A study on nosocomial pathogens in ICU with special reference to multiresistant *Acinetobacter baumannii* harbouring multiple plasmids


Department of Microbiology, University of Pune, *Microbial Sciences Division, Agharkar Research Institute, **Department of Microbiology, KEM hospital & +Institute of Bioinformatics & Biotechnology & Department of Microbiology, University of Pune, Pune, India

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**Background & objectives**: Antibiotic resistant bacterial nosocomial infections are a leading problem in intensive care units (ICU). Present investigation was undertaken to know antibiotic resistance in *Acinetobacter baumannii* and some other pathogens obtained from clinical samples from ICU causing nosocomial infections. Special emphasis was given on plasmid mediated transferable antibiotic resistance in *Acinetobacter*.

**Methods**: The clinical specimens obtained from ICU, were investigated to study distribution of nosocomial pathogens (272) and their antibiotic resistance profile. *Acinetobacter* isolates were identified by API20NE system. Antimicrobial resistance was studied with minimum inhibitory concentration (MIC) by double dilution agar plate method. The plasmid profile of 26 antibiotic resistant isolates of *Acinetobacter* was studied. Curing of R-plasmids was determined in three antibiotic resistant plasmid containing *A. baumannii* isolates. Plasmid transfer was studied by transformation.

**Results**: Major infections found in ICU were due to *Acinetobacter baumannii*, *Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Staphylococcus aureus* and *Streptococcus pyogenes*. The infection rate was maximum in urinary tract (44.4%) followed by wound infections (29.4%), pneumonia (10.7%) and bronchitis (7.4%). *Acinetobacter* isolates displayed high level of antibiotic resistance (up to 1024µg/ml) to most of antibiotics. More than 90 per cent isolates of *Acinetobacter* were resistant to a minimum of 23 antibiotics. Plasmid profile of *Acinetobacter* isolates showed presence of 1-4 plasmids. Ethidium bromide cured plasmids pUPI280, pUPI281, pUPI282 with curing efficiencies 20, 16 and 11 per cent respectively while acridine orange cured plasmids pUPI280, pUPI281 with curing efficiencies 7 and 18 per cent retrospectively. Transformation frequency of *E. coli* HB101 with pUPI281 was 4.3×10⁴ transformants/µg plasmid DNA.

**Interpretation & conclusions**: *A. baumannii* was found to be associated with urinary tract infections, respiratory tract infections, septicemia, bacteraemia, meningitis and wound infections. *A. baumannii* displayed higher resistance to more number of antibiotics than other nosocomial pathogens from ICU. Antibiotic sensitivity of *A. baumannii* cured isolates confirmed plasmid borne nature of antibiotic resistance markers. Transfer of antibiotic resistant plasmids from *Acinetobacter* to other nosocomial pathogens can create complications in the treatment of the patient. Therefore, it is very important to target *Acinetobacter* which is associated with nosocomial infections.

**Key words** *Acinetobacter baumannii* - antibiotic resistance - ICU - nosocomial infections - plasmid curing
Antimicrobial resistance in nosocomial infections is increasing with both morbidity and mortality greater when infection is caused by drug resistant organisms. This increase is due to overuse and misuse of antimicrobial agents, immunosuppressed patients and exogenous transmission of bacteria, usually by hospital personnel. Nosocomial infections are typically exogenous, the source being any part of the hospital ecosystem, including people, objects, food, water and air in the hospital. These infections are opportunistic and microorganisms of low virulence can cause disease in hospital patients whose immune mechanisms are impaired. The outcome is that many antibiotics can no longer be used for the treatment of infections caused by such organisms and the threat to the usage of other drugs increases.

*Acinetobacter* is most frequently isolated bacterium in clinical specimens. Members of genus *Acinetobacter* are Gram-negative, non-motile, non-spor forming encapsulated coccobacilli belonging to family *Neisseriaceae*. It is an opportunistic pathogen found to be associated with a wide spectrum of infections including nosocomial pneumonia, meningitis, endocarditis, skin and soft tissue infections, urinary tract infections, conjunctivitis, burn wound infections and bacteremia. *Acinetobacter baumannii* is the commonest isolate from Gram-negative sepsis in immunocompromized patients, posing risk for high mortality. Outbreaks of *Acinetobacter* infections are linked to contaminated respiratory equipment, intravascular access devices, bedding materials and transmission via hands of hospital personnel. During recent years, *A. baumannii* has become a significant pathogen especially in intensive care units. It typically colonizes skin and indwelling plastic devices of the hospitalized patients. Persistence of endemic *A. baumannii* isolates in ICU seems to be related to their ability for long-term survival on inanimate surfaces in patients’ immediate environment and their widespread resistance to the major antimicrobial agents.

Multidrug resistance of *Acinetobacter* isolates is a growing problem and has been widely reported. Resistance in *Acinetobacter* to majority of commercially available antimicrobials (aminoglycosides, cephalosporins, quinolones and imipenem) raises an important therapeutic problem. The presence of resistance plasmids (R-plasmids) is a significant feature of this organism. More than 80 per cent of *Acinetobacter* isolates carry multiple indigenous plasmids of variable molecular size. The plasmids present in *Acinetobacter* can be readily transferred experimentally to other pathogenic bacteria by transformation and conjugation. Also *Acinetobacter* acquires R plasmids from various pathogenic bacteria as well. *Acinetobacter* has the capacity to serve as a potential reservoir of transmissible drug resistance genes especially in nosocomial environment. In *Acinetobacter* associated nosocomial infections, the major problem encountered is the readily transferable antimicrobial resistance expressed by this organism.

The growing number of nosocomial infections and rapid increase in antibiotic resistant *Acinetobacter* isolates has prompted us to investigate incidence and prevalence of antibiotic resistant *Acinetobacter* isolated in 2003 from different clinical samples from ICU of KEM hospital, Pune, India. Antibiotic resistance pattern, plasmid profile, plasmid curing as well as plasmid transfer study in *A. baumannii* isolates were carried out to confirm the plasmid borne nature of antibiotic resistant markers.

**Material & Methods**

*Clinical specimens*: Bacterial resistance to several antibiotics was studied in 272 different bacterial pathogens (Gram-positive and Gram-negative) from clinical samples (urine, pus, sputum, blood, etc.) from King Edward Memorial Hospital (KEM Hospital, affiliated to the University of Pune), Pune, from February 2003 to December 2003. Clinical samples including urine (150), sputum (85), pus (64), blood (73), peritoneal fluid (26), Foley’s tip (32), abdominal washing (38), bronchial washings (10), and CSF (5) were collected from variety of patients from intensive care unit. Clinical samples were investigated to find the distribution of nosocomial pathogens in causing different opportunistic infections and their antibiotic resistance profile.

**Identification of Acinetobacter and other nosocomial pathogens**: Clinical isolates of *Acinetobacter, Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Staphylococcus aureus and Streptococcus pyogenes* were identified on basis of morphological, cultural and biochemical characteristics. *Acinetobacter* was identified on basis of five preliminary tests viz., Gram staining, capsule staining, motility, oxidase and catalase tests. Phenotypic identification was performed by biochemical tests. Chromosomal DNA transformation assay of Juni was used to confirm Genus *Acinetobacter*. *A. baumannii* isolates (26) were confirmed by using API2ONE system.
Control strains and culture conditions: Antibiotic resistance pattern was studied in 26 isolates of *A. baumannii*. All the isolates resistant to multiple antibiotics were screened for presence of plasmids. Control strains used for antibiotic resistance included *E. coli* (RP4), *E. coli* (R751), *E. coli* (HB101), *A. calcoaceticus* MTCC127, *A. calcoaceticus* MTCC1271 and *A. calcoaceticus* MTCC1425. Control strains used for plasmid profile studies included *P. aeruginosa* (RIP64), *E. coli* (PRK2013), *S. typhi* (R136), *E. coli* K12 (pBR322), *E. coli* K12 (RP4) and *E. coli* V517 provided by Microbial Type Culture Collection (MTCC), Institute of Microbial Technology, Chandigarh, India. Cultures were grown aerobically at 37°C, with constant shaking at 150 rpm for 16-18 h.

Chemicals and culture media: Antibiotic powders were obtained from Parke-Davis, Ltd. Mumbai, India. Antibiotic discs, chemicals and media were purchased from Hi-Media, Mumbai, India. EDTA and other chemicals used in plasmid isolation and purification studies were purchased from Qualigens (India). Cultures were grown in Luria-Bertani (L-B) broth for all experiments.

Determination of resistance to antibiotics: Antibiotic resistance profile was determined by Kirby Bauer disc diffusion method on Mueller Hinton (MH) agar plates (Hi-media, Mumbai)25. Discs were consistently tested for efficacy against standards strains recommended by National Committee for Clinical Laboratory Standards (NCCLS)26 as well as others with known antimicrobial susceptibility pattern. Results were interpreted as per cent sensitive (%S) and per cent resistant (%R) isolates derived using NCCLS26 and WHO breakpoints26,27.

Determination of minimal inhibitory concentration (MIC) of antibiotics: Antibiotic susceptibility testing of 26 *A. baumannii* isolates to 27 antibiotics belonging to different groups was carried out on MH agar. MIC was determined by double dilution agar plate method28. It was determined according to NCCLS (now Clinical Laboratory Standards Institute, CLSI) guidelines26,29. Concentration range of each antibiotic used was 1 µg/ml to 1024 µg/ml.

Isolation and purification of plasmid DNA: Plasmid isolation was done using modified Kado and Liu30 and Sambrook method31. Standard strains having plasmids of known molecular weight were run with each set. Cultures were grown aerobically in L-B medium31, at 37°C, 150 rpm for 16-18 h. Following modifications were included in the standard protocol. In Kado and Liu’s method cell pellet was suspended in 100 µl E-buffer (20 mM tris-acetate and 2 mM sodium salt of EDTA, pH 7.9) followed by addition of 200-400 µl lysing buffer (3% SDS and 50 mM tris, pH 12.6 adjusted with 2N NaOH). Heat treatment at 65°C for 90 min ensured complete lysis. There were no modifications in lysis procedure for Sambrook method. Phenol: chloroform extraction (protein precipitation) was done for both the methods. Nucleic acid precipitation for both the methods was done with equal volume isopropanol. Plasmid pellet thus obtained was dissolved in 30 µl TE (10 mM tris, 1 mM EDTA, pH 8) buffer. Agarose gel electrophoresis was performed on 0.8 per cent (w/v) agarose gels prepared in TAE buffer30 (40mM tris acetate and 2 mM sodium EDTA, pH 7.9 adjusted with glacial acetic acid). Plasmid profiles were documented under UV light in Gel Documentation System (Alpha Innotech Corp., USA).

Determination of molecular weight of plasmid: Molecular weights of plasmids from *A. baumannii* isolates were determined by comparing with standard plasmids, pBR322 (4.36 kb), PRK2013 (47 kb), RP4 (57 kb), RIP64 (135 kb) and R136 (59 kb). Images of gels were captured on Alpha Imager gel documentation system and molecular weight of test plasmids was determined by comparing them with standard plasmids using the software provided in gel documentation system. For reproducibility testing, comparison of plasmids with standard plasmids was done thrice and an average of 2 readings obtained for each isolate was affirmed as the final molecular weight of plasmid. V517 series of plasmids (*E. coli* V517, MTCC 131) was used as plasmid molecular weight standard.

Curing of antibiotic resistance: The plasmid curing was performed in *A. baumannii* A23 (pUPI280), *A. baumannii* A24 (pUPI281), *A. baumannii* A26 (pUPI282) (all three plasmids identified in present study) and standard plasmid containing strains *E. coli* K12 (RP4) and *E. coli* K12 (pBR322) by method as described by Deshpande et al.32. The percentage curing efficiency was expressed as number of colonies with cured phenotype per 100 colonies tested. The physical loss of plasmid in the cured derivative was confirmed by agarose gel electrophoresis of the plasmid DNA preparation of respective cultures. Antibiotic sensitive cured colonies were also tested for loss of resistance to antibiotics by disc diffusion assay. The experiment was performed in duplicate.

Plasmid transfer by transformation: HB101 of *E. coli* was used as host for transformation experiments.
Competent cells of *E. coli* HB101 were prepared using calcium chloride method\(^\text{31}\). Transformation experiments were performed by “heat shock method”\(^\text{31}\) using plasmid pUPI281 (Ap\(^\text{r}\), Gm\(^\text{r}\), Km\(^\text{r}\)) from *A. baumannii* A24 and competent cells of *E. coli* HB101 as recipient. Transformation efficiency was calculated as number of transformants per µg of plasmid DNA.

### Results

**Nosocomial infections in intensive care unit:** A total of 272 bacterial isolates were obtained from clinical specimens like blood, urine, pus, sputum, CSF, peritoneal fluid, abdominal washing and Foley’s catheter tube. These were identified as *Acinetobacter* (36), *A. baumannii* (28), *A. junii* (8) *E. coli* (74), *K. pneumoniae* (52), *P. aeruginosa* (36), *S. aureus* (47) and *S. pyogenes* (27) (Table I). Maximum numbers of pathogens were isolated from urine, pus and sputum. *E. coli* was found to be most predominant isolate found from ICU. Urine was most common source of *Acinetobacter*. From 36 isolates of *Acinetobacter*, 28 were identified and confirmed as *A. baumannii* and 8 as *A. junii* by API20NE system. Urinary tract infections (43.38%) were most predominant infections (Table I). Other infections detected were septicemia (1.84%), pneumonia (10.66%), wound infections (29.41%), bronchitis (7.35%), tuberculosis (0.74%), bacteraemia (5.15%) and meningitis (1.5%). The commonest were pneumonia (7.35%), tuberculosis (0.74%), bacteraemia (29.41%), wound infections (29.41%), Other infections detected were septicemia (1.84%) as *Acinetobacter* from ICU. Urine was most common source of *E. coli* pathogens were isolated from urine, pus and sputum.

### Antibiotic resistance patterns in clinical isolates of *A. baumannii*

Twenty six *A. baumannii* isolates with high antibiotic resistance were identified and tested against 27 antibiotics from different groups. A wide range of concentrations of antibiotics (1-1024 µg/ml) was tested against *A. baumannii*. Majority of isolates tolerated more than 512 µg/ml of antibiotic from all the groups and most showed high level of resistance to multiple antibiotics. More than 80 per cent isolates of *A. baumannii* were highly resistant to β-lactam antibiotics tested except...
ceftazidime and ceftriazone whereas 54 and 61.6 per cent resistance was observed at MIC more than 512 µg/ml. Less than 5 per cent isolates could be inhibited at 128 µg/ml in β-lactam group antibiotics. All A. baumannii isolates were resistant to penicillin and cefuroxime at 512-1024 µg/ml. More than 90 per cent isolates were resistant to ampicillin, amoxicillin, and piperacillin at 512-1024 µg/ml (Fig. 1). Cefuroxime showed maximum level of resistance in cephalosporin group. Resistance of Acinetobacter to quinolones was less as compared to aminoglycosides and β-lactam antibiotics (Fig. 2). 100 per cent resistance was observed to nalidixic acid at 512-1024 µg/ml. More than 80 per cent isolates were resistant to ciprofloxacin and norfloxacin at 512-1024 µg/ml. Resistance level was low to ofloxacin and sparfloxacin as compared to other antibiotics of this group.

Among aminoglycosides, 5 antibiotics were tested (Fig. 3). More than 80 per cent isolates were resistant to aminoglycoside antibiotics except tobramycin where 65.3 per cent resistance was observed at MIC more than 512 µg/ml. High level of resistance (MIC 512-1024) was detected for amikacin and streptomycin. For clindamycin 92 per cent isolates were resistant at 512-1024 µg/ml. Resistance to tetracycline was high as compared to doxycycline of same group. At 512-1024 µg/ml tetracycline more than 96 per cent isolates of A. baumannii were resistant; 65 per cent isolates were resistant to chloramphenicol at 512-1024 µg/ml in phenolics group (Fig. 4). For erythromycin, 54 per cent

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isolates were resistant at 512-1024 µg/ml. For polymyxin B. baumannii isolates were resistant only up to 128 µg/ml. For doxycycline, rifampicin and trimethoprim resistance level was low as compared to other antibiotics.

Plasmid profile in A. baumannii: Multiple plasmids were found in all isolates of A. baumannii. Plasmid number found in 26 isolates of A. baumannii was in the range from 1 to 5. In 9 isolates sharp plasmids were observed (Fig. 5). They were used for further genetic experiments. Sambrook method was found to be better since it showed sharp plasmid bands than Kado and Liu method. Molecular sizes of all plasmids ranged from 4 to 50kb by comparing with standard plasmids pBR322 (4.36 kb), pRK2013 (47 kb), RP4 (57kb), RIP64 (135kb) and R136 (59 kb) and E. coli (V517) (Fig. 5).

Curing of antibiotic resistance: Plasmid curing by ethidium bromide and acridine orange was detected in A. baumannii A23, A24 and A26 (Table III). Ethidium Bromide cured plasmids pUPI280, pUPI281, pUPI282 with curing efficiencies 20, 16 and 11 per cent respectively while acridine orange was able to cure plasmids pUPI280, pUPI281 with curing efficiencies 7 and 18 per cent respectively. Acridine orange was unable to cure plasmid RP4 from E. coli and pUPI282 from A. baumannii A26. The plasmid cured isolates of A. baumannii and reference strains showed absence of plasmid on agarose gel electrophoresis which clearly confirmed their plasmid elimination.

Plasmid transfer by transformation: Plasmid pUPI281 (Ap, Gm, Km) was transferred from A. baumannii A24 to E. coli HB101 by transformation. Frequency of transformation of E. coli HB101 with pUPI281 was observed to be 4.3×10^4 transformants/µg plasmid DNA.
Discussion

There are several reports on outbreaks of multidrug resistant Acinetobacter baumannii in an ICU33-35. In ICU critically ill patients are always at higher risks of developing nosocomial infections with antibiotic resistant strains. The emergence and spread of multidrug resistant A. baumannii and its genetic potential to carry and transfer diverse antibiotic resistant determinants pose a major threat in hospitals 36.

In the present study, most common bacterial pathogens in ICU acquired infections were Acinetobacter, Pseudomonas, Klebsiella, E. coli, Staphylococcus and Streptococcus. Infection rate was highest in urinary tract followed by wound infections, pneumonia and bronchitis. Urinary tract infection was higher as compared to other studies which ranged from 13 to 19 per cent37. The foremost causes of urinary tract infections in hospitals are *E. coli*, *P. aeruginosa*, *Klebsiella*, *Proteus*, *Enterococci* and *Candida*38. In this study total numbers of organisms isolated from urine were 118. *E. coli* were most predominant organisms in causing UTI, it did not display high level of resistance to antibiotics. In a study by Hsueh et al38, the most frequent isolates from UTI were *Candida* spp. (23.6%) followed by *E. coli* (18.6%) and *P. aeruginosa* (11%). Singh et al37, showed presence of *E. coli*, *P. aeruginosa*, *Proteus mirabilis* and *Enterococcus faecalis* in equal proportion in causing UTI. In the present study *Enterococcus* and *Candida* were not isolated.

*Staphylococcus* was predominant in causing wound infections. Other organisms detected were *Pseudomonas*, *Klebsiella*, *E. coli*, *Acinetobacter* and *Streptococcus*. These results were comparable with previous findings39,40. Isolation rate of *Staphylococcus* was maximum in causing respiratory tract infections (RTIs) in the present study. Other organisms causing RTIs included *Streptococcus*, *Klebsiella*, *E. coli*, *Pseudomonas* and *Acinetobacter*. In a previous report *A. lwoffii* and *A. junii* were isolated from upper respiratory tract of healthy humans42, while in this study *A. baumannii* was found to be associated with tuberculosis and bronchitis. In a study by Singh et al37, most frequent isolates causing RTIs were *Klebsiella* (24.48%), followed by *Proteus* (18.33%) and *E. coli* (12.24%).

Other nosocomial infections included bacteraemia septicemia and meningitis. In the present study, isolation of *Pseudomonas* and *Acinetobacter* in blood

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### Table III. Curing of R-plasmids in clinical isolates of *A. baumannii* with EtBr and acridine orange

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<td>11</td>
<td>256</td>
<td>—</td>
</tr>
<tr>
<td><em>E. coli</em> K 12</td>
<td>RP4</td>
<td>Ap, Te, Km</td>
<td>Ethidium Bromide</td>
<td>128</td>
<td>14</td>
<td>64</td>
<td>—</td>
</tr>
<tr>
<td><em>E. coli</em> K12</td>
<td>pBR322</td>
<td>Ap, Te</td>
<td>Ethidium Bromide</td>
<td>128</td>
<td>23</td>
<td>128</td>
<td>14</td>
</tr>
</tbody>
</table>

SIC, Subinhibitory concentration. Total 300 clones tested. —, Below detection limit (none of the 300 clones tested showed curing of plasmid)

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![Fig. 5. Plasmid profile of standard strains and clinical isolates of *A. baumannii* harbouring R-plasmids.](image-url)
stream infections along with *Staphylococcus* suggests possibilities of sepsis resulting from nosocomial infections. Kapil\(^4\) has reported outbreak of bacteraemia due to *A. baumannii* in leukaemia patients in a tertiary care hospital in Delhi. In our study organisms causing meningitis were *Pseudomonas*, followed by equal proportions of *Acinetobacter* and *E. coli*. Wroblewska *et al*\(^4\) reported outbreak of nosocomial meningitis caused by *A. baumannii* in neurosurgical patients.

*Acinetobacter* is reported for about 10 per cent of nosocomial infections in ICU patients\(^5\). In this study *Acinetobacter* was isolated in a significant proportion from clinical samples in ICU infections, and multidrug resistant *Acinetobacter* isolates were found to be associated with almost all types of nosocomial infections like UTIs, RTIs, septicaemia, bacteraemia, meningitis and wound infections. In a recent study by Prashanth and Badrinath\(^6\) reported multidrug resistant *Acinetobacter* responsible for majority of infections. Presence of multidrug resistant plasmid harbouring *A. baumannii*, causing all types of nosocomial infections could lead to therapeutic problems.

All bacterial isolates showed high frequency of resistance to multiple antibiotics but maximum resistance was observed in *Acinetobacter* isolates. *Acinetobacter* isolates have a propensity to readily develop resistance to second and third generation antibiotics such as cefotaxime, ciprofloxacin, and giving rise to therapeutic problems\(^7\). As higher generation antibiotics are being developed to overcome problem of resistance against available antibiotics, bacteria are developing mechanisms to resist newer antimicrobials. In this study *A. baumannii* isolates showed resistance to both old and new generation antibiotics.

Member of genus *Acinetobacter* have been shown to be resistant to β-lactam and aminoglycoside antibiotics\(^8,9\) and thought to be a reservoir of antibiotic resistant genes in hospital environment. However, *Acinetobacter* isolated from healthy skin exhibited higher susceptibility to antibiotics as compared to clinical and environmental isolates\(^10\). A correlation between metal and antibiotic resistance has been established among clinical and environmental isolates\(^11\). In the present study all isolates of *A. baumannii* were found resistant to clinically achievable levels of most commonly used antibiotics. For relatively new antibiotics such as broad spectrum cephalosporins (cephotaxime, cepazidime, ceftriazone) and tobramycin slightly less resistance was observed. Partial susceptibility was observed for quinolones like ofloxacin, sparfloxacin, lomefloxacin and other antibiotics. Maximum susceptibility was detected against polymyxin B. Despite the rising clinical importance of *A. baumannii* compared to other nosocomial pathogens, this organism has been widely overlooked.

The major problem encountered by ICU clinicians relates to readily transferable antibiotic resistance expressed by *Acinetobacter*. *A. baumannii* has the ability to acquire resistance to many major classes of antibiotics\(^12\). Multiple antibiotic resistance in *Acinetobacter* was reported previously but plasmid borne nature of antibiotic resistance has been reported only in a few cases in India\(^13\). Clinical isolates of *Acinetobacter* harbour plasmids of different molecular sizes ranging from 15-56kb\(^14\). We found plasmids having molecular sizes 4-50kb.

Elimination of plasmid from antibiotic resistant *A. baumannii* and antibiotic sensitivity of *A. baumannii* cured isolates confirmed plasmid borne nature of antibiotic resistance markers. In three isolates of *A. baumannii*, plasmid elimination was observed by using conventional curing agents like acridine orange and ethidium bromide. The cured isolates showed very low MIC values as compared to original isolates. Physical loss of plasmid from cured strains showed plasmid borne nature of antibiotic resistance markers.

Transferable plasmid mediated antibiotic resistances poses a great threat as it can achieve much larger dimension due to wide and rapid dissemination. This transferable resistance is carried on R-plasmids\(^15\). The clinical *A. baumannii* isolate as well as unrelated environmental *A. baumannii* isolate had a similar carbapenem resistance plasmid suggesting spread of this genetic character\(^16\). A single plasmid which acts as vector of resistance genes can carry a number of genes coding for multiple drug resistance. In the present study, *A. baumannii* isolates harbouring R-plasmids were found resistant to multiple antibiotics. Transfer of antibiotic resistant plasmids to other nosocomial pathogens can create complications in the treatment of patients. Thus *Acinetobacter* needs to be considered as an important pathogen and steps must be taken to contain *Acinetobacter* nosocomial infections.

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References


