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Fifteen-Year Clinical Trends in Resistance of Inpatients *Pseudomonas aeruginosa*: Implications for Antimicrobial Therapy

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Abstract

Background: *P. aeruginosa* is a major nosocomial pathogen. This long-term surveillance study is to evaluate the frequency and antimicrobial susceptibility trends of *P. aeruginosa* over a 15-year period. **Methods:** We conducted a retrospective single center study of *P. aeruginosa* isolates using the VITEK 2 system between 2010 and 2024. Data was analyzed as frequency, ratio, and resistance trends. *P. aeruginosa* susceptibility was examined for the available antipseudomonal agents. Trends were assessed by Poisson Generalized Linear model, and visualized. Effect size was measured as rate ratio (RR), and P-value considered < 0.05 as significant. **Results:** A total of 1728 isolates were identified over the fifteen years, *P. aeruginosa* was 9.12% of gram-negative bacteria, ICU contributed to 12.9% of the isolates. There was a decreasing frequency trend (RR = 0.98, P < 0.000). Also, there was a reduction in resistance rates except levofloxacin (RR = 1.04, 95%C.I. 0.97 - 1.11, P = 0.30). Carbapenems were almost stationary (RR = 1.008, P ≥ 0.54). Carbapenems resistance *P. aeruginosa* rate was 40.7%, and the Extended Spectrum β-Lactamases producers (ESBL) was 6.7%. **Conclusion:** Resistant *P. aeruginosa* rates are high for carbapenems and fluoroquinolones with a concerning susceptibility and MIC distribution. Though, the current study demonstrated a marginal decreasing resistance trend for the other antimicrobials. The findings underscore the importance of continuous local surveillance to inform empirical therapy and reinforce antimicrobial stewardship strategies in resource-limited settings.

Keywords

P. aeruginosa, Resistance Trends, Antimicrobial Susceptibility, Antibacterials, Resistance Mechanisms

1. Introduction

Pseudomonas aeruginosa is a gram-negative ubiquitous microorganism that survives in all health care areas of hospitals, its successful persistence is attributed to its metabolic versatility and robustness in causing infections [1]. A correlation between environmental and clinical isolates including their antimicrobial susceptibility were demonstrated [2]. Typically, *P. aeruginosa* is associated with clinical infections in immunocompromised individuals, patients with structural lung disease, and those with cystic fibrosis, and it is linked to high mortality rates [3].

The increasing resistance of *P. aeruginosa* initially emerged started as tolerance to multiple antimicrobials classes and was subsequently driven by continued antimicrobial selection pressure, resulting in the evolution of resistant strains to a number of antimicrobials. This phenomenon is particularly notable in patients with chronic lung disease driven by antibacterials therapy exposure pressure [4]. In a nosocomial setting, the wide scale use of carbapenems has contributed to rising rates of carbapenem-resistant *P. aeruginosa* (CRPa), leading to inappropriate therapy, treatment failures, and excess mortality [5] [6].

Fortunately, in China Southeast single center, time-interrupted series analysis comparing two periods (2006-2007 and 2016-2017) demonstrated a decline in resistance patterns over time, associated with loss of *oprD*, the principle carbapenem-resistance in *P. aeruginosa*. This reduction in CRPa coincided with a national antimicrobials stewardship directive focusing on reducing the daily defined dose (DDD) in tertiary care hospitals [7].

The present study aims to evaluate *P. aeruginosa* antimicrobial susceptibility patterns, and resistance trends over a 15-year period (2010-2024).

2. Materials and Methods

2.1. Pseudomonas Identification (General Outlines)

Clinical specimens were processed in the Specialty Hospital (Amman, Jordan) clinical microbiology laboratory in accordance with the standard operating procedures for isolating the gram-negative bacteria. Isolates were recovered on routine media and preliminarily identified by Gram stain and biochemical/phenotypic screening. Species identification and antimicrobial susceptibility testing (AST) were performed using the VITEK® 2 automated system (bioMérieux) following the manufacturer's instructions for preparation of the colonies and antibiotics cards for *P. aeruginosa*. MICs were interpreted using the clinical laboratory standard institute (CLSI M100 2024 breakpoints). Quality control was performed daily with appropriate reference strains, and any discrepant or unusual results were confirmed by repeating the test procedure or manual identification with AST performed by Kurby-Baur diffusion disc or E-test.

2.2. Data Collection

At large, the laboratory receives and processes specimens from both the inpatients

and outpatients, covering different sources. Here, the inpatients isolates were solely studied, focusing on *P. aeruginosa*. All specimens are processed for identification and antimicrobial susceptibility testing by VITEK® 2 automated system (*bioMérieux*, *Marcy-L'Etoile*, *France*). The system maintains a multi-year database with a backup repository. Data was exported as text files and formatted into Microsoft Excel sheet (*Microsoft Corporation*). The total number of isolates was 33418.

Patients related metadata collected included hospital location, specimen type, specimen source, specimen date, collection date, testing date, organism name, its bio-number, probability and confidence level. Bacteria isolate-related variables included AST, minimum inhibitory concentration, resistance enzyme profiles, and additional Advanced Expert System (AES) interpretations of resistance mechanisms across antimicrobials classes.

2.3. Ethics Statement

The specialty Hospital Institutional Review board (IRB), reviewed the study procedure based on a detailed letter from the principal investigator. The study did not involve direct human subjects' participation but used data retrieved from the archived VITEK 2 database. The IRB approval was granted on 5 October 25, approval number IRB 5\1\t\123322.

2.4. Statistical Analysis

Data Excel sheet was converted as Comma Separated Variables (CVS) file and imported into R (R Foundation for Statistical Computing) for analysis, using the RStudio/Posit Integrated Development Environment (IDE), and RStudio (IDE)/Posit Integrated Development Environment [8] [9]. Data was cleaned and systematized; specimen source information was used to complete missing fields for specimen type within the same case, and specimen type was used for analysis.

A subset data frame containing all *P. aeruginosa* isolates was generated for descriptive analysis, frequency distributions, tabulation, and graphical visualization of resistance patterns, yearly trends, and isolated distribution rates. A generalized linear model (GLM) with a Poisson distribution was applied to assess resistance trends over the 15-year period. Exponentiation GLM coefficients were used to calculated effect sizes, rate ratios (RRs), 95% Confidence Interval, and P-value. A P-value < 0.05 was considered statistically significant.

3. Results

There was year-to-year variability in the frequency of *Ps. aeruginosa* isolates. In 2010 and 2011 the number of isolates was 90 and 94, respectively, followed by a progressive increase to 111 in 212 and 150 in 2015, and then a subsequent decline to 119 in 2024 (**Table 1**). There were 1728 inpatient *P. aeruginosa* isolates, of which 223 (12.9) originated from the intensive care unit (ICU) and 1505 (87.1%) from general wards. The distribution of specimen sources (**Table 2**) showed that

most isolates were recovered from unspecified abscesses and body fluids (n = 647, 38.7%), followed by urine (n = 528, 31.7%), bronchial/upper respiratory specimens (n = 158, 9.5%), lungs/pleural samples (BAL/pleura; n = 80, 4.8%), ears (n = 72, 4.3%), skin and soft tissue (n = 65, 3.9%), blood (n = 91, 5.4%), bone (n = 11, 0.7%), abdomen (n = 8, 0.5%), vaginal specimens (n = 5, 0.3%), central venous catheter (n = 2, 0.1%), and cerebrospinal fluid (n = 3, 0.2%).

The overall proportion of *P. aeruginosa* was 9.12% of all GNB, and 5.17% of all isolates, showing a significant decreasing yearly trend (Slope = −0.02, RR (rate ratio) = 0.98, P < 0.000). The GNB trend showed minimal change (Slope = 0.007, RR = 1.007, P = 0.000). The proportion of *P. aeruginosa* among GNB demonstrated a downward trend over time (Slope = −0.04, RR = 0.96, P = 0.04), while its proportion among all isolates exhibited a slight upward trend (Slope = 0.04, RR = 1.04, P < 0.000). An additional decrease trend was seen in the ratio of *P. aeruginosa* among all isolates (Slope = −0.08, RR = 0.962, P = 0.002). Temporal trends for *P. aeruginosa* among GNB are visualized in Figures 1(a)-(c).

Table 1. Frequency and rates of inpatient isolates of *P. aeruginosa* with trends (for ratios out of gram-negative bacteria (GNB) and out of total isolates) for the years (2010-2024).

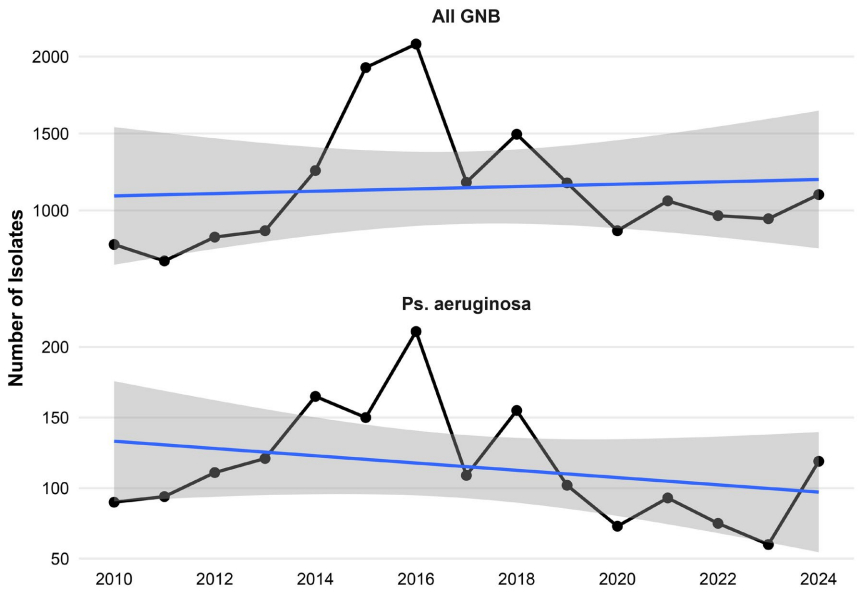
	***P. aeruginosa* Yearly Counts (N) and Ratios (%)**				
Year	**All Isolates[1] (N)**	**GNB[2] (N)**	***P. aeruginosa* (N)[3]**	***P. aeruginosa*/GNB[4] (%)**	***P. aeruginosa*/All[5] (%)**
2010	1097	779	90	11.6	8.2
2011	1037	672	94	14.0	9.1
2012	1218	827	111	13.4	9.1
2013	1214	868	121	13.9	9.8
2014	1637	1259	165	13.1	10.1
2015	2992	1928	150	7.8	5.0
2016	3710	2081	211	10.1	5.7
2017	2559	1183	109	9.2	4.3
2018	3406	1495	155	10.4	4.5
2019	3287	1179	102	8.7	3.1
2020	2870	868	73	8.4	2.5
2021	3183	1062	93	8.8	2.9
2022	1620	966	75	7.8	4.6
2023	1539	946	60	6.3	3.9
2024	2005	1103	119	10.8	5.9

P. aeruginosa Total Number in Fifteen Years = 1728; Total Ratio from 17,216 GNB Isolates = 9.12%; Total Ratio from All 33,418 Isolates = 5.17%. [1]Trends in for all isolates counts over years showed minimal increase (Slope = 0.037, RR = 1.04, P < 0.000); [2]Trends in GNB was horizontal, no increase or decrease (Slope = 0.007, RR = 1.007, P = 0.000); [3]Trends for *P. aeruginosa* counts over years, it showed decline (Slope = −0.0224, RR = 0.98, P < 0.000); [4]Trends in the yearly ratio of *P. aeruginosa* to yearly GNB (Slope = −0.039, RR = 1.04, P = 0.037); [5]Trends in the yearly ratio of *P. aeruginosa* to yearly all isolates (Slope = −0.077, RR = 0.962, P = 0.002).

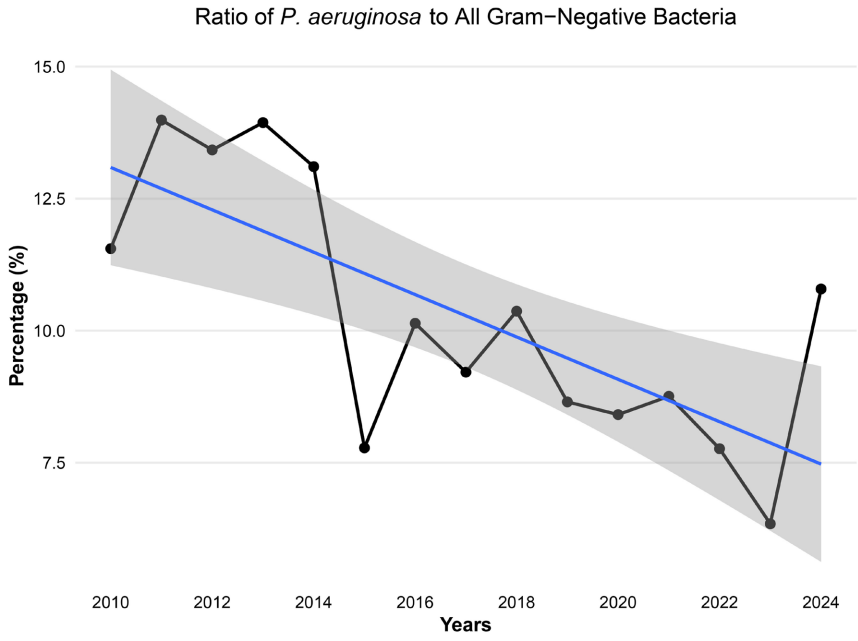
Table 2. Location and source of the inpatients' isolate of *P. aeruginosa*.

Pseudomonas aeruginosa	N[1]	(%)	Comments
Total Count	1728	(100)	Inpatients
Patient Location			
Floors	1505	(87.1)	Isolates including Pathogens
Intensive care unit	223	(12.9)	
Specimen Source			
Urine	472	(28.3)	Voided urine
Respiratory	158	(9.5)	Sputum and tracheal
Blood[2]	91	(5.4)	Aerobic/Anaerobic Bottles
Ear	72	(4.3)	Swabs
Skin and Soft Tissue	65	(3.9)	Including pressure ulcers
Lung and Pleura[2]	80	(4.8)	Sterile sites
Urine-Catheter	56	(3.4)	Foley's Catheter
Bone[2]	11	(0.7)	Sterile sites (biopsies/swabs)
Abdomen[2]	8	(0.5)	Sterile sites (Peritoneum)
Vaginal	5	(0.3)	High Vaginal Swabs
Cerebrospinal fluid[2]	3	(0.2)	Sterile sites
Central Venous Catheter[2]	2	(0.1)	Sterile sites
Abscess and Fluid	647	(38.7)	Unspecified

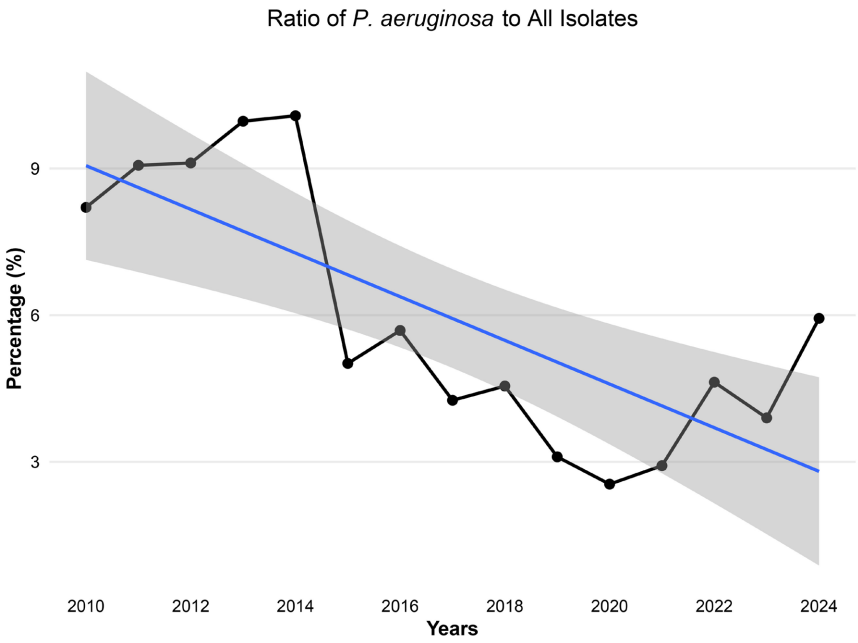
[1]N (%): Number and percent of isolates. [2]Supposedly sterile sites n = 195, isolated from pleural spaces and broncho-alveolar lavage (BAL).



(a)



(b)



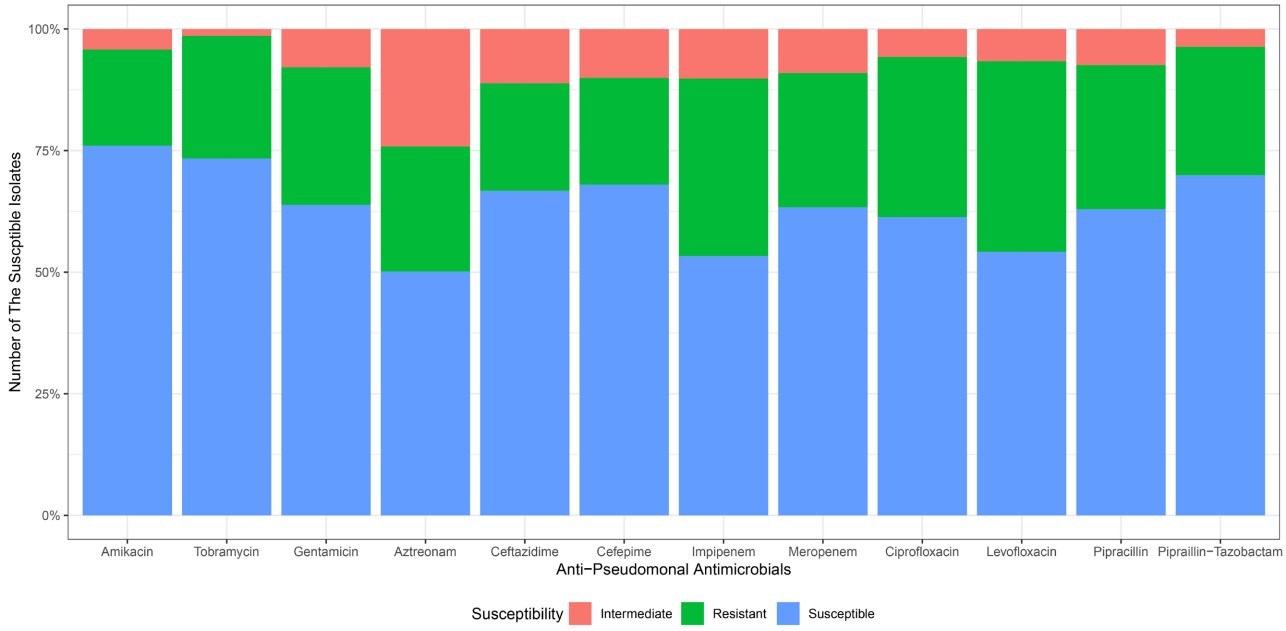
(c)

Figure 1. (a) Frequency distribution of all gram-negative bacteria and *P. aeruginosa*; (b) ratio of *P. aeruginosa* to all gram-negative bacteria; (c) ratio of *P. aeruginosa* to all lisolates.

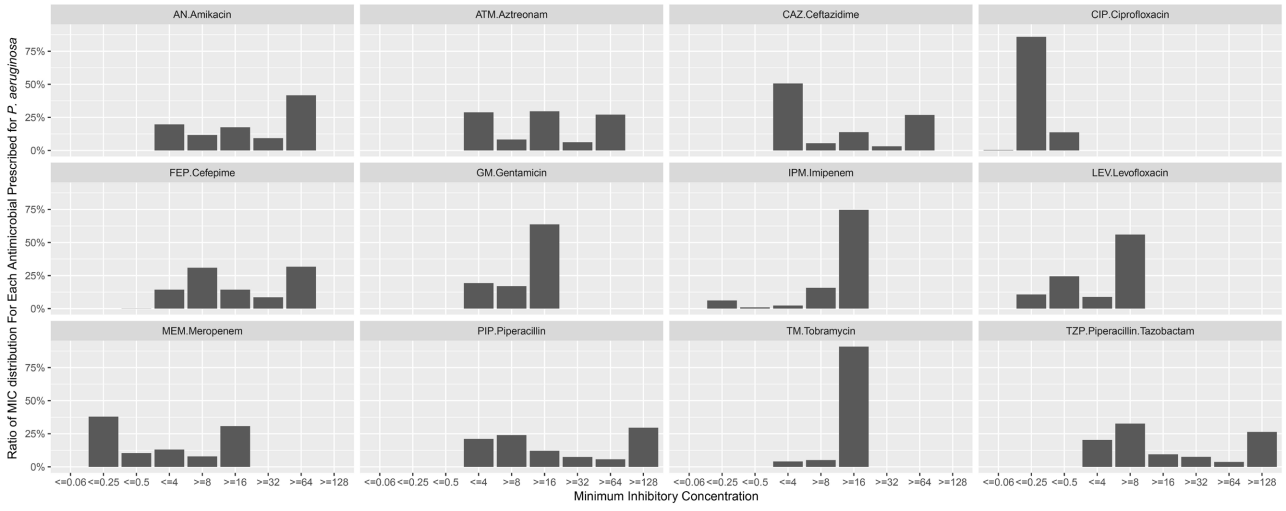
P. aeruginosa susceptibility showed no significant annual variability for the susceptible, intermediate and resistance categories (**Figure 2(a)**). Based on CLSI 2024 MIC breakpoints, MIC distribution showed the following resistance: Ceftazidime 33.3%, Cefepime 32%, Aztreonam 49.4% (breakpoint > 8 μgm/ml); amikacin 24% (breakpoint > 16 μgm/ml); Gentamicin 36.1% and Tobramycin 27% (breakpoint >

4 µgm/ml); imipenem 46.7% and Meropenem 36.7% (breakpoint > 2 µgm/ml). Levofloxacin 45.8% (breakpoint > 2 µgm/ml); Ciprofloxacin 38.7% (breakpoint > 1 µgm/ml); Piperacillin-Tazobactam 30.1% and Piperacillin 37% (breakpoint > 16 µgm/ml) (**Figure 2(b)**). Across the 15-year period, susceptibility patterns showed a notable proportion of resistant isolates, with relatively few intermediate strains (**Figure 3**; detailed antibacterial resistance trends and effect sizes in **Figures 3(b)-(c)**).

Colistin was not included in VITECK 2 testing because CLSI recommends broth microdilution as the reference method [10] which was only recently implemented in our laboratory.

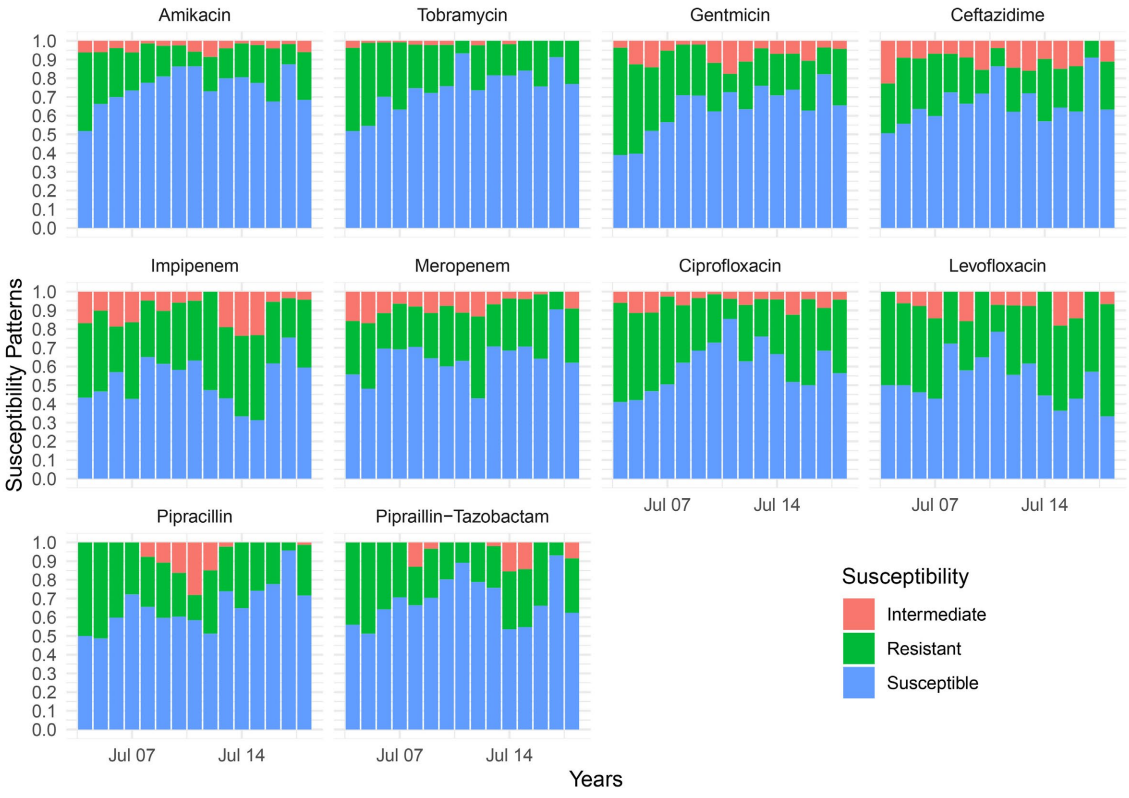


(a)

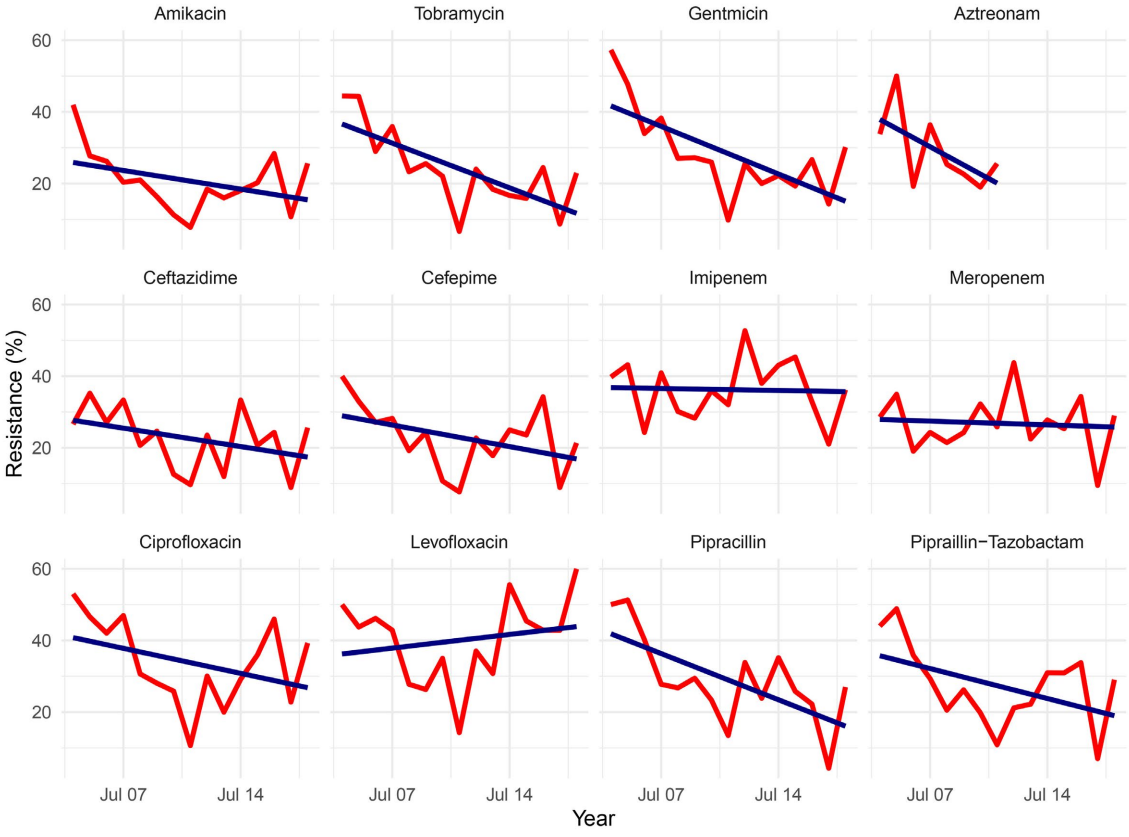


(b)

Figure 2. (a) All tested isolates of *P. aeruginosa* and their susceptibility for antimicrobials during the fifteen years (2010-2024); (b) the minimum inhibitory concentration for the tested antimicrobials against *P. aeruginosa* (2010-2024).


Amikacin
Tobramycin
Gentmicin
Ceftazidime
Impipenem
Meropenem
Ciprofloxacin
Levofloxacin
Pipracillin
Pipraillin−Tazobactam
Susceptibility Patterns
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
Jul 07
Jul 14
Susceptibility
Intermediate
Resistant
Susceptible
Years

(a)


Amikacin
Tobramycin
Gentmicin
Aztreonam
Ceftazidime
Cefepime
Imipenem
Meropenem
Ciprofloxacin
Levofloxacin
Pipracillin
Pipraillin−Tazobactam
Resistance (%)
60
40
20
Jul 07
Jul 14
Year

(b)

Antibiotics	slope	Std.Error	RR	CI_low	CI_high	P_value
Amikacin	-0.035	0.016	0.966	0.936	0.996	0.0287
Tobramycin	-0.100	0.015	0.905	0.879	0.932	0.0000
Gentamicin	-0.086	0.015	0.918	0.891	0.945	0.0000
Aztreonam	-0.106	0.040	0.899	0.832	0.973	0.0080
Ceftazidime	-0.037	0.015	0.964	0.936	0.992	0.0136
Cefepime	-0.043	0.015	0.958	0.930	0.986	0.0041
Imipenem	0.008	0.013	1.008	0.983	1.034	0.5383
Meropenem	0.008	0.015	1.008	0.979	1.038	0.5938
Ciprofloxacin	-0.040	0.013	0.961	0.937	0.986	0.0021
Levofloxacin	0.035	0.034	1.036	0.969	1.107	0.3033
Pipracillin	-0.077	0.017	0.926	0.896	0.957	0.0000
Pipracillin-Tazobactam	-0.050	0.015	0.951	0.924	0.980	0.0009

(c)

Figure 3. *P. aeruginosa* (a) susceptibility, (b) resistance trends, (c) effect size, odds ratio (RR), and significance for several agents (2010-2024).

Each antimicrobial demonstrated a calculated resistance rate ratio (RR) (**Figure 3(c)**), e.g. levofloxacin and carbapenems showed increasing resistance trends, although these were not statistically significant: levofloxacin (RR = 1.04, 95% C.I. 0.97 - 1.11, P = 0.30), Imipenem (RR = 1.008, 95% C.I. (0.98 - 1.03), P = 0.54), and meropenem (RR = 1.008, 95% C.I. (0.98 - 1.04), P = 0.59). All remaining antimicrobials showed significant decreasing resistance trends, including Ciprofloxacin (RR = 0.96, 95% C.I. (0.94 - 0.99), P = 0.002), ceftazidime showed decrease in *P. aeruginosa* resistance (RR = 0.96, 95% C.I. (0.94 - 0.99), P = 0.014), cefepime (RR = 0.96, 95% C.I. (0.93 - 0.97), P = 0.004), gentamycin (RR = 0.92, 95% C.I. (0.89 - 0.95), P < 0.000), tobramycin (RR = 0.91, 95% C.I. (0.88 - 0.93), P <0.000), amikacin (RR = 0.97, 95% C.I. (0.94 - 1.0), P = 0.03), aztreonam (RR = 0.90, 95% C.I. (0.83 - 0.97), P = 0.008), piperacillin (RR = 0.93, 95% C.I. (0.9 - 0.96), P < 0.000) and piperacillin-Tazobactam (RR = 0.95, 95% C.I. (0.92 - 0.98), P = 0.001) (**Figure 3(c)**).

The Advanced Expert System (AES) identified several carbapenems-resistant mechanisms in *P. aeruginosa*. Carbapenemases-producing *P. aeruginosa* (CRPa) showed a significant annual reduction in resistance (RR = 0.98, P < 0.000) (**Figure 4**). Additional mechanisms included Metallo-β-lactamase or OXA-type enzymes (n = 61, 3.5%), impermeability (n = 637, 36.9%), efflux (MexAB; n = 4, 0.2%), and high level cephalosporinase activity (n = 52, 3.0%).

Extended Spectrum β-lactamases (ESBL-producing) *P. aeruginosa* (clavulanate inhibited) accounted for 42 isolates (2.4%) as a single resistance mechanism, and an additional 74 isolates (4.3%) combines ESBL production with impermeability, giving a total of 116 ESBL-producing isolates (6.7%). Aminoglycoside-resistant *P.*

aeruginosa affected multiple agents simultaneously (n = 936, 55.2%), including amikacin, gentamicin, netilmicin, and tobramycin, while gentamicin-only resistance was observed in 29 isolates (1.7%). Fluoroquinolone resistance occurred in 702 isolates (40.6%), and wild-type susceptibility patterns were noted in 994 isolates (57.5%).

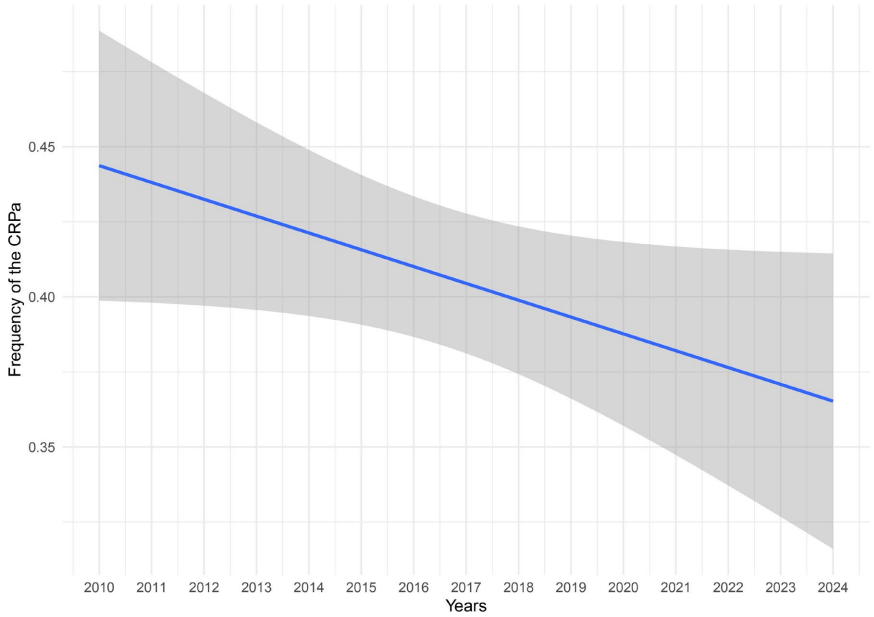


Figure 4. Trends of the carbapenem-resistant *P. aeruginosa* (CRPa) over fifteen years (2010-2024).

4. Discussion

Our study shows that *P. aeruginosa* contributed to 5.17% of hospital bacterial, and 9.12% of GNB isolates (**Table 1**). A total of 87.1% of isolates originated from general wards and 12.9% from the ICU, yet most of the studies emphasize the ICU as a major reservoir for cross-transmission and environmental contamination particularly in burn units [11]-[13].

Although most isolates were urinary (28.3%), followed by respiratory (14.3%), a considerable number were recovered from blood (5.4%) and sterile sites (n = 195, 11.3%) together comprising 16.7% of all isolates, underscoring the organism's potential virulence. The other anatomical sites are frequently colonized; however true infection could not always be determined due to the lake of clinical data [14].

Hospital *P. aeruginosa* strains are typically more resistant to antimicrobials including β-lactams, fluoroquinolones, aminoglycoside, and aminoglycosides, resistance tend to increase under antimicrobial selection pressure [15]. Although our isolates were derived exclusively from inpatients, Momenah *et al.* reported comparable susceptibility profiles between community and hospital isolates, contradicting the commonly held assumption [14]. Moreover, despite the higher virulence attributed to Hospital acquired *P. aeruginosa*, one study found no significant mortality difference when compared with community-acquired infections;

greater organ dysfunction occurred in hospital-acquired sepsis (P = 0.05), but mortality did not differ significantly (P = 0.38). The authors concluded that the presence or absence of specific virulence factors, rather than infection origin, was more influential [16]. Nonetheless, delayed appropriate therapy in *P. aeruginosa* sepsis remains strongly associated with increased mortality [17].

In our data the overall frequency of GNB seems to be stationary over the fifteen years period, increasing numerically until declining during the COVID-19 pandemic (**Figure 1**). In contrast, *P. aeruginosa* showed significant decline in frequency and rate ratio over time, including during the pandemic (RR = 0.96, P = 0.04). A similar reduction in multidrug resistant *P. aeruginosa* following the onset of COVID-19 was previously reported and attributed to stricter infection control practices, shifts in antibacterials prescribing, and possibly a clinical focus on viral management rather than bacterial infections [18].

Resistance rates to antipseudomonal agents were consistently high throughout the study period, with many MIC distributions exceedingly more than twice the susceptibility breakpoints (**Figure 2(a)**, **Figure 2(b)**). The average resistance rate (including the intermediate susceptibility categories) was 36.4% (range: 24 - 49.9, **Figure 2(a)**).

Individual antimicrobials susceptibilities are examined separately, *P. aeruginosa* resistance (including intermediate susceptibility) to carbapenems and quinolones were higher than to others agents (**Figure 3(a)**). Resistance trends (**Figure 3(b)**) demonstrated that carbapenems remained minimally increased and largely stable over time (RR = 1.008, P > 0.5). Levofloxacin resistance increased over time (including intermediate isolates), although this was not statistically significant, yet the effect size suggests epidemiological relevance (RR = 1.04, P > 0.5). When considering only resistant isolates, levofloxacin resistance increased (Slope = 1.035, RR = 1.04, P = 0.303), while ciprofloxacin resistance significant decreased over time (Slope = −0.04, RR = 0.96, P = 0.002), this relatively small effect size (**Figure 3(b)**, **Figure 3(c)**) may still have meaningful implications at the hospitals or population level [19]. Recently, similar to our isolates, a study reported a discordant fluoroquinolones susceptibility in *P. aeruginosa* for both quinolones; ciprofloxacin-susceptible and levofloxacin-resistant *P. aeruginosa* were reported and linked to efflux pump over expression rather than quinolone target mutation [20]. Hitherto, antipseudomonal quinolones resistance has been observed several decades ago, and they recorded high rate between 1991-2000 and were associate with higher mortality [21] [22].

CRPa are currently a major health care challenge in nosocomial infections, with an alarming high rate up to 30% worldwide [23]. Our study showed an even higher rate of 40.7%. CRPa results from several mechanisms, most prominent the enzymes carbapenemases production. After excluding other AES-interpreted mechanisms, we observe a small but statistically significant decline in resistance (RR = 0.98, P < 0.000) (**Figure 4**). CRPa bacteremia was reported to be associated with high mortality approaching 50%, particularly among ICU patients, those with

prolonged hospitalization, invasive devices, or multiple comorbidities [24] [25].

P. aeruginosa also expresses ESBL and the inducible AmpC cephalosporinase (derepression). Additional resistance mechanisms include MexAB efflux pump, the inducible MexXY efflux pump not identified in this study, porin loss or reduce impermeability around 4.3%, or isolated but clavulanate inhibited ESBL around 2.4% [26] [27]. The coexistence of several resistance mechanisms increases the likelihood of the initial inappropriate therapy and is associated with the poorer outcomes and higher mortality, particularly in blood stream infections [28].

In this era of rising bacterial resistance to commonly prescribed antimicrobials, available treatment options have become increasingly limited. Several new agents targeting resistant *Pseudomonas aeruginosa* have been introduced in recent years, including cefolozane-tazobactam, ceftazidime-avibactam, imipenem-cilastatin-relebactam, cefepime-zidebactam, cefiderocol, and the investigational agent murepavadin. Although these agents may continue to provide effective therapeutic options in the coming decade, their use imposes a substantial economic and logistical burden on low- and lower-middle-income countries [29] [30].

Our study aims to provide clinically meaningful interpretation of *P. aeruginosa* resistance patterns and long-term trends. These findings highlight the need to strengthen antimicrobial stewardship, enhance prescription-control policies, and enforce antimicrobial privilege systems [31]. The main limitation of our study is its retrospective, single-center design and the lack of comprehensive clinical data, which prevented us from correlating isolates, susceptibility patterns, and patient outcomes to provide more informed recommendations for managing the increasing antimicrobial resistance.

5. Conclusion

Despite a modest overall decline in *P. aeruginosa* resistance trends—except for levofloxacin and carbapenems—overall resistance rates remain high, and CRPa rates remain continues to rise at an alarming rate higher in our region than what is being observed in many other countries. Hospitals should address the unconventional antimicrobial prescribing practices observed at institutional levels by enforcing antimicrobial restriction and stewardship policies, alongside strong infection-prevention programs. Although our study shows a modest decline in resistance over time, more aggressive and targeted interventions are needed to achieve clinically meaningful reductions with large effect sizes.

Acknowledgements

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Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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