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Original Research Article

Impact of the revised CLSI M100 (36th edition, 2026) minimum inhibitory concentration breakpoints on antimicrobial susceptibility interpretation and multidrug-resistance classification of clinical *Pseudomonas aeruginosa* isolates: a retrospective study

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ABSTRACT

Background: Susceptibility interpretation depends on the breakpoint applied to the measured minimum inhibitory concentration (MIC). The CLSI M100 36th edition (2026) introduced revised criteria for *Pseudomonas aeruginosa*, including lower fluoroquinolone breakpoints, removal of the gentamicin breakpoint, restriction of amikacin reporting to urinary isolates and removal of the colistin-susceptible category. Authors assessed how reinterpreting archived MICs altered susceptibility categorisation and multidrug-resistant (MDR)/extensively drug-resistant (XDR) classification.
Methods: Archived VITEK-2 MIC results for 200 non-duplicate clinical *P. aeruginosa* isolates (December 2020–January 2025) were interpreted twice using the laboratory's previously reported interpretations and the 2026 CLSI criteria. Categorical agreement, susceptibility percentages (Wilson 95% CIs) and MDR/XDR proportions (Magiorakos framework) were compared and paired changes were tested using the exact McNemar test.
Results: Of the 1,871 drug–isolate results interpretable under both schemes, 285 (15.2%) changed categories. According to the 2026 criteria, susceptibility ranged from 35.8% (levofloxacin) to 54.1% (piperacillin/tazobactam). Reinterpretation resulted in increased susceptibility for several β-lactams and ciprofloxacin, mainly through resistant-to-susceptible reassignment (McNemar $p \leq 0.015$). Colistin lost its susceptible category, leaving 91.9% intermediate and 8.1% resistant strains, without any MIC changes. Overall, 38.5% of the isolates were non-MDR, 10.0% were MDR and 51.5% were XDR strains.
Conclusions: Under CLSI M100 36th edition, reinterpretation reclassified roughly one in seven results, with a net susceptibility gain for several β-lactams and ciprofloxacin and removal of the colistin susceptible category, while most isolates remained XDR. Laboratories should revalidate these breakpoints and account for colistin and aminoglycoside reporting changes in the antibiograms.

Keywords: Antimicrobial susceptibility testing, CLSI breakpoints, Minimum inhibitory concentration, Multidrug resistance, *Pseudomonas aeruginosa*

INTRODUCTION

Pseudomonas aeruginosa is a leading cause of healthcare-associated infections and is intrinsically resistant to many antimicrobial agents.[1,2] It is designated as a critical-priority pathogen for which new therapeutic and stewardship strategies are urgently needed.[2,3] Its capacity to acquire additional resistance through mutation and horizontal gene transfer, including depression of chromosomal AmpC and up-regulation of efflux systems, makes reliable antimicrobial susceptibility testing (AST) central to patient management and to the surveillance of resistance trends.[4-7] The clinical significance of an AST result is defined by the interpretive breakpoint applied to the measured MIC.

Breakpoints are periodically revised to reflect updated pharmacokinetic–pharmacodynamic modelling, MIC distributions and clinical outcome data.[8,9] Because a change in a breakpoint or a change in reporting convention can move an isolate between the susceptible (S), intermediate (I) and resistant (R) categories without any change in the underlying MIC, such revisions can alter reported susceptibility rates, cumulative antibiograms and the classification of isolates as multidrug-resistant (MDR) or extensively drug-resistant (XDR).[10]

Over successive editions of the M100 document, CLSI has revised the interpretive criteria for *P. aeruginosa*. The current CLSI M100 36th edition (2026) incorporates lower fluoroquinolone breakpoints, removal of the gentamicin breakpoints, restriction of amikacin reporting to urinary isolates, a revised piperacillin/tazobactam breakpoint and reporting of colistin without a susceptible category, while retaining netilmicin and ticarcillin-clavulanate as provided in the supplemental agents table in the CLSI document; the antipseudomonal cephalosporin and carbapenem numeric breakpoints are largely unchanged.[1,11] Understanding the practical effect of these revisions requires applying the current criteria to a defined set of isolates and comparing the resulting interpretations with those previously reported, an approach also relevant where laboratories choose between CLSI and EUCAST systems.[9,12,13]

Therefore, we performed a retrospective breakpoint-impact analysis of 200 clinical *P. aeruginosa* isolates. Our objectives were to quantify the categorical reclassification produced by the CLSI M100 36th edition breakpoints, compare susceptibility percentages for the antipseudomonal agents before and after the revision and determine the proportion of isolates classified as MDR or XDR under the current criteria. This comparison quantifies the cumulative effect of transitioning from the laboratory's previously reported interpretations to the current consolidated breakpoints.

METHODS

Study design and setting

This retrospective study was based on the archived laboratory records. Clinical isolates of *P. aeruginosa* were recovered, identified and tested for antimicrobial susceptibility at the diagnostic microbiology laboratory of Nazareth Hospital, Shillong, Meghalaya, during routine patient care between January 2021 and January 2025.

These isolates, along with their laboratory records, were subsequently retrieved and transferred to the Department of Microbiology, Assam Don Bosco University, Guwahati, Assam, for further study. In the analysis reported here, the recorded MIC values and susceptibility results of all non-duplicate isolates from this period were reinterpreted; no new susceptibility test were performed. Species-level identification was performed at the diagnostic laboratory using the VITEK-2 Compact system (bioMérieux, Marcy-l'Étoile, France).

Specimen and isolate selection

Consecutive (total) sampling was used: every identified, non-duplicate *P. aeruginosa* isolate recorded in the diagnostic laboratory database for the study period was included, with one isolate per patient episode, yielding 200 isolates. Isolates were obtained from tracheal aspirate, wound swab, urine and pus specimens submitted for culture and sensitivity testing.

Demographic details (age and sex), specimen type and ward information were retrieved from laboratory records. Isolates that could not be confidently identified as *P. aeruginosa*, duplicate isolates from the same patient episode and records with incomplete susceptibility data were excluded.

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing was performed by the diagnostic laboratory using the MIC method on the VITEK-2 Compact system (bioMérieux) with appropriate Gram-negative susceptibility cards. The panel comprised piperacillin/ tazobactam, ceftazidime, cefoperazone/ sulbactam, cefepime, imipenem, meropenem, amikacin, gentamicin, netilmicin, ciprofloxacin, levofloxacin, colistin, ticarcillin/clavulanic acid and fosfomycin susceptibility tests.

Routine quality control was maintained using *P. aeruginosa* ATCC 27853, in accordance with CLSI recommendations.[14] The recorded MIC values were retrieved for this study and the agents were referred to by their International Non-proprietary Names. Because CLSI and EUCAST recommend that colistin susceptibility be determined by reference broth microdilution and consider automated systems, including the VITEK-2, to be unreliable for this agent, the colistin MIC values are used here only to illustrate the effect of the reporting-convention change and not as definitive measures of colistin activity.

Breakpoint interpretation

Each recorded MIC was interpreted twice against an identical numeric value: first using the interpretations previously reported by the laboratory (the "prior" criteria, as recorded in the archive) and then using the CLSI M100 36th edition (2026) criteria. Under the 2026 criteria, the applied MIC breakpoints (µg/ml; S/I/R) were piperacillin/tazobactam ≤16/32/≥64 (revised from a prior resistant breakpoint of ≥128), ceftazidime ≤8/16/≥32, cefepime ≤8/16/≥32, imipenem ≤2/4/≥8, meropenem ≤2/4/≥8, ciprofloxacin ≤0.5/1/≥2 (lowered from ≤1/2/≥4) and levofloxacin ≤1/2/≥4 (lowered from ≤2/4/≥8).

Amikacin (≤16/32/≥64) was reported for urinary isolates only. Gentamicin, which lost its CLSI *P. aeruginosa* breakpoint, was not assigned a category under the 2026 scheme; netilmicin (≤8/16/≥32) and ticarcillin-clavulanate (≤16/2/32/2–64/2/≥128/2) were retained as supplemental ("Other") agents.

Colistin was reported without a susceptible category under the 2026 criteria (MIC ≤2 µg/ml intermediate; ≥4 µg/ml resistant), whereas the previous scheme retained a susceptible category (≤2 µg/ml). Cefoperazone/sulbactam and fosfomycin have no CLSI *P. aeruginosa* breakpoint and were interpreted only under the prior/local criteria. The complete set of prior and 2026 breakpoints is presented in Table 5.

Definition of multidrug and extensive drug resistance

Isolates were classified as non-MDR, MDR or XDR following the international expert-proposal criteria of Magiorakos et al applied to the CLSI M100 36th edition (2026) interpretation.[4] The antimicrobial categories relevant to P. aeruginosa were antipseudomonal penicillin/β-lactamase-inhibitor combinations (piperacillin/tazobactam and ticarcillin-clavulanate), antipseudomonal cephalosporins (ceftazidime and cefepime), carbapenems (imipenem and meropenem), fluoroquinolones (ciprofloxacin and levofloxacin), aminoglycosides (amikacin and netilmicin), polymyxins (colistin) and phosphonic acids (fosfomycin).

MDR was defined as acquired non-susceptibility (intermediate or resistant) to at least one agent in three or more categories and XDR as non-susceptibility to at least one agent in all but two or fewer categories (evaluated where at least five categories were tested). Isolates that did not meet the MDR threshold were classified as non-MDR.

Data handling and statistical analysis

Interpretations were generated by applying the defined breakpoints to the recorded MICs in a spreadsheet-based interpretation model, with each result carried forward from its corresponding numerical MIC. The categorical agreement between the prior and 2026 interpretations was expressed as the number and percentage of drug–isolate results whose category changed and the direction of movement was tabulated per agent. Susceptibility, intermediate and resistance percentages were calculated per agent over the isolates with interpretable results and 95% confidence intervals (CIs) were derived using the Wilson method. Censored MIC values (for example, ≤ or ≥ a given concentration) were conservatively interpreted at the relevant breakpoint.

Because each isolate was interpreted under both breakpoint sets, the paired change in susceptible-versus-non-susceptible status was tested for each agent using the exact (binomial) McNemar test, which was restricted to isolates evaluable under both schemes. A two-sided p value<0.05 was considered statistically significant.

Because a separate McNemar test was performed for each antimicrobial agent, these paired comparisons were treated as descriptive rather than confirmatory and no formal correction for multiple testing was applied; the p-values should therefore be read as exploratory indicators of the direction and consistency of change rather than as tests of a single pre-specified hypothesis. Data were compiled in Microsoft Excel and analysed using standard statistical software.

RESULTS

Isolate and patient characteristics

A total of 200 non-duplicate clinical P. aeruginosa isolates were analysed. The patients comprised 117 males (58.5%) and 83 females (41.5%); the median age was 44.5 years (range, 3–89 years). The most common specimen type was tracheal aspirate (90 isolates, 45.0%), followed by wound swabs (48, 24.0%), urine (36, 18.0%) and pus (26, 13.0%). Patient and specimen characteristics are summarised in Table 1.

Categorical reclassification under the 2026 breakpoints

Of the 1,871 drug–isolate results that were interpretable under both the prior and 2026 criteria, 285 (15.2%) categorical assignments changed when the 2026 breakpoints were applied. Unlike a simple breakpoint-lowering, the shifts were bidirectional and affected most of the patients. The single largest movement was for colistin (84 results changed), driven almost entirely by 77 susceptible-to-intermediate reassignments that followed the removal of the susceptible category. Among the β-lactams, ceftazidime (34 results changed), cefepime (31), meropenem (31) and imipenem (30) showed substantial movement, predominantly resistant-to-susceptible (ceftazidime, 19; imipenem, 18; cefepime, 16; meropenem, 16 results moving R→S).

The fluoroquinolone levels changed in 21 (ciprofloxacin) and 28 (levofloxacin) samples, respectively. These shifts arise from two distinct sources. For agents whose 2026 numeric breakpoints were revised, such as fluoroquinolones (lowered ciprofloxacin and levofloxacin criteria), piperacillin/tazobactam (revised resistant breakpoint) and colistin (removal of the susceptible category), reclassification was a direct consequence of the breakpoint change.

For the antipseudomonal cephalosporins and carbapenems, whose 2026 numeric breakpoints are essentially unchanged from prior CLSI values, the observed movement reflects differences between the interpretations originally recorded in the archive and a strict, consistent application of the current criteria, rather than any change in the breakpoint itself. The directions and magnitudes of the shifts per agent are listed in Table 3.

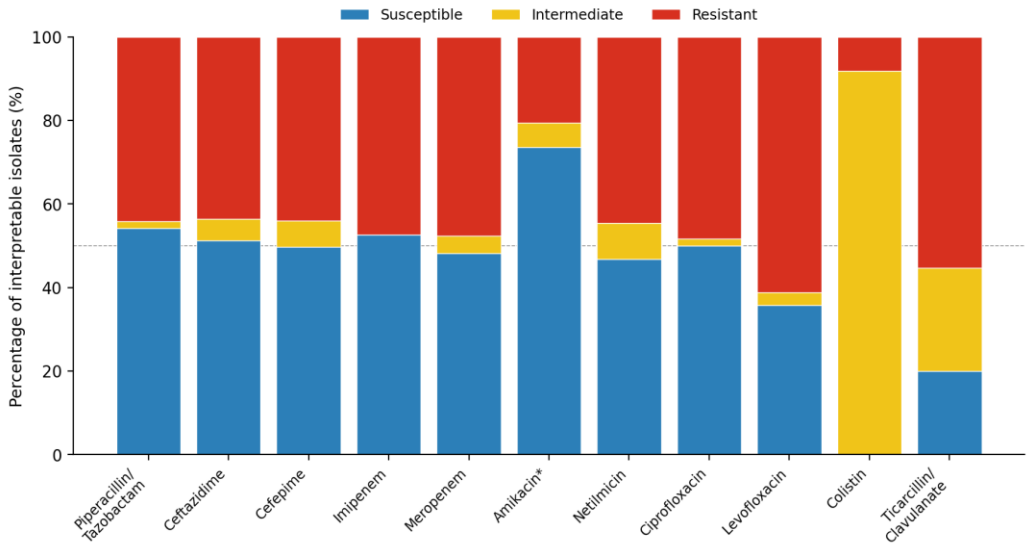

Figure 1: Antimicrobial susceptibility profile of the 200 clinical Pseudomonas aeruginosa isolates under the CLSI M100 36th edition (2026) breakpoints. Stacked bars show the percentage of interpretable isolates categorised as susceptible, intermediate and resistant for each agent with a 2026 P. aeruginosa breakpoint. Amikacin (*) is shown only for urinary isolates. Colistin has no susceptible category under the 2026 criteria; therefore, its bar comprises intermediate and resistant categories only.

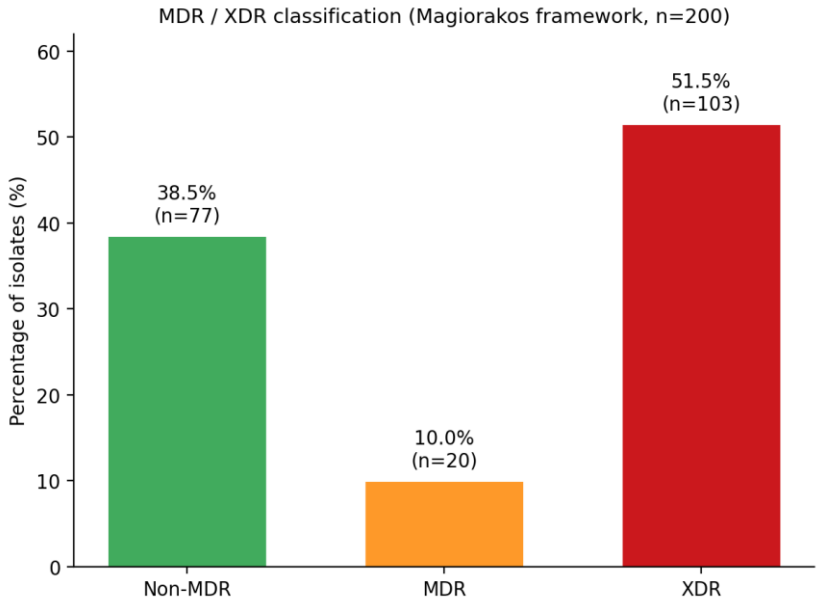

Figure 2: Distribution of non-MDR, MDR and XDR *Pseudomonas aeruginosa* isolates under CLSI M100 36th edition (2026) interpretations, classified using the Magiorakos framework. Under the 2026 criteria, 38.5% of isolates were non-MDR, 10.0% MDR and 51.5% XDR; thus, the majority met the criteria for extensive drug resistance.

Susceptibility under prior and 2026 breakpoints

Under the 2026 criteria, susceptibility (with Wilson 95% CIs) was 54.1% for piperacillin/tazobactam (46.9–61.2%), 51.3% for ceftazidime (44.3–58.2%), 49.7% for cefepime (42.7–56.8%), 52.7% for imipenem (45.5–59.8%), 48.2% for meropenem (41.3–55.2%), 50.0% for ciprofloxacin (42.8–57.2%) and 35.8% for levofloxacin (29.3–42.7%). Compared with the interpretations previously reported, susceptibility increased for piperacillin/tazobactam (46.7% to 54.1%; McNemar exact $p<0.001$), ceftazidime (44.2% to 51.3%; $p=0.015$), cefepime (41.1% to 49.7%; $p=0.006$) and ciprofloxacin (41.0% to 50.0%; $p<0.001$) and more modestly for imipenem (46.5% to 52.7%; $p=0.061$), meropenem (44.4% to 48.2%; $p=0.265$) and levofloxacin (34.7% to 35.8%; $p=0.557$).

Amikacin, reported for all isolates under the prior criteria (66.7% susceptible), was reported for urinary isolates only under the 2026 criteria, of which 73.5% (n=34) were susceptible. Gentamicin (prior susceptibility, 60.0%) and fosfomycin had no 2026 *P. aeruginosa* breakpoint and netilmicin (46.7% susceptible) and ticarcillin-clavulanate (20.0% susceptible) were interpreted as supplementary agents. Colistin retained no susceptible category under the 2026 criteria: 91.9% of isolates tested as intermediate (MIC ≤2 µg/ml) and 8.1% as resistant, compared with 42.0% susceptible, 47.3% intermediate and 10.6% resistant under the prior scheme, entirely reflecting the reporting-convention change rather than any change in MIC. The susceptibility percentages for all agents under both breakpoint sets are presented in Table 2 and the susceptibility profile under the 2026 criteria is shown in Figure 1.

Multidrug and extensive drug resistance

Under the Magiorakos framework applied to the 2026 interpretations, 77 isolates (38.5%) were non-MDR, 20 (10.0%) were MDR and 103 (51.5%) were XDR; thus, 123 isolates (61.5%) met the criteria for MDR or worse, with XDR being the predominant category. XDR was frequent across specimen types and was highest in tracheal aspirate isolates (56 of 90, 62.2%); frank MDR was concentrated in male patients (17 of 20 MDR isolates). The overall distribution and breakdown by sex and specimen are summarised in Table 4 and illustrated in Figure 2.

Table 1: Demographic and specimen characteristics of the 200 *Pseudomonas aeruginosa* isolates.

Characteristic	Category	Number (n)	(%)
Sex	Male	117	58.5
	Female	83	41.5
Age (in years)	Median (range)	44.5 (3–89)	—
Specimen type	Tracheal aspirate	90	45.0
	Wound / swab	48	24.0
	Urine	36	18.0
	Pus	26	13.0
Total		200	100.0

Table 2: Antimicrobial susceptibility of *P. aeruginosa* under prior and CLSI M100 36th edition (2026) breakpoints.

Antimicrobial agent	Tested n (prior)	Tested n (2026)	Prior %S	2026 %S	Prior %R	2026 %R
Piperacillin/tazobactam	182	181	46.7	54.1	49.5	44.2
Ceftazidime	197	195	44.2	51.3	54.3	43.6
Cefoperazone/sulbactam	193	—	40.9	—	52.3	—
Cefepime	192	191	41.1	49.7	49.0	44.0
Imipenem	185	184	46.5	52.7	51.9	47.3
Meropenem	198	195	44.4	48.2	50.5	47.7
Amikacin	186	34	66.7	73.5	25.3	20.6
Gentamicin	185	—	60.0	—	18.9	—
Netilmicin	186	184	46.2	46.7	45.7	44.6
Ciprofloxacin	188	184	41.0	50.0	54.3	48.4
Levofloxacin	196	193	34.7	35.8	60.7	61.1
Colistin	188	186	42.0	0.0	10.6	8.1
Ticarcillin/clavulanate	151	150	17.9	20.0	57.6	55.3
Fosfomycin	12	—	16.7	—	75.0	—

%S, percentage susceptible; %R, percentage resistant.

Table 3: Categorical reclassification of results under the CLSI M100 36th edition (2026) breakpoints by agent.

Antimicrobial agent	R→S	R→I	I→S	I→R	S→I	S→R	Total changed
Piperacillin/tazobactam	12	0	1	2	0	0	15
Ceftazidime	19	8	0	1	0	6	34
Cefepime	16	1	5	3	0	6	31
Imipenem	18	0	2	1	0	9	30
Meropenem	16	0	2	2	2	9	31
Amikacin	2	0	0	0	0	0	2
Netilmicin	1	1	0	0	0	1	3

Continued.

Antimicrobial agent	R→S	R→I	I→S	I→R	S→I	S→R	Total changed
Ciprofloxacin	14	0	4	2	0	1	21
Levofloxacin	14	0	1	2	0	11	28
Colistin	0	6	0	1	77	0	84
Ticarcillin/clavulanate	3	1	1	0	1	0	6
Total	115	17	16	14	80	43	285

R, resistant; I, intermediate; S, susceptible.

Table 4: MDR/XDR classification of the 200 *P. aeruginosa* isolates under the CLSI M100 36th edition (2026) interpretations, overall and by sex and specimen.

Classification	Overall n (%)	Male	Female	Tracheal aspirate	Swab	Urine	Pus
Non-MDR	77 (38.5)	40	37	26	16	18	17
MDR	20 (10.0)	17	3	8	7	2	3
XDR	103 (51.5)	60	43	56	25	16	6
Total	200 (100.0)	117	83	90	48	36	26

Table 5: CLSI M100 breakpoints (µg/ml) for *P. aeruginosa* applied in this study: prior to CLSI M100 36th edition (2026).

Drug	Prior S	Prior I	Prior R	2026 S	2026 I	2026 R
Piperacillin/tazobactam	≤16	32–64	≥128	≤16	32	≥64
Ceftazidime	≤8	16	≥32	≤8	16	≥32
Cefepime	≤8	16	≥32	≤8	16	≥32
Imipenem	≤2	4	≥8	≤2	4	≥8
Meropenem	≤2	4	≥8	≤2	4	≥8
Amikacin	≤16	32	≥64	≤16*	32*	≥64*
Gentamicin	≤4	8	≥16	—	—	—
Netilmicin	≤8	16	≥32	≤8	16	≥32
Ciprofloxacin	≤1	2	≥4	≤0.5	1	≥2
Levofloxacin	≤2	4	≥8	≤1	2	≥4
Colistin	≤2	—	≥4	—	≤2	≥4
Ticarcillin/clavulanate	≤16/2	32/2–64/2	≥128/2	≤16/2	32/2–64/2	≥128/2

*Amikacin is reported for urinary isolates only under the 2026 criteria.

DISCUSSION

In this retrospective analysis of 200 clinical *P. aeruginosa* isolates, reinterpreting the archived MICs under the CLSI M100 36th edition (2026) criteria changed the interpretive category of 15.2% of the results across the panel. Contrary to the expectation that only fluoroquinolones would be moved, reclassification affected most agents and was largely bidirectional. For β-lactams and ciprofloxacin, the net effect was a gain in susceptibility, predominantly driven by resistant-to-susceptible reassignment.

As noted in the results, only part of the overall reclassification is attributable to genuine breakpoint revisions (fluoroquinolones, piperacillin/ tazobactam and colistin); for cephalosporins and carbapenems, whose numeric breakpoints are essentially unchanged, the movement reflects a mismatch between the interpretations originally recorded in the archive and a strict application of the current criteria. Therefore, the headline figure of 15.2% should be understood as a combined measure of breakpoint-driven change and prior-interpretation inconsistency, not as a pure breakpoint effect. This distinction underscores the value of periodically re-auditing archived AST records according to current interpretive criteria.

The co list in findings warrant particular emphasis because they account for the single largest block of reclassification. The 2026 criteria define no susceptible category for colistin, so isolates previously reported as susceptible are now reported as intermediate without any change in the underlying MIC.[1] In the data, this moved 77 isolates from susceptible to intermediate, leaving 91.9% of isolates in the intermediate band and 8.1% resistant. This is an interpretive artefact of the reporting convention rather than a change in colistin activity and it should be explained clearly in cumulative antibiograms so that a fall in reported colistin susceptibility is not misread as emerging resistance.[10] Because CLSI and EUCAST recommend reference broth microdilution for colistin and regard automated MICs as unreliable, the colistin results here are illustrative of the reporting change only and should not guide therapy.

The overall burden of resistance in this population was high and was not reduced by revision. Carbapenem resistance approached half of isolates (imipenem 47.3%, meropenem 47.7% resistant) and fluoroquinolone resistance exceeded 60% for levofloxacin.[15,16] Under the 2026 criteria the majority of isolates were XDR (51.5%) and only 38.5% were non-MDR, consistent with contemporary surveillance of *P. aeruginosa* from respiratory and other clinical sources.[17,18] These rates reflect the well-recognised difficulty of treating pseudomonal infection in settings with substantial antimicrobial pressure.[8,19-21] The revision also changes the agents that feed the MDR/XDR classification system. Removal of the gentamicin breakpoints, restriction of amikacin to urinary isolates and retention of netilmicin as a supplemental agent alter the composition of the aminoglycoside category, while the colistin reporting change affects the polymyxin category.[4,12] Laboratories adopting the 2026 criteria should therefore be aware that both the reported susceptibility of individual agents and the categories that drive MDR/XDR classification shift under the new rules, even where the underlying MICs are unchanged.

Resistance mechanisms were not characterised in this analysis, which was based on VITEK-2 minimum inhibitory concentration (MIC) values reinterpreted under current breakpoints rather than on any assay designed to detect specific β-lactamases. Automated MIC patterns cannot confirm the underlying enzyme and CLSI provides no validated ESBL confirmatory test for P. aeruginosa. Prospective studies using validated confirmatory methods, such as the modified carbapenem-inactivation test or molecular detection of bla genes encoding extended-spectrum and metallo-β-lactamases, are required to define the mechanisms underlying resistance observed in this population.

The study had some limitations. It was retrospective and conducted at a single centre and the isolate set reflects local epidemiology and referral patterns; the magnitude of reclassification may differ elsewhere depending on the local MIC distribution. Susceptibility testing was performed by a diagnostic laboratory using a single automated system (VITEK-2 Compact) and we reinterpreted the archived MIC values instead of retesting the isolates. The "prior" interpretations were those recorded in the laboratory archive, whose exact provenance (software version and expert system rules) could not be fully reconstructed, which limited the mechanistic interpretation of individual category shifts.

Automated MICs may differ from reference broth microdilution and for colistin, broth microdilution is recommended; therefore, the colistin results should be viewed with caution. Because many VITEK MIC results are censored at the ends of the tested range, some isolates could not be assigned to a single dilution, although this did not affect the categorical interpretation of the reported breakpoints. Several agents (cefoperazone/sulbactam, fosfomycin) lack CLSI *P. aeruginosa* breakpoints and could not be categorised under the 2026 scheme. Finally, the analysis was interpretive and did not incorporate clinical outcomes, which are the ultimate arbiters of breakpoint validity.

Despite these limitations, this study provides a clear and practical message. Transition to the CLSI M100 36th edition breakpoints reclassify a meaningful minority of *P. aeruginosa* results across the whole panel, with a net gain in susceptibility for several β-lactams and ciprofloxacin and a large reporting-driven shift for colistin, while leaving the substantial underlying burden of resistance most isolates being XDR unchanged. Where resistance to first-line agents is confirmed, newer options such as ceftolozane/ tazobactam and other agents active against resistant *P. aeruginosa* may warrant consideration, guided by local susceptibility data.[22-24] Laboratories should validate the revised breakpoints on their own systems, update cumulative antibiograms accordingly and communicate the colistin and aminoglycoside reporting changes to clinicians.

CONCLUSION

Application of the CLSI M100 36th edition (2026) breakpoints to a fixed set of clinical *P. aeruginosa* isolates reclassified approximately one in seven interpretable results across the panel, with a net gain in susceptibility for several β-lactams and ciprofloxacin and removal of the colistin susceptible category, which reassigned most isolates to the intermediate band. Under the revised criteria, most isolates were XDR and only a minority were non-MDR. This revision refines the interpretation of borderline MICs and alters the reporting conventions without masking the substantial resistance present in this population. A prospective multicentre evaluation linked to clinical outcomes and molecular confirmation of carbapenemase mechanisms would further define the impact of these breakpoints.

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