

Multidrug resistant and extensively drug resistant *Acinetobacter baumannii* hospital infection associated with high mortality: A retrospective study in the Pediatric Intensive Care Unit

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Abstract

Background Multidrug resistant (MDR) and extensively drug resistant (XDR) *Acinetobacter baumannii* presents challenges for clinical treatment and causes high mortality in children. We aimed to assess the risk factors for MDR/XDR *Acinetobacter baumannii* infection and for overall mortality in this patient population.

Methods This retrospective study included 102 pediatric patients who developed MDR/XDR *Acinetobacter baumannii* infection in the pediatric intensive care unit (PICU) of Shanghai Children's Hospital in China from December 2014 to May 2018. Clinical presentations and outcome of the patients were analyzed. The primary outcome was overall mortality. Secondary outcomes included the 28-day mortality and the length of hospital stay.

Results Of the 102 patients (63 males and 39 females; mean age: 51.79 months), the overall mortality rate was 29.41%. 18(17.64%) had bloodstream infections; 4(3.92%) for which cerebrospinal fluid (CSF) cultures were positive; 14(13.73%) of them got positive cultures in aseptic fluid; 10 (9.8%) had central catheter-associated bloodstream infections; lower respiratory isolates (56/102) accounted for 54.9% of all patients. Non-survival patients appeared to have a lower NK cell activity 5.87%(2.43%, 8.93%) vs. 8.45%(4.51%, 14.61%), $P = 0.011$), higher CD4 + T cell ratio (35.32% (29%, 47.81%) vs. 31.97% (20.52%, 38.22%), $P = 0.045$), and higher serum level of interleukin-6 (IL-6, 235.51(0.1, 4172.77) pg/ml vs. 0.1(0.1, 110.92) pg/ml, $P = 0.028$), interleukin-8 (IL-8, 22.73(3.14, 540.12) vs. 0.1(0.1, 25.85)pg/ml, $P = 0.03$), and interleukin-10(10.82(0.1, 83.29)pg/ml vs. 6.05(0.1, 21.81)pg/ml) were observed. Multivariate logistic analysis indicated that high serum level of BUN (RR, 1.216, 95%CI, 1.27-2.616; $P = 0.001$) and high serum level of Cr (RR, 1.823, 95%CI, 0.902-0.980; $P = 0.004$) were associated with high risk of overall mortality in MDR/XDR *Acinetobacter baumannii* infected patients.

Conclusion MDR/XDR- *Acinetobacter baumannii* is an important opportunistic pathogen that causes nosocomial infection in PICU with a rather high mortality. The incidence increased in recent years, ineffective management, immune dysfunction, acute kidney injury contributed to the risk of death.

Introduction

Acinetobacter baumannii is a Gram-negative coccobacillus that has a remarkable ability to acquire antibiotic resistance which embarrassed for decades for causing persistent nosocomial infections^[1] The mortality rate varies from 18.26% to 88.7% depending on the infection source^[2,3]. The propensity of *Acinetobacter baumannii* to be multidrug-resistant (MDR) or extensively drug-resistant (XDR) presents therapeutic challenges^[4,5]. Invasive operations such as intra trachea mechanical ventilation, inserted invasive devices, intensive care unit stay, recent surgery, use of broad-spectrum antibiotics, ineffective management, and septic shock at diagnosis are reported as risk factors for colonization or infection by MDR *Acinetobacter baumannii* and higher mortality^[6,7].

The incidence of infections due to MDR *Acinetobacter baumannii* in Southeast Asia were higher compared to other areas^[8,9], According to a five-year case-control study from Southwest China, it was the severity of illness (high APACHE II score and MODS) that highlighted the mortality of patients with nosocomial *Acinetobacter baumannii* bacteremia^[10]. The problems for pediatrician in China are polymyxins cannot be obtained, tigecycline only became available in late 2016, and had limited experience in manage these young patients^[11,12].

In pediatric critically ill patients, underlying diseases, immune deficiency, and invasive operations all contributed to the higher incidence of multidrug resistant *Acinetobacter baumannii* infection. Multidrug resistant *Acinetobacter baumannii* infection is responsible for a high mortality rate among neonates in the NICU^[13]. According to a report from Seoul National University Children's Hospital, carbapenem nonsusceptibility, neutropenia, and prolonged ICU stay as independent risk factors for mortality due to *Acinetobacter baumannii* bacteremia in PICU patients^[14]. However, no study has examined these risk factors for mortality or clinical features due to MDR/XDR *Acinetobacter baumannii* infection in critically ill pediatric patients in China. Thus, the objective of this study is to assess the outcome and risk factors in children with hospitalized MDR/XDR *Acinetobacter baumannii* infection in PICU.

Material And Methods

This retrospective study included pediatric patients who developed multidrug resistant and extensively drug resistant *Acinetobacter baumannii* infection in the PICU of Shanghai Children's Hospital in China from December 2014 to May 2018. We defined colonization as positive culture results from clinical isolated samples without clinical manifestation of infection.

Identification of MDR/XDR-*Acinetobacter baumannii* was confirmed by positive blood, sputum, aseptic fluid or catheter culture as well as symptoms and signs of infection. Susceptibility testing to the tested antibiotics was determined according to the Clinical and Laboratory Standards Institute (CLSI) interpretive criteria, and MDR/XDR-*Acinetobacter baumannii* isolates were subjected to minimum inhibitory concentration (MIC) for colistin^[15]. MDR was defined as acquired non-susceptibility to at least one agent in three or more antimicrobial categories, XDR was defined as non-susceptibility to at least one agent in all but two or fewer antimicrobial categories (i.e. bacterial isolates remain susceptible to only one or two categories) and PDR was defined as non-susceptibility to all agents in all antimicrobial categories^[16]. We defined blood stream infection, ventilator associated pneumonia (VAP), central catheter-associated bloodstream infection, meningitis, intraabdominal infection, and urinary tract infection according to IDSA and CLSI criteria^[17,18]. Tracheal aspirate specimens were qualified on a quantification (cfu/mL) from the culture, the colony quantification less than 1×10^3 cfu/mL was considered as possible colonization.

Clinical conditions and outcomes

Clinical, biological and treatment data were obtained retrospectively from the patients' medical records. Underlying illnesses at the time of admission to the PICU were classified and pediatric risk of mortality (PRISM) scores were measured at the time of PICU admission. The overall mortality was defined as the survival status when the patient discharged. The 28-day mortality was defined as the survival status after diagnosis with MDR/XDR *Acinetobacter baumannii* infection. The primary outcome was overall mortality. Secondary outcomes included the 28-day mortality and the length of hospital stay.

Statistical analysis

Continuous variables were presented as mean±standard deviation for normal distribution data and as median (interquartile range) for abnormal distribution data. Comparative analysis was conducted using an independent sample nonparametric test. The *chi-square* test or *Fisher's* exact test was conducted. The Mann-Whitney test was used for continuous variables. Variables showing $p < 0.05$ in a univariate analysis were included in a multivariate analysis, which was performed by stepwise logistic regression. All statistical analyses were performed with the Statistical Package for the Social Sciences, version 17.0 (SPSS Inc., Chicago, IL, USA).

Results

Clinical manifestations

During the study period of December 2014 and May 2018, a total of 102 episodes of MDR/XDR *Acinetobacter baumannii* in 102 patients were identified, their demographics and clinical features were summarized in Table 1. The overall incidence of MDR/XDR *Acinetobacter baumannii* was 0.48 cases/1000 patient-days, and we observed a continually increased distribution of cases during the study period. In our study, the overall mortality was 29.4% (30/102), while the 28-day mortality was 15.69% (16/102). Both the incidence and mortality were growing higher (Table 2). In 18 cases (17.65%), surveillance blood cultures became positive after clinical manifestation appeared, while 10 catheter (9.8%), 4 cerebrospinal fluid (3.92%) and 14 (13.73%) aseptic fluid were identified MDR/XDR *Acinetobacter baumannii* positive. 19 (18.63%) episodes had tracheal aspirate cultures positive, and the other 37 (36.27%) had positive sputum cultures for MDR/XDR *Acinetobacter baumannii*. The mean age was 51.79 months and male predominated (61.76%). 51 patients had surgery (50%) and 70 treated with corticosteroids (68.6%), all these patients received broad-spectrum antibiotics treatment for more than one week. More patients in non-survival group inserted arterial catheters (96.67% vs. 80.56%, $P = 0.036$), endotracheal intubation (96.67% vs. 79.17%, $P = 0.027$) and experienced operation (66.67% vs. 43.06%, $P = 0.03$).

Organ function and immune status of MDR/XDR *Acinetobacter baumannii* infected patients

Table 3. listed the organ function of patients at diagnose of MDR/XDR *Acinetobacter baumannii* infection, as the concern of the importance of immunologic function in the process of anti-inflammatory pathophysiological, the indicators of immune function as well as serum cytokine levels were recorded in

Table 4. It seems that no significant differences lies in the organ functions at diagnose of MDR/XDR *Acinetobacter baumannii* infection, but non-survival patients appeared to have a lower NK cell activity 5.87%(2.43%, 8.93%) vs. 8.45%(4.51%, 14.61%), $P = 0.011$), higher CD4⁺ T cell ratio (35.32% (29%, 47.81%) vs. 31.97% (20.52%, 38.22%), $P = 0.045$), at the same time, a higher serum level of interleukin-6 (IL-6, 235.51(0.1, 4172.77) pg/ml vs. 0.1(0.1, 110.92) pg/ml, $P = 0.028$), interleukin-8 (IL-8, 22.73(3.14, 540.12) vs. 0.1(0.1, 25.85)pg/ml, $P = 0.03$), interleukin-10 (10.82(0.1, 83.29) pg/ml vs. 6.05(0.1, 21.81)pg/ml) was noticed, which revealed a stronger inflammatory response in this group of patients.

Drug resistance results of MDR/XDR *Acinetobacter baumannii*

Bacteriological findings are summarized in Table 6. (Antimicrobial resistance in 102 *Acinetobacter baumannii* clinical isolates). MDR/XDR *Acinetobacter baumannii* was resistant to carbapenems, aminoglycosides, most cephalosporins and sulfa drugs. Their resistance rates were more than 75%. Only tigecycline, polymyxin had a high sensitivity to MDR/XDR *Acinetobacter baumannii* (86.3%, 92.2%). The resistance rates of cefoperazone-sulbactam was 41.2%, while had an intermediary rate of 47.1%.

Outcomes

All the patients received active antibiotic treatment within 7 days. Regarding treatment given within 7 days, based on the in vitro susceptibility test, cefoperazone/sulbactam, tigecycline, polymyxin were the recommended antimicrobial agents for XDR *Acinetobacter baumannii* eradication. Before 2017, the patients received cefoperazone/sulbactam combined with carbapenems after appearance of MDR/XDR *Acinetobacter baumannii*. During January to December 2017, as tigecycline became available, 28 patients were treated by tigecycline combined with cefoperazone/sulbactam or fosfomycin. Although 63.3% patients in the non-survival group treated by tigecycline (15.2% in survival group), it seems that a delayed applying of this antibiotic ($0.56 \pm 0.27d$ vs. $7.07 \pm 4.93d$ after positive culture reports received, $p = 0.0025$). Totally, 72 patients (70.59%) improved and 30 patients (29.41%) dead.

Discussion

Acinetobacter baumannii, an aerobic, gram-negative bacillus which is widely distributed in nature, is notorious for its remarkable Ability to acquire antibiotic resistance. As a result, it causes persistent hospital-acquired infections^[1]. In our present study, the incidence density of MDR/XDR- *Acinetobacter baumannii* at our facility was 0.48 cases/1000 patient-days, which was lower than the incidence of 0.47 cases/1000 patient-days from reported in ICU patients^[19] and lower than the incidence of another PICU that literature reports^[2]. In our study, more arterial catheters inserted and higher rate of mechanical ventilation were observed in non-survival patients, and more surgical operations were operated in this group of patients. These results indicated that patients characterized by more severe underlying diseases and more complex medical history will cause severe infection of *Acinetobacter baumannii* while MDR/XDR *Acinetobacter baumannii* strains appeared.

From our single-center data, ventilator-associated pneumonia (VAP) occupied the major complication in pediatric critically ill patients who MDR/XDR *Acinetobacter baumannii* infection with an incidence rate of 54.9% (56/102). Both blood stream and CNS infection caused relative high mortality. Due to an increase in invasive operations as well as the severity of primary conditions of critically ill patients, the overall incidence of MDR/XDR *Acinetobacter baumannii* infection increased.

MDR/XDR *Acinetobacter baumannii* is resistant to most pediatric antimicrobial agents, including penicillins, most cephalosporins, carbapenems, aminoglycosides, and sulfa drugs. XDR *Acinetobacter baumannii* tends to develop resistance to multiple antimicrobial agents through degrading enzymes targeting β -lactams, increasing the expression of Ampc enzyme, modifying enzymes targeting aminoglycosides and alteration to the binding sites for quinolones, producing OXA-23 carbapenem enzyme, decreasing the expression of outer membrane pore channel protein, efflux pump system hyperactivity, and loss of PBPs^[20]. In our study, the susceptibility results showed that the drug resistance rates of MDR/XDR *Acinetobacter baumannii* to beta-lactam antibiotics were more than 75% except for cefoperazone/sulbactam (42%). This phenomenon could be explained that sulbactam may be combined with the important PBPs or change the outer membrane permeability of G⁻ bacteria, thus making the beta-lactamase leakage while increasing the opportunity of other antibacterial drugs into biomass^[21]. As the contribution of sulbactam, another antibiotic contains sulbactam-ampicillin/sulbactam, showed lower drug resistance rate (79.4%) than other beta-lactam antibiotics. During the period of neither tigecycline nor polymyxin was available, a combination therapy of cefoperazone/sulbactam or fosfomycin and carbapenems was applied.

Since the safety of children's medication has been put in an important place, our choice of antibiotics faced a lot of limitations. Although no evidence in the literature that combination therapy is prior to single drug for infection with MDR/XDR *Acinetobacter baumannii*, some in vitro studies have shown that certain drug combinations are synergistic^[22,23]. In the study by Singkham-In U et al^[24], the combination of 1× MIC of imipenem (16-64 mg/L, and 128mg/L of isolate A10) and 1× MIC of fosfomycin (128-256mg/L) showed synergism and bactericidal effect against most *Acinetobacter baumannii* isolates. Fosfomycin, when combined with imipenem, may enhance the inhibition of bacterial cell wall synthesis. So, from this point of view, the combination of fosfomycin and carbapenems in the past few years benefited a number of our patients. In our study, from Jan 2017, 28 patients received tigecycline treatment. Though it seemed that 63.3% patients in the non-survival group treated by tigecycline while 15.2% in survival group, what cannot be ignored was the initiation of tigecycline treatment in non-survival group was 6.5 day later than survival group positive culture report received, which might have an impact on higher treatment failure rate.

Acinetobacter baumannii was transported together by infiltrating neutrophils. Go Kamoshida et al.^[25] found that *Acinetobacter baumannii* exploits human neutrophils by adhering to and inducing IL-8 release for bacterial portage. *Acinetobacter baumannii* stimulation IL-8 plays a critical role in enhancing the migration of *Acinetobacter baumannii*-adhering neutrophils, the migration of *Acinetobacter baumannii*

was suppressed when the infiltration of neutrophils was suppressed by inhibiting IL-8^[26]. Through Toll-like receptor 4 (TLR4) and CD14, *Acinetobacter baumannii* lipopolysaccharide leads the production of the neutrophil chemotactic factor IL-8 and the proinflammatory cytokine TNF- α ^[27]. In our MDR/XDR *Acinetobacter baumannii* infected patients, a higher serum level of IL-8 (15.25 (1.62, 47.215)pg/mL vs. 0.1(0.1, 22.99)pg/mL) was detected in the non-survival group, this phenomenon might explain that *Acinetobacter baumannii* in this group seems to spread throughout the body more easily.

Studies have shown that purified TLR2 ligands from *Acinetobacter baumannii* are immunostimulatory^[28]. CD4⁺ T cells play important role in the Th1/Th2 paradigm, participate in inflammation^[29], AbOmpA of *Acinetobacter baumannii* (AbOmpA) is a major porin protein in the outer membrane and is partly responsible for apoptosis of eukaryotic cells. Jun SikLee et al^[30] co-cultured CD4⁺ splenic T cells with AbOmpA-treated DCs, and found AbOmpA directs CD4⁺ T cell differentiation towards a Th1 response. In our research, the patients in non-survival group appeared a higher CD4⁺ T cell ratio than survival patients, which revealed that a persistent neutrophil activation and accumulation in tissue that caused inflammation spread.

Our study had several limitations. First, because data were collected retrospectively from medical records, some parameters had to be inferred from the charts. Susceptibility testing had limitations, fosfomycin was not included, which resulted in a lack of antibiotic susceptibility evidence when we made treatment decisions.

In conclusion, our study suggests that MDR/XDR- *Acinetobacter baumannii* is an important opportunistic pathogen that causes nosocomial infection in PICU with a rather high mortality. The incidence increased in recent years, ineffective management, immune dysfunction, AKI contributed to the risk of death.

Abbreviations

MDR:multidrug resistant

XDR: extensively drug resistant

PICU: pediatric intensive care unit

CSF: cerebrospinal fluid

CLSI: Clinical and Laboratory Standards Institute

PRISM \square : pediatric risk of mortality \square

AKI: acute kidney injury

ALT: alanine transaminase

Cl: cardiac index

BUN: blood urea nitrogen

Cr: creatinine

MIC: minimum inhibitory concentration

VAP: ventilator-associated pneumonia LA: lactate

EF: left ventricular ejection fraction

PaO₂/FiO₂: the ratio of the partial pressure of oxygen in arterial blood (PaO₂) to the inspired oxygen fraction (FiO₂)

TLR4: Toll-like receptor 4

Declarations

Ethical approval and consent to participate: Not applicable

Conflict of Interest: Not applicable.

Availability of data and materials: Our present study was a retrospective observational study. All the data were obtained from medical records of patients. The raw data was submitted as a supplementary data.

Competing interests: The authors have declared that no competing interests exist.

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Author's contributions: YCZ and YJS designed the study. JYS, TS managed data and its quality. JYS, YJS and CXW performed the statistical analysis. FW, YPZ and HJM participated in the data interpretation. JYS, TS, YC drafted the manuscript. CXW and YCZ contributed substantially to its revision. All authors read the manuscript carefully and approved the final version.

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Tables

Table 1 The Basic Characteristics of MDR/XDR *Acinetobacter baumannii* patients

Characteristics		Survivors	Nonsurvivors	P value
		n=72(70.59%)	(n=30(29.41%)	
Age, month (M(Q1,Q3))		30 (9.6, 88.75)	42 (9.825, 102.75)	0.404
Male/Female (n=)		44/28	19/11	0.835
Underlying disorders				
	Pneumonia(n%)	61, 84.7%	25, 83.3%	0.862
	Meningitis(n%)	1, 1.38%	6, 20%	<0.001*
	Leukemia/	6, 8.33%/	6, 20%/	<0.001
	Solid tumor(n%)	10, 13.89%	11, 33.3%	
	Trauma(n%)	3, 4.17%	0, 0%	0.553
	Sepsis(n%)	65, 90.27%	25, 83.33%	0.321
	Peritonitis(n%)	8, 11.11%	8, 26.67%	0.049*
	Cardiovascular disease(n%)	8, 11.11%	5, 16.67%	0.627
	Hepatic disorder(n%)	5, 6.94%	2, 6.67%	0.813
	Others(n%)	10, 13.89%	5, 16.67%	0.718
WBC count(*10 ⁹ /L)	M(Q1,Q3)	8 (6.01, 13.69)	5.1 (1.64, 14.78)	0.747
Neutropenia(*10 ⁹ /L)	M(Q1,Q3)	4.89 (3.57, 9.23)	3.37 (0.61, 11.05)	0.717
CRP(mg/L)	M(Q1,Q3)	37 (10, 74)	27 (16, 94.5)	0.298
PCT (ng/L)	M(Q1,Q3)	0.48 (0.18, 5.08)	1.56 (0.53, 8.27)	0.505
Lactate(mmol/L)	M(Q1,Q3)	2.2 (1.53, 3.48)	2.2 (2, 3.5)	0.955
PRISM	mean±SD	6.73±5.19	8.3±4.76	0.159
Shock		18, 25%	16, 53.33%	0.006*
PaO ₂ /FiO ₂	mean±SD	269±112.76	220.97±147.55	0.084
Exposure factors				
	Steroid (n=, %)	49, 68.06%	21, 70%	0.289
	Immune suppressor (n=, %)	11, 15.27%	8, 26.67%	0.263
	Length of hospital day(day,M(Q1,Q3))	24 (14, 48.75)	31.5 (24.75, 41.5)	0.606
	Length of PICU day(day,M(Q1,Q3))	14(8,23.5)	22.5(13.25, 41)	0.459
	Length of mechanical ventilation(day, M(Q1,Q3))	6 (1, 15)	10 (6, 25)	0.212
	Central vein catheter (n=, %)	69, 95.83%	30, 100%	0.256
	Arterial catheter (n=, %)	58, 80.56%	29, 96.67%	0.036*
	Intra trachea intubation (n=, %)	57, 79.17%	29, 96.67%	0.027*
	Operation (n=, %)	31, 43.06%	20, 66.67%	0.03*

*p<0.05

Table 2 Type of initial infection and the infection trends over the years

	2014-2015	2016	2017	2018
	(n =15)	(n =18)	(n =34)	(n =35)
Bloodstream (n)	4	3	5	6
Central nervous infection(n)	1	0	1	2
Pneumonia (n)	4	8	23	21
Central-catheter	0	3	3	4
associated (n)				
Aseptic fluid (n)	6	4	2	2
Incidence (%)	14.71%	17.65%	33.33%	34.31%
Overall mortality (n%)	6[40%]	4[22.22%]	7[20.59%]	13[38.24%]

Table 3. Organ function of patients at diagnose of MDR/XDR Acinetobacter baumannii infection

Organ	Survivors	Nonsurvivors	P value
Cardiovascular			
MAP(mmHg) (mean±SD)	64.02±12.99	58.13±15.21	0.052
EF(%) (mean±SD)	65.14±7.06	66.78±3.09	0.251
CI(L/min.m2) ((M(Q1,Q3))	3.8 (3.55, 4.25)	4 (3, 6.1)	0.342
LA(mmol/L) (mean±SD)	3.12±2.9	3.09±2.42	0.955
Respiratory			
PEEP(cmH2O) (mean±SD)	4.84±1.31	6.24±2.02	<0.001*
Tidal volume(ml/kg) (mean±SD)	8.11±0.92	7.98±0.83	0.571
PaO2/FiO2 (mean±SD)	269±112.76	269±113.55	0.084
Plateau pressure (cmH2O) ((M(Q1,Q3))	10 (9, 12)	11 (10, 13)	0.684
Hepatic			
TBIL(mmol/L) ((M(Q1,Q3))	6.26 (4.14, 9.76)	10.86(6.64, 18.85)	0.443
ALT(U/L) ((M(Q1,Q3))	25.5 (14, 48)	20 (11.75, 34.75)	0.7
Renal			
BUN(mmol/L) (mean±SD)	4.22±2.89	8.18±9.52	0.002*
Cr(umol/L) ((M(Q1,Q3))	22 (17, 36.25)	21 (17.75, 33.25)	0.391
Nervous			
Glasgow(mean±SD)	10.97±2.93	10.03±3.14	0.152
Gastric-intestine			
Intra-abdominal pressure(cmH2O) (mean±SD)	7.46±4.15	8.95±5.01	0.227

*p<0.05

Table 4. The immunologic function and serum cytokines levels in the patients

Characteristics	Survivors	Nonsurvivors	P value
NK (%) (M(Q1,Q3))	8.45(4.51, 14.61)	5.87 (2.43, 8.93)	0.011*
IgG(g/L) (M(Q1,Q3))	10.85 (5.88, 13.38)	8.98 (5.97, 16.65)	0.926
IgA(g/L) (M(Q1,Q3))	0.66 (0.29, 1.41)	0.55 (0.37, 1.91)	0.691
IgM(g/L) (M(Q1,Q3))	0.96 (0.55, 1.39)	0.67 (0.27, 1.06)	0.026*
CD4+(%) (M(Q1,Q3))	31.97 (20.52, 38.22)	35.32 (29, 47.81)	0.045*
CD8+(%) (M(Q1,Q3))	23.53 (15.7, 37.17)	29.26 (20.79, 43.67)	0.35
CD19+(%) (M(Q1,Q3))	26.66 (9.42, 39.46)	16.65 (9, 28.24)	0.15
IL-6(pg/ml) (M(Q1,Q3))	0.1 (0.1, 110.92)	235.51 (0.1, 4172.77)	0.028*
IL-8(pg/ml) (M(Q1,Q3))	0.1 (0.1, 25.85)	22.73 (3.14, 540.12)	0.03*
IL-10(pg/ml) (M(Q1,Q3))	6.05 (0.1, 21.81)	10.82 (0.1, 83.29)	0.04*

*p<0.05

variable	OR (95%CI)	P value
BUN (mmol/L)	1.823 (1.270-2.616)	0.001*
Cr (mmol/L)	0.94 (0.902-0.980)	0.004*
P < 0.05*		

Table 5
Logistic regression of risk factors

Table 6. Antimicrobial resistance in 102 *Acinetobacter baumannii* clinical isolates

Antibiotics	Resistance	Intermediary	Sensitive
	n=%	n=%	n=%
Gentamicin	102, 100%	0, 0%	0, 0%
Amikacin	89, 41.1%	3, 2.9%	10, 9.8%
Ceftriaxone	92, 90.2%	10, 9.8%	0, 0%
Cefepime	96, 94.1%	6, 5.9%	0, 0%
Ceftazidime	95, 93.1%	7, 6.9%	0, 0%
SMZCo	95, 93.1%	2, 1.9%	5, 4.9%
Ampicillin/sulbactam	81, 79.4%	11, 10.8%	10, 9.8%
Cefoperazone/sulbactam	42, 41.2%	48, 47.1%	12, 11.7%
Piperacillin/tzobartan	94, 92.2%	6, 5.9%	2, 1.9%
Imipenem	97, 95.1%	2, 1.9%	3, 2.5%
Meropenem	94, 92.2%	2, 1.9%	6, 5.9%
Tetracycline	3, 2.9%	13, 12.75%	88, 86.3%
Polymyxin	8, 7.8%	1, 0.98%	94, 92.2%
Minocycline	53, 51.96%	38, 37.3%	11, 10.78%

Supplementary Files

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