RESEARCH PAPER

A Retrospective Study on the Prevalence and Antibiotic Susceptibility Pattern of Acinetobacter baumannii in Various Clinical Samples in a Tertiary Care Hospital

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ABSTRACT

Background: Previously regarded as a relatively harmless environmental organism, Acinetobacter baumannii has now been recognized as one of the most troublesome bacteria in hospitals, especially concerning patients who are very ill. With the increasing resistance of this bacterium to various kinds of drugs, the number of antibiotics effective against it is becoming limited. This study aimed at estimating the frequency of clinical specimens with A. baumannii in our hospital and the response of the strains isolated from these specimens to antibiotics prescribed on various wards and ICUs.

Methods: We retrospectively reviewed four months of susceptibility data for Acinetobacter spp. from September through December 2023. We used our WHONET-5 database at our hospital to extract this data regarding the response of A. baumannii to antibiotics based on susceptibility testing done via the [Kirby-Bauer Disk Diffusion method using an automated system].

Results: Sixty-five Acinetobacter isolates were logged in total, and 40 of these (61.5%) turned out to be A. baumannii complex; a quarter of that group — 10 isolates — met the criteria for multidrug resistance. Nearly all of the resistant strains traced back to patients in the ICU or the post-surgical ward. Men made up the larger share of positive cultures, 28 of 40 (70%), against 11 women (27.5%); one further isolate (2.5%) came from a six-year-old child. Urine and catheter-tip samples supplied over half the isolates (53.8%), with pus/wound swabs (15.4%), respiratory specimens (12.3%), miscellaneous sources (10.8%), and blood (7.7%) making up the rest. Resistance ran high against penicillins (46.0–100%), third- and fourth-generation cephalosporins (36.0–61.5%), carbapenems (34.4–82.8%), and fluoroquinolones (39.3–46.7%), whereas colistin retained full activity against every isolate tested, and tigecycline and amikacin remained effective in 93.4% and 87.7% of cases respectively.

Conclusion: A. baumannii continues to pose a genuine challenge on our wards, having grown resistant to several first-line drug classes, yet colistin, tigecycline, and amikacin still cover most isolates. Keeping watch on local resistance trends, prescribing antibiotics more carefully, and tightening infection-control routines will all matter if this narrowing set of options is to be preserved.

Keywords: *Acinetobacter baumannii; multidrug resistance; antimicrobial susceptibility; nosocomial infection; colistin; tigecycline; intensive care unit*

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1. INTRODUCTION

Acinetobacter is a genus of Gram-negative coccobacilli that neither ferment sugars nor swim under their own power; taxonomists currently recognise 27 named species within it, alongside a handful of provisional ones still awaiting formal description [1,2]. Of these, A. baumannii is by far the one clinicians encounter most, and it carries the greatest clinical weight of the group [1]. Textbooks used to describe it as little more than a bystander organism — low in virulence, rarely worth worrying about. That picture no longer holds. Over roughly the last forty years the organism has turned into one of the more persistent hospital-acquired pathogens anywhere in the world, and its heaviest toll falls on patients already critically unwell in intensive care [1,3,4].

Part of what makes A. baumannii so hard to shake off is its sheer hardiness: it can sit on a bedrail or ventilator tubing for days without moisture, and it seems able to pick up — or switch on — resistance genes faster than many of its Gram-negative relatives [3,5]. The upshot has been a steady rise in strains classed as multidrug-resistant (MDR), extensively drug-resistant (XDR), or, less commonly, pandrug-resistant (PDR), categories set out in an internationally agreed framework [6]. When MDR A. baumannii takes hold — as ventilator-associated pneumonia, a catheter-linked urinary infection, bloodstream infection, or a wound gone bad after surgery — patients tend to stay in hospital longer, cost more to treat, and, in the ICU especially, face a higher chance of dying from the episode [7,8].

India, like many other resource-constrained settings, tends to carry a higher burden of antibiotic resistance due to a number of factors including the tendency to prescribe antibiotics empirically, heavy use of invasive devices such as catheters and ventilators, and inadequate infectious disease control programs [9-11]. Resistance patterns can be so variable, even from one hospital or unit to another, that local surveillance for the prevalence of A. baumannii and the antibiotic susceptibility profile of that organism is critical for guiding clinicians in their choice of empirical therapy and helping stewardship teams target their interventions. With this need in mind, we undertook a review of our own laboratory's Acinetobacter data, for the purpose of learning the rate at which A. baumannii was reported in our isolates and obtaining antibiotic susceptibility patterns among the different types of specimens.

Aim: To determine the prevalence and antimicrobial susceptibility pattern of A. baumannii isolates recovered from clinical samples of patients admitted to various wards and ICUs of a tertiary care hospital.

2. MATERIALS AND METHODS

2.1 Study Design, Setting, And Period

We ran a retrospective review of laboratory surveillance records in the Department of Microbiology, Tagore Medical College & Hospital, Tamil Nadu. Every Acinetobacter isolate logged from diagnostic specimens received by the laboratory over a four-month window — September to December 2023 — was pulled for analysis.

2.2 Sample Collection and Organism Identification

We took in one isolate per patient (duplicates were excluded) across the usual range of specimen types sent for routine work-up: urine, catheter tips, pus and wound swabs, blood, respiratory samples (sputum, bronchoalveolar lavage fluid, endotracheal aspirates), and a miscellaneous group covering other body fluids. Species/complex-level identification relied on [conventional biochemical testing / an automated identification platform], with every result logged into the laboratory's WHONET-5 surveillance system.

2.3 Antimicrobial Susceptibility Testing

Susceptibility was worked out using [disc diffusion on Mueller–Hinton agar by the Kirby–Bauer method / an automated susceptibility platform — the author should specify which], and zones/MICs were read against the CLSI breakpoints in force at the time. The panel covered penicillins (ampicillin/ampicillin-sulbactam), third- and fourth-generation cephalosporins (ceftazidime, cefepime), carbapenems (imipenem, meropenem), fluoroquinolones (ciprofloxacin, levofloxacin), the aminoglycosides amikacin and gentamicin, tigecycline, and colistin [12]. Because CLSI has not published a species-specific breakpoint for tigecycline against A. baumannii, we fell back on the FDA/manufacturer breakpoint set for Enterobacterales as a working reference — a compromise we return to in the discussion of this study's limitations.

2.4 Definition of Multidrug Resistance

An isolate was called multidrug-resistant if it showed non-susceptibility to at least one drug in three or more of the following groups: penicillin/beta-lactamase-inhibitor combinations, cephalosporins, carbapenems, fluoroquinolones, and aminoglycosides — the interim consensus definition set out by Magiorakos and colleagues [6].

2.5 Data Extraction and Statistical Analysis

For each isolate we pulled the specimen type, the admitting ward or unit, the patient's age and sex, and the full susceptibility profile from WHONET-5. Findings are reported descriptively, as counts and percentages for categorical variables; because the dataset covers a single short surveillance window rather than a comparative or longitudinal design, no formal hypothesis testing was attempted.

2.6 Ethical Considerations

Because the work drew only on de-identified laboratory surveillance data — nothing that involved contacting patients or altering their care — it proceeded under institutional clearance [Institutional Ethics Committee approval number and date to be inserted by the author]. No patient-identifying information was accessed at any stage.

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3. RESULTS

3.1 Prevalence of Acinetobacter Baumannii

The four-month window yielded 65 Acinetobacter isolates in total, one per patient. Forty of these, or 61.5%, keyed out as A. baumannii complex, and of that subset, 10 isolates — a quarter — met our threshold for multidrug resistance.

3.2 Specimen-Wise Distribution

Table 1 breaks the 65 isolates down by where they came from. Urine and catheter tips contributed more than any other source, accounting for just over half of the total, with pus/wound swabs and respiratory specimens making up most of the remainder.

Clinical specimen	No. of isolates (n = 65)	Percentage (%)*
Urine / catheter tip	35	53.8
Pus / wound swab	10	15.4
Respiratory tract (sputum, BAL fluid, ET secretions)	8	12.3
Miscellaneous (body fluids, tissue, others)	7	10.8
Blood	5	7.7
Total	**65**	**100.0**

**Percentages recalculated from reported isolate counts (n/65); see editorial note above.*

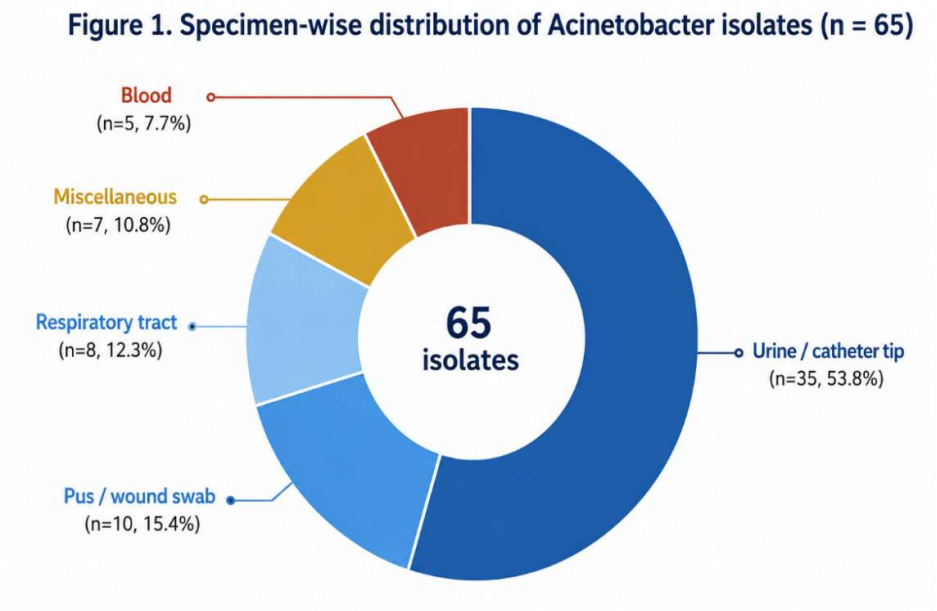

Figure 1. Specimen-wise distribution of Acinetobacter isolates (n = 65). Urine and catheter-tip specimens accounted for over half of all isolates.

3.3 Demographic Profile

Looking at the 40 patients from whom A. baumannii was isolated, men outnumbered women by a wide margin — 28 (70%) against 11 (27.5%) — with a single additional isolate (2.5%) coming from a six-year-old child rather than an adult.

3.4 Ward-Wise Distribution of Multidrug-Resistant Isolates

Every one of the 10 MDR isolates traced back to a patient in either the ICU or the post-surgical ward — a pattern that fits what has been reported elsewhere, where critical illness, indwelling devices, and recent surgery tend to go hand in hand with picking up resistant Acinetobacter.

3.5 Antimicrobial Susceptibility Pattern

Table 2 sets out how the isolates as a group performed against the antibiotic panel. Resistance was substantial against penicillins, the extended-spectrum cephalosporins, carbapenems, and fluoroquinolones, while colistin met no resistance at all, and both tigecycline and amikacin held up well.

Antimicrobial agent / class	Resistance (%)	Sensitivity (%)
Penicillins (ampicillin/ampicillin-sulbactam)	46.0 – 100.0	—
Third- and fourth-generation cephalosporins (ceftazidime, cefepime)	36.0 – 61.5	—
Carbapenems (imipenem, meropenem)	34.4 – 82.8	—
Fluoroquinolones (ciprofloxacin, levofloxacin)	39.3 – 46.7	—
Aminoglycosides (amikacin)	—	87.7
Tigecycline	—	93.4
Colistin (polymyxin E)	0.0	100.0

Looking specifically at the MDR subset, colistin worked against every one of them when re-tested with the higher-tier panel, and amikacin came out ahead of gentamicin as the more reliably active aminoglycoside.

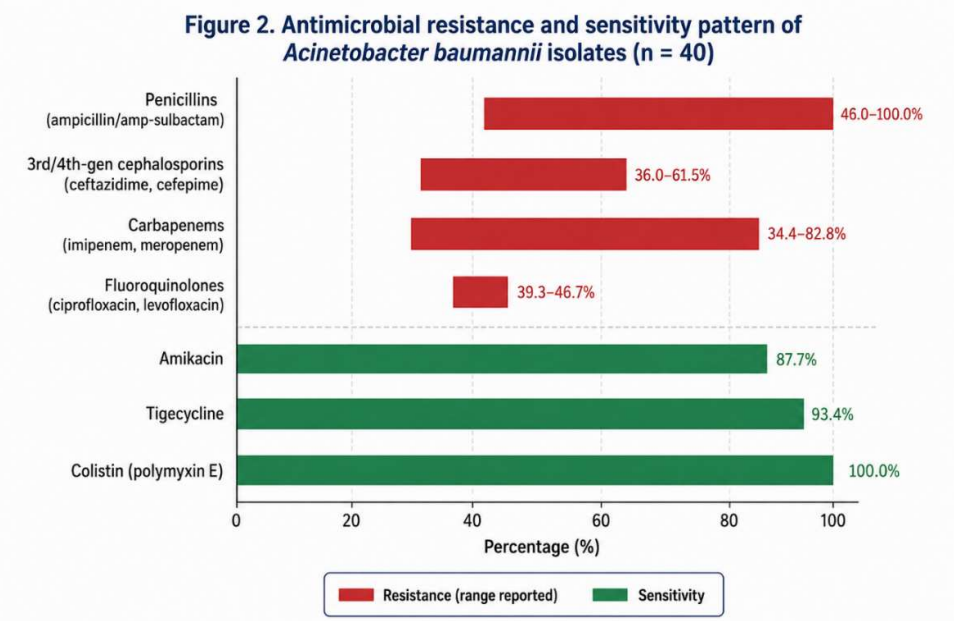

Figure 2. Antimicrobial resistance and sensitivity pattern of A. baumannii isolates (n = 40). Bars for the four resistant drug classes show the reported resistance range; bars for amikacin, tigecycline, and colistin show percentage sensitivity.

4. DISCUSSION

Slightly above three out of five Acinetobacter isolates were found to be A. baumannii, with twenty-five percent of these meeting the definition of multidrug resistant, numbers that correlate well with those from other Indian tertiary facilities and global surveillance databases on an organism that is being called a serious hospital threat [1,3,9,11]. It is not surprising that urine and catheter samples accounted for most of the cultures; A. baumannii is known to colonize indwelling urinary catheters and cause urinary tract infection in hospitalized patients [2,9].

All of the multidrug-resistant strains were found in patients in the intensive care unit or post-operative ward; this is consistent with prior research of infections in hospital patients. The use of invasive devices, lengthy hospital stays, heavy exposure to antibiotics, and immunosuppression created the ideal conditions for the appearance of resistant A. baumannii [1,4,7,8]. The majority of cultures came from male patients (70%)—this finding is also seen in many other hospitals; still, it is difficult to determine whether this high percentage represents true susceptibility or simply reflects demographics of hospitalized patients and their pre-existing conditions.

Resistance was prevalent among penicillins, cephalosporins of the newer generation, compounds of the carbapenem class and fluoroquinolones in the present study; these findings mirror the resistance mechanisms A. baumannii is known to exhibit, namely, efflux pumps, OXA-type and metallo-beta-lactamases, modified or absent porins, and mutations in fluoroquinolone target sites [3,5]. Excessive use of antibiotics, the capacity of this microbe to persist on surfaces, its capacity for gene modification, and the condition of patients provided contributing factors for the emergence of drug resistance in tertiary hospitals [1,3,5].

On a more positive note, there were no reports of resistance to colistin in any of the culture samples; furthermore, tigecycline and amikacin have proven effective in suppression of multidrug-resistant strain infections among patients. Colistin is held in reserve for use when other drugs are ineffective because it has serious side effects of renal failure and neurological dysfunction [13]. Tigecycline — chemically a derivative of minocycline — performed well in vitro here too, though it is worth flagging that CLSI has not settled on a species-specific breakpoint for A. baumannii, so susceptibility figures for this drug should be read with some caution rather than taken at face value [1]. Amikacin outperformed gentamicin among the aminoglycosides, matching what others have found; even so, drugs in this class are generally better used alongside another agent than alone in Gram-negative bloodstream infection, given their limited tissue penetration, potential for kidney and ear toxicity, and generally weaker outcomes as monotherapy [14].

Where treatment options are running thin, combining agents — colistin with a carbapenem, say, or colistin with tigecycline — has been suggested as a way to squeeze out better outcomes in MDR A. baumannii infection, and such regimens are worth discussing with microbiology and infectious-disease colleagues, particularly for XDR strains [7,13]. But no antibiotic regimen substitutes for good infection control: hand hygiene, isolating or cohorting colonised patients, thorough environmental cleaning, sensible limits on catheter and ventilator use, and a functioning stewardship programme all remain central to keeping MDR A. baumannii from spreading further [1,3,15].

5. LIMITATIONS

A few caveats apply here. The data come from one centre and cover just four months, so how well these findings travel to other settings — or hold up across seasons and longer stretches of time — is uncertain. Being a retrospective pull from laboratory records rather than a clinical study, we could not tell colonisation apart from true infection, nor track what happened to these patients in terms of treatment or survival. We also did not attempt any molecular work — no testing for blaOXA-23-like, blaNDM, or direct evidence of efflux-pump activity — so the resistance mechanisms discussed above remain inferred rather than confirmed in this particular isolate set. And with only 10 MDR isolates to work with, any subgroup patterns within that group should be read as suggestive rather than conclusive. A larger, prospective study spanning several centres and incorporating molecular resistance testing would go further toward settling these questions.

6. CONCLUSION

A. baumannii continues to establish itself as a significant nosocomial threat at this centre, with a notable share of isolates crossing into multidrug-resistant territory — overwhelmingly among ICU and post-surgical patients. Even though resistance to penicillins, cephalosporins, carbapenems, and fluoroquinolones ran high, colistin remained active against essentially every isolate and both tigecycline and amikacin retained strong activity, leaving real options on the table for treating MDR disease. Taken together, these results argue for keeping local resistance surveillance running, tightening up antibiotic prescribing habits, and holding the line on infection-control practice, alongside clear guidance to clinicians about when therapy is likely to fail in patients carrying multidrug-resistant A. baumannii.

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