropenem and ciprofloxacin.

During his admission there were two further surgical procedures performed primarily to change the VAC dressing. Thirteen days after admission further exposure of the wound revealed that the wound was clean but there were still areas of necrosis along the wound edges. Minor debridement was performed and the VAC dressing replaced. Sixteen days after admission the VAC dressing was again replaced and the wound reviewed. It was noted that there was significant improvement but still small areas of necrotic tissue both proximally and distally. There were areas of necrotic tissue attached to the fascia but all the musculature was viable. A further minor debridement was performed with removal of small areas of necrotic tissue and fascia. Samples were sent for microscopy, culture and sensitivity and the wound was washed with hydrogen peroxide. During the latter two weeks of his admission he became clinically jaundiced with worsening hepatic and renal function and his condition continued to deteriorate despite appropriate antibiotics and surgical interventions and he developed hepatic, circulatory, respiratory and haematological failure.

During the patient's admission his blood glucose levels fluctuated between 4 and 8 mmol/l apart from a 24 h period when he was significantly inotrope dependent and had a single glucose reading of 10 mmol/l requiring insulin. Three weeks after this initial presentation, he continued to require significant vasopressor support and significant tissue from the lower limbs had been lost. The decision by the intensive care and surgical teams was that in light of his co-morbidities and worsening multiple organ failure ongoing medical care constituted medical futility and a decision was made to palliate.

### 3. Discussion

Our case highlights the fact that *A. baumannii* can cause fatal soft tissue infections. Whilst *A. baumannii* infections were initially found to be non-fatal, there are an increasing number of reports confirming increased mortality and morbidity associated with multi drug resistant *Acinetobacter* infections.<sup>5</sup> The case we present shows similarity with other case reports of *A. baumannii* infection published in literature.

The incidence of multi drug resistant *Acinetobacter* infections is increasing. In Australia, the first report of a hospital acquired drug resistant *A. baumannii* strain within an intensive care unit was in 1996 from Western Australia.<sup>6</sup> Since then there has been an emergence of carbapenem resistance in *A. baumannii* recovered from blood cultures in Australia.<sup>7</sup> A recently published study undertaken by Marshall et al.,<sup>8</sup> within three major teaching hospitals in Melbourne found the rate of colonization of ICU patients with *Acinetobacter* spp. to be steadily rising from an estimated 3–5 percent over its five year duration.

Over the last few decades it has been noted that many strains of *A. baumannii* have developed resistance to many currently available antibiotics and the mechanisms involved with this multi-drug resistance have been extensively described by Fournier et al.<sup>9</sup> This research suggested the acquisition of genetic material was responsible for the rapid development of these strains to display multi drug resistance. This genetic material carried clusters of up to 52 distinct genes involved in encoding both enzymatic and non-enzymatic resistance to several antibiotic families at once – including anti-pseudomonal carbapenems and cephalosporins, fluoroquinolones and aminoglycosides.

Reflecting this rapid rise in antimicrobial resistance, a systematic review conducted by Falagas<sup>5</sup> suggested an increase in mortality in patients who acquired *A. baumannii* when compared with matched controls without acquisition. Despite reports of SSTI caused by *A. baumannii* in war trauma patients,<sup>10</sup> its role in non-traumatic SSTI is not well defined.<sup>4</sup> *A. baumannii* is being recovered more frequently in association with SSTI.<sup>4</sup>

Managing *Acinetobacter* infections remains difficult due to the array of resistance and the pathogens ability to develop new and ongoing resistance.<sup>11</sup> Antimicrobial agents that are typically active against *Acinetobacter* infections are the carbapenems, amikacin, sulbactam, colistin, rifampicin and tetracyclines. In the setting of broad spectrum antimicrobial resistance, colistin usually retains activity despite it being considered by some to be overly neuro or nephrotoxic for routine clinical use. Colistin in association with rifampicin appears to be relatively safe and effective in treating critically ill patients with infections caused by multidrug-resistant *A. baumannii*.<sup>12</sup>

In the case presented, *A. baumannii* colonization of the superficial pre-tibial abrasions on the patient's left leg resulted in bacteraemia, extensive cellulitis and myositis. The patient under discussion had several risk factors including chronic liver disease and morbid obesity. Although he had been treated initially on empirical broad spectrum antibiotics, blood cultures

revealed MDR *A. baumannii*, that was resistant to his initially prescribed antibiotics. Despite more targeted antibiotic therapy and surgical debridements he continued to deteriorate and developed multiple organ failure that eventually led to his death.

A wide range of other pathogens such as Nocardia, atypical mycobacteria (*M. fortuitum*, *M. marinum*, *M. ulcerans*), Sporothrix, Cryptococcus, agents of mycetoma (Pseudallescheria, Blastomyces, Phialophora) and Leishmania cause unusual skin and soft tissue infections.

Relapsing or persisting skin and soft tissue infections after injury with biological materials should also prompt consideration of other pathogens including atypical mycobacteria, even in non-immune compromised hosts. Insidiously, *Mycobacterium marinum* infection can occur without any contact to fish tanks or swimming pools. There have been case reports of patients with arthritis receiving immunosuppressive therapy who have been infected with atypical mycobacteria. One patient developed a granulomatous infection of the right thumb and forearm due to *M. marinum* and was successfully treated with clarithromycin and ethambutol.<sup>13</sup> There have also been cases of atypical soft tissue infection after cosmetic surgery. A case of *Mycobacterium cheloneae* infection in the buttock after combined liposuction and lipoinjection has been described.<sup>14</sup> The real possibility of contamination from operating room equipment was the potential etiologic factor of this infection. The difficulty confirming the diagnosis was solved by specific culturing techniques. Successful treatment with limited debridement and irrigation combined with prolonged specific antibiotic therapy effected a long-term cure. Non-tuberculous mycobacterial skin infections have an increasing incidence and in immunocompetent patients, they usually follow local trauma. There have been reports of cutaneous *Mycobacterium abscessus* infection following mesotherapy where the lesions were successfully treated with a combination of clarithromycin, ciprofloxacin, and doxycycline.<sup>15</sup> Another rare cause of soft tissue infection and necrotizing fasciitis is infection with *Vibrio vulnificus*. This occurs mostly in immunocompromised patients with hepatic disease, diabetes mellitus, chronic renal insufficiency and adrenal insufficiency.<sup>16</sup> The authors conclude that early and appropriate diagnosis for *V. vulnificus* infection should be made, especially in patients presenting with atypical clinical findings and early fasciotomy within 24 h remains the highest priority and decreases mortality. A further rare cause of skin and soft tissue infection is the pathogen *Clostridium septicum* and an association exists between colon carcinoma and *C. septicum* infection, especially bacteremia.<sup>17</sup> The diagnosis of soft tissue infection remains difficult and the assessment of wound fluid lactate concentration might be helpful for confirming the suspicion of soft tissue infection, particularly when clinical signs are atypical.<sup>18</sup> Atypical mycobacterial skin infections are difficult to diagnose due to their non-specific histopathology and presence of few bacteria. Therefore, these infections are often not recognized. There is data to suggest that real-time PCR is useful in detecting mycobacterial infections in undiagnosed formalin-fixed/paraffin-embedded skin samples and that the application of molecular approaches would improve the diagnoses of mycobacterial skin infections.<sup>19</sup> Atypical mycobacterial infections should be suspected in patients who develop late-onset skin and soft tissue infection after cutaneous injury, injection, and surgical intervention, particularly if they do not respond to conventional antibiotic treatment. The lesions are often successfully treated with a combination of clarithromycin, ciprofloxacin, and doxycycline. In patients with complicated skin and skin structure infections there is evidence that tigecycline is as effective as vancomycin plus aztreonam. Furthermore limited evidence shows effectiveness in patients with resistant *Acinetobacter* infection in intensive care units, and the possibility that the use of tigecycline may reduce length of hospital stay.<sup>20</sup>

The surgical management of the patient was aggressive with a total of 4 procedures during his hospital admission with histology and repeated microbiology obtained to guide antimicrobial therapy. Despite subsequent VAC dressing changes and clinical improvement of the wound with minimal necrosis the patient continued to deteriorate. The worsening multiorgan failure despite ongoing source control suggests overwhelming sepsis with progression to severe sepsis and septic shock. This response to infection would be consistent with the patients confirmed liver cirrhosis. A review of patients with complicated skin and soft tissue infections (cSSTI) confirmed susceptibility in patients with co-morbidities (42.6%), prior surgical intervention (43.4%), more severe infections such as bacteraemia (51.6%) affected fascia (49.0%) and in patients admitted to intensive care unit (56.2%).<sup>21</sup> Despite sonography being a frequent initial investigation a study has found no significant correlation between ultrasound findings and clinical management in patients with soft tissue infections.<sup>22</sup> The early diagnosis of necrotizing soft tissue infection is challenging, but the key to successful management of patients with necrotizing soft tissue infection are early recognition and complete surgical debridement. Early initiation of appropriate broad-spectrum antibiotic therapy must take into consideration the potential pathogens. Critical care management such as the initial fluid resuscitation, end-organ support, pain management, nutrition support, and wound care are all important aspects of the care of patients with necrotizing soft tissue infection. Soft tissue reconstruction should take into account both functional and cosmetic outcome.<sup>23</sup>

Importantly in patients with necrotizing soft tissue infection, a delay of surgical treatment of >12 h is associated with an increased number of surgical debridements and higher incidence of septic shock and acute renal failure.<sup>24</sup> Because aggressive radical debridement of all poorly perfused tissue is required, patients frequently suffer from massive skin defects, which often requires autograft skin grafting or myocutaneous flaps. However, options are limited in patients with limited autograft donor availability, or questionable underlying wound bed viability. There have been reports of synthetic devices leading to favourable outcomes when large wound defects exist.<sup>25</sup> Our patient had alcoholic liver disease with cirrhosis and morbid obesity that may have predisposed him to *Acinetobacter* infection. Cirrhosis is considered an immunocompromised state that leads to a variety of infections, which then account for an approximate 30% mortality.<sup>26</sup> Cirrhosis-associated immune dysfunction syndrome (CAIDS) is a multifactorial state of systemic immune dysfunction which decreases a patient's ability to clear to clear cytokines, bacteria, and endotoxins from circulation. Furthermore porto-systemic shunting, whereby blood is increasingly directed away from the liver, and reduced RE cells in patients with cirrhosis, allow less bacteria and endotoxins to be cleared by the liver from circulation.<sup>26</sup> Monocyte spreading, chemotaxis, bacterial phagocytosis, and bacterial killing are significantly reduced in cirrhosis compared with controls. Patients with cirrhosis have decreased neutrophil mobilization and phagocytic activity, a phenomenon that correlates with severity of liver disease.<sup>27</sup> Chronic oedema and increased bacterial translocation predispose patients with cirrhosis to soft tissue infections, which constitute approximately 11% of infections.<sup>28</sup> Necrotizing fasciitis is predominantly caused by gram-negative bacilli.<sup>29</sup> Necrotizing fasciitis in cirrhotic patients rarely develops from an obvious portal of entry in the extremities suggesting a potential pathway of bacterial translocation and bacteremia leading to soft tissue infections.<sup>30</sup> Broad spectrum antibiotic treatment is essential in managing patients with soft tissue infections complicating liver cirrhosis and they are invariably resistant to therapy as a consequence of the described pathophysiology. Surgical intervention for deep infections may be needed and multiple interventions are often required in these

profoundly immune compromised patients. In spite of adequate management the mortality remains high.

#### 4. Conclusion

*A. baumannii* is emerging as an important cause of severe, life-threatening cellulitis and other SSTI and may be associated with a high mortality despite early aggressive and appropriate treatment. SSTI in patients with pre-existing immunocompromised state such as cirrhosis may carry a very high mortality in spite of aggressive and appropriate management.

#### Conflict of interest

None of the authors have commercial association or financial involvement that might pose a conflict of interest in connection with this article.

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#### Ethical approval

Consent could not be obtained from the patient due to his clinical condition. This patient had no next of kin to consent for publication of this case report. We have therefore requested the Human Research Ethics Committee (HREC) of our hospital to review this case report for publication. Our HREC approved this case report for publication in the current form. A copy of the HREC approval letter is available for review by the Editor-in-Chief of this journal on request.

#### Author contributions

Aqsa Ali: Data collection, Review of Literature and drafting of the manuscript.

John Botha: Concept, Review of Literature, Writing and reviewing of Manuscript.

Ravindranath Tiruvoipati: Review of Literature, reviewing and revising manuscript.

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