Case Report

Case Report Bullous impetigo associated with Abiotrophia defectiva in an immunocompetent adult

Heather M. Anderson,1 Cathy Miller,1,2 Earl Kemp1,2 and Mark K. Huntington1,2

Correspondence
Mark K. Huntington
mark.huntington@usd.edu

1Sioux Falls Family Medicine Residency Program, Center for Family Medicine, Sioux Falls, SD 57105, USA
2Department of Family Medicine, University of South Dakota Sanford School of Medicine, Sioux Falls, SD 57105, USA

Infection of humans by Abiotrophia defectiva, a nutritionally variant streptococcus, most commonly takes the form of endocarditis, though a variety of other manifestations ranging from central nervous system abscesses to orthopaedic infections have been seen. We report here what we believe is the first case of bullous impetigo associated with this organism.

Introduction

Although Abiotrophia species have been implicated in a broad range of infectious conditions, they have not been associated with infections of the skin. We report here a case of bullous impetigo associated with Abiotrophia defectiva in an immunocompetent individual.

Case report

A 64-year-old male presented to his personal physician with bullae on his right foot. He first noticed a painless ‘blood blister’ between toes after wearing new shoes. Noting this, he continued his usual activities. Over the next 48 h, another blister had developed, and his foot swelled significantly.

Medical history was significant for hypertension and hyperlipidaemia; his medications included metoprolol, simvastatin and aspirin. He had no recent dental care. He was married, had a very active lifestyle, no drug allergies, no use of alcohol or illicit drugs, and an 80 pack-year smoking history prior to quitting 7 years earlier.

He was afebrile with three 1 cm diameter bullae on the superior aspect of his right foot, one of which was macerated. Oedema extended to his ankle and was warm to the touch, with normal sensation and full pulses. He was diagnosed with cellulitis and begun on cephalexin (500 mg orally four times per day), topical triple antibiotic and a non-compressive dressing. Follow-up was planned in 3 days.

The following day (Saturday) after four doses of cephalexin, he presented to the emergency department with progression of the bullae. He had no pain. He was afebrile, well-appearing, had no cardiac murmurs and had a benign examination except for his right lower extremity. One of the bullae had progressed to a diameter of 3 cm with the other two bullae as previously described. The foot was erythematous with oedema extending to the mid-calf. Good pulses and normal capillary refill were present. Wound and blood cultures were collected. A complete blood count and basic metabolic panel were normal. He was diagnosed with bullous impetigo and given single doses of vancomycin and piperacillin–tazobactam intravenously in the emergency department. Sulfamethoxazole–trimethoprim (800–160 mg – two tabs orally two times per day) was added to the cephalaxin, and he was discharged for outpatient follow-up.

The culture of the wound grew a small amount of Abiotrophia defectiva, and no other organisms. Identification was via an automated commercial system employing 43 tests of carbon source, enzymic activities and resistance (Vitek 2 GP card; bioMérieux) (Pincus, 2006). The blood culture showed no growth. Antibiotic susceptibilities were not reported, per the clinical laboratory’s protocol for these organisms. The patient completed a 10 day course of antibiotics with resolution of the oedema, erythema and bullae; slight discoloration of the skin continued.

Three days later, he presented to an acute care clinic (unconnected to either his medical home or the hospital emergency) with new bullae, oedema and erythema in the right foot (Fig. 1). He was restarted on cephalaxin and sulfamethoxazole–trimethoprim as before. He followed up with his personal physician in 2 days; his examination was unchanged since the acute care visit, and antibiotics were changed to levofloxacin (750 mg orally daily). The following day, with new bullae and increasing discoloration, an infectious disease specialist was consulted by his personal physician and treatment was again changed, this time to dicloxacillin (500 mg orally every 6 h for 14 days). His symptoms slowly improved and ultimately resolved.
Discussion

Originally identified as a ‘nutritionally variant streptococcus’, and initially classified as Streptococcus defectivus, A. defectiva is part of the human oral microflora, where its presence in dental biofilm appears to correlate with a decreased risk of dental caries (Kanasi et al., 2010).

A variety of infections by this organism have been reported. The majority (58%) present as endocarditis, with bacteremia and sepsis composing another 26% (Christensen & Facklam, 2001). Endocarditis is commonly associated with embolic phenomena, valvular destruction and treatment failure (Christensen & Facklam, 2001; Lin & Hsu, 2007; Kiernan et al., 2008; Kohok et al., 2011). Other serious endovascular infections have also been reported (Vargiami et al., 2010).

Central nervous system infections, most commonly brain abscesses but also meningitis, have been reported (Cerceo et al., 2004). These have been linked to embolic phenomena, neurosurgical instrumentation and immunosuppression (Cerceo et al., 2004; Kohok et al., 2011).

Ophthalmological infections have been encountered, ranging from keratitis to endophthalmitis (Horstkotte et al., 2010).

A. defectiva has caused orthopaedic infections, including prosthesis infection, septic arthritis, discitis and sacroiliitis (Wilhelm et al., 2005; O’Connor et al., 2008; Cassir et al., 2011). It has been implicated in conditions such as sinusitis, peritonitis, parapneumonic effusion and even haemophagocytic syndrome (Buckingham et al., 2003; Paju et al., 2003; Arslan et al., 2006; Kiernan et al., 2008).

Antibiotic resistance is a significant problem, due to β-lactams, erm(B) and mef(A). Resistance to cephalosporins, carbapenems, macrolides, penicillins and tetracyclines, and intermediate susceptibility to chloramphenicol, have been observed (Zheng et al., 2004). Fluoroquinolone resistance is reported in the related Abiotrophia elegans, as well (Zheng et al., 2004).

To the best of our knowledge, this current report represents the first case of bullous impetigo, or any other skin infection, associated with this organism. This attribution, however, is not without difficulty. The complicated bimodal clinical course of this infection, along with the use of multiple antibiotics, presents significant limitations to firmly establishing the role of Abiotrophia. For example, it is possible that cephalaxin administered prior to obtaining the culture suppressed the in vitro growth of more typical organisms of impetigo. Additionally, isolation of this organism from the skin rather than a sterile body site potentially diminishes the strength of the claim of this organism as the solely responsible agent. Conventional phenotypic bacterial identification methods, rather than the more specific 16S rRNA typing, were employed in our clinical laboratory. While identification by the former is 97% specific (Pincus, 2006), sole reliance upon it represents another potential limitation of this report.

Following the perceived failure of the initial treatment with the cephalosporin, coverage was added for community-acquired meticillin-resistant Staphylococcus aureus, a fairly common cause of treatment failures in the local area. While the abandonment of the fluoroquinolone following recrudescence may have been premature, the use of an extended-spectrum penicillin is consistent with reported susceptibilities of these organisms.

Beyond the novel manifestation of Abiotrophia infection, this case highlights a deficiency in health-care delivery in our community. For this single infection, the patient sought care from multiple providers at multiple facilities. The result was difficulty in objectively tracking the progress of his condition and a delay in definitive treatment due to reinitiation of a demonstrated ineffective regimen. Although the ultimate outcome was favourable, it is easy to imagine how it might not have been. This case illustrates the importance of continuity of care in acute conditions in addition to the chronic conditions for which the value of continuity is more widely recognized.

Fig. 1. Bullous impetigo. Photograph taken at the time of recrudescence, with healing base of bullae on the lateral-dorsal aspect of the foot (upper panel), and involvement of the intertriginous regions (lower panel).
Bullous impetigo can be added to the litany of conditions with which *Abiotrophia* may be associated. It must be considered in the differential diagnosis of skin infections not responding to antibiotic therapy targeting the more common aetiologies of impetigo.

**References**


