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Case Report

Facial ulcerations due to *Acinetobacter baumannii*: Vessel thrombosis with bacterial mycelia

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ABSTRACT

A 14-year-old girl presented with a 2-week history of progressive facial ulcerations that did not respond to cephalexin and topical dexamethasone. Biopsy on the ulcer showed rod-shaped bacteria and actinomycetes-like mycelia in the vessel walls and within thrombi. Tissue culture yielded *Acinetobacter baumannii*, which was resistant to cephalexin. A favourite outcome was achieved with minocycline treatment. This is the first case report of *A. baumannii*-related vasculitis.

Introduction

Acinetobacter baumannii is an emerging prevalent and significant pathogen, most commonly associated with respiratory infection, bacterimia or secondary skin infections [1] which can occur in community or health care associated settings. Acinetobacter skin and soft tissue infections outside of the traumatic wound setting are rare occurrences. *A. baumannii* related vasculitis confirmed by pathology has never been reported, though necrotizing fasciitis, a type of vasculitis, has been described with widespread tissue necrosis causing significant morbidity and mortality [2–6]. Herein, we report a case of progressive facial ulcers caused by *A. baumannii*-related vasculitis in a young immunocompetent girl.

Case report

A 14-year-old girl living in Hebei, China presented with a 2-week history of progressive ulcers in her right preauricular skin. These lesions initiated as swelling and nodules with severe itching after a precursor of facial palsy, which was treated topically with herbal plaster. The nodules soon developed to progressive ulcers and kept enlarging. She had a medication history with cephalexin and topical dexamethasone in local clinic after suspected with cellulitis and vasculitis, however, without improvement. The girl had no history of immune disorders and diabetes mellitus. There is no family history of lymphoma, vasculitis or metabolic disorders.

On examination, the patient was afebrile. Her blood investigations and liver and renal function tests were within normal limits. Nodules and ulcers with marked boundary were seen on her right preotic skin (Fig. 1A). Little pus was seen within the ulcers. Biopsy was sampled on the ulcer for bacteriological and fungal culture and for histopathological examination (Fig. 1B).

Histopathology revealed vasculitis with thromboses, granulomatous inflammatory infiltration with lymphocytes, macrophages and a few multinucleate giant cells (Fig. 2A). Grocott's methenamine silver (GMS) staining showed actinomycetes-like mycelia within vessel walls and thrombi (Fig. 2B) while they could be seen as clusters or chains from the epidermis to the subcutaneous tissues (Fig. 2C and D). Mycological culture was negative for fungi while bacterial culture was positive with creamish white colonies, later identified as bacterium. The bacterium was identified using VITEK II automatic bacteria identification system and the drug susceptibility done by the Kirby-Bauer method. Antimicrobial susceptibility testing results were determined in accordance to the United States performance standards for antimicrobial susceptibility testing CLSI-M100.S21. The organism was sensitive to minocycline, meropenem, levofloxacin and ceftazidime, intermediate to ceftriaxone and resistant to cefaclor, cephalexin and aztreonam.

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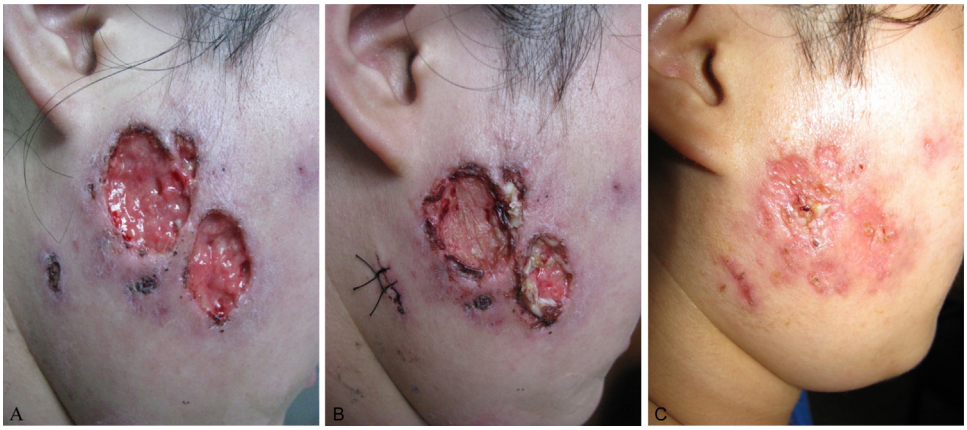

Fig. 1. (A–C) Nodules and ulcers without pus and marked boundary in the right preotic skin in a 14-year-old Chinese girl associated with *Acinetobacter baumannii*-related vasculitis (A: At presentation; B: A week later; C: Eleven days later).

The patient was treated with minocycline at the dosage of 50 mg twice daily and topical mupirocin cream. Ulcers and swelling began to shrink soon after treatment administration and healed within 3 weeks (Fig. 1B and C). She remains well two year after and the facial palsy has never relapsed.

Discussion

Our case was characterized by progressive face ulcerations, no respondence to antibiotics (cephalexin) treatment, positive tissue culture for *A. baumannii*, a giant cell vasculitis unresponsive to topical corticosteriods, bacteria seen as bacilli, mycelia or clusters in the vessel walls, thrombi and among the tissues and a favourite outcome using other antimicrobial to which the organism was found to be sensitive to. In this case, it was not possible to differentiate clinically between immune, infection or malignancy related vasculitis as all present as red, boggy nodules initially, which can progress to necrotic ulcers. Little purulence in the ulcers was found so that possibility of bacterial etiology could have been easily overlooked. However, as bacteria were seen in vessel wall and thrombus, together with giant cell granulomas and inflammatory cell infiltration, a bacterial etiology causing facial ulceration was suspected.

Minocycline has been used in the treatment of vasculitis although the underlying mechanisms are unclear [7]. Reduction of

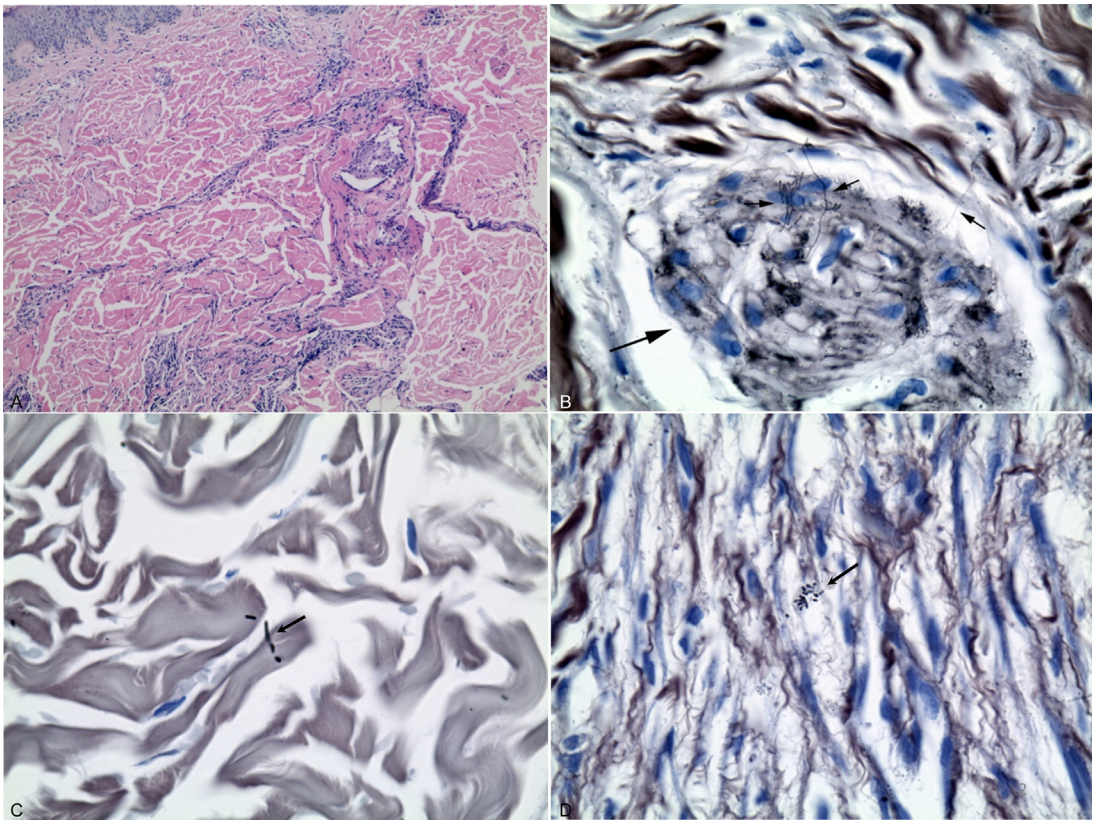

Fig. 2. (A) Histology with H&E staining showed giant-cell vasculitis with thromboses, inflammatory infiltrate with lymphocytes, macrophages, and multinucleate giant cells (A, 100×); (B–D) Gomori's methenamine silver nitrate staining showed bacteria within thrombosis (long arrow) and vessel wall in as mycelia, chains and rod about 1 μm width (short arrows, 1000×).

cytokines or pro-inflammatory protein expression, suppression of T cell proliferation, activation and cell cycle progression are all the described anti-inflammatory properties of the compound [8]. Given that the anti inflammatory properties of minocycline having an effect in the case, findings of bacterial elements in tissue support the activities of minocycline to vasculitis by adding its antibiotic property.

A. baumannii has been reported previously as the causative organism of necrotizing fasciitis (NF), a type of vasculitis commonly associated with destructive, sometimes fatal, outcomes due to infection-induced arterial thrombosis. The condition can be caused by single organisms such as *Streptococcus pyogenes* (Group A beta-hemolytic streptococcus), or the salt water living *Vibrio vulnificus* as examples [9], even *E. coli* has been reported [10] and can also be associated with multi bacterial processes as a Fournies's gangrene. The incidence of *A. baumannii* associated necrotizing fasciitis varied from 2 percent to 19 percent with a mortality of 50 percent but other type of vasculitis with the organism has not been reported [2–6]. In our case, detection of bacteria in histopathology confirmed that the growth in culture was significant, not just superficial colonization.

Vasculitis is an inflammatory disorder of the blood vessel with multiple etiologies and bacterial infection is presumed as one of the causes. Sran et al. [11] reported cutaneous nodules caused by *Mycobacterium chilean* related vasculitis. Like our case, the organism was demonstrated in tissue and confirmed by culture. Antimicrobial therapy alleviated the symptoms. Perez et al. [12] described a *Mycoplasma pneumoniae* associated vasculitis in a 28-year-old woman. Infection was suspected by histologic examination showing leukocytoclastic vasculitis with endothelial cell swelling, fibrinoid changes of vessel walls, a neutrophilic perivascular infiltrate with leukocytoclasis, and extravasated erythrocytes. Serology indicated *M. pneumoniae* infection that was underscored by treatment with erythromycin, although indomethacin was also used. In 1996, van Putten [13] reported patients of Wegener's granulomatosis associated with *Staphylococcus aureus* infection. Their clinical manifestations and c-ANCA tilters fluctuated in accordance with severity of lower respiratory tract infections with *S. aureus*, but they improved after sulfamethoxazole/trimethoprim therapy, to which the pathogen was sensitive. In animals, a lethal midline granuloma-like case with *Nocardia* infection was confirmed by pathology [14]. In our case, positive tissue culture, bacteria seen in pathology, and favourite outcome with minocycline could confirm the infection associated vasculitis. It was especially crucial in this case that bacteria, subsequently identified as *A. baumannii*, were visualized histopathologically to confirm the organism as the causative agent of the progressive facial ulcerations.

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