ACINETOBACTER BAUMANNII MENINGITIS
A Case Report

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INTRODUCTION

Acinetobacter baumannii causes various serious nosocomial infections worldwide. Bacterial meningitis is a common complication after neurosurgical operation, and the percentage of A. baumannii meningitis is growing, especially the one resisting multiple drugs [1]. The neurosurgical patients have a high risk to suffer from bacterial meningitis caused by A. baumannii with potentially fatal consequences. The meningitis caused by A. baumannii is well recognized and has been described worldwide. Most case reports about the meningitis were associated with external ventricular drainage (EVD), cerebrospinal fluid (CSF) leaking, or head trauma [2-5]. This post-surgical meningitis is especially severe, because the selection of the antibiotic depends not only on the susceptibility of A. baumannii, but also on the penetration of the chosen antibiotic through the blood-brain barrier [6-7]. We report here the case of a male patient that acquired meningitis with A. baumannii following neurosurgical operation.

CASE PRESENTATION

We report the case of a 44-year-old male patient who presented to our department with a cranial trauma.

His physical exam revealed: coma (Glasgow score 9); afibrile, right hemiplegia, left frontal cranial-cerebral lesion.

Further inquiries included: leukocytosis: 30000/mm3 with N 90%; anemia: Hb 10 g/dl; ESR: 120 mm/h; hypoproteinemia: total protein 5.8g/dl. CT scan indicated areas of hypodensity interesting frontal lobe and signs of left ventriculitis (Figure 1).

Five days following the neurosurgical intervention the patient presented fever, altered state, confusion and neck stiffness.

We then performed a lumbar puncture; the examination of CSF revealed a purulent liquid with PN 95%. The direct exam showed diplococci and Gram-negative coccobacilli. From CSF culture it was identified as A. baumannii.

The antibiogram revealed that A. baumannii was resistant to: amoxicilline + clavulanic acid, cefoxitine, ceftriaxone, cefuroxime, amikacin, ticarcilline, ciprofloxacine, meropenem and tienam.

The patient was given pathogenic therapy, vancomycin and colistin. He remained febrile without improving...
his neuropsychological status. We also noticed the dehiscent cranial wound. Repeated lumbar cultures after 3 and 7 days respectively, showed the persistence of *A. baumannii*, which indicated the failure of the treatment.

Unfortunately, the patient died on day 32 from hospital admission.

In this case, some factors could have led to acquired *A. baumannii* meningitis, such as: admission to ICU, the neurosurgical intervention, the presence of venous and urinary catheters. Unfortunately, we were not able to test the sensibility of this bacterium to colistin, which proved to be effective with multidrug-resistant *A. baumannii*.

**DISCUSSION**

*Acinetobacter baumannii* is a polymorphic Gram-negative coccobacillus and tolerates long periods in both moist and dry conditions, and the easy acquisition of drug resistance makes it a great challenge in disease control.

The epidemiological profile suggests that it is of low virulence and the disease is dependent on significant host immunological impairment. It spreads through contact with contaminated medium (soil, meat and water), open wounds, skin, contact with patient, ventilators, sinks, bed rails, humidifiers.

Risk factors are represented by hospitalized patients and people with chronic lung disease, diabetes and weakened immune system. It seems plausible that this ubiquitous organism has acquired a vast array of pathogenesis islands to deal with this diversity [8]. Therefore, management of infections due to *A. baumannii* has become a real public health issue in many countries.

Virulence factors that influence the pathogenesis of *A. baumannii* are its surface motility on solid/semisolid media and the ability to form biofilm on abiotic or biotic surfaces. They also include the outer membrane protein A of *Acinetobacter baumannii* (OmpA), phospholipases, membrane polysaccharide components, penicillin-binding proteins (PB 7/80) and β-lactamase PER-1, metal acquisition system and outer membrane vesicles.

Intracranial infections including ventriculitis and meningitis caused by *A. baumannii* in the neurosurgery setting have led to challenging situations. The percentage of intracranial infection caused by *A. baumannii* in postoperative infection continuously increased in recent years. Regarding the frequency of meningitis with *A. baumannii*, in a review of four studies including 281 adult patients with hospital-acquired meningitis, 3.6% had meningitis due to *Acinetobacter* spp. [8-11]. One study on nosocomial meningitis in children reported that *Acinetobacter* accounted for 11.2% (20/178) of cases [12]. In USA and Taiwan, *Acinetobacter* ranked fifth place in nosocomial meningitis [8,10], while in Turkey it was the leading cause of Gram-negative post-neurosurgical meningitis [13,14].

Risk factors for post-neurosurgical meningitis are craniotomy, cerebrospinal leakage, incision infection, prolonged duration of surgery, surgery of sinus, external and internal ventricular drain, lumbar puncture, head trauma [15]. The median time to develop *Acinetobacter* meningitis after a neurosurgical procedure is 12 days (range 1-40 days) [16].

Meningitis which develops within 3 months after neurosurgical operation is defined as “post-neurosurgical meningitis” [17].

Post-neurosurgical *A. baumannii* meningitis was diagnosed after meeting the following criteria:

1) CSF culture: presence of *A. baumannii*;
2) CSF modifications: WBC increased, protein elevating and glucose decreasing;
3) The patient had fever (≥ 38°C), headache, vomiting, confusion, irritability or meningeal irritation;
4) The patient had a neurosurgical operation within 3 months [18].

Regarding treatment of *A. baumannii* meningitis, carbapenems used to be the empirical drugs for choice. However, more than 30% of *A. baumannii* strains were resistant to at least three kinds of antibiotics in many general hospitals [19]. Its resistance in the Middle East in general, and specifically in Lebanon, is increasing and many strains were found to be carbapenem resistant. In a study including hospitals from different Middle Eastern countries, it was found that *A. baumannii* isolated from a Lebanese hospital carried the oxa-58, oxa-23 and oxa-72
genes conferring the carbapenems resistance [20]. Another study, conducted in Hôpital-Dieu University Hospital in Beirut, showed an alarming increase in imipenem resistance among A. baumannii isolates from 7.7% in 2006 to 35.4% in 2009. In Lebanon, resistance was given by predominance of OXA-23, OXA-24, OXA-58, and OXA-143 and by spread of NDM-1 and GES-11 [21-28].

Colistin, an old antibiotic, was introduced in clinical use from 1950s, and abrogated in 1980s due to serious renal toxicity and neurovirulence. Maar tens et al. compared colistin with carbapenems and tobramycin, and found that colistin was still effective for A. baumannii in resistance of other antibiotics, and no difference in renal toxicity was revealed among these antibiotics [29]. Rolain et al. indicated that colistin worked through modifying the negative charges of outer membrane in Gram-negative bacteria [30].

This synergistic combination was studied also in Lebanon, at Saint George Hospital, Beirut, where it showed that combining colistin with carbapenems had high rates of additive effects [31]. The reason for the increased synergy with meropenem than imipenem might be that most carbapenemases target with greater affinity imipenem as compared to meropenem. The synergistic or additive effect might be influenced by the ability of colistin to disrupt the bacterial outer membrane and increase its permeability for carbapenems [25-36] and therefore stop the cross-linking of the new synthesized polymers. Combination therapy delays the emergence of bacterial resistance and specifically the rapidly developing resistance and heteroresistance toward colistin.

CONCLUSION

We recommend an increased awareness of the significant role of the multidrug-resistant bacteria Acinetobacter baumannii in nosocomial infections in the field of neurosurgery. It represents a serious challenge for epidemiologists, infection control professionals, and clinicians.

REFERENCES


