Case Report

A rarely isolated micro-organism, *Budvicia aquatica*, cultured from urine of a patient with Guillain–Barré SyndromeHanna Tomczak¹ and Piotr Smuszkiewicz²

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Introduction: As a pathogen, *Budvicia aquatica* is a rarely cultured micro-organism. It was first isolated in the Czech Republic in 1983. It can chiefly be found in the waters of streams, rivers, wells and swimming pools, but it may cause infections in immunocompromised patients.

Case presentation: On 4 October 2013, a 67-year-old male patient was admitted to the Intensive Care Unit from the Neurological Department due to a deterioration of his general state and the occurrence of acute respiratory failure in the course of Guillain–Barré Syndrome. A urine culture was routinely prepared when his urinary catheter was exchanged on 2 November 2013, and more than 10^5 c.f.u. *B. aquatica* ml⁻¹ was cultured, although no symptoms of infection were diagnosed. The patient did not require antibiotic therapy.

Conclusion: It is possible that the absence of symptoms of infection resulted from the immunomodulatory effect of IgG, which the patient had received to treat the underlying disease, and to the regular exchange of his urinary catheter.

Keywords: *Budvicia aquatica*; Guillain–Barré syndrome; urine.

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Introduction

As a pathogen, *Budvicia aquatica* is a rarely cultured micro-organism. It was first isolated in the Czech Republic in 1983 (Bouvet *et al.*, 1985), and is found chiefly in streams, rivers, wells and swimming pools (Schubert & Groeger-Söhn, 1998). It can cause infections in immunocompromised patients (Corbin *et al.*, 2007). *B. aquatica* is a Gram-negative rod of the family Enterobacteriaceae, and can be observed under a microscope in the form of long curved shapes (Aldova *et al.*, 1985; Zdorovenko *et al.*, 2011).

Case report

On 4 October 2013, a 67-year-old male patient was admitted to the Intensive Care Unit (ICU) from the Neurological Department due to the deterioration of his general state and the occurrence of acute respiratory failure in the course of Guillain–Barré syndrome (GBS).

His medical history revealed several episodes of alcoholic polyneuropathy, paroxysmal atrial fibrillation, hypertension and ischaemic heart disease.

On admission to the ICU, the patient was analgosedated, intubated, mechanically ventilated and supported with infusion of catecholamine, and suffering from cardiopulmonary instability.

A neurological examination did not reveal meningeal symptoms. Paresis of the upper limbs was diagnosed (second degree on the Lovett scale). There was a flaccid paraplegia of the lower limbs and tendon reflexes were abolished in all the four limbs; no pathological symptoms were diagnosed. Mechanical ventilation was continued (biphasic positive airway pressure, followed by continuous positive airway pressure), as well as a catecholamine infusion. A supply of IgG was implemented at a dose of 30 g per day, which was initiated in the Neurological Department.

On admission, materials for routine microbiological tests were collected: swabs from the nasal vestibule and from the anus to test if the patient was a carrier of pathogens, bronchoalveolar lavage (BAL) and blood. *Streptococcus viridians* and *Staphylococcus hominis* were cultured from the BAL at 10^3 c.f.u. ml⁻¹. As there were symptoms of infection, amoxicillin/clavulanic acid was administered according to the antibiogram (10–16 October 2013). *Veillonella* spp. were then cultured from the blood, so

Abbreviations: BAL, bronchoalveolar lavage; GBS, Guillain–Barré Syndrome; ICU, Intensive Care Unit.

metronidazole was included (18–24 October 2013). In another sample from the BAL collected on day 11 of hospitalization in the ICU, *Enterobacter cloacae* and *Staphylococcus epidermidis* were cultured. These were sensitive to trimethoprim/sulfamethoxazole, which was administered from 21 to 29 October 2013. The patient's general state improved. There were negative results in two follow-up urine cultures on days 3 and 16 of hospitalization in the ICU.

In another urine culture, which was routinely carried out when a urinary catheter was exchanged on 2 November 2013, *B. aquatica* was cultured at more than 10^5 c.f.u. ml⁻¹. The strain was cultured in the form of grey–white colonies on Columbia agar with 5 % sheep blood (bioMerieux), whereas on MacConkey medium (bioMerieux) the colonies were pink. In contrast, on UTI chromogenic medium (Argenta), which is used for urine cultures in routine microbiological diagnostics, large cherry-coloured colonies were cultured, which were different from all others that had been analysed before. A microscope slide was prepared. The presence of long, curved, Gram-negative rods was observed in the Gram-stained preparation. The micro-organisms were identified using a VITEK automated system (bioMerieux) with a GN card, which is dedicated to Gram-negative micro-organisms. The results indicated that the organism isolated was *B. aquatica*, with a probability of 92 %. Overall, 64 biochemical traits were assessed. The sensitivity to antibiotics was tested using an AST 258 card (bioMerieux) for *Enterobacteriaceae* cultured from urine. The strain did not continue growing. In view of this fact, an Etest (bioMerieux) was used on Mueller–Hinton agar (bioMerieux), but growth did not continue. Therefore, an attempt to carry out tests on a medium with 5 % sheep blood was made, because the micro-organisms proved to be very demanding. The strain started growing and it was possible to assess its sensitivity. The MIC values ($\mu\text{g ml}^{-1}$) for individual antibiotics were as follows: amikacin, MIC=6 [sensitive (S)]; amoxicillin/clavulanic acid, MIC=24 [resistant (R)]; cefepime, MIC=0.064 (S); ceftazidime, MIC=0.125 (S); ciprofloxacin, MIC=0.064 (S); gentamicin, MIC=3 [intermediate (I)]; imipenem, MIC=0.50 (S); levofloxacin, MIC=0.064 (S); meropenem, MIC=0.094 (S); tigecycline, MIC=0.75 (S); and trimethoprim/sulfamethoxazole, MIC >256 (R). The results were interpreted according to the European Committee on Antimicrobial Susceptibility Testing criteria.

Thus, the strain was resistant to amoxicillin/clavulanic acid and trimethoprim/sulfamethoxazole, which the patient had received previously. No clinical or laboratory symptoms of a urinary tract infection were observed: on 1 November 2013, the procalcitonin level was 0.14 ng ml⁻¹, whereas on 5 November 2013, it was 0.09 ng ml⁻¹; the C-reactive protein level increased from 24.3 mg l⁻¹ on 1 November 2013 to 41.7 mg l⁻¹ on 2 November 2013; and leukocyte levels were normal.

During hospitalization, paroxysmal atrial fibrillation was observed. This reached a value of 150 min⁻¹ and was

treated successfully with amiodarone and digoxin. On day 8 of hospitalization, the patient underwent echocardiography with the following results: ejection fraction about 50 %; abnormal valves having calcified aortic cusps with reduced mobility and mitral regurgitation; first-degree portal venous phase; and abnormal left ventricular wall contractility (apical akinesis). The diagnosis was benign aortic regurgitation and segmental disorders of left ventricular wall contractility.

On day 10 of hospitalization, the patient underwent bronchofiberscope-controlled percutaneous tracheostomy. Due to the absence of neurological improvement, plasmapheresis therapy was started on day 14 (the patient underwent eight plasmapheresis procedures). On day 26, the patient was weaned from mechanical ventilation.

On day 30, the patient was transferred to the Neurological Department. The patient was discharged in a stable general state. He was conscious, with good contact, was logical and was breathing independently and efficiently. A cuffless tracheostomy tube with a disposable inner cannula was left in the patient's trachea. His circulation system was stable. He had no fever and was nourished orally. The mobility of the patient's upper limbs improved, but there was less improvement in the mobility of his lower limbs. Because *B. aquatica* was cultured from his urine collected in the ICU, the patient's urine was collected for culture again, although the patient did not show any symptoms of infection. No significant bacteriuria was found in the urine.

Discussion

In ICU patients, the severe clinical state and invasive procedures (e.g. endotracheal intubation, tracheotomy, arterial and venous cannulation, urinary catheterization, parenteral nutrition, renal replacement therapy) favour the development of infections. Catheters and drains, which are entered into the patient without anatomical barriers, can become colonized in a short time and may be sources of infection.

The long immobilization of patients resulting from paresis and paralysis in GBS predisposes them to pneumonia and urinary tract infections (Nq *et al.*, 1995).

The results of our observations indicated that *B. aquatica* is a fastidious micro-organism, shown by the fact that it did not grow on antibiogram cards in the VITEK automated system or on Mueller–Hinton agar, which was used to test the sensitivity to antibiotics. The bacteria began to grow only on medium enriched with 5 % sheep blood. *B. aquatica* was sensitive to almost all antibiotics except amoxicillin/clavulanic acid and trimethoprim/sulfamethoxazole. The patient previously received trimethoprim/sulfamethoxazole. More than 10^5 c.f.u. *B. aquatica* ml⁻¹ was cultured from the patient, but no symptoms of infection were diagnosed. However, the infection parameters (C-reactive protein, procalcitonin and leukocytes)

increased slightly. This state was classified as urinary catheter colonization. In the following days of hospitalization, no symptoms of infection were observed and no more *B. aquatica* was cultured from consecutive urine samples. The patient did not require antibiotic therapy. It is possible that the absence of symptoms of infection resulted from the immunomodulatory effect of IgG, which the patient had received to treat the underlying disease, and to the regular exchange of his urinary catheter.

The literature provides one report on a *B. aquatica* infection, which led to sepsis in an immunocompromised patient (Corbin *et al.*, 2007). GBS belongs to a group of diseases whose pathophysiology encompasses the entire immune system, because nerves become impaired during the course of the disease as a result of autoimmune mechanisms. Thus, GBS should be treated as an independent factor responsible for a higher predisposition of patients to infection.

So far, *B. aquatica* has been related to an aquatic environment. However, it seems that in favourable conditions this pathogen may also develop in patients with diseases of the immune system.

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