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Original Research Article

Prevalence and Antibiotic Susceptibility Pattern of Multidrug-Resistant *Pseudomonas aeruginosa* in a Tertiary Hospital, South Sumatera, Indonesia

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Abstract

Background: *Pseudomonas aeruginosa* is a Gram-negative bacterium that causes opportunistic and healthcare-acquired infections. Its ability to rapidly acquire antibiotic resistance complicates treatment, prolongs hospitalization, and increases morbidity, mortality, and healthcare costs.

Objective: This study aimed to determine the prevalence and antibiotic susceptibility pattern of multidrug-resistant *P. aeruginosa* at Dr. Mohammad Hoesin General Hospital, South Sumatera, Indonesia.

Methods: A descriptive observational study with a cross-sectional design was conducted using data from the Clinical Microbiology Laboratory and Medical Records Installation of Dr. Mohammad Hoesin General Hospital, Palembang, from May 2022 to April 2023. Data were analyzed univariately and presented through tables, figures, and descriptive summaries.

Results: A total of 465 hospitalized patients were found to be infected with *P. aeruginosa*. Adult patients aged 18–65 years were the most affected (71%). The leading primary diagnoses were diabetic foot ulcer, pneumonia, and sepsis. Most patients were hospitalized for 8–14 days (25.2%). Antibiotic susceptibility testing showed the highest sensitivity to amikacin (94.2%), while the lowest was observed with meropenem and ceftazidime. The resistance profile included 24.7% MDR, 6.7% XDR, 0.9% probable PDR, and 2.4% DTR isolates.

Conclusion: *P. aeruginosa* infections were predominantly found in adult patients with prolonged hospitalization. Amikacin remains the most effective agent against *P. aeruginosa*, while meropenem and ceftazidime showed poor efficacy. The presence of MDR, XDR, PDR, and DTR strains highlights the urgent need for rational antibiotic use and strengthened antimicrobial stewardship programs.

Keywords: *Pseudomonas aeruginosa; Hospitalized patients; Antibiotic resistance; Multidrug resistance.*
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INTRODUCTION

Pseudomonas aeruginosa is a Gram-negative bacterium that causes opportunistic infections, particularly in immunocompromised individuals.[1,2] It is the most clinically significant *Pseudomonas* species, widely distributed in nature and frequently found in moist hospital environments.[3,4] Its adaptability enables colonization of multiple anatomical sites, while pathogenicity is enhanced by extended-spectrum β-

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lactamase (ESBL) production, acquisition of resistance mechanisms, secretion of virulence factors, and biofilm formation, which together protect the organism from antimicrobial exposure and host immune responses, leading to persistent and difficult-to-treat infections.[5–7]

A major challenge in managing *P. aeruginosa* infections is its intrinsic resistance to multiple antibiotic classes, which occurs independently of prior antimicrobial exposure.[8] This resistance is mainly attributed to low outer membrane permeability, multidrug efflux pump systems, and chromosomally encoded β-lactamases such as AmpC, all of which reduce antibiotic penetration and activity.[8,9] Consequently, *P. aeruginosa* is naturally non-susceptible to several antimicrobial agents, including non-antipseudomonal penicillins, first- and second-generation cephalosporins, macrolides, tetracyclines (including tigecycline), glycopeptides, lincosamides, trimethoprim–sulfamethoxazole, and chloramphenicol, substantially limiting therapeutic options and contributing to its classification as a difficult-to-treat pathogen.[9–11]

P. aeruginosa is a major cause of healthcare-associated infections (HAIs), accounting for approximately 10%–15% of cases overall and up to 20% in intensive care units (ICUs).[12] These infections are associated with increased morbidity, mortality, and healthcare costs; the Centers for Disease Control and Prevention (CDC) report approximately 32,600 multidrug-resistant (MDR) cases and 2,700 related deaths annually in the United States.[13] At Dr. Mohammad Hoesin General Hospital, Palembang, 34.6% of isolates were MDR, largely linked to inappropriate antibiotic use.[14,15] Compared with other pathogens, *P. aeruginosa* infections are associated with poorer outcomes, including higher inflammation, mortality, and reduced quality of life.[16] In Indonesia, carbapenem resistance among *P. aeruginosa* isolates reached 51.2% in 2021, predominantly among hospitalized patients, further complicating clinical management.[17]

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Given these challenges, continuous local surveillance of antimicrobial susceptibility patterns is essential. This study aims to evaluate the antibiotic susceptibility profile of *P. aeruginosa* isolates to support rational antibiotic use, strengthen infection prevention strategies, and improve clinical outcomes among inpatients at Dr. Mohammad Hoesin General Hospital, Palembang.

Pathology between May 2022 and April 2023. All specimens were collected following hospital standard operating procedures to ensure specimen quality and clinical relevance. Analysis was performed on laboratory isolates; however, to minimize duplication bias, only the first isolate per patient was included for resistance pattern analysis. Although the data were obtained from routine laboratory services, this approach is widely accepted for antimicrobial resistance surveillance and forms the basis of national and global programs such as WHO GLASS. This study received ethical approval (Protocol No.: 193-2023).

The primary variable assessed was the antibiotic susceptibility pattern of *P. aeruginosa.* Susceptibility testing was performed using the microbroth dilution with automatic Vitek 2, according to CLSI M100, 32nd edition.[18] The antibiotics tested covered multiple pharmacological groups, including aminoglycosides, cephalosporins, carbapenems, and fluoroquinolones. Results were interpreted as sensitive, intermediate, or resistant. Quality control was performed using *P. aeruginosa* ATCC 27853 according to CLSI recommendations. Data were analyzed descriptively, with sensitivity profiles presented as frequencies and

Table 1. Demographic profile

Variable	n (%)	Mean ± SD	Median (Min-Max)
Age (Year)		43.73 ± 21.52	48.34 (3[a] – 83)
Children (0–17)	63 (13.5)		
Adults (18–65)	330 (71)		
Elderly (>65)	72 (15.5)		
Duration of Hospitalization (Days)		20.54 ± 17.37	15 (1 – 107)
1–7	110 (23.7)		
8–14	117 (25.2)		
15–21	71 (15.3)		
22–28	54 (11.6)		
29–35	42 (9)		
>35	71 (15.3)		
Total	465 (100%)		

Note:
[a] Age in days

Table 2. Primary diagnosis

Variable	Frequency (n)	Percentage (%)
Primary Infection	**119**	**25.7**
Integumentary system	**51**	**11**
Diabetic foot ulcer (DFU)	28	
Surgical wound infection	9	
Cellulitis	3	
Decubitus ulcer	3	
Abscess	2	
Open wound infection	2	
Exfoliative dermatitis	1	
Exposure keratitis	1	
Necrotizing fasciitis	1	
Skin necrosis	1	
Nervous system and sensory organs	**25**	**5.4**
Chronic suppurative otitis media (CSOM)	8	
Hydrocephalus	5	
Encephalitis	2	
Meningoencephalitis	2	
Transverse myelitis	2	
Chronic wasting disease (CWD)	1	
Endophthalmitis	1	
Meningitis	1	
Auricular perichondritis	1	
Tetanus	1	
Corneal ulcer	1	
Respiratory system	**21**	**4.5**
Pneumonia	11	
Empyema	4	
Acute respiratory distress syndrome (ARDS)	3	
Bronchiectasis	1	
Respiratory failure	1	
Rhinosinusitis	1	
Digestive system	**5**	**1.1**
Dyspepsia	2	
Gastroenteritis	2	
Stomatitis	1	
Musculoskeletal system	**4**	**0.9**
Osteomyelitis	3	
Arthritis	1	
Genitourinary system	**2**	**0.4**
Urinary tract infection (UTI)	2	
Systemic infection	**11**	**2.4**
Sepsis	11	
Secondary Infection	**44**	**9.5**
Non-Infectious	**302**	**64.9**
Total	465	100

percentages. Findings were summarized in tables and figures to illustrate resistance trends and provide evidence-based support for antibiotic selection and infection control strategies.

RESULTS

A total of 465 clinical specimens from hospitalized patients with confirmed *Pseudomonas aeruginosa* infections were analyzed between May 2022 and April 2023. All collected samples were tested for antibiotic susceptibility testing (AST).

Demographic Profile of *P. aeruginosa*-Infected Patients

The demographic profile of hospitalized patients diagnosed with *P. aeruginosa* infection is summarized in Table 1.

Most *Pseudomonas aeruginosa* infections occurred in adults aged 18–65 years (71%), with fewer cases in elderly (15.5%) and pediatric (13.5%) patients. The mean patient age was 43.7 years (range: 3 days–83 years). The highest incidence was seen in patients hospitalized for 8–14 days (25.2%), with an average hospital stay of 20.5 days (range: 1–107 days).

Primary Diagnosis of *P. aeruginosa*-Infected Patients

The distribution of primary diagnoses among hospitalized patients with *P. aeruginosa* infection is summarized in Table 2.

Among inpatients with *Pseudomonas aeruginosa* infection, the majority had a primary (admitting) diagnosis of non-infectious diseases (64.9%), followed by infectious diseases (25.7%), while *P. aeruginosa* was identified as a secondary infection in 9.5% of cases. In patients with admitting diagnosis of non-infectious diseases, culture were primarily indicated in patients with prolonged hospitalization, invasive procedures, indwelling medical devices, or clinical deterioration suggestive of healthcare-associated infection. By system, the integumentary system was most affected (11%), with diabetic foot ulcer as the leading diagnosis, followed by the nervous/sensory organs (5.4%, mainly chronic suppurative otitis media), the respiratory system (4.5%, mainly pneumonia), the hematological system (2.4%, mainly sepsis), and smaller proportions in the digestive, musculoskeletal, and genitourinary systems.

Antibiotic Susceptibility Pattern of *P. aeruginosa*

The findings on the antibiotic resistance and sensitivity patterns of *P. aeruginosa* are summarized in Table 3.

Pseudomonas aeruginosa isolates showed the highest sensitivity to amikacin (94.2%), followed by gentamicin (86.5%) and cefepime (81.7%). Moderate resistance was observed against meropenem (18.9%) and ceftazidime (18.1%).

Resistance Patterns of *P. aeruginosa*

To standardize resistance terminology, international consensus defined multidrug-resistant (MDR), extensively drug-resistant (XDR), and pandrug-resistant (PDR) organisms in 2013, with the later addition of difficult-to-treat resistance (DTR). MDR refers to non-susceptibility to at least one agent in three or more antimicrobial classes; XDR indicates susceptibility to only one or two classes; and PDR denotes resistance to all available agents.[19] In 2018, the Infectious Diseases Society of America (IDSA) defined DTR *P. aeruginosa* as strains resistant to key antipseudomonal agents, including ceftazidime, cefepime, piperacillin–tazobactam, carbapenems, fluoroquinolones, and aztreonam.[20]

The distribution of *P. aeruginosa* resistance profiles—classified as PDR, XDR, MDR, and Non-MDR—is illustrated in Figure 1.

Among *P. aeruginosa* isolates, resistance profiles showed 0.9% probable PDR, 6.7% XDR, 24.7% MDR, and 67.7% non-MDR. After accounting for overlap, the overall prevalence was 32.3% MDR, 7.6% XDR, and

Table 3. Antibiotic susceptibility pattern of *P. aeruginosa*

No.	Antibiotics	Sensitive		Intermediate		Resistance	
		n	%	n	%	n	%
1.	Amikacin	438	94.2	6	1.3	21	4.5
2.	Gentamicin	402	86.5	15	3.2	48	10.3
3.	Cefepime	380	81.7	43	9.2	42	9
4.	Ciprofloxacin	379	81.5	15	3.2	71	15.3
5.	Piperacillin-Tazobactam	365	78.5	40	8.6	60	12.9
6.	Meropenem	356	76.6	21	4.5	88	18.9
7.	Ceftazidime	349	75.1	32	6.9	84	18.1
8.	Aztreonam	289	62.2	99	21.3	77	16.6

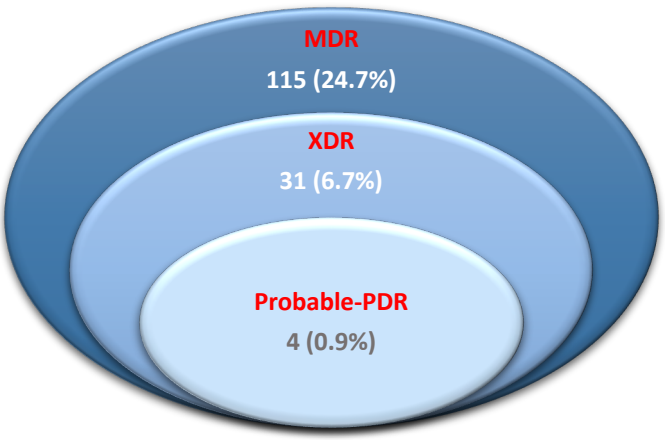

Figure 1. Profile of PDR, XDR, MDR, and non-MDR of *P. aeruginosa*

0.9% PDR. Additional classification into DTR and non-DTR groups is shown in Figure 2.

Based on qualitative analysis, 11 patients (2.4%) were classified as having DTR, while the remaining 454 patients (97.6%) were categorized as non-DTR.

DISCUSSION

Demographic Profile of *P. aeruginosa*-Infected Patients

Most *Pseudomonas aeruginosa* infections occurred in adults (71%), followed by elderly patients (15.5%), while children accounted for the lowest proportion (13.5%). These findings are consistent with Primasari *et al.* (2020), who reported that 78.67% of cases were in adults aged 18–65 years.[21] The higher burden among adults may be linked to their greater involvement in productive activities and frequent environmental exposure, which increases the likelihood of contact with *P. aeruginosa*.

However, Dharmayanti and Sukrama (2019) at Sanglah General Hospital, Denpasar, found the highest infection rate in children (0–5 years) at 33%.[22] Similarly, Fabian *et al.* (2020) at Dr. Soetomo Regional Hospital, Surabaya, observed toddlers as the most affected group (38%). This vulnerability may be attributed to their immature adaptive immune system, given their limited exposure to pathogens. Such immaturity is marked by inefficient phagocyte migration to infection sites, low cytokine production, and reduced levels of serum IgM, IgA, and IgE.[23]

In the present study, the mean age of patients with *P. aeruginosa* infection was 43.73 years (range: 3 days–83 years). This is higher than the average age reported by Hosu *et al.* (2021), which was 32.8 years (range: 6 days–84 years).[24] Conversely, Zhang *et al.* (2020) found a higher mean age of 60.5 years in patients with *P. aeruginosa* (range: 10 months–95 years).[25] At Felegehiwot Referral Hospital, the mean age was 29 years,[26] while Gill *et al.* (2016) reported 36.2 years.[27]

The elderly were the second most affected group in this study, likely due to immunosenescence—age-related decline in immune function—that increases susceptibility to infection. With aging, both innate and adaptive immunity deteriorate. Innate immune impairments include reduced phagocytic function of neutrophils and NK cells, as well as decreased macrophage activity and antigen presentation. Adaptive immunity is weakened by thymic involution, which lowers CD4 and CD8 lymphocyte production, thereby reducing the effectiveness of immune responses.[28,29] Because this study population consisted of hospitalized patients, many with compromised immunity and comorbidities, variations in age distribution across studies may reflect differences in patients' immune status and disease severity.[30]

The highest proportion of *Pseudomonas aeruginosa* infections was found among patients hospitalized for 8–14 days (25.2%), followed by those staying 1–7 days (23.7%), 15–21 days (15.3%), >35 days (15.3%), 22–28 days (11.6%), and 29–35 days (9%). Similar results were reported by Kristiningrum *et al.* (2023), who noted that 62% of multidrug-resistant organism (MDRO) cases occurred in patients hospitalized for 1–15 days, while 38% were treated for more than 16 days.[31] In contrast, Nair *et al.* (2017) found that healthcare-associated infections (HAIs) were most common in patients with hospital stays exceeding 30 days (35.21%).[32]

In this study, the average length of stay for patients infected with *P. aeruginosa* was 20.54 ± 17.37 days. This is comparable to Deni and Pangalila (2019) at Royal Taruma Hospital, Jakarta, who reported a mean hospitalization of 29.77 ± 18.23 days for patients infected with *P. aeruginosa* and *A. baumannii* in the ICU.[33]

It is widely recognized that the risk of HAIs increases with longer hospital stays, underscoring the need to minimize treatment duration. Prolonged hospitalization increases patient exposure to healthcare workers, invasive devices, and contaminated hospital environments, all of which may harbor *P. aeruginosa* biofilms.[34] Importantly, extended stays are not solely the result of *P. aeruginosa* infections, but also depend on a patient's immune status and the severity of their underlying illness. Patients with compromised immunity or more severe conditions generally require longer treatment courses.[35]

Primary Diagnosis of *P. aeruginosa*-Infected Patients

At Dr. Mohammad Hoesin General Hospital, Palembang, between May 2022 and April 2023, the most frequent primary diagnoses among inpatients with *Pseudomonas aeruginosa* infection were diabetic foot ulcers (19 cases), pneumonia (11 cases), and sepsis (11 cases), with integumentary system involvement reaching 11%. These results align with Estiningsih *et al.* (2016) at Dr. Soeradji Tirtonegoro General Hospital, Klaten, who found that most infants infected with *P. aeruginosa* developed sepsis (54.3%) and pneumonia (17.4%).[36] Similarly, Wijaksana *et al.* (2019) at Arifin Achmad Regional Hospital, Pekanbaru, identified *P. aeruginosa* as the third most frequent cause of sepsis (16.22%),[37] while Aziz *et al.* (2020) at Hasan Sadikin General Hospital, Bandung, reported it as the second most common Gram-negative pathogen causing pneumonia.[38]

As an opportunistic bacterium, *P. aeruginosa* is capable of infecting multiple organ systems—including the integumentary, urinary, sensory, and cardiorespiratory systems—and is responsible for a range of infections such as pneumonia, meningitis, urinary tract infections, peritonitis, osteomyelitis, bloodstream infections, and surgical site infections.[39,40] Notably, around 90% of skin and soft tissue infections—including surgical wounds and diabetic foot ulcers—are linked to *P. aeruginosa* due to its strong biofilm-forming ability. These biofilms impair wound healing, increase resistance to host defenses and antibiotics, and worsen clinical outcomes.[10] The pathogen is also frequently isolated in diabetic patients with ulcers or gangrene, where poor blood circulation limits immune responses and delays tissue recovery.[41]

Thus, early detection plays a key role in preventing biofilm development and subsequent antibiotic resistance.

Another critical characteristic of *P. aeruginosa* is its ability to persist in nutrient-deficient environments, which enables contamination of medical devices, especially respiratory equipment. Inhalation of aerosols from contaminated devices allows bacterial colonization of the respiratory tract, leading to infections such as pneumonia—particularly in ICU patients. These infections often trigger uncontrolled immune responses, progressing to sepsis.[42,43] *P. aeruginosa* is also the most frequently isolated bacterium from ventilator tubing, making it a major cause of ventilator-associated pneumonia (VAP). Saputra *et al.* (2019) at Sanglah General Hospital, Denpasar, found that most multidrug-resistant *P. aeruginosa* cases were diagnosed as VAP,[44] while Reynolds and Kollef (2021) reported even higher rates of 32–42.8%.[45] The pathogenesis of VAP begins with bacterial adherence to the mucosal barrier via flagella and type IV pili, triggering local inflammation. Once attached, *P. aeruginosa* deploys its type III secretion system (T3SS) to damage host tissues, block phagocytosis, and disrupt endothelial barriers, allowing rapid dissemination within the host.[46]

Antibiotic Susceptibility Pattern of *P. aeruginosa*

Susceptibility testing was conducted on 8 antibiotics—amikacin, gentamicin, cefepime, ciprofloxacin, piperacillin–tazobactam, meropenem, ceftazidime, and aztreonam. The results demonstrated that *Pseudomonas aeruginosa* exhibited the greatest sensitivity to amikacin (94.2%) and gentamicin (86.5%). These findings are in line with Sutrisno (2020), who also identified amikacin as the most effective drug against *P. aeruginosa.*[14] Comparable trends were seen in Anggraini *et al.* (2018) at Arifin Achmad Regional Hospital, Pekanbaru, with sensitivity rates of 76.9% for amikacin and 54.5% for gentamicin,[47] as well as in Perez *et al.* (2019), who reported rates of 62.3% and 51%, respectively.[48]

P. aeruginosa in this study demonstrated its highest susceptibility to aminoglycosides, especially amikacin and gentamicin. The retained susceptibility of XDR *Pseudomonas aeruginosa* to aminoglycosides reflects the unique ribosomal target of this antibiotic class—the bacterial 30S subunit—which is structurally and functionally distinct from the targets of β-lactams and fluoroquinolones. Aminoglycosides inhibit protein synthesis by binding the 30S ribosome, whereas resistance to β-lactams and fluoroquinolones predominantly arises through β-lactamase production, penicillin-binding protein modifications, and mutations in DNA gyrase/topoisomerase IV that do not directly affect ribosomal binding. Because these dominant resistance mechanisms do not overlap with aminoglycoside action, many XDR isolates may still respond to aminoglycosides in the absence of specific resistance determinants such as aminoglycoside-modifying enzymes or 16S rRNA methyltransferases, the latter of which directly alter the ribosomal binding site and confer high-level aminoglycoside resistance.[49,50] Although aminoglycosides demonstrated high susceptibility, their clinical use should be interpreted cautiously, as pharmacodynamic constraints and limited tissue penetration restrict their effectiveness as monotherapy outside urinary tract infections.

Ciprofloxacin also showed good sensitivity (81.5%), though this was lower than the 100% susceptibility reported by Dharmayanti and Sukrama (2019) at Sanglah General Hospital, Denpasar.[22] Conversely, Anggraini *et al.* (2018) noted lower sensitivity at 48.8%.[47] As a fluoroquinolone, ciprofloxacin works by inhibiting DNA replication through the blockage of DNA gyrase (topoisomerase II) and topoisomerase IV, resulting in double-stranded DNA breaks and growth arrest. Resistance commonly develops through mutations in *gyrA* and *parC*, or by efflux pump overexpression, reducing intracellular drug levels. Importantly, ciprofloxacin can penetrate biofilms during their early stages, improving treatment outcomes in biofilm-related infections. Nevertheless, its extensive clinical use has driven increasing resistance and cross-resistance to other antibiotic classes, including aminoglycosides and β-lactams, often through shared mechanisms like efflux pumps and co-selection pressures.[51,52]

In contrast to intrinsic resistance observed for certain agents, this study demonstrated resistance to key antipseudomonal β-lactams, with meropenem and ceftazidime resistance rates of 18.9% and 18.1%, respectively, among 465 *Pseudomonas aeruginosa* isolates. Similar trends have been reported globally, with surveillance data indicating declining susceptibility to both agents. The INFORM program from United States medical centers reported resistance rates of 11.5% for meropenem and 17.5% for ceftazidime, while a global meta-analysis identified a pooled meropenem resistance prevalence of 34.7%, highlighting the growing threat of carbapenem non-susceptibility.[53,54] Substantial regional variation has also been documented, with studies from Egypt reporting meropenem and ceftazidime resistance rates of 53.1% and 85.7%, respectively.[55]

National data from Indonesia further support moderate β-lactam resistance among *P. aeruginosa* isolates. A systematic review reported ceftazidime resistance of approximately 35% and carbapenem resistance, including meropenem, of 36%.[56] Cross-sectional surveillance from Sanglah General Hospital, Denpasar, identified meropenem resistance among intensive care unit isolates, with several multidrug-resistant (MDR) subsets exhibiting markedly reduced susceptibility to ceftazidime.[57] Similarly, an observational study conducted in Jakarta reported lower susceptibility rates to ceftazidime (69.5%) and meropenem (76.25%) among *P. aeruginosa* isolates recovered from pneumonia patients, suggesting a trend toward increasing resistance.[58] These findings are consistent with reports from Arifin Achmad Regional Hospital, Pekanbaru, which demonstrated decreased susceptibility to both agents in MDR *P. aeruginosa*.[47]

Comparable resistance patterns have been observed internationally. Surveillance studies from Spain reported meropenem resistance rates of up to 37.6% and ceftazidime resistance of approximately

33.4% among intensive care unit isolates, while genome-based surveillance from Pakistan showed that nearly 65% of isolates were resistant to at least one carbapenem.[59,60] Multinational surveillance studies further confirm persistent non-susceptibility to both ceftazidime and meropenem across regions, reflecting geographic variability in resistance mechanisms.[53,61]

Resistance Patterns of *P. aeruginosa*

The resistance profile of hospitalized patients with *P. aeruginosa* infections in this study showed 0.9% classified as probable PDR, 2.4% as DTR, 6.7% as XDR, and 24.7% as MDR. These proportions are comparable with Santoro *et al.* (2020), who reported 27.84% MDR, 2.09% XDR, and 0.19% PDR in Italy.[62] Gill *et al.* (2016) similarly noted 50% MDR, 2.3% XDR, and no PDR isolates.[27] Resistance rates between 2017 and 2022 have declined, with MDR decreasing from 27.2% to 14.8%, XDR from 15.2% to 5.9%, and DTR from 4.2% to 2.1%.[63] The classification of probable PDR in this study reflects the limited number of antibiotic classes tested at Dr. Mohammad Hoesin General Hospital, Palembang.

Specifically, when excluding overlap with XDR and PDR, 32.3% of patients were infected with MDR strains. This proportion is slightly lower compared to Sutrisno (2020), who reported 34.6%,[14] and Saputra *et al.* (2021) with 36.6%.[44] Other studies reported comparable rates, such as Spagnolo *et al.* (2021) in Spain (26.2%),[42] and Hosu *et al.* (2021) in Africa (36.8%).[24] These findings also align with global INFORM surveillance, which documented MDR prevalence between 11.5–24.7% and XDR between 9–11.2%.[64] MDR *P. aeruginosa* is usually hospital-acquired, either from environmental persistence or contaminated medical devices, with resistance driven by mutations and horizontal gene transfer. Risk factors include recent antibiotic exposure and inappropriate antibiotic use, both of which encourage adaptive resistance.[65,66]

The World Health Organization (WHO) recognized *P. aeruginosa* as a critical priority pathogen in 2017, underscoring the urgent need for novel antibiotics due to escalating resistance rates.[24] Similarly, the Infectious Diseases Society of America (IDSA) highlighted that this bacterium shows the highest rates of DTR compared with other pathogens, being resistant to many first-line agents such as ceftazidime, cefepime, piperacillin–tazobactam, imipenem–cilastatin, meropenem, ciprofloxacin, levofloxacin, and aztreonam.[67] Although imipenem–cilastatin and levofloxacin were not tested due to type of ast card that has been used, their resistance profiles were inferred from meropenem and ciprofloxacin results.[18]

Multiple molecular mechanisms collectively drive the emergence of DTR *P. aeruginosa*, including reduced OprD porin expression, AmpC hyperproduction, efflux pump activation, and mutations in penicillin-binding proteins.[20] These resistance determinants substantially compromise the effectiveness of available antimicrobials, resulting in a 40% higher mortality risk among patients infected with DTR strains, particularly because treatment options are severely limited and often rely on suboptimal alternatives.[68] Compounding this problem, *P. aeruginosa* possesses intrinsic resistance to aminoglycosides, fluoroquinolones, and β-lactams, a characteristic that contributes significantly to its high morbidity and mortality burden.[69] The increasing occurrence of MDR isolates in hospitalized populations further constrains therapeutic choices and complicates clinical management.[70] Taken together, these interrelated processes reinforce one another, enabling the pathogen to persist despite aggressive therapy and underscoring the critical need for enhanced antimicrobial stewardship, rapid diagnostic tools, and novel agents capable of targeting these multifactorial resistance pathways.

CONCLUSION

Pseudomonas aeruginosa infections at Dr. Mohammad Hoesin General Hospital, Palembang, were most prevalent among adult patients, particularly those with prolonged hospital stays. The respiratory tract was the most common source of isolates, while diabetic foot ulcer, pneumonia, and sepsis were the leading primary diagnoses. Antibiotic susceptibility testing demonstrated that aminoglycosides, especially amikacin, remain the most effective agents, whereas the lowest susceptibility rates were observed for meropenem and ceftazidime. The detection of MDR, XDR, PDR, and DTR strains underscores the growing challenge of antimicrobial resistance and its implications for patient outcomes. These findings emphasize the importance of rational antibiotic prescribing, early detection, strict infection control measures, and strengthened antimicrobial programs to limit resistance development and improve treatment success. Further molecular surveillance is recommended to better characterize resistance mechanisms and guide future antimicrobial strategies.

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