Research Article

Prevalence and resistance pattern of Acinetobacter species in PICU and NICU in a tertiary care Paediatric hospital in Bangalore

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Abstract

Objectives: Acinetobacter species are one of the most frequent nosocomial pathogen and can cause a wide range of infections, including bacteremia, pneumonia, urinary tract infection, peritonitis, etc. This organism is becoming resistant to a large group of antibiotics, especially β-lactam antibiotics and also carbapenems. **Aim:** To determine the prevalence of Acinetobacter species in the patients of NICU and PICU of a tertiary care paediatric hospital and also to study their resistance pattern. **Materials and Methods:** This is a retrospective study done over a period of 12 months from January 2016 to December 2016. The Acinetobacter species isolates by all the clinical samples from NICU and PICU were identified by colony characteristics and biochemical reactions. The resistance patterns of these isolates were identified using various antibiotics by Kirby-Bauer disc diffusion test as per CLSI guidelines. Their antibiogram data and a clinical correlation was made to assess their pathogenic status and mode of acquisition. **Results:** Acinetobacter species was isolated in 280(30.7%) samples out of 911(17.75%) culture positive isolates from a total of 5131cultures from NICU and PICU. Maximum isolates were from Tracheal aspirate 93 (57%) followed by pus (52.71%) and blood 88(19.4%). The organism showed high rate of resistance to cefazolin (96.5%) ampicillin (91.8%), amoxyclav (85.2%) ceftriaxone (88.5%), piperacillin (82.9%), ceftazidime (77.5%), amikacin (75.2%) and ciprofloxacin (86.9%). The organism showed moderate resistance to Imipenem(68%) , meropenum (65%) and colistin (60%). **Conclusion:** In this study, Acinetobacter species was resistant to many drugs including imepenum and meropenum and there was a significant relationship between patients on mechanical ventilation, length of hospital stay and drug resistance.

Key words: Acinetobacter species, Prevalence, Resistance patterns

Introduction

Hospital acquired infections are a major challenge to patient safety. It is estimated that, a total of 1.7 million hospital acquired infections occurred (4.5 per 100 admissions every year), and almost 99,000 deaths were associated with a hospital acquired infection, making hospital acquired infections the sixth leading cause of death in the United States Hospital acquired infections are most commonly associated with invasive medical devices or surgical procedures [1].

Acinetobacter spp. have emerged as particularly important organisms in intensive care units (ICUs), and this is probably related, at least in part, to the increasingly invasive diagnostic and therapeutic procedures used in hospital ICUs in recent years[2]. Global data reveals that multidrug-resistant Acinetobacter baumannii is emerging as a common hospital-and community-acquired infection that is difficult to treat. It is a very resistant and aggressive organism that infects patients with weakened defenses like ICU patients and those with invasive devices [3].

In large surveillance studies from the United States, between 5 and 10% of cases of ICU-acquired pneumonia were due to Acinetobacter baumannii. Clinical isolates of *Acinetobacter* species initially retained at least partial susceptibility against the 3rd and 4th generations viz cephalosporins, fluoroquinolones, semisynthetic aminoglycosides, carbapenems and 100%

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susceptibility to imipenem. However, during late 1980 and 1990s, worldwide emergence and spread of *Acinetobacter* strains resistant to imipenem further limited therapeutic alternatives.

This organism has multiple mechanisms for resistance including an impermeable outer membrane, enzymes which breakdown of antibiotics especially AmpC β-lactamases, class D OXA-type and class B metallo-β-lactamases which allow the organism to resist carbapenems, porin channels alterations as well as efflux pumps, and other genetic changes that may lead to resistance to fluoroquinolones.

All A. baumannii strains are chromosomally encoded AmpC cephalosporinases also known as Acinetobacter-derived cephalosporinases (ADCs). Extended-spectrum β-lactamases (ESBLs) from the Ambler class A group have also been described for A. baumannii, but assessment of their true prevalence is hindered by difficulties with laboratory detection, especially in the presence of an Amp C. More recent focus has been on VEB-1, which disseminated throughout hospitals in France (clonal dissemination) and was also recently reported from Belgium and Argentina (VEB-1a).

Other ESBLs identified in A. baumannii include TEM-92 and -116 from Italy and The Netherlands, respectively, and SHV-12 from China and The Netherlands. Also, CTX-M-2 and CTX-M-43 have been described from Japan and Bolivia, respectively [4].

Rational use of antimicrobial agents is critically important to prevent *Acinetobacter* infections as well as to avoid poor outcomes.

Therefore early detection of such organisms is necessary for timely implementation of strict infection control practices and treatment with alternative antimicrobials.

Materials and Methods

Source of data: This is a retrospective study done over a period of 12months from January 2016 to December 2016. The Acinetobacter species isolates by all the clinical samples from NICU and PICU patients were included in the study. The study was conducted in the department of Microbiology, Indira Gandhi institute of child health, Bangalore.

Inclusion Criteria: Acinetobacter species isolated by all the clinical samples from NICU and PICU patients were included in the study

Exclusion criteria

- Samples which yielded other organisms other than Acinetobacter species were not included in the study.
- Samples which were not collected under aseptic conditions and also which were inadequate in quantity were rejected

Methodology

This is a retrospective study done over a period of 12months from January 2016 to December 2016. The Acinetobacter species isolates by all the clinical samples from NICU and PICU patients were included in the study. These isolates were identified from samples collected under aseptic conditions which were inoculated on MacConkey agar & Blood agar.

The plates were incubated aerobically at 37^0C for 24-48 hrs. Presumptive identification was done on the basis of colony characteristics, Gram staining, catalase test, oxidase test, nitrate reduction test, oxidative/ fermentative test.

On MacConkey agar colonies of *A. baumannii* appeared as non-lactose fermenter and on blood agar colonies were about 1 to 2 mm in diameter, non-pigmented, domed, and muciod, with smooth to pitted surfaces. *A. baumannii* were oxidase negative and non motile.

All these species of *Acinetobacter* were then screened for antibiotic sensitivity by Kirby- Bauer disk-diffusion method on Muller Hinton Agar according to CLSI (Clinical Laboratory Standard Institute) guidelines.

Clinical details of all patients whose cultures were positive for *Acinetobacter* species were collected. All the data were analysed.

Results

A total number of cultures obtained were 5131 from patients admitted in NICU and PICU in our hospital. In this 911 (17.75%) were culture positives and 280 (30.7%) were Acinetobacter species isolated out of which 106 (37.8%) from NICU and 174 (62.1%) from PICU.

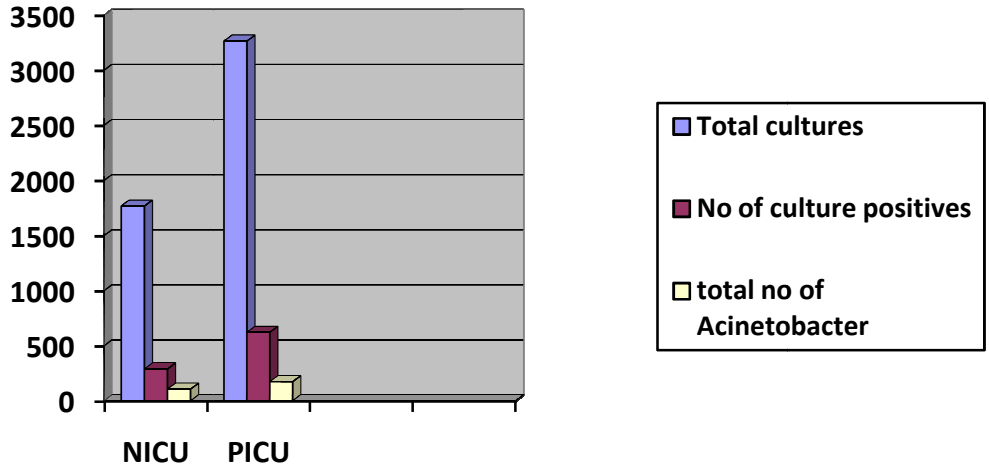

Fig-1: Total number of culture positives and Acinetobacter species in NICU and PICU

Acinetobacter species were identified by colony morphology and biochemical tests. On MacConkey agar colonies of *A. baumannii* appeared as non-lactose fermenter and on blood agar colonies were about 1 to 2 mm in diameter, non-pigmented, domed, and muciod, with smooth to pitted surfaces. *A. baumannii* were oxidase negative and non motile.

Acinetobacter species was majorly isolated from Tracheal aspirate followed by pus and blood. In the total 3750 samples of blood cultures sent, 452(12%) were culture positive in that 88(19.4%) were Acinetobacter species. The total pus cultures were 197, the culture positive were 129(29.89%) and in that Acinetobacter species was 68(52.71%). In Tracheal aspirate out of 235 cultures 163(69.36%) were culture positives and Acinetobacter was 93(57%). Acinetobacter was also isolated from Endotracheal tip 16(80%) and CSF 9 (30%).

Table-1: Distribution of Acinetobacter species in different clinical specimens.

Type of isolate	Total number of isolates	Total number of culture positives	No of Acinetobacter isolate
Blood	3750	452(12%)	88(19.4%)
Pus	197	129(29.89%)	68(52.71%)
CSF	489	30(6.13%)	9(30%)
Tracheal aspirate	235	163(69.36%)	93(57%)
Urine	420	217(51.66%)	6(2.7%)
E.T tip	40	20(50%)	16(80%)

Table -2: Distribution of Acinetobacter species in 2016 in our hospital.

	Jan	Feb	Mar	April	May	June	July	Aug	Sep	Oct	Nov	Dec	Total
Blood	4	7	7	4	2	8	4	7	9	4	20	12	88
Pus	3	4	6	8	3	2	7	5	10	3	13	4	68
URINE	0	0	0	0	0	1	3	1	0	0	1	0	6
ET tube	2	0	2	0	2	3	0	1	0	2	2	2	16
Tracheal aspirate	6	10	4	3	9	7	11	11	4	10	10	8	93
CSF	0	0	2	1	1	0	2	0	1	0	0	2	9

The distribution of acinetobacter species throughout the year in various months showed maximum isolation during November and December months.

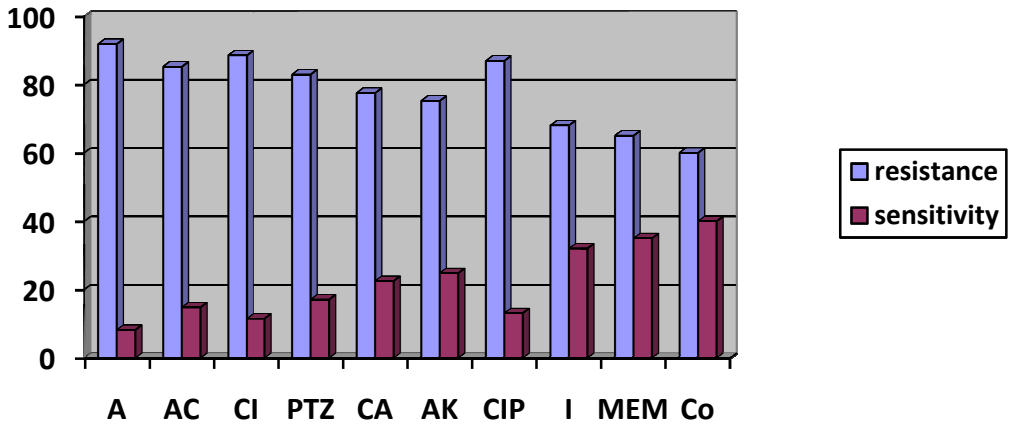

Fig-2: Antibiotic sensitive and resistance pattern of Acinetobacter species for various drugs

The organism showed high rate of resistance to cefazolin (96.5%), ampicillin (91.8%), amoxyclav (85.2%), ceftriaxone (88.5%), piperacillin (82.9%), ceftazidime (77.5%), amikacin (75.2%) and ciprofloxacin (86.9%). The acinetobacter species also showed resistance to the higher drugs like Imipenem (68%) meropenum (65%) and even colistin (60%).

Discussion

Acinetobacter baumannii is a ubiquitous gram-negative *bacillus* that is commonly associated with aquatic environments [5]. Being an opportunistic pathogen; it has been shown to colonize the skin and mucous membranes of the respiratory system of infected individuals [6]. Severe nosocomial infections due to *A. baumannii* are frequently found in the intensive care units (ICUs), which can cause ventilator-associated pneumonia (VAP), septicemia, secondary meningitis, endocarditis, infections of the skin, soft tissues, urinary tract, and those originating from prosthetic devices [7-10].

Regard to the rapid development of resistance against various antimicrobial agents due to the high ability of natural genetic transformation and the potential for widespread dissemination because of the ability to survive on environmental surfaces, *A. baumannii* has currently surpassed other bacteria as the second most commonly isolated glucose non-fermenter in clinical laboratories after *Pseudomonas aeruginosa* with high mortality rates of 41% [5].

Carbapenems, particularly imipenem, are currently the first choice in the treatment of *A. baumannii* infections [11]. In 1991, the first nosocomial, carbapenem-resistant *A. baumannii* (CR-AB) strain was reported from the USA. Several mechanisms responsible for resistance to carbapenems in CR-AB have been described: production of carbapenemases such as oxacillinases (OXA enzymes), decreased outer-membrane permeability caused by the loss or reduced expression of 29 kDa and 33 kDa porins, and alterations in penicillin-binding proteins and efflux pumps [12-14].

In our study the emergence and spread of Acinetobacter species was investigated from our hospitalized patients in NICU and PICU. As we analysed the data more number of Acinetobacter species were isolated from PICU than NICU. This may be due to the maximum patient load in PICU and also the maximum handling of the patient compared to patients in NICU. Here there is a significant difference in the patient and nurse ratio which is one nurse for 2 patients (1:2) in NICU and in PICU it is one nurse for 8 patients (1:8), which may also be the major contributing factor.

Acinetobacter species were isolated from all the samples like tracheal aspirate, blood, pus, urine, CSF and Et tips. But the maximum numbers were isolated from Tracheal aspirate and minimum number was isolated from urine. This is in contrast to the study by Shrivastava, et al in which the maximum isolation of Acinetobacter species was from urine than the respiratory secretions [15]. In their study Out of 83 samples which revealed Acinetobacter, 23 (27.7%) were urine, 48 (57.8%) were blood and 12 (14.4%) were respiratory samples [15]. Another study by Anitha

M et al showed maximum isolation of Acinetobacter species in respiratory secretions than urine, i. e 46% from respiratory secretions and only 24% from urine which is in concordance to our study [16].

Acinetobacter strains which are among the most important nosocomial pathogens survive for a long time by colonization in different environments, on the surfaces of mechanical devices used in hospitals, patients and hospital staff. [17].

Acinetobacter spp. is the second most common non-fermenting bacteria after Pseudomonas species that are isolated from human specimens, especially among nosocomial infections [18]. In our study Acinetobacter species showed high rate of resistance to cefazolin (96.5%) ampicillin (91.8%), amoxyclav (85.2%) ceftriaxone (88.5%), piperacillin (82.9%), ceftazidime (77.5%), amikacin (75.2%) and ciprofloxacin (86.9%). Imipenem was also found resistance (68%) and meropenum was (65%). This was in concordance with a study by Rahbar et al, were determined that, A.baumannii shows high percentage of resistance to ceftriaxone (90.9%), piperacillin (90.9%), ceftazidime (84.1%), amikacin (85.2%), and ciprofloxacin (90.9%)[19].

Carbapenems have been thought as the agents of choice for serious A. baumannii infections. But in our study the Acinetobacter species also showed resistance to imepenum (68%) and meropenum (65%). The organisms showed resistance to the colistin (60%) also. The more number of resistant to carbapenams and colistin were isolated from Tracheal specimens and sensitive organisms were isolated from urine.

Conclusion

Resistance to carbapenems and colistin by Acinetobacter species is a significant alarming sign that to in pediatric hospitals. This stresses upon rational use of antibiotics and also newer therapeutic strategies, strict infection control measures and also to decrease the patients nurse ratio.

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