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Clinical and Antimicrobial Resistance Patterns of Acinetobacter Species: Implications For Future AI-Enabled Hospital Surveillance

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ABSTRACT

Background: Acinetobacter species has surfaced as a significant hospital pathogen and are getting significant importance due to its increasing medicine resistance. They cause outbreaks in intesive care units and health care units. This growing resistance burden highlights the need for improved surveillance approaches in hospital settings. The increasing availability of hospital microbiology and antimicrobial susceptibility data supports future AI-enabled surveillance, with machine-learning and predictive analytics helping identify emerging resistance trends and high-risk multidrug-resistant patterns.

Material and method: A cross-sectional study was conducted to determine the frequency and antibiotic resistnace pattern of Acinetobacter spp isolated from different clinical samples collected from cases admitted in various wards and intensive care units of the hospital over a period of 2 year (t November 2020 to December 2022).

Results: Out of 3715 samples, out of these, 120 (3.23%) Acinetobacter spp were isolated. These had been isolated from urine, pus, blood and from and various other body fluids. 59.68% was from general ward and 40.32% was from ICU. Samples received in Microbiology laboratory during the study period. Maximum resistance was seen to Ceftazidime 77.50%, followed by Levofloxacin 76.67%. Out of 120 Acinetobacter isolates 60.83% were multidrug resistance.

Conclusions: *Acinetobacter* isolates showed substantial multidrug resistance, highlighting the need for judicious antibiotic use, strict hand hygiene, and effective infection-control practices. These resistance patterns may also support future AI-enabled surveillance, machine-learning and data-mining approaches for early warning and antimicrobial stewardship.

Keywords: Acinetobacter spp; nosocomial infection, antibiotic resistance; intensive care units; multidrug resistance; AI-enabled hospital surveillance; machine learning; predictive analytics.

INTRODUCTION

A Dutch microbiologist, by name Martinus Willem Beijerinck discovered in 1911, an aerobic, Gram-negative, non-fermentative bacterium we now know to be of the genus Acinetobacter.[1] The genus Acinetobacter are Gram-negative, rigorously aerobicnon-fermenting,non-fastidious,non-motile, catalase-positive and oxidase negative coccobacillary bacteria. They prefer wettish terrain and can fluently attained from soil, water, food and sewage.[2] They're generally considered to be opportunistic pathogens, and of recent have been reported to beget a number of outbreaks of nosocomial infections in rehabilitated cases like septicemia, pneumonia, crack sepsis, endocarditis, meningitis and urinary tract infections(UTI).[3, 4] similar infections are frequently extremely delicate for the clinician to treat because of the wide resistance of these bacteria to the major group of antibiotics. further than two third of Acinetobacter infections are due to Acinetobacter baumanii. Acinetobacter baumanii causes health care associated infections.[5- 8] Acinetobacter baumanii also has the capability to form biofilms, which may play a part in the process of colonization.[9] Acinetobacter associated infections represent a tough challenge to control in oppressively ill cases especially those in ICU. Acinetobacter species have the capacity to acquire resistance to nearly all presently being antimicrobial agents.[10] Despite the adding significance and frequence of multidrug resistant Acinetobacter infections, numerous clinicians and microbiologists still warrant an appreciation of significance of these organisms

because of their confused taxonomic status.[11] Because of their adding significance of nosocomial infections and multidrug resistant pattern, farther study is warranted. This growing burden of antimicrobial resistance highlights the need for more systematic approaches to hospital surveillance and early detection of changing resistance patterns. The increasing availability of hospital microbiology and antimicrobial susceptibility data also creates opportunities for future use of artificial intelligence and machine learning in hospital antimicrobial resistance surveillance. Such approaches may help identify resistance trends, detect emerging multidrug-resistant patterns, and support early warning and antimicrobial stewardship in healthcare settings.[12] Future AI-enabled surveillance may further use predictive analytics and automated pattern recognition to identify unusual resistance profiles, detect possible resistance clusters, and support timely infection-control decisions. Data-mining approaches may also help analyse accumulated microbiological records to recognise changing antimicrobial resistance trends across hospital settings.[13] Variables routinely generated in such microbiological surveillance, including Acinetobacter species, specimen type, hospital location, antimicrobial susceptibility profile, and multidrug-resistance status, may serve as structured inputs for future machine-learning-based resistance-risk assessment and automated hospital surveillance.[14] In the present study attempt was made to find out the frequence of Acinetobacter isolates attained from colorful clinical samples collected from cases admitted in colorful ICUs and wards by phenotypic identification scheme and also determine their antimicrobial vulnerability pattern and to consider the relevance of these findings for future AI-enabled hospital surveillance. The clinical and resistance patterns generated through such studies may therefore provide useful structured information for the future development of machine-learning-assisted hospital surveillance and data-driven antimicrobial resistance monitoring.[15]

Material and system This study was conducted in the Department of Microbiology, Government Medical College Nagpur, Maharashtra and included Acinetobacter species insulated from colorful clinical samples over a period of two time(November 2020 to December 2022). A aggregate of 3715 clinical samples similar as pus, urine, blood, catheter tips, tracheal aspirate, foam and other body fluids were collected from cases, admitted in ICU and different wards of sanitarium. The samples entered in the laboratory were invested on 5 Sheep Blood Agar and Mac Conkey agar and incubated overnight aerobically at both 37 ◦ C. All isolates attained were further reused and linked by routine microbiological and biochemical tests. In case of urine samples, the isolates were subordinated to biochemical tests only if the colony count was significant(> 105 CFU/ ml) and if set up significant, urine samples were invested on Cysteine Lactose Electrolyte Deficient(CLED) agar. All isolates attained were further reused and linked by standard routine microbiological processes. Genus Acinetobacter was linked by Gram staining as Gram negative coccobacilli, colony morphology(Non Lactose- fermenting, glistering, small mucoid colonies),non-motile, oxidase negative, catalase positive, TSI response(K/ K) and citrate application test positive. Speciation of Acinetobacter was done on the base of glucose oxidation(OF test), heamolysis on blood agar, growth at 37 °C and 44 °C, citrate application, Arginine decarboxylation, Glucose application, as shown in(Table 1).[16]

Table 1. Speciation scheme of Acinetobacter species.12

Name of test	**Acinetobacter species**				
	A. baumannii	A. lwoffii	A. haemolyticus	A. junii	A. radioresistens
Gram staining	Gram negative cocci or coccobacilli				
Urease	V	V	-	-	-
10 % Lactose	F	NF	NF	NF	NF
Citrate utilisation	+	-	-	-	-
OF glucose	+	-	V	-	-
Haemolysis	-	-	+	-	-
Gelatine hydrolysis	-	-	+	-	-
Growth at 42° C	+	-	-	-	-

Growth at 37° C	+	+	+	+	+
Arginine hydrolysis	+	-	+	+	+

After identification by phenotypic methods, antibiotic susceptibility was performed for each isolate by the Kirby-Bauer disc diffusion method on Mueller-Hinton agar using 0.5 MacFarland turbidity standard and comparing zone sizes with control strain Pseudomonas aeruginosa ATCC 27853.[17] The antimicrobial agents used were-ceftazidime (30µg), cefepime (30µg)), ceftazidime and clavulinic acid, piperacillin-tazobactam (100µg)/10µg)), imipenem (10µg)), meropenem (10µg), gentamicin (10µg), amikacin (30µg), cotrimoxazole (25µg), ciprofloxacin (5µg)), levofloxacin, norfloxacin (30µg) and nitrofurantoin (300) (for urinary isolates), and colistin (10µg). Antibiotic susceptibility results were interpreted by measuring the zone diameters produced and correlating them with the CLSI standards.[18]

Results

During the study period, 3715 were positive for bacterial growth of which 120 (3.23%) were culture positive for Acinetobacter spp.

The isolation rate of Acinetobacter spp. was maximum in General ward 94 (78.33%) followed by ICU 26(21.67%). It has been observed that there was a higher incidence of Acinetobacter infection in males (52.50%) then females (47.50%).

Acinetobacter spp. was more common in patient with age group of 21-30 years with an incidence of (40%).

Among various clinical samples received in the laboratory the isolation rate of Acinetobacter spp was maximum from tracheal aspirate sample 32(26.66%), followed by pus 22.50%, blood 13.33%, pleural fluid 12.50%, urine 7.50%, bronchoalveolar lavage 6.667%, sputum 5.83%, ascitic fluid 2.50% isolates, central venous pressure line tip 1.68% and only 0.83% isolate from cerebrospinal fluid (Table 2)

Table 2. Specimen distribution of Acinetobacter isolates. (n=120)

S. no	Specimen	No of isolates (%)
1.	Tracheal aspirate	32(26.66%)
2.	Pus	27 (22.50%)
3.	Blood	16(13.33%)
4.	Pleural fluid	15(12.50%)
5.	Urine	09 (7.50%)
6.	Bronchoalveolar lavage	08 (6.67%)
7.	Sputum	07 (5.83%)
8.	Ascitic fluid	03 (2.50%)
9.	Central venous pressure line tip	02 (1.68%)
10.	Cerebrospinal fluid	01 (0.83%)
11.	Total	120 (100%)

In our study we had found out that the most common infection caused by Acinetobacter is Pneumonia 39.17%, followed by skin and soft tissue infection 22.50%. It has been found that Acinetobacter is also responsible for other infections also such as septicaemia 15%, pleural effusion 12.50%, urinary tract infection 7.50%, ascites 2.50% and meningitis 0.83%. (Figure 1)

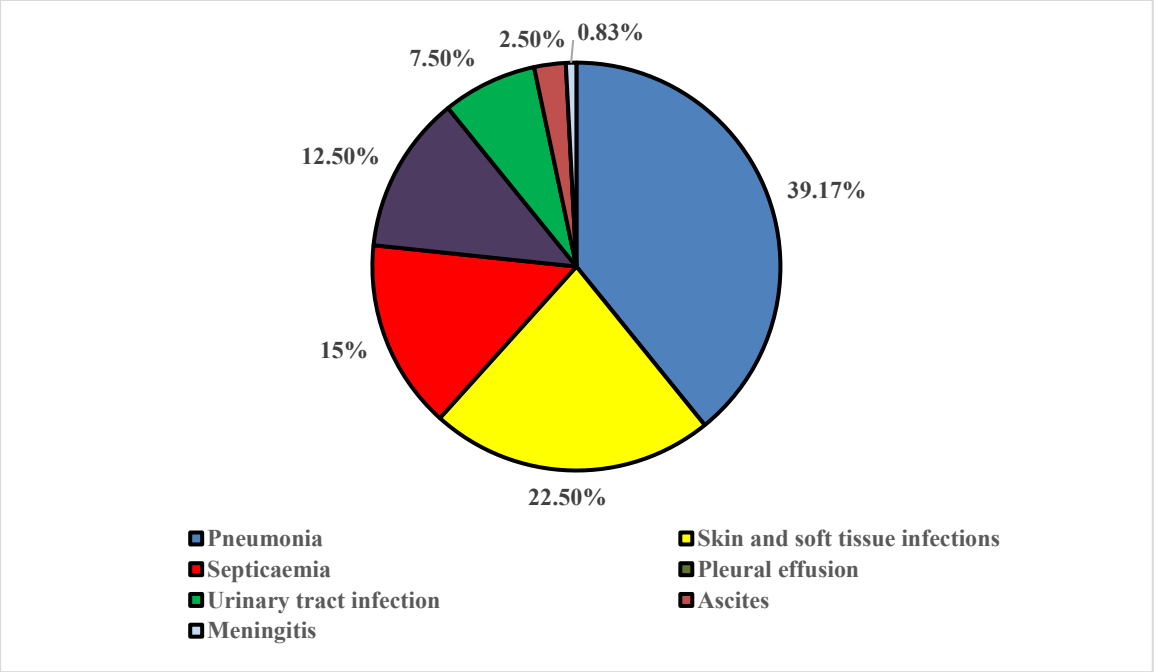

Figure 1: Infections caused by Acinetobacter spp (n=120)

The most predominant species of Acinetobacter isolated was A. baumannii 54.17% followed by A. lwoffii 37.50%, A. haemolyticus 4.17%, A. radioresistens 2.50% and A. junii 1.66% (Table 3).

Table 3. Species distribution of Acinetobacter isolates. (n=120)

S.no	Species	Total
1.	A.baumannii	65(54.17%)
2.	A.lwoffii	45(37.50%)
3.	A.haemolyticus	05(4.17%)
4.	A.radioresistens	03(2.50%)
5.	A.junii	02(1.66%)
	Total	120(100%)

Maximum number of isolates of Acinetobacter Species from wards was A.baumannii (65) followed by A. lwoffii (36), A. haemolyticus (03), A. radioresistens (03) and A. junii (02).
(Figure 2)

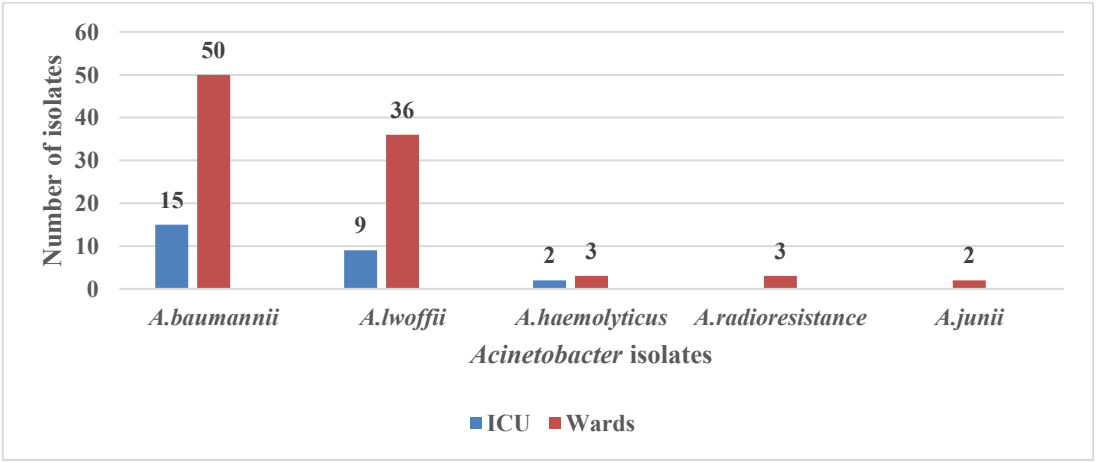

Figure 2: Species wise distribution of Acinetobacter isolates in various hospital wards (n=120)

Resistance pattern of Acinetobacter species to different antimicrobials showed Maximum resistance to Ceftazidime 77.50%, followed by Levofloxacin 76.67%, cefepime 70.83%, gentamicin 69.17%, tobramycin 66.67%. Minimum resistance was seen to Trimethoprim- sulfamethoxazole 48.33%, followed by imipenem 50.00% and minocycline 60.83% (Table 4)

Group	Antimicrobial agent	Resistant isolates number (%)
Group I	Ceftazidime	93(77.50%)
	Ciprofloxacin	92(76.67%)
	Gentamycin	83(69.17%)
	Tobramycin	80(66.67%)
	Levofloxacin	78(65.00%)
	Imipenem	60(50.00%)
Group II	Cefepime	85(70.83%)
	Piperacillin tazobactam	75(62.50%)
	Amikacin	77(64.17%)
	Minocycline	73(60.83%)
	Trimethoprim- sulfamethoxazole	58(48.33%)

The number of drug-resistant Acinetobacter isolates which were multi drug resistant (MDR) was 73 (60.83%). All MDR isolates were resistant to at least one agent in three or more antimicrobial categories; penicillins, cephalosporins, aminoglycosides, fluoroquinolones and carbapenems.

It has been found out that out of 60 imipenem resistance isolates 5.00% were sensitive, 3.33% intermediate resistance and 91.67% were resistant.(Figure 3)

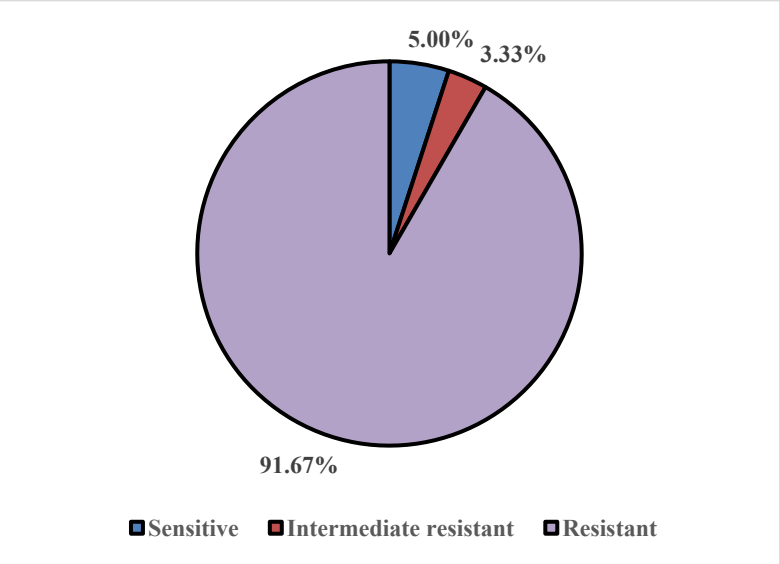

Figure 3: Imipenem Minimum Inhibitory Concentration (MIC) among carbapenem resistant isolates by E strip (n=60)

Discussion

Acinetobacter is an emerging pathogen, and over the last three decades it has proven to be particularly difficult to treat by healthcare services. Acinetobacter species, once considered to be a part of the commensal flora of humans, now pose a serious challenge, especially in health-care settings where it is now an important cause of life-threatening infections. Acinetobacter causes many infections including ventilator-associated pneumonia, meningitis, urinary tract infection, bone/wound infections, and the most serious type is the bacteraemia. Acinetobacter spp has emerged as a one of the cause of ICUs infections due to its ability to survive on inanimate surfaces for prolonged time periods extending from 3 days to 5 months, facilitates its spread in health-care settings thus, it can be easily detected on various common and routine use health care set up items as sinks, floors, cupboards, bed linens, mattresses, bed rails, curtains, hospital trolleys, and ventilation equipment such as respirators and AMBU bags.[19]

Acinetobacter can cause a wide variety of clinical syndromes, most prominently respiratory tract infections but also bacteraemia, skin and soft tissue infections, urinary tract infections, osteomyelitis and intracranial infections. Due to the limited therapeutic options for multidrug-resistant Acinetobacter infections, prevention of health care- associated transmission is critical to prevent morbidity.[20]

Multi-drug-resistant Acinetobacter baumannii isolates are key pathogens that contribute to the global burden of antimicrobial resistance. The problem continues to escalate, with many hospitals worldwide now reporting outbreaks caused by multidrug resistant strains of Acinetobacter. The most clinically significant pathogen is Acinetobacter baumannii because of the rapidity with which it develops antimicrobial resistance (including strains resistant to all commercially available antibiotics).[20]

In this context, routinely generated hospital microbiology data may also have value for future AI- and machine-learning-based surveillance, where patterns of species distribution, specimen type, hospital location, and antimicrobial resistance could be analysed to identify emerging resistance trends and high-risk patterns.[12] Predictive analytics and data-mining approaches may further help recognise unusual resistance combinations, changing hospital-level trends, and possible clusters of multidrug resistance, thereby supporting earlier surveillance alerts and more focused infection-control responses.[13] Against this background, the present study provides clinically relevant resistance data that may inform such future surveillance approaches.

In our study, a total number of 120 (3.23%) Acinetobacter strains were isolated from 3715 clinical specimen. Similar finding of 3% and 3.36% of total organisms isolated was reported by Dash et al [21] in Odisha and Gupta et al [22] in Pune respectively.

Of the 120 Acinetobacter isolates found in our study, 94 (78.33%) Acinetobacter strains were from patients admitted in wards. Islahi et al [23] (2014) and Mindoli et al [24] (2010) showed findings 73.91% and 73% isolation of Acinetobacter respectively from general wards similar to our study. This is probably related to increasingly invasive diagnostic procedures used, greater quantity of broad-spectrum antimicrobials used and prolonged duration of stay in hospital.[21] In contrast to our study, very lower rates of 18.7% were reported by Jaggi et al [25] (2012).

Out of 120 isolates, 23 (21.67%) Acinetobacter were responsible for ICU infections in our study. Sinha et al22 (2013) reported 22.14% which nearly similar to our study findings and Dash et al [21] (2013) reported 45.20% rates from ICU. This variation can be attributed to the varying prevalence rates of different Acinetobacter spp in the hospital environment and the community in different geographical areas.[21]

In the present study, Acinetobacter infections were found in both the sexes and also in all age groups, but the most common age group is 20-30 years. It was observed that Acinetobacter isolates were more common in males (58.82%) as compared to females and male to female ratio was 1.1:1. This may be due to the fact that the males report more frequently to the hospitals compared to females.

Dash et al [21] (2013) in their study reported male: female ratio 1.08:1 and Mindolli et al [24] (2010) reported male to female ratio 1.6:1.

Currently at least 31 genome species have been described within the genus Acinetobacter. Some species such as A. johnsonii, A. lwoffi and A. radioresistens seem to be natural inhabitants of human skin and may also be commensals in human oropharynx and vagina. A. lwoffii has been more commonly associated with meningitis than other species and A. junii rarely causes ocular infection and bacteremia.[16]

In our study, we have found that out of the 120 Acinetobacter isolates, A. baumannii (54.17%) was the most common species isolated from various clinical samples, it is also most commonly associated with various infections followed by A. lwoffii (37.50%), A. haemolyticus (4.17%), A. radioresistance (2.50%) and A. junii (1.66%).

In the present study, maximum Acinetobacter isolates were from tracheal aspirate 32(26.66%), followed by pus 27(22.50%), blood 16(13.33%), pleural fluid 15(12.50%), urine 09(7.50%), bronchoalveolar lavage 08 (6.667%), sputum 07(5.83%), ascitic fluid 3(2.50%) isolates, central venous pressure line tip 02(1.68%) and only 1(0.83%) isolate from cerebrospinal fluid.

Gupta et al [22] predominantly isolated Acinetobacter strains from blood samples 41 (36.9%) followed by pus 25 (22.5%), respiratory samples 16 (14.4%), urine 13 (11.7%), other body fluids 10 (9%) and various catheter tips 6 (5.4%). Apoorva et al. [26] found maximum number of Acinetobacter isolates from respiratory

samples (35.78%) followed by pus (32.84%). Pooja et al. [24] also isolated 25.6% of the Acinetobacter isolates from respiratory tract.

This indicates that Acinetobacter infections were most frequently involved in the respiratory tract of intubated patients. The higher incidence of isolation of Acinetobacter sp. from respiratory tract samples has also been previously well documented by various researchers worldwide.[25]

There are three major factors possibly contributing to the persistence of A. baumanii in the hospital environment, i.e., resistance to major antimicrobial drugs, resistance to dessication, and resistance to disinfectants. Resistance to antibiotics may provide certain A. baumanii strains with a selective advantage in an environment, such as the modern ICU, when microorganisms are confronted with extensive exposure to antimicrobials.[21]

We have studied the antimicrobial resistance pattern among 120 Acinetobacter isolates by Kirby-Bauer disc diffusion method and recorded maximum resistance to Ceftazidime (77.50%), followed by Levofloxacin (76.67%), cefepime (70.83%), gentamicin (69.17%), tobramycin (66.67%). Minimum resistance was seen to Trimethoprim- sulfamethoxazole (48.33%), followed by meropenem (53.33%) and minocycline (60.83%).

Prashanth et al [27] (2006) in his study, recorded high levels of resistance for ampicillin (85%), cefazolin (92.2%), gentamicin (63%), piperacillin (88%) and norfloxacin (69%). A study conducted by Joshi et al [34] (2006), reported high levels of resistance to tobramycin (97.9%) followed by cefazolin (96.5%), cefuroxime (95.4%), piperacillin (92.8%), cefoperazone (85.1%) and cefotaxime (82.2%).

This wide variation can be due to various factors like patients' co-morbid conditions, compliance of infection control programs, type of strains, their survival in the environment and further colonization of the patients. The resistance patterns detected in Acinetobacter could reflect the antibiotic misuse and lack of regulations on the over-the-counter sale in some parts of the World. Our study suggested that due to the increasing resistance of Acinetobacter, we should judiciously use antibiotics by making an attempt to distinguish colonization from infections and treatment should be only given to the clinically confirmed Acinetobacter infections and not merely colonization.[21]

In our study, out of 120 isolates, 73 (60.83%) isolates were found to have multiple drug resistance. However, Dash et al [21] (2013) recorded MDR in 54.7% isolates of Acinetobacter. Whereas, Badave et al (2015) in their study found very high MDR isolates 90.3%. Similarly, Mahajan et al. [28] (2011) and Tripathi et al. [29] (2014) has reported multidrug resistance of 70% and 89.71% respectively.

The observed MDR and antimicrobial-resistance patterns illustrate the type of structured laboratory information that could be incorporated into future AI-enabled hospital surveillance systems. Machine-learning approaches may potentially support automated recognition of changing resistance profiles and early identification of high-risk MDR patterns, thereby complementing antimicrobial stewardship and infection-control activities.[12] In future surveillance frameworks, supervised learning could support risk classification of resistant isolates, while unsupervised approaches could assist in identifying previously unrecognised resistance clusters within routinely accumulated hospital data.[15] Explainable AI may also improve clinical usefulness by showing which microbiological features contribute most strongly to surveillance alerts or risk patterns.[14]

More specifically, variables generated in this study, including species identity, specimen source, hospital location, antimicrobial susceptibility profile, multidrug-resistance status, and carbapenem resistance, could form structured inputs for future AI-assisted surveillance models.[14] Integration of these variables may enable automated risk stratification, detection of emerging resistance clusters, and generation of early-warning signals for infection-control teams. Such systems could also support data-driven antimicrobial stewardship by prioritising high-risk resistance patterns for timely clinical review.[13] Carbapenem resistance is particularly relevant in this context because it represents an important high-risk resistance pattern for hospital surveillance.

In our study, we have found that there are 60 (50%) isolates which shows resistance to imipenem. These isolates by disc diffusion were further evaluated for Minimum inhibitory concentration (MIC) by E-strip and we have found 5.00% were sensitive, 3.33% intermediate resistance and 91.67% were resistant. [30-33]

Chakraborty et al [34-35] showed 11.36 % resistance to carbapenem in the year 2011. This could be explained by their stringent antibiotic policies and judicious use of carbapenems in their countries. Dash et al [21] in his study conducted in the year 2013 showed 19% resistance to carbapenem. Similar study conducted by Tripathi et al [29] showed 40% resistance to carbapenem in year 2014.

Conclusions

These days the rate of sequestration of Acinetobacter spp indicated by various studies indicates its part as nosocomial pathogen, especially in critically ill cases admitted in ICUs. It's a great challenge for the croakers to treat MDR Acinetobacter spp. In our study, Acinetobacter were resistant to utmost generally used antibiotics. Emergence of carbapenem resistance is worrisome. Rational use of antibiotic is necessary to help microbial resistance. Though the organism has developed multidrug resistance, it has largely remained susceptible to cleansers and antiseptics. A strict control of the sanitorium terrain, hand hygiene and

optimizing judicious use of antibiotics is recommended in order to reduce the resistance rates and also to reduce the MDR frequency in the sanitorium. Beyond these immediate infection-control implications, the resistance patterns identified in this study may also have value for future data-driven surveillance.

The clinical and antimicrobial resistance patterns identified in this study may also support the future development of AI-enabled hospital surveillance systems for monitoring emerging resistance trends, identifying high-risk multidrug-resistant patterns, and strengthening antimicrobial stewardship and infection-control practices. Integration of such routinely generated microbiological data with machine-learning, predictive analytics, and data-mining approaches may further support automated resistance monitoring, early-warning systems, and data-driven clinical surveillance. Such integration may also enable future risk stratification and explainable AI-assisted identification of the microbiological factors contributing to high-risk resistance patterns.

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References

1. Nemec A, Musilek M, Maixnerova M et al. Acinetobacter beijerinckiisp. nov. and Acinetobacter gyllenbergiisp. Nov., hemolytic organisms isolated from humans. Int J Syst Evol Microbiol. 2009; 59:118-24.
2. Gerner – Smidt P. Taxonomy and epidemiology of Acinetobacter infections. Rev Med Microbiol. 1995; 6:186-97.
3. Tower K J. Clinical importance and antibiotic resistance of Acinetobacter spp. J Med Microbiol. 1997; 46: 721-46.
4. Levi I, Rubinstein E. Acinetobacter infections-overview of clinical features. In: Bergogne-Berezin I, Joly-Guilloo MI, Towner KJ, editors. Acinetobacter: microbiology, epidemiology, infections, management. Boca Raton, CRC Press. 1996; 101-15.
5. Davis Ka, Moran KA, McAllister CK et al. Multidrug resistant Acinetobacter extremity in soldiers. Emer Infect Dis. 2005; 11(8); 1218-24.
6. Oncul O, Keskin O, Akar HV et al. Hospital-acquired following the 1999 Marmara earthquake. J Hosp Infect. 2002; 51(1):47-51.
7. Young LS, Sabel AL, Price CS et al. Epidemiologic, clinical and economic evaluation of an outbreak of multidrug resistant Acinetobacter baumanii infections in a surgical
8. intensive unit. Infect Control Hosp Epidemiol. 2007; 28(11): 1247-54.
9. VanLooveren M, Goossens H. ARPAC steering Group. Antimicrobial resistance of Acinetobacter species in Europe. Clin Microbial Infect. 2004; 10(8): 684-704.
10. Rajmohan G, Srinivasan VB, Gebreyes WA. Blocide- tolerant multidrug-resistant Acinetobacter baumaniic linical strains are associated with higher biofilm formation. J Hosp Infect. 2009; 73(3): 287-9.
11. Murray CK, Hospenthal DR. Acinetobacter infection in the ICU. Crit Care Clin. 2008; 24: 237-48.
12. de la Lastra JM, Wardell SJ, Pal T, de la Fuente-Nunez C, Pletzer D. From data to decisions: leveraging artificial intelligence and machine learning in combating antimicrobial resistance–a comprehensive review. Journal of medical systems. 2024 Aug 1;48(1):71.
13. Baddal B, Taner F, Uzun Ozsahin D. Harnessing of artificial intelligence for the diagnosis and prevention of hospital-acquired infections: a systematic review. Diagnostics. 2024 Feb 23;14(5):484.
14. Kim JI, Maguire F, Tsang KK, Gouliouris T, Peacock SJ, McAllister TA, McArthur AG, Beiko RG. Machine learning for antimicrobial resistance prediction: current practice, limitations, and clinical perspective. Clinical microbiology reviews. 2022 Sep 21;35(3):e00179-21.
15. Sakagianni A, Koufopoulou C, Feretzakis G, Kalles D, Verykios VS, Myrianthefs P. Using machine learning to predict antimicrobial resistance—a literature review. Antibiotics. 2023 Feb 24;12(3):452.
16. Raina D, Sharma N, Mahawal B et al. Speciation and antibiotic resistance pattern of Acinetobacter spp in a tertiary care hospital in Uttarakhand. Int Journ of Med Reaserch & Health Sci. 2016; 5(4): 89-96.
17. Koneman Elmer, Jr., Winn Washington, Allen Stephen, Janda Williams, Procop Gary, Schreckenberger Paul, Woods Gail. Koneman's Color Atlas and Textbook of Diagnostic Microbiology. 6th Edition. Philadelphia: Lippincott, Williams & Wilkins; 2006:386-87
18. Tille PM. Bailey and Scott's Diagnostic Microbiology, 13th edition. St. Louis, Missouri: Elsevier; 2014:193-231.
19. Clinical Laboratory Standards Institutes (CLSI). Performance standards for antimicrobial susceptibility

testing. 26th ed. CLSI supplement M100S. Wayne, PA: Clinical and Laboratory Standards Institute. 2022 Jan;36(1):40-256
20. Nandi D, Arjuna A. Acinetobacter main cause of hospital acquired infections: A review. Asian J Pharm Clin Res 2017; 10:53-6.
21. Dash M, Padhi S, Patnaik S, Mohanty I, Misra P. Frequency, risk factors and antibiogram of Acinetobacter species isolated from various clinical samples in a tertiary care hospital in Odisha, India. Avicenna J Med. 2013;3(4):97-102.
22. Gupta N, Gandham N, Jadhav S, Mishra RN. Isolation and identification of Acinetobacter species with special reference to antibiotic resistance. J Nat Sc Biol Med. 2015;6:159-62.
23. Islahi S et al. Prevalence and resistance pattern of Acinetobacter species in hospitalized patients in a tertiary care centre. Journal of Evolution of Medical and Dental Sciences 2014; Vol. 3, Issue 17, April 28; Page: 4629-4635.
24. Mindolli P B et al. Identification and Speciation Of Acinetobacter And Their Antimicrobial Susceptibility Testing Al Ameen J Med Sci (2010)3(4):345-349
25. Jaggi N, Sissodia P. Sharma L. Acinetobacter baumannii isolates in a tertiary care hospital: Antimicrobial resistance and clinical significance. Journal of Microbiology and Infectious Diseases/2012; 2 (2): pg 57-83
26. Apoorva T, Atul R, Rukadikar et al. Clinical and Antimicrobial Profile of Acinetobacter spp at a tertiary care hospital in central India. J of Med and Dent. Sci/eISSN- 2278-4802/pISSN-2278-4748; 2014; 3(29): 8102-8.
27. Prashanth K. Badrinath S. Nosocomial infections due to Acinetobacter species: clinical findings, risk and prognostic factors, Indian Journal of Medical Microbiology, (2006) 24 (1):39-44
28. Gomathy Mahajan, Sheevani Sheemar, Shashi Chopra et al.Carbapenem Resistance and Phenotypic Detection of Carbapenamases in Clinical isolates of A.baumannii.Indian J of Med sciences 2011;65(1):18-25.
29. Tripathi P C., Gajbhiye S R., Agrawal G N. Clinical and antimicrobial profile of Acinetobacter spp: An emerging nosocomial superbug. Adv Biomed Res 2014,3:13: pg 1-6
30. Berges L, Rodriguez-Villalobos H, Deplano A, Struelens MJ. Prospective evaluation of imipenem/EDTA combined disc and E test for detection of metallo-ß-lactamase-producing Pseudomonas aeruginosa. J Antimicrob Chemother 2007; 59:812-13
31. Sinha N, Agarwal J, Srivastava S, Singh M. Analysis of carbapenem- resistant Acinetobacter from a tertiary care setting in North India. Indian Journal of Medical Microbiology. (2013) 31(1): 60-63
32. Singla P, Sikka R, Deep A et al. Pattern of antimicrobial resistance in clinical isolates of Acinetobacter species at a tertiary level health care facility in Northern India. J Evol Med Dental Scien. 2013; 2: 159-65.
33. Kaur A, Gupta V. Chhina D. Prevalence of metallo- ß-lactamase- producing (MBL) Acinetobacter species in a tertiary care hospital. IRAN J. MICROBIOL. Vol. 6, No. 1 (February 2014), 22-25.
34. Joshi SG, Litake GM, Satpute MG, Telang NV, Ghole VS, Niphadkar KB. Clinical and demographic features of infection caused by Acinetobacter species. Ind J Med Sci. 2006;60:351-60.
35. Chakraborty B, Banerjee D. Acinetobacter baumannii: no more a choosy intruder? Indian J Med Sci. 2011 Aug: 65(8):344-8