

MULTIDRUG-RESISTANT *ACINETOBACTER BAUMANNII* FROM NOSOCOMIAL URINARY TRACT INFECTION: A CASE REPORT

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ABSTRACT

The worldwide emergence of multidrug-resistant (MDR) *Acinetobacter baumannii* and high morbidity and mortality associated with this infection, present a challenge to clinicians. Emergence of resistance to multiple classes of antibiotics has limited the therapeutic options. In this case report, we describe the clinical presentation, risk factors, laboratory evaluation and therapeutic outcome of a 70-year-old patient who developed nosocomial urinary tract infection caused by MDR *A. baumannii* due to prolonged stay in intensive care unit, exposure to broad spectrum antibiotics and indwelling urinary catheter.

Keywords: *Acinetobacter baumannii*, Urinary tract infection, Nosocomial infection, Antimicrobial resistance.

INTRODUCTION

Acinetobacter baumannii is an oxidase negative, Gram-negative coccobacillus and it is ubiquitous in nature [1]. Its ability to form biofilm and survive in the environment for extended periods in adverse conditions and in the presence of antibiotics are the factors contributing to its successful colonization in hospital environment and on medical devices [2,3]. Chromosomally encoded cephalosporinase, high level of efflux pump expression combined with a minimally permeable cell membrane confer intrinsic resistance to several commonly used antibiotics [3]. In addition, *A. baumannii* is notorious for acquiring transferrable drug resistance by horizontal transfer of plasmids. Consequently, high end antibiotics are often the only drugs, which remain sensitive and are considered for therapy despite their higher cost and toxicity.

CASE REPORT

A 70-year-old male patient was admitted to the surgical ward with complaints of abdominal pain, nausea and vomiting of 2 days' duration along with decreased frequency of micturition. Patient was not known to have diabetes, hypertension, and tuberculosis. Emergency laparotomy was proposed as hollow viscus perforation was suspected, but deferred due to the poor general condition of the patient. Bilateral flank drains were inserted, and there was a bilious drain. 3 days later, an emergency laparotomy with omental patch closure with feeding jejunostomy was performed for duodenal perforation.

On the second post-operative day, pus swab yielded growth of - *Klebsiella pneumoniae* and *Escherichia coli*, which were both sensitive to imipenem, piperacillin - tazobactam and amikacin. However, the central line tip and blood culture were sterile. On the 15th post-operative day, urine sample was sent for culture and sensitivity. Urine was turbid, uncentrifuged deposit showed few pus cells and epithelial cells along with many bacilli. The specimen was also plated on to blood agar and cysteine lactose electrolyte deficient (CLED) agar. After 24 hrs of incubation, mucoid, dome-shaped colonies were seen on blood agar and mucoid non-lactose fermenting colonies were seen on CLED agar (Fig. 1a and b).

Preliminary examination of the isolate revealed that it was a Gram-negative cocco-bacilli, non-motile, catalase positive and oxidase

negative. This isolate was subjected to further biochemical reactions as per standard microbiological procedures and identified by conventional methods as - *Acinetobacter* spp [4]. This isolate was resistant to the entire panel of first-line drugs used for urinary tract infections (UTI) - amikacin, nalidixic acid, nitrofurantoin, co-trimoxazole, ceftriaxone and norfloxacin (Fig. 2).

Thus, the isolate was subjected to further testing by the imipenem, colistin and polymyxin-B and susceptibility were observed for colistin and polymyxin-B only (Fig. 3).

The isolate was subjected to further analysis by Vitek-2 compact by strictly following the manufacturer's instructions. The isolate was identified as *A. baumannii* complex. Antibiotic susceptibility testing was done using AST-N090 Vitek card, and it showed resistance to - aminoglycosides - gentamicin and tobramycin, fluoroquinolones - ciprofloxacin and levofloxacin, piperacillin - tazobactam and to imipenem. The resistance exhibited to the carbapenems qualified it to be called carbapenem-resistant multidrug-

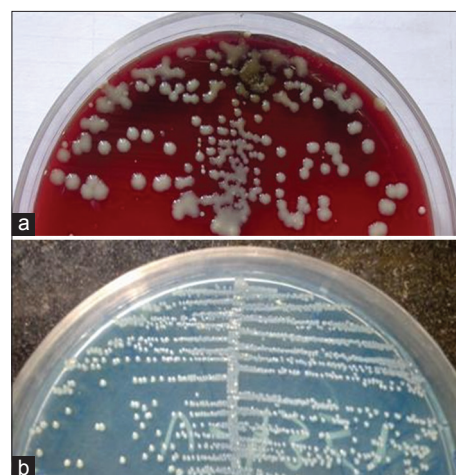


Fig. 1: (a and b) Colonies of *Acinetobacter baumannii* on blood agar (above) and cysteine lactose electrolyte deficient agar (below)

resistant (MDR) *Acinetobacter* spp. It showed intermediate susceptibility to tigecycline, the drug of choice in these situations (Fig. 4).

The patient was treated with colistin. Patient showed good clinical response to antibiotic therapy and was discharged.

DISCUSSION

A. baumannii is defined as a MDR phenotype if it exhibits resistance to three or more groups of antibiotics belong to anti-pseudomonal cephalosporins (ceftazidime or cefepime), anti-pseudomonal



Fig. 2: Antibiogram of *Acinetobacter baumannii* by disk diffusion method showing resistance to all first line antibiotics



Fig. 3: Antibiogram of *Acinetobacter baumannii* by disk diffusion method showing resistance to imipenem and sensitivity to polymyxin-B and colistin

bioMerieux Customer: System #:		Laboratory Report		Printed Oct 25, 2011 10:53 CDT Printed by: labsuper	
Isolate Group: U4351-1					
Bionumber: 0201010103500210 Selected Organism: <i>Acinetobacter baumannii</i>					
Susceptibility Information	Card: AST-N090		Lot Number: 390204820		Expires: Jul 23, 2012 13:00 CDT
	Completed: Oct 4, 2011 18:50 CDT		Status: Final		Analysis Time: 7.00 hours
Antimicrobial	MIC	Interpretation	Antimicrobial	MIC	Interpretation
Ampicillin/Sulbactam	>= 32	R	Amikacin		
Ticarcillin	>= 128	R	Gentamicin	>= 16	R
Piperacillin	>= 128	R	Tobramycin	>= 16	R
Piperacillin/Tazobactam	>= 128	R	Ciprofloxacin	>= 4	R
Ceftazidime	>= 64	R	Levofloxacin	>= 8	R
Ceftriaxone	>= 64	R	Tetracycline	>= 16	R
Cefoperazone/Sulbactam	>= 64	R	Tigecycline	4	I
Cefepime	>= 64	R	Colistin		
Imipenem	>= 16	R	Trimethoprim/Sulfamethoxazole	>= 320	R
Meropenem					
+= Deduced drug * = AES modified ** = User modified					
AES Findings:		Last Modified: Sep 5, 2011 10:31 CDT		Parameter Set: Copy of CLSI+Natural Resistance	
Confidence Level:	Consistent				

Fig. 4: Antibiogram of *Acinetobacter baumannii* by vitek-2 method

carbapenems (imipenem or meropenem), ampicillin/sulbactam, fluoroquinolones or aminoglycosides [1,5]. Whereas, pan-resistant *A. baumannii* shows resistance to polymyxin-B and/or colistin in addition to resistance to all above mentioned groups of antibiotics [1].

MDRA *A. baumannii* infections occur in patients with immunosuppression, serious underlying diseases, those subjected to invasive procedures, treated with broad-spectrum antibiotics or had indwelling catheters and prolonged stay in hospital/intensive care unit [5] infections due to *A. baumannii* are frequently found in nosocomial settings, where they are implicated in ventilator-associated pneumonia, UTIs and bacteraemia. Carbapenems have been used for treating infections caused by MDR *A. baumannii*. However, resistance to this group is now fast emerging. The prevalence of carbapenem - resistant *A. baumannii* causing UTI varies widely. Taneja *et al.* reported 22.3% of urinary isolates of *A. baumannii* were resistant to carbapenem while MDR and pan-resistant *A. baumannii* were 41.5% and 3.5% respectively [6]. In another study, 40.2% and 22.3% urinary isolates were MDR and pan-resistant respectively [7].

Colistin and tigecycline are antibiotic of choice in carbapenem - resistant MDR *A. baumannii* infections [5,6]. However, use both these drugs are associated with significant organ toxicity [8]. In this case, the patient had multiple risk factors, i.e. old age, long duration of hospital stay, exposure to broad-spectrum antibiotics and urinary catheterization. Especially, catheterization might have favoured biofilm mode of growth and the urinary colonization with additional antibiotic resistance. Furthermore, the isolate had intermediate susceptibility to tigecycline. Tigecycline is a newer antibiotic and the first member of glycylicycline. Resistance to tigecycline in *A. baumannii* varies from 0% to 66% [6]. In contrast, colistin is an older drug, introduced in the 1960s [8]. However, severe nephrotoxicity and neurotoxicity precludes its routine clinical use [8]. In our patient, colistin was the only therapeutic choice, and it showed good response. Currently, few studies have reported successful outcome with colistin administered locally at the site of infection, as aerosol inhalation for pneumonia and continuous urinary irrigation for UTI [8,9]. However, further research is essential to support its clinical use.

In conclusion, *A. baumannii* is an important opportunistic agent of nosocomial UTI, especially in patients with longer hospitalization, antibiotic exposure, urinary catheterization and decreased immunity. High antimicrobial resistance and patient co-morbidities limit therapeutic choices. Hence, alternative therapeutic options are urgently needed to treat a patient with *A. baumannii* infection.

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