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Case Report
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Necrotizing Fasciitis with Thrombus due to Multidrug Resistant *Acinetobacter baumanii* Infection

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Abstract

This report describes a 53 year old man who died of necrotizing fasciitis caused by multidrug-resistant *Acinetobacter baumannii* infection, characterized by fever, leg pain and swelling. He was treated with cefotaxine, vancomycin and levofloxacin in a local hospital, but did not respond. Physical examination showed high fever and flaring red indurate swelling with severe tenderness along his right leg. Five sets of fascia yielded multidrug-resistant *A. baumannii*. Pathology showed extensive necrosis with thrombi and inflammatory cell infiltration, in which rod-shaped bacteria were seen in macrophages. Immunohistochemical analysis confirmed that most macrophages had NK/T cell markers. The patient developed toxic shock due to multiple organ failure and died before receiving potent antibiotic treatment. Necrotizing fasciitis requires urgent treatment of infection related thrombosis while multidrug resistance and NK/T cells may obscure the nature of infection.

Keywords: Necrotizing fasciitis; *Acinetobacter baumanii*; Multidrug-resistance; NK/T-cells

Introduction

Necrotizing fasciitis is a fulminant infection of skin, soft tissue and the deep fascia, commonly caused by the virulent forms of *Streptococcus pyogenes*, often referred to as "flesh-eating bacteria". However, in addition to virulence, multidrug-resistant forms of bacteria such as *Klebsiella pneumonia* [1], *Aeromonas hydrophila* [2], *Proteus mirabilis*, *Escherichia coli* [3] and *A. baumannii* [4] are also important factor leading to spread of infection, misdiagnosis and treatment failure. We here report a fatal case of necrotizing fasciitis with thrombus due to multidrug resistant *Acinetobacter baumanii* infection.

Case Presentation

A 53-year-old man was hospitalized due to 1-month history of right leg swelling with excruciating pain and a fever of 39.5°C. The lesion was first noted as a papulo-vesicle on the right thigh after a mosquito bite. It spread gradually to the entire leg with diffuse swelling and induration (Figure1A). He had a medication history with cephathiamidine, vancomycin, and levofloxacin after suspected with cellulitis due to increased white blood cells, however, without improvement of his condition. He had complained of weakness, night sweats, and weight loss for two weeks prior to admission in our hospital. He had no history of immune disorders, no diabetes milieus and no hypertension. On examination, the patient appeared pale and sweaty, with a temperature of 39.8°C, a pulse of 72 beats per min, a respiration of 24 breaths per min, and a blood pressure of 150/100 mmHg. There was obvious reddish-purple indurate swelling with severe tender spread out from abdominal wall, buttock to the right side low extremity. An erosion of 6 cm × 2 cm was seen on the posterior region of thigh (Figure 1). His white blood cell count was 7.6×10^9 cells/L with 75% band cells, platelets of 83 gram/L with elevate ESR, a sodium level of 104 mmol/L, a potassium level of 2.8 mmol/L, a alanine aminotransferase level of 94 IU/L, a glutamic oxaloacetic transaminase level of 424 IU/L and a plasma albumin level of 28 g/L. Urinalysis showed proteinuria (++/mL). Magnetic resonance imaging examination showed hyperdensity and thickening of the right leg. Histology showed vasculitis with widespread necrosis from dermis to fascia. Giant cell infiltration was seen along vessels with thromboses and artery inclusion, followed by angiodestruction and onion-skin lesions (Figure 2A, 2B). With Periodic Acid-Schiff staining (PAS), bacteria could been seen as rod-like, mycelia, or in clusters among tissues and within macrophages or multinucleate giant cells, surrounded by pleomorphic cell infiltration (Figures 2B-2D). However, immunohistochemistry analyses of these inflammatory cells were positive for CD68, CD3, CD56, LCA (Leukocyte

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Common Antigen). The expression of Ki-67 was 30% higher than the normal threshold. He was suspected as having necrotizing fasciitis and given immediately with cefoperazone sodium and sulbactam sodium, etimicin, and immunoglobulin (10 g/d for 3 d). However, little improvement had been gained with the anti-infective therapy. Bacterial and mycological cultivations from the bullous liquid and erosion surface secretion had repeatedly shown negative results. He was then given the administration of methylprednisolone (80 mg/d) for detection of NK/T cells. The condition of the patient deteriorated rapidly. The entire leg turned to black with numerous blisters and bullae. The affected limb was then incised and drainage with 2,600 ml dark bloody fluid. He later went to toxic shock and multi-organ failure and deceased before potent antibiotics were administrated. Five sets of fascia yielded multidrug-resistant *A. baumannii* with sensitivity only to imipenem and meropenem, which had never been used for him. Diagnosis of necrotizing fasciitis caused by *A. baumbanii* was then suspected.

Discussion

Necrotizing fasciitis is a life-threatening soft tissue infection and most commonly caused by virulent forms of group *A. streptococci* that is often referred to as "flesh-eating bacteria". Multidrug-resistance *Escherichia coli* has been reported as another "flesh-eating bacteria" in NF in 2006 and since when the number of NF due to *E. coli* has been increasingly reported. These infections could also cause acute constant necrotizing fasciitis with multi-organ failure and toxic shock syndrome [5-8]. *Acinetobacter baumannii* is most commonly associated with community-acquired respiratory infections, or secondary skin infections. Skin and soft tissue infection with *Acinetobacter* after mosquito bites are rare occurrences. *A. baumanii* is famous for the multidrug-resistant nature or even pan-resistant to antibiotics that might led to fulminant infection [3]. This microorganism has been reported previously as the pathogen of necrotizing fasciitis with a incidence of 2% to19%, but with a mortality of 50% [2,3]. Monomicrobial cultures of *A. baumannii* in tissue or/and within blood may confirm the causative agents. Natural Killer (NK)/T-cells is the major immune cell subsets in infections [4], might been considered as malignance, and the situation might lead to misunderstanding of their anti-infection activities. We have detected NK/T-cells infiltration patients with Mucor irregularis infection, which had been misdiagnosed previously with "NK/T-cell lymphoma" and which had achieved complete remission with amphotericin B [5,6]. The NK/T-cells disappeared after mere antifungal therapy and were induced in mice [8]. Herein we present another case of infection with NK/T-cell infiltration, which was a fulminant necrotizing fasciitis caused by multidrug-resistant *Acinetobacter baumanii*. The delayed recovery of bacteria interfered assessment of these immune response cells with a consequence of misdiagnosis. NK/T-cells is the major immune cell subsets in necrotizing fasciitis [4] and aggregation of which may be seen in a variety of infections, such as Mucor irregularis and *Rhizopus arrhizus* infection [5,6]. They disappeared after mere antifungal therapy [5-11]. Recently, Schmidt demonstrated that both interleukin-2 prestimulated and unstimulated human NK cells could damage *Rhizopus oryzae* hyphae that is in correspondent with mucoramycosis [12]. Our investigation showed that Mucor irregularis and *Rhizopus arrhizus* could induce NK/T cell infiltration (CD3+, CD8+, CD56+, TIA1+, GZMB+, PRF+), proliferation (Ki67+), and angioinvasion in patients and in mice. Thrombosis, such as myocardial infarction, is the direct cause of acute necrosis and needs urgent treatment. The thrombus caused by infection needs thrombolytic treatment on the basis of effective anti infection, so as to prevent tissue necrosis as soon as possible.

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