

Discernment of *Acinetobacter* Species in World Scenario

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Abstract

The morphology of *Acinetobacter* species is Gram-negative, nonmotile, nonfermenting, Strictly aerobic, Oxidase negative, and Catalase positive. It usually results from an infectious agent and is nonpathogenic in healthy people. *A. baumannii* can survive, for a long time and spread quickly in a hospital setting. *Acinetobacter baumannii* can cause many infections, the most common of which are bloodstream infections and nosocomial pneumonia in severe patients. These infections have a high mortality rate & the global emergence of antibiotic-resistant strains from multiple classes has reduced the number of drugs that still have activity against this pathogen. The lack of *A. baumannii* isolates that produce carbapenemase in addition to having a minimal inhibitory concentration of imipenem greater than carbapenem resistance. The overview of the prevalence of *Acinetobacter* in the Indian scenario and world scenario. Comparison between carbapenem resistance and multidrug resistance. According to the studies, carbapenem resistance is rapidly increasing. Infection with *Acinetobacter* is linked to high mortality and morbidity. According to our review, patients who have an infection with *A. baumannii* may be at an increased risk of dying. However, due to the confounding factors of illness severity, inappropriate impractical antimicrobial treatment, and small sample size, cautious interpretations are necessary. We covered the main factor that make *A. baumannii* such a prevalent nosocomial pathogen in this review, including such as its virulence components, desiccation resistance, and carbapenem resistance mechanisms. With the emergence of extended resistance to even more recent antibiotics, *Acinetobacter* species are rapidly proliferating. They can develop resistance much more quickly than other gram-negative organisms.

Keywords: *A.baumannii*, Desiccation, Morbidity, Carbapenem, Resistance, Extended, Antimicrobial

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Citation: Meshram N, Meshram S. Discernment of *Acinetobacter* Species in World Scenario. J Pure Appl Microbiol. 2023;17(3):1400-1409. doi: 10.22207/JPAM.17.3.11

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INTRODUCTION

Gram-negative, Non-motile, Nonfastidious, Nonfermenting, Oxidase negative, Catalase positive and Strictly aerobic bacteria are the characteristics of *Acinetobacter*.¹ *Acinetobacter* species usually appear mucoid, smooth, lemon yellow to greyish-white colonies, though some isolates produce a diffusible brown pigment.² It is oxidase negative, which can be used as a quick confirmatory method to distinguish *Acinetobacter* species from other nonfermenting bacteria. In the traditional nitrate reduction test, most strains are not able to reduce Nitrate-to- Nitrite.³ *Acinetobacter baumannii* can cause many infections, the most common of which are bloodstream infections and nosocomial pneumonia in severe patients. These infections have a high mortality rate & the global emergence of antibiotic-resistant strains from multiple classes has reduced the number of drugs that still have activity against this pathogen.⁴ One of the most problematic pathogens in healthcare today has just recently been identified as *Acinetobacter baumannii*.¹ It usually results from an infectious agent and is non-pathogenic in healthy people.⁵

A. baumannii can survive for a long time and spread quickly in a hospital setting.⁶ The two ways it is spread are through direct- contact with infected people or through indirect- contact with a contaminated environment. The airborne route is a major factor in the spread of *A. baumannii* infections in the hospital.⁷ Among the ESKAPE pathogens, *A. baumannii* is one of the most difficult.⁸ *A. baumannii* has a remarkable capacity for developing antibiotic resistance quickly, which within a few decades led to multidrug resistance.⁹ Factors that Increase Drug Resistance Long-term, i.e.(Central- venous- Catheterization, Urinary- catheterization, Prior- exposure) to potent antibiotics and invasive procedures are all risk factors for *Acinetobacter* colonization and infection.¹⁰ Gram-negative bacteria, particularly nosocomial pathogens like *Acinetobacter*, showed a high and steadily rising rate of Carbapenem-resistance.¹¹ *A. baumannii* resistance rates in hospitals in North America increased, going from "1.0% in 2003 to 58.0%" in 2008, according to data gathered. *Acinetobacter* species that are carbapenem-resistant seriously endanger

public health in Europe. Higher than 70 percent of any and all invasive *Acinetobacter* species isolates in Southern and Eastern Europe were carbapenem-resistant.¹² Carbapenem Resistance for *A. baumannii* is more common than 50% in many hospitals, especially in Intensive care unit settings, as well as emergence & developed quickly in South and Southeast Asia in 2017.¹³ Imipenem nonsusceptibility was linked to "ISAb1" upstream of the acquired carbapenemase *bla*_{OXA-23}, *bla*_{OXA-40}, *bla*_{OXA-58} or the intrinsic carbapenemase *bla*_{OXA-51}. Eight different clusters of isolates, including European clones First, second, and third, were formed. The most prevalent and significant group, with 246 isolates, was European clone II i.e USA.¹⁴ Prevalence- of specific genes in isolates of *A. baumannii* resistant to Imipenem of MBL-positive rate of OXA-51 was 44(97.77), OXA-23 was 44(100) and *bla*_{NDM} were 4(100).¹⁵ The mechanisms that make *A. baumannii* resistant to carbapenem put the drug's effectiveness in danger.

Mechanism of *Acinetobacter baumannii*'s resistance to carbapenem

The carbapenem resistance mechanism of *A. baumannii* can be divided into four groups:

Penicillin-binding protein changes

Due to the downregulation of the Penicillin-binding protein, *A. baumannii* strains exhibit low drug affinities and exhibit carbapenem resistance.¹⁶ In addition to the production of carbapenemases, the absence of 732-kDa PBP in *A. baumannii* isolates with imipenem minimum inhibitory concentrations (MICs) greater than 4 mg/L was associated with carbapenem resistance.¹⁷ PBPs play a role in only low-level CRAB main fact that is the mutations altering their production level or binding affinity cause resistance in beta-lactam antibiotics.¹⁸

Outer membrane porins are lost

Membrane permeability caused by reduced porin expression or porin mutation is another way that *Acinetobacter* species resist carbapenem. OMPs & porin channels are typically responsible for carrying antimicrobial agents into cells.¹⁹ Multiple of OMPs are involved in the transfer of beta-lactams across the membrane of *A. baumannii*.¹³

Effluent pumps being turned on

Efflux pumps (EP) may also influence *A. baumannii* susceptibility to carbapenems.²⁰ Efflux systems actively remove several antibiotics by pumping them out of the cell, in contrast to OMPs which are linked to antibiotic uptake. This causes multidrug resistance.²¹ Three of the five Efflux Pump families that are known to increase bacterial resistance—the Multidrug & Toxic compound extrusion (MATE) family, the Major facilitator superfamily (MFS), and the Resistance Nodulation cell division (RND) family most frequently found in pathogens.¹⁸

Synthesis of carbapenem-hydrolyzing beta-lactamases

The significant Carbapenem resistance mechanism in *A. baumannii*, entails the inactivation or enzymatic activity of carbapenems. Carbapenemase enzymes, which also are commonly found on plasmids and are highly infectious, usually carry out this process.²² Classes A, B, C, and D of *A. baumannii* molecular b-lactamase enzymes have been identified based on their own preferred substrate and catalytic domain (from Ambler classification system). Class C enzymes hydrolyze cephalosporins, whereas

carbapenemase belongs to classes A, B, and D. Metallo-b-lactamases (MBLs), also known as beta-lactamases, are enzymes that activate & disrupt the beta-lactam ring in the presence of an H₂O molecule & a Zn²⁺ (a divalent cation). Contrarily, non-Metallo carbapenemases such as Beta-lactamases from classes A, C, and D require serine for catalytic activity as shown in Figure.²³

Review

Indian scenario of *Acinetobacter* species and carbapenem-resistant

Neeraj Goel conducted a study on the relationship-between the use of antibiotics and the emergence of resistance in non-fermenters in tertiary care hospitals between 2002 and 2008. Blood cultures were positive 15,465 isolates (22% positivity rate) were grown from 69,010 blood samples. The non-fermenters among the 1525 isolates included 754 isolates of *A. baumannii*. Consuming carbapenem was associated with *A. baumannii*'s development of resistance ($r = 0.756$, $P = 0.049$), but other antimicrobials among non-fermenters. *A. baumannii* and carbapenem resistance were 74%.²⁵

A study by Jana M Swenson et al. stated that *Acinetobacter* spp. antimicrobial susceptibility

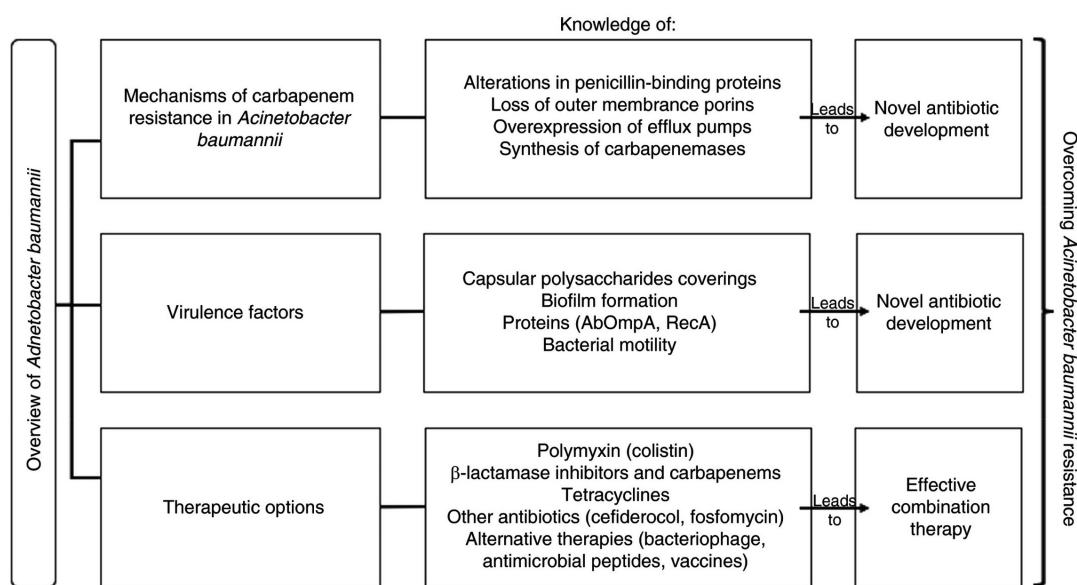


Figure. Important features in *Acinetobacter baumannii* such as its virulence factors and mechanisms of carbapenem resistance, as well as important therapeutic options for decreasing the frequency and chances of outbreaks caused by this pathogen (adapted from Lee et al.).²⁴

testing using NCCLS broth microdilution and disc diffusion test. The 196 isolates were identified. Of the 196 isolates, 149 (76%) belonged to the *Acinetobacter calcoaceticus-baumannii* complex (genomospecies 1, 2, 3, and 13).²⁶

Sadia Shakoor et al. conducted an in vitro study of Tigecycline & another tetracycline against carbapenem-resistant *Acinetobacter* species in 2006 in a tertiary care facility in Karachi, Pakistan. A total of 100 *Acinetobacter* spp. isolates were examined. In 98% of cases, *Acinetobacter* spp. was carbapenem-resistant.²⁷

M. Johanson described the need for improved antimicrobial and infection control stewardship in the Vietnamese intensive care unit in 2007. An average of 811 defined daily doses of antibiotics were consumed. In these studies, antibiotic consumption is correlated with the isolation of gram-negative bacteria is 80% of isolated bacterial strains. The antibiotic most commonly used were third-generation cephalosporins, followed by ampicillin, amoxicillin, and carbapenems. The *Acinetobacter* species were the most frequently pathogenic in blood cultures. The most frequently isolated bacterium from either the respiratory tract or every another source put together was *Acinetobacter*. In 79%, 80%, and 89% of cases, the *Acinetobacter* species were less susceptible to imipenem, ciprofloxacin, and ceftazidime, respectively.²⁸

Ali Faisal Saleem performed research on pan-resistant *Acinetobacter* infection in neonates in Karachi, Pakistan in 2008. *Acinetobacter* was grown in 122 cultures from 78 neonates during the 5 year study. Positive cultures were found in the following locations: (blood n = 57, trachea n = 55, tissue/wound/body fluids n = 4, eye n = 4, urine n = 1, and CSF n = 1.71%)of *Acinetobacter* isolates (87/122; only sensitive to Polymyxin) had pan-resistance.²⁹

A.Y. Kruse conducted a cohort study on neonatal bloodstream infection in a pediatric hospital in Vietnam in 2009-2010. Pathogenic isolates were responsible for 56% of the BSIs. The majority of pathogenic isolates recovered were gram-negative bacteria, the most common being *Acinetobacter* spp.³⁰

Murali Alagesan et al. conducted a single-center study from 2009 to 2013 to examine the Gram-negative blood culture isolates over ten

years and changes in susceptibility patterns. Only the Critical care unit yielded 4128 of *A. baumannii* isolates, 75% of which were carbapenem-resistant and only susceptible to polymyxin E and tigecycline.³¹

In 2010-2011, Gomty Mahajan et al. conducted research on carbapenemase phenotypic detection and carbapenem resistance in clinical isolates of *Acinetobacter baumannii*. By using the disc diffusion method, meropenem resistance was detected in 42 isolates (31.81%). Using the modified Hodge test, 47.6% of the samples were carbapenemase positive, and the EDTA disc synergy test revealed that 19% of the samples were phenotypical MBL producers (EDS). Most of these 42 isolates, came from patients admitted to ICU. Four *A. baumannii* complex isolates demonstrated resistance to all known medications, including tigecycline and polymyxin B.³²

Nguyen Thi Khanh Nhu determined that carbapenem-resistant *Acinetobacter baumannii* was the main cause of ventilator-associated pneumonia in ICU patients in 2010 in a hospital for infectious diseases in southern Vietnam. In the first eight years of the time series, 29% of all isolates per year were from *Acinetobacter* species. This proportion fell to 23% in 2008 before rising to 35% and 45%, respectively, in 2009 and 2010. The percentage of *Acinetobacter* spp. isolates increased by 6.6% yearly over the course of the study (OR 1.066, P = 0.022, 95% CI 1.02-1.12).³³

The Carbapenem-resistant isolates of *A. baumannii* were molecularly characterized by Atul Khajuria et al. between 2011 and 2013 in the ICU of a tertiary care facility in central India. By using the disc diffusion method, it was discovered that 155 out of 368 (42.11%) isolates of *A. baumannii* had decreased susceptibility to imipenem. 130 (83.87%) of the 155 tested isolates had MIC values for imipenem & meropenem that ranges from 16 to 64 mg per liter, according to Clinical laboratory standard institute breakpoints. 93 (60%) of the 155 isolates were found to produce carbapenemase, according to the Modified Hodge test. Out of 155 isolates, 89 (57.41%) tested positive for DDST, 73 (47.09%) for CDST, and 105 (67.74%) for MBL (IP/IPI) E-test. 47/105 (44.76%) of the 105 samples had the *bla*_{OXA-51} gene, 55/105 (52.38%) had the *bla*_{OXA-23} gene, and 15/105 (14.28%) had the *bla*_{OXA-58} gene.³⁴

In 2011, Shahzeera Begum conducted research on the frequency of multidrug-resistant *Acinetobacter baumannii* in a clinical sample from a tertiary care hospital in Islamabad, Pakistan. MDR prevalence in *A. baumannii* was found to be 100%. The antibiotic susceptibility profile revealed that tigecycline or minocycline were the most effective antibiotics against *A. baumannii*. Metallo-β-lactamase, and carbapenemase were produced by almost all *A. baumannii* isolates. Although ampC was present in 41.76% of the isolates, none of them produced ESBLs. Tetracycline is moderately effective against *A. baumannii*, as determined by the antibiogram as well as minimal inhibitory concentrations (MICs).³⁵

At Nepal's National Institute of Neurological & Allied Science in 2011–2012, Ganesh Thapa investigated nosocomial isolates and their drug-resistant pattern in ICU patients. 149 (79.67%) of the 187 total isolates were Gram-negative, and 121 (81.2%) of them were MDR. *Acinetobacter* spp. were the most frequently isolated Gram-negative bacteria, representing 58 (38.9%) of all isolates, 46 (79.31%) of which were MDR. With 23 isolates (15.4%), 20 of which were MDR (86.95%), *K. oxytoca* came in second. Similar to this, of the 38 total Gram-positive isolates, MDR isolates were discovered in 21 (55.2%) of them.³⁶

A systematic review of *Acinetobacter* species-caused urinary tract infections in hospitalized patients was done by Sanjeev et al. Of the 2240 culture-positive samples, 46 UTI patients had *Acinetobacter* spp. isolated from them. Imipenem came in second with 69.5%, followed by Tigecycline (91%), Meropenem (67.3%), and Gatifloxacin (63%), which had the lowest susceptibility. *Acinetobacter* species infection had spread throughout the bodies of the six patients who passed away. The most frequent risk factor for severe and widespread infection was mechanical ventilation.³⁷

According to a 2013 study by Reyes Martin peria et al., quantitative real-time PCR enables quick detection of antibiotic resistance in *A. baumannii*. Forty-eight clinical isolates of *A. baumannii* with a wide range of MICs of amikacin, colistin, ciprofloxacin, & imipenem were examined for growth using a real time PCR assay that targets a highly conserved region of the (outer membrane protein A) gene. 184 of 192

determinants underwent broth microdilution as a result (95.8%).⁶

In 2013 and 2014, Shirota Shrestha performed molecular epidemiology on isolates of the multidrug-resistant *A. Baumannii*, revealing the emergence of a new epidemic clonal lineage. Among the 246 *Acinetobacter* spp. C isolates were examined, and 129 distinct XDR/MDR isolates were discovered, including 122 *A. baumannii* isolates, 6 *A. calcoaceticus* isolates, and 1 *A. Berezina* isolate. The 122 *A. baumannii* isolates included 109 XDR and 13 MDR strains.³⁸

Identification and isolation of *Acinetobacter* species with an emphasis on antibiotic resistance, according to a Neetu Gupta et al. study in 2015. Of the 3298 infected samples, 111 (3.36%) contained *acinetobacter*. The most prevalent (72%) was the *Acinetobacter calcoaceticus & baumannii* (Acb) complex. The most resistant drug was piperacillin, which had a resistance rate of 55 percent, followed by Ceftazidime (46%) & Ceftriaxone (46%). Antibiotic resistance and isolation rates were increased in the hospital's intensive care units. "Extended-spectrum beta-lactamases and Metallo Beta Lactamases" production was suspected in 31.5% and 14.4%, respectively, of the isolates.³⁹

One of the studies was conducted on an environmental sample. A 2017 study claims that the discovery of antibiotic-resistant *Acinetobacter baumannii* in a variety of hospital environments as a potential source of *Acinetobacter* infection transmission Zahra Shamsizadeh et al. The approach Samples of air, water, and inanimate surfaces from four hospitals were examined for the presence of three of the most common OXA type carbapenemase encoding genes. The findings of this study revealed that 2% (1/42) of water samples, 17percent, (7/42) of surface samples, &11% (7/64) of air samples all contained *A. baumannii*.⁴⁰

The 2016 study "Emergence of carbapenem-resistant *Acinetobacter* in a Temperate North Indian State" by N.K. Bali et al. 165 non-duplicate *Acinetobacter* strains were examined during the study period. The proportion of imipenem-resistant isolates in blood samples (the number is 72) was significantly increased than the proportion of imipenem-sensitive isolates (n=twenty six, 36.1 percent). *Acinetobacter*

Table. Epidemiology of *Acinetobacter* species and Carbapenem-resistant

No.	Author	Year	%(<i>Acinetobacter</i> resistant isolates)	<i>Acinetobacter</i> isolates	Population screened	Sample type	Ref.
1.	Neeraj Goel	2002-2008	0-74%(754)	1,525	Inpatient	Blood sample	25
2.	Jana.M.Swe-nson	2004	76%(149)	196	Inpatient	All clinical sample	26
3.	Sadia Shakoor	2006	98%	100	Inpatient	All clinical sample	27
4.	M.Johanson	2007	69%	170	Intensive care unit	All clinical sample	28
5.	Ali Faisal	2008	89%	122	Intensive care unit	All clinical sample	29
6.	A.Y.Kruse	2009-2010	43%	58	Inpatient	Blood sample	30
7.	Murall Alagesan	2009-2013	67-74%(332)	4128	Inpatient	Blood sample	31
8.	Gomty Mahajan	2010-2011	42%(132)	42	Inpatient	All clinical sample	32
9.	N.T.K.Nhu	2010	80%	35	Intensive care unit	All clinical sample	33
10.	Atul Khajuria	2011-2013	42%(362)	368	Intensive care unit	All clinical sample	34
11.	Shahzeera B	2011	100%	91	Inpatient& outpatient	Wound sample	35
12.	Ganesh Thapa	2011-2012	17%	58	Intensive care unit	All clinical sample	36
13.	Sanjeev H	2013	91%(2240)	2240	Inpatient	All clinical sample	37
14.	Shorita Shrestha	2013-2014	98%	122	Inpatient & outpatient	All clinical sample	38
15.	Neetu Gupta	2015	72%(111)	3298	Intensive care unit	All clinical sample	39
16.	N.K.Bali	2016	36.1%(65)	165	Inpatient	Blood sample	41
17.	Velma Rebica	2018	90%(399)	622	Inpatient	All clinical sample	42

baumannii was found to be responsible for 65 of the carbapenem-resistant isolates.⁴¹

According to Velma Rebica's et al. study from 2018, *Acinetobacter* species are important in hospital settings. Because of this, 399 (62.18%) of the 622 isolates came from inpatients and 223 (37.82%) came from outpatients. Higher than 90 percent of the isolates exhibited resistance to ampicillin, amoxicillin clavulanic acid, ceftazidime, ceftriaxone, and amikacin.⁴²

A study titled MDR pattern of *Acinetobacter* spp. isolated from clinical specimens was published in 2021 by Zeleke Ayenew et al. 102 *Acinetobacter* spp. strains from various clinical samples were analyzed as a result. Blood (23.5%), pus (33.3%), urine (15.6%), and bodily fluid (11.7%) were the next most common sources of fluids. The antimicrobial resistance patterns for Piperacillin tazobactam is 67.8%, Ciprofloxacin 59.4%, Ceftazidime 82.1%, Ceftriaxone 87.1%, Cefepime 80.0%, and Meropenem are 12.5% were all significantly ascending. But for tobramycin (from 56.5% to 42.8%), Amikacin (from 42.1% to 31.4%), and antimicrobial resistance decreased. *Acinetobacter* species had average rates of 56.7% multidrug resistance and 71.6% Carbapenem non-susceptibility.⁸

A study on the frequency & mechanism of the carbapenem-resistant gene in *Acinetobacter baumannii* was done in 2022 by Komal Khalid et al. The OXA-51-like gene was discovered to be present in 45 (97.82%) of the *Acinetobacter baumannii* strains, and the OXA-23-like gene was discovered to be present in 44 (95.65%) of these strains. Additionally, four (9.09%) Carbapenem-resistant *Acinetobacter baumannii* (CRAB) isolates tested positive for the New Delhi MBL, or *bla*_{NDM} gene. All *bla*_{NDM} isolates co-expressed OXA-23-like, whereas only one carbapenem-resistant *Acinetobacter baumannii* isolate that has been *bla*_{NDM} positive lacked OXA-51-like.¹⁵

A study on Prevalence of multidrug-resistant *Acinetobacter baumannii* in a critical care setting: A tertiary teaching hospital experience in 2021 by Thabit Alotaibi et al. The total sample were identified by 198 patient with *Acinetobacter baumannii*. The prevalence of *Acinetobacter baumannii* is 3.37% and overall mortality rate is 40.81%. According to the analysis of the care given to patients with *Acinetobacter baumannii*

infections, 65 patients received colistin alone, 18 received carbapenems, and 22 received both carbapenems and colistin. Patients with *Acinetobacter baumannii* infections spent an average of 20.25 days in the hospital. Our research revealed that the survival rates among patients receiving carbapenems had higher rates of death than those receiving colistin.⁴³

A study on Epidemiology and outcomes associated with carbapenem-resistant *Acinetobacter baumannii* and carbapenem-resistant *Pseudomonas aeruginosa*: a retrospective cohort study in 2022 by Amanda vivo et al. The 90-day mortality rates for CRAB and CRPA were 30.3% and 24.5%, respectively, and the inpatient post-LOS was 26 and 27 days in 1,048 and 8,204 different patients, respectively. In comparison to urine cultures, positive blood cultures in patients with CRAB (OR 6.98, 95% CI 3.55-13.73) and CRPA (OR 2.82, 95% CI 2.04-3.90) were linked to higher odds of 90-day mortality. Higher Charlson scores were related to higher odds of 90-day mortality in patients with CRAB and CRPA blood cultures. Blood cultures were linked to a lower LOS compared to urine cultures among patients from inpatient care settings in CRAB and CRPA. All the above studies are mentioned in Table.⁴⁴

DISCUSSION

The pathogen *Acinetobacter* is a nosocomial one. Infectious disease experts are worried about its potential to develop antimicrobial drug resistance and infect healthy hosts. A study from India of *Acinetobacter* species from 2002-2022 ranges from 3% to 100%.

Acinetobacter prevalence in Maharashtra was found to be only 3%, according to a 2015 study by Neetu Gupta et al. Blood 41 is (36.9%), pus 25 is (22.5%), respiratory sample 16 is (14.4%), urine 13 is (11.7%), and other clinical samples containing *Acinetobacter* species isolation profiles. These are all the samples taken from the Intensive care unit. The most common patients with *Acinetobacter* bacteremia are those who are critically ill and are admitted to intensive care units (ICUs), as these patients frequently need repeated invasive procedures, a prolonged hospital stay, and broad-spectrum antimicrobial therapy.⁴⁵ In earlier studies, the majority of *Acinetobacter* isolates

from our study came from blood samples and intensive care units.⁴⁶

A study by Sanjeev et al. in Mangalore isolated a higher percentage, i.e. 100% of *Acinetobacter* isolates in Urinary tract infections of Hospitalized patients. All samples are urine samples.³⁷

Hydrolyzing enzymes like Metallo beta-lactamases and OXA carbapenemases may play a role in carbapenem resistance. Imipenem and Meropenem are two examples of the drugs of choice in the past for treating infections caused by *Acinetobacter* species.⁴⁷ The Antibiotic sensitivity pattern of Neetu Gupta et al. is Amikacin 47(42%), Ciprofloxacin 26(23%), Imipenem 24(22%), Piperacillin 61(55%), Ceftazidime 51(46%), Cefepime 49(44%) and Ceftriaxone 51(46%).³⁹ The Antibiotic sensitivity pattern of Sanjeev H et al. is Imipenem 32(69.5%), Meropenem 31(67.3%), Piperacillin Tazobactam 08(18%), Cefepime 08(18%), Ceftazidime 08(18%) and Ceftriaxone 08(18%).³⁷

Acinetobacter isolates showing Carbapenem resistance from 2002 to 2018 varies between 74-90%, but a study by Bali et al. in Srinagar show less percentage of Carbapenem-resistant, i.e. 36%. They have collected all clinical samples from the ICU. The antibiogram of the 164 different organisms that make carbapenemase. Ceftriaxone, ceftazidime, amikacin, piperacillin-tazobactam, and ciprofloxacin.⁴¹

In the current study, it was discovered that there were significantly more *Acinetobacter* spp., 40, which is (24.4%); (P=0.009). It has been reported that this organism frequently causes infections and that these infections are becoming harder to treat because the majority of *A. baumannii* recovered from patients is MDR.⁴⁸

The world studies from 2006-2014 show 43% to 100% resistance, whereas a study by Ganesh Thapa et al. 2011-2012 in Nepal shows less carbapenem resistance 17%. *Acinetobacter* isolates from all clinical samples in the Intensive care unit. The Antibiotic profile pattern is Imipenem 17.24, Ciprofloxacin 82.75, Cefepime 86.20, Cefoxitime 82.75, Cotrimaxole 93.83, and amikacin 67.24.³⁶

The review shows that in all the above studies, Carbapenem resistance of *Acinetobacter* isolates is in the rapidly increasing order.

CONCLUSION

Infection with *Acinetobacter* is linked to high mortality and morbidity. According to our review, patients who have an infection with *A. baumannii* may be at an increased risk of dying. However, due to the confounding factors of illness severity, inappropriate impractical antimicrobial treatment, and small sample size, cautious interpretations are necessary.

We covered the main factors that make *A. baumannii* such a prevalent nosocomial pathogen in this review, including such as its virulence components, desiccation resistance, and carbapenem resistance mechanisms. With the emergence of extended resistance to even more recent antibiotics, *Acinetobacter* species are rapidly proliferating. They can develop resistance much more quickly than other gram-negative organisms .

ACKNOWLEDGMENTS

None.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHORS' CONTRIBUTION

Both authors listed have made a substantial, direct and intellectual contribution to the work, and approved it for publication.

FUNDING

None.

DATA AVAILABILITY

All datasets generated or analyzed during this study are included in the manuscript.

ETHICS STATEMENT

Not applicable .

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