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Original Research Article

Evaluation of In-Vitro Activity of Plazomicin against Carbapenem Resistant Gram-Negative Bacilli Isolates from a Tertiary Care Hospital in Western India

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Abstract

Background: Carbapenem-resistant Gram-negative bacilli (CR-GNB) significantly compromise therapeutic options in clinical practice. Plazomicin, a next-generation Aminoglycoside, has been structurally optimized to evade most AMEs and retain activity against MDR Enterobacterales, may offer improved activity over conventional aminoglycosides, yet data from the Indian context are limited.

Methods: In this cross-sectional study conducted at a tertiary care cancer hospital in western India from December 2024-January 2025, 100 non-duplicate CR-GNB clinical isolates were evaluated. Antimicrobial susceptibility testing was performed using VITEK-2. Carbapenem isolates were further evaluated for expression of Carbapenemase genes and in vitro susceptibility to Plazomicin and its comparison to other Aminoglycoside.

Results: Plazomicin susceptibility (42%) was significantly higher than amikacin (18%), gentamicin (33%), and tobramycin (13.3%) ($p<0.001$). Isolates co-producing NDM with OXA-48 demonstrated elevated plazomicin MICs and resistance while those producing KPC or OXA-48 demonstrated a relatively higher proportion of susceptibility. Klebsiella pneumoniae showed MIC_{50}=>256, MIC_{90}=>256; Escherichia coli MIC_{50}=1, MIC_{90}=>256; Pseudomonas aeruginosa MIC_{50}=12, MIC_{90}=>256; and Enterobacter cloacae MIC_{50}=0.75, MIC_{90}=1. Plazomicin retains potent activity against isolates harboring single carbapenemase genes, while co-production of multiple resistance determinants- especially NDM with OXA-48 markedly diminishes susceptibility and results in elevated MIC values.

Conclusions: Plazomicin shows superior in vitro activity compared to conventional aminoglycosides against selected CR-GNB isolates. Resistance genotypes markedly influence plazomicin susceptibility, highlighting the role of molecular characterisation in guiding antimicrobial therapy.

Keywords: Carbapenem-resistant Gram-negative bacilli (CR-GNB); Plazomicin; Aminoglycoside Modifying Enzyme (AME); Carbapenemase gene.

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Introduction

Carbapenem-resistant Gram-negative bacilli (CR-GNB) have emerged as one of the most formidable global health challenges, primarily due to their extensive spread, limited therapeutic options, and association with high morbidity and mortality [1]. The most common pathogens implicated include Klebsiella pneumoniae, Escherichia coli, Pseudomonas aeruginosa, and Acinetobacter baumannii. These organisms acquire resistance through multiple mechanisms such as the production of carbapenemase enzymes, altered porin channels, and active efflux pumps [2]. Among these, carbapenemase production- particularly New Delhi metallo-β-lactamase (NDM), oxacillinase-48 (OXA-48), and Verona integron-encoded metallo-β-lactamase (VIM)- remains the most clinically significant as it confers high-level resistance to most β-lactams, including last-line Carbapenems [3]. Infections caused by CR-GNB are frequently associated with poor clinical outcomes, prolonged hospital stays, and increased healthcare costs [4]. The therapeutic armamentarium against these pathogens remains limited, often restricted to older and more toxic drugs such as Colistin, Tigecycline, or Fosfomycin. Alarmingly, resistance to these agents is also being increasingly reported [5]. Hence, there is an urgent need to identify newer antimicrobials with reliable activity against multidrug-resistant (MDR) Gram-negative bacteria. Aminoglycosides continue to play a vital role in the management of Gram-negative infections because of their rapid

bactericidal activity and proven synergy with β-lactams [6]. However, the widespread occurrence of Aminoglycoside modifying enzymes (AMEs) and 16S rRNA methyltransferases has significantly reduced their clinical utility [7]. Plazomicin, a next-generation Aminoglycoside derived from sisomicin, has been structurally optimized to evade most AMEs and retain activity against MDR Enterobacterales [8]. Its antibacterial effect is mediated through binding to the 30S ribosomal subunit, resulting in inhibition of protein synthesis and bacterial cell death. Importantly, Plazomicin remains active against many strains resistant to conventional Aminoglycoside, although resistance may still emerge through 16S rRNA methylation mediated by armA and rmtB genes [9].

The U.S. Food and Drug Administration (FDA) approved Plazomicin in 2018 for the treatment of complicated urinary tract infections (cUTIs) based on results from the EPIC trial [10]. Since then, its potential use in severe infections such as bacteremia, pneumonia, and intra-abdominal infections has been under investigation [11]. Several studies have reported lower minimum inhibitory concentrations (MICs) of Plazomicin compared to conventional Aminoglycoside, particularly against carbapenem-resistant Enterobacterales [12]. In countries such as India, where NDM and OXA-48 producers are highly prevalent, Plazomicin may represent a viable alternative to Colistin for treating bloodstream and urinary infections [13]. However, given the geographical variability of resistance determinants, it is essential to assess the performance of new antimicrobials in local epidemiological settings.

The present study was therefore designed to evaluate the in vitro activity of Plazomicin against carbapenem-resistant Gram-negative bacilli isolated from clinical specimens at a tertiary care oncology hospital. The study also aimed to compare the efficacy of Plazomicin with conventional Aminoglycoside and to analyze correlations between susceptibility profiles and underlying resistance genes. Such data are vital to support antimicrobial stewardship initiatives and guide rational therapeutic decision-making in high-risk clinical environments.

Materials and Methods

Study Design and Setting: This prospective cross-sectional in vitro study was conducted in the Department of Microbiology, at a tertiary care cancer hospital in Mumbai, over a two-month period from December 2024 to January 2025. The study included clinically significant, non-duplicate carbapenem-resistant Gram negative bacilli isolated from hospitalized patients. Only the first isolate from each patient was included to avoid duplication.

Bacterial Identification and Antimicrobial Susceptibility Testing: All isolates were initially identified and subjected to antimicrobial susceptibility testing using the VITEK-2 Compact system (bioMérieux, France), following the manufacturer's instructions [14]. The isolates that exhibited resistance to any of the carbapenem agents were further evaluated for expression of Carbapenemases genes and in vitro susceptibility to Plazomicin and its comparison to other Aminoglycoside.

Plazomicin Susceptibility Testing: Susceptibility to Plazomicin was determined by Epsilometer test (E-test, concentration range: 0.016–256 μg/mL) using Mueller–Hinton agar, as per the CLSI M100 (34th edition) guidelines [15]. Interpretive breakpoints were Susceptible (S): ≤2 μg/mL, Intermediate (I): 4 μg/mL and Resistant (R): ≥8 μg/mL. Escherichia coli ATCC 25922 was used as the quality control strain.

Detection of Carbapenemase Genes: Carbapenemase gene detection was performed using the NG-Test® CARBA 5 (RESIST-5 O.K.N.V.I.) kit (NG Biotech, France), a rapid lateral flow immunoassay designed to identify the five major carbapenemase genes- OXA-48-like, KPC, NDM, VIM, and IMP [16].

Data Compilation and Statistical Analysis: MIC values were recorded for all isolates, and MIC_{50} and MIC_{90} values were calculated by arranging the MICs in ascending order and identifying concentrations that inhibited 50% and 90% of isolates, respectively. Overall susceptibility rates for Plazomicin and other Aminoglycosides were expressed as percentages. Statistical analysis was performed using Microsoft Excel and chi-square test to compare susceptibility differences between Plazomicin and other Aminoglycosides. Associations between genotypic (NDM, OXA-48, etc.) and phenotypic resistance profiles were analyzed to assess genotype-phenotype correlations. A p-value of <0.05 was considered statistically significant.

Results

A total of 100 non-duplicate carbapenem-resistant Gram-negative bacilli (CR-GNB) were included in the study. The majority of isolates were Klebsiella pneumoniae (n = 45; 45%) and Escherichia coli (n = 34; 34%), followed by Pseudomonas aeruginosa (n = 15; 15%) and Enterobacter cloacae (n = 6; 6%). The predominance of K. pneumoniae and E. coli reflects the high burden of carbapenem-resistant Enterobacterales in the hospital setting, while P. aeruginosa and E. cloacae contributed to a smaller but clinically relevant proportion of infections.

Table 1: Comparative Susceptibility Profile of Aminoglycosides

Antibiotic	S	R	I	p-value
PLAZOMICIN	42 (42%)	56 (56%)	2 (2%)	**<0.001**
AMIKACIN	18 (18%)	80 (80%)	2 (2%)	
GENTAMICIN	33 (33%)	67 (67%)	0 (0%)	
TOBRAMYCIN	2/15 (13.3%)	13/15 (86.7%)	0/15 (0%)	

The comparative in vitro susceptibility of the 100 carbapenem-resistant Gram-negative isolates to Plazomicin, Amikacin, Gentamicin, and Tobramycin is summarized in Table 1.

Plazomicin demonstrated the highest overall activity, with 42% of isolates being susceptible, 56% resistant, and 2% showing intermediate susceptibility. In comparison, Amikacin exhibited a markedly lower susceptibility rate of 18%, with 80% of isolates resistant and 2% in the intermediate category. Gentamicin showed moderate activity, with 33% of isolates susceptible and 67% resistant, while none were categorized as intermediate. Tobramycin displayed the least activity, with susceptibility observed in only 13.3% of isolates.

Overall, Plazomicin retained superior in vitro efficacy compared with the conventional Aminoglycoside ($p < 0.001$), particularly against isolates resistant to Amikacin, Gentamicin, and Tobramycin.

Table 2: Resistance Genotype Correlation with Plazomicin Susceptibility

GENE	TOTAL (n=100)	S (n=42)	R (n=56)	I (n=2)
NDM	63	29 (46%)	32 (50.8%)	2 (3.2%)
NDM, OXA-48	23	5 (21.7%)	18 (78.3%)	0(0%)
NDM, KPC	3	3 (100%)	0 (0%)	0(0%)
OXA-48	10	4 (40%)	6 (60%)	0(0%)
KPC	1	1 (100%)	0(0%)	0(0%)

The correlation between resistance genotypes and in vitro Plazomicin susceptibility among the isolates is summarized in Table 2. Among the 63 isolates harboring the NDM gene alone, 29 (46%) were susceptible, 32 (50.8%) resistant, and 2 (3.2%) showed intermediate susceptibility.

In isolates co-producing NDM and OXA-48 (n = 23), susceptibility to Plazomicin was markedly reduced, with only 5 (22%) susceptible and 18 (78%) resistant. Interestingly, all three isolates co-harboring NDM and KPC were fully susceptible to Plazomicin. Of the 10 isolates harboring OXA-48 alone, 4 were susceptible and 6 resistant. The single isolate carrying KPC alone also exhibited susceptibility to Plazomicin.

Overall, Plazomicin activity appeared variable among isolates carrying NDM-type carbapenemases, whereas those producing KPC or OXA-48 demonstrated a relatively higher proportion of susceptibility.

Table 3: Organism-wise Resistance Gene Profile, Plazomicin Susceptibility, MIC_{50} and MIC_{90}

Organism	Gene	Total	S	R	I	MIC_{50}	MIC_{90}
K. pneumoniae		**45**	**17**	**28**	**0**	>256	>256
	NDM	16	10	6	0	0.75	>256
	NDM,OXA-48	17	1	16	0	>256	>256
	NDM,KPC	2	2	0	0	0.38	0.5
	OXA-48	9	3	6	0	>256	>256
	KPC	1	1	0	0	0.5	0.5
E. coli		**34**	**18**	**15**	**1**	1	>256
	NDM	27	12	14	1	3	>256
	NDM, OXA-48	5	4	1	0	1	>256
	NDM,KPC	1	1	0	0	1	1
	OXA-48	1	1	0	0	1	1
Ps. aeruginosa		**15**	**2**	**12**	**1**	12	>256
	NDM	15	2	12	1	12	>256
E. cloacae		**6**	**5**	**1**	**0**	0.75	1
	NDM	5	5	0	0	0.75	1
	NDM, OXA-48	1	0	1	0	>256	>256

The organism-wise resistance gene profile, Plazomicin susceptibility, and corresponding MIC_{50} and MIC_{90} values are summarized in Table 3. Among Klebsiella pneumoniae isolates (n = 45), 17 were susceptible and 28 resistant. Isolates harboring NDM alone showed partial susceptibility

(10/16), whereas those co-producing NDM and OXA-48 (16/17) were mostly resistant, with MIC_{90} >256 µg/ml. In contrast, isolates with NDM/KPC and KPC alone retained full susceptibility, demonstrating low MIC_{50}/MIC_{90} values (0.38–0.5 µg/mL). Among Escherichia coli isolates (n = 34), 18 were susceptible, 15 resistant, and one showed intermediate susceptibility.

NDM-positive E. coli isolates (n=27) demonstrated 12 susceptible, 14 resistant, and one intermediate result, with MIC_{50} of 3 µg/mL and MIC_{90} >256 µg/mL. Isolates co-producing NDM and OXA-48, or carrying single OXA-48 or NDM/KPC genes exhibited variable susceptibility. Among Pseudomonas aeruginosa isolates (n = 15), all carrying NDM, resistance was predominant (12/15), with MIC_{50} of 12 µg/mL and MIC_{90} >256 µg/ml. Among Enterobacter cloacae isolates (n = 6), five were susceptible and one was resistant, with MIC_{50} and MIC_{90} values of 0.75 µg/mL and 1 µg/mL, respectively.

All five isolates carrying NDM alone were susceptible, whereas the single isolate co-harboring NDM and OXA-48 was resistant, showing MIC values exceeding 256 µg/ ml. Overall, these findings indicate that Plazomicin retains potent activity against isolates harboring single carbapenemase genes, while co-production of multiple resistance determinants- especially NDM with OXA-48 markedly diminishes susceptibility and results in elevated MIC values.

Discussion:

This study provides a comprehensive evaluation of the in vitro activity of Plazomicin against 100 carbapenem-resistant Gram-negative bacilli (CR-GNB) isolated from clinical samples at a tertiary care hospital in western India. The isolates included Escherichia coli, Klebsiella pneumoniae, Enterobacter cloacae, and Pseudomonas aeruginosa. The findings highlight the potential of Plazomicin as a promising addition to the antimicrobial armamentarium in the era of escalating multidrug resistance (MDR). Plazomicin demonstrated superior overall activity (42% susceptibility) compared to conventional Aminoglycoside - Amikacin (18%), Gentamicin (33%), and Tobramycin (13.3%), suggesting its potential as an effective therapeutic alternative against MDR Enterobacterales and Pseudomonas spp., where older Aminoglycoside often fail. International studies have reported even higher susceptibility rates of Plazomicin globally, Gizam İnce et al. observed Plazomicin susceptibility in 71.7% of carbapenem-resistant isolates, markedly higher than Gentamicin (45%) and Amikacin (51.7–56.7%, per CLSI and EUCAST breakpoints) [17]. Fleischmann et al. and Castanheira et al. also demonstrated the superior in vitro activity of Plazomicin against MDR aerobic Gram-negative bacilli, even where other aminoglycoside were ineffective [18,19]. This difference is likely attributable to the high prevalence of 16S rRNA methyltransferases (RMTases) among carbapenem-resistant Enterobacterales (CRE). These enzymes confer high-level resistance to all Aminoglycoside, including Plazomicin [20]. Furthermore, co-carriage of multiple resistance determinants- particularly NDM with OXA-48 appears to substantially reduce Plazomicin efficacy in our setting.

In our isolates, blaNDM was the most frequent carbapenemase gene, followed by blaOXA-48, consistent with observations by Essam et al. [21]. Tawfick et al. also reported the predominance of NDM enzymes (68.9%) over OXA-48 (32.6%) and KPC (1.5%) [22]. These findings reinforce the global dominance of NDM-type carbapenemases, particularly in regions with high carbapenem use and antimicrobial pressure.

Correlation of resistance genotypes with Plazomicin activity revealed distinct patterns. Isolates carrying NDM alone showed 46% susceptibility, whereas those co-producing NDM and OXA-48 exhibited markedly reduced susceptibility (21.7%). Isolates with OXA-48 alone demonstrated 40% susceptibility, while those carrying KPC or NDM with KPC retained full susceptibility.

These results are consistent with international data. Essam et al. reported Plazomicin activity in 31.4% of isolates overall, including 21% among blaNDM-positive and 41% among blaOXA-48-positive strains [21]. Fleischmann et al. also showed reduced activity in blaNDM (35.7%) and blaOXA-48-like (50%) producers, but higher susceptibility among blaCTX-M (68.6%) and blaKPC (94.9%) isolates [18].

Our findings are similar to this trend, reduced susceptibility with NDM and OXA-48 co-producers but preserved activity in KPC producers. Interestingly, isolates carrying NDM alone in our study showed higher susceptibility than OXA-48 producers, possibly because a substantial proportion of NDM isolates (n=23) were also co-producers of OXA-48 with very low susceptibility, and the total number of NDM-only isolates (n=58) was far greater than OXA-48-only (n=10), affecting the apparent distribution. The reduced Plazomicin activity against NDM-positive isolates again reflects the co-expression of 16S rRNA methyltransferases (16S-RMTases) to likely confer high-level pan-Aminoglycoside resistance (HL-PAR). Castanheira et al. found overall $MIC_{50/90}$ = 0.25/128 µg/mL for carbapenem-resistant Enterobact eriaceae, with potent activity against blaKPC ($MIC_{50/90}$ = 0.25/2 µg/mL) and reduced

activity against blaOXA-48-like ($MIC_{50/90}$ = 0.25/16 µg/mL) [23]. Fleischmann et al. similarly reported Plazomicin activity against KPC producers ($MIC_{50/90}$ = 0.5/2 µg/mL) but uniform resistance among blaNDM-positive isolates (MIC_{90} >128 µg/mL) [18]. Gizem İnce et al. observed high resistance in carbapenem-resistant K. pneumoniae ($MIC_{50/90}$ = 0.5/>256 µg/mL), whereas E. coli blaCTX-M producers remained largely susceptible ($MIC_{50/90}$ = 2/4 µg/mL) [17]. For E. cloacae, our findings of excellent Plazomicin activity agree with Castanheira et al., who noted sustained efficacy even among carbapenem-resistant strains [23]. For P. aeruginosa, our results align with Castanheira et al. ($MIC_{50/90}$ = 4/16 µg/mL across 103 isolates), emphasizing Plazomicin's limited role against NDM-positive Pseudomonas [23]. The high resistance observed among NDM/OXA-48 co-producers may be because of additional mechanisms such as 16S-RMTase production and Aminoglycoside-modifying enzymes (AMEs). Several studies have linked high-level Plazomicin resistance (MIC ≥64 µg/mL) to RMTase activity. Hidalgo et al. associated blaNDM with rmtF, while Taylor et al. reported that 93.4% of RMTase-producing isolates co-carried blaNDM [24,25]. Similarly, Pargasam et al. found that 48% of NDM-producing E. coli and 35% of OXA-48-like K. pneumoniae harbored AMEs with RMTases, rendering them resistant to Plazomicin [26]. Our study did not directly assess RMTase genes, which remains a limitation.

Conclusion:

This study highlights the variable in vitro activity of Plazomicin against carbapenem-resistant Gram-negative bacilli isolated from a tertiary care cancer hospital in western India. Plazomicin demonstrated superior overall efficacy compared to conventional Aminoglycoside such as Amikacin, Gentamicin, and Tobramycin. However, its activity was markedly influenced by the underlying resistance genotypes.

The presence of blaNDM, particularly in combination with blaOXA-48 was associated with high-level resistance and elevated MIC values, whereas isolates harboring blaKPC or blaOXA-48 alone retained appreciable susceptibility. Excellent activity was observed against Enterobacter cloacae and selected Klebsiella pneumoniae isolates, while Pseudomonas aeruginosa showed limited response.

These findings underscore the importance of molecular characterization in predicting Plazomicin efficacy and guiding antimicrobial therapy in high-risk clinical settings. Although Plazomicin remains a valuable therapeutic option against selected multidrug-resistant Enterobacterales, the emergence of co-existing carbapenemases and 16S rRNA methyltransferases may limit its utility. Continued surveillance and larger clinical studies are warranted to define its role in managing infections caused by highly resistant pathogens.

References:

1. World Health Organization. WHO bacterial priority pathogens list, 2024
2. Nordmann P, Naas T, Poirel L. Global spread of Carbapenemase-producing Enterobacteriac eae. Emerg Infect Dis. 2011 Oct;17(10):1791-8. doi: 10.3201/eid1710.110655. PMID: 22000347; PMCID: PMC3310682.
3. Kraiem AG, Zorgani A, Elahmer O, Hammami A, Chaaben BM, Ghenghesh KS. New Delhi metallo-β-lactamase and OXA-48 carbapenemases in Gram-negative bacilli isolates in Libya. Libyan J Med. 2015 Aug 19;10(1):29206. doi: 10.3402/ljm.v10.29206. PMID: 26294290; PMCID: PMC4543448.
4. Li Q, Zhou X, Yang R, Shen X, Li G, Zhang C, Li P, Li S, Xie J, Yang Y. Carbapenem-resistant Gram-negative bacteria (CR-GNB) in ICUs: resistance genes, therapeutics, and prevention - a comprehensive review. Front Public Health. 2024 Mar 27;12:1376513. doi: 10.3389/fpubh.2024.1376513. PMID: 38601497; PMCID: PMC11004409.
5. Cassir N, Rolain JM, Brouqui P. A new strategy to fight antimicrobial resistance: the revival of old antibiotics. Frontiers in microbiology. 2014 Oct 20;5:551.
6. Vakulenko SB, Mobashery S. Versatility of aminoglycosides and prospects for their future. Clinical microbiology reviews. 2003 Jul;16(3):430-50.
7. Jouybari MA, Ahanjan M, Mirzaei B, Goli HR. Role of aminoglycoside-modifying enzymes and 16S rRNA methylase (ArmA) in resistance of Acinetobacter baumannii clinical isolates against aminoglycosides. Revista da Sociedade Brasileira de Medicina Tropical. 2021 Jan 29;54:e05992020.
8. Shaeer KM, Zmarlicka MT, Chahine EB, Piccicacco N, Cho JC. Plazomicin: a next-generation aminoglycoside. Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy. 2019 Jan;39(1):77-93.
9. Gür D, Hasdemir U, Çakar A, Çavuşoğlu İ, Çelik T, Aksu B, Plazomicin Study Group. Comparative in vitro activity of plazomicin and older aminoglyosides against Enterobacterales isolates; prevalence of aminoglycoside modifying enzymes and 16S rRNA methyltransferases. Diagnostic microbiology and infectious disease. 2020 Aug 1;97(4):115092.
10. Wagenlehner FM, Cloutier DJ, Komirenko AS, Cebrik DS, Krause KM, Keepers TR, Connolly LE, Miller LG, Friedland I, Dwyer JP. Once-daily plazomicin for complicated

urinary tract infections. New England Journal of Medicine. 2019 Feb 21;380(8):729-40.
11. Słabisz N, Leśnik P, Janc J, Fidut M, Bartoszewicz M, Dudek-Wicher R, Nawrot U. Evaluation of the in vitro susceptibility of clinical isolates of NDM-producing Klebsiella pneumoniae to new antibiotics included in a treatment regimen for infections. Frontiers in Microbiology. 2024 Apr 5; 15:1331628.
12. Clark JA, Burgess DS. Plazomicin: a new aminoglycoside in the fight against antimicrobial resistance. Therapeutic advances in infectious disease. 2020 Sep; 7:204993612 0952604.
13. Achaogen, Inc. A Study of Plazomicin Compared with Colistin in Patients with Infection Due to Carbapenem-Resistant Enterobacteriaceae (CRE) (CARE) [Internet]. Bethesda (MD): National Library of Medicine (US); 2013 Oct [cited 2025 Dec 9]. Identifier NCT01970371. Available from: clinicaltrial s.gov.
14. bioMérieux. VITEK® 2 – automated microbial identification and antibiotic susceptibility system. bioMérieux; 2025.
15. Clinical and Laboratory Standards Institute (CLSI). Performance Standards for Antimicrobial Susceptibility Testing. 34th ed. CLSI supplement M100. Wayne, PA: CLSI; 2024.
16. NG Biotech. NG-Test® CARBA 5 – rapid immunochromatographic assay for five carbapenemases. NG Biotech; Guipry, France; 2020.
17. Gizam ince et al. Comparison of in vitro activities of plazomicin and other aminoglycosides against clinical isolates of Klebsiella pneumoniae and Escherichia coli. J AntimicrobChemother 2021; 76: 3192–3196 doi:10.1093/jac/dkab331Advance Access publication 9 September 2021
18. Fleischmann WA, Greenwood-Quaintance KE, Patel R. In vitro activity of plazomicin compared to amikacin, gentamicin, and tobramycin against multidrug-resistant aerobic gram-negative bacilli. Antimicrobial agents and chemotherapy. 2020 Jan 27;64(2):10-128.
19. Castanheira M, Sader HS, Mendes RE, Jones RN. Activity of plazomicin tested against Enterobacterales isolates collected from US hospitals in 2016–2017: effect of different breakpoint criteria on susceptibility rates among aminoglycosides. Antimicrobial Agents and Chemotherapy. 2020 Apr 21;64(5):10-128.
20. Alfieri A, Di Franco S, Donatiello V, Maffei V, Fittipaldi C, Fiore M, Coppolino F, Sansone P, Pace MC, Passavanti MB. Plazomicin against multidrug-resistant bacteria: A scoping review. Life. 2022 Nov 22;12(12):1949.
21. Essam S, Nawar N, ElBashaar M, Soliman M, Abdelfattah M. In Vitro Activity of Plazomicin among Carbapenem-resistant Enterobacteriaceae. Open Access Macedonian Journal of Medical Sciences. 2021 Nov 25;9(A):1203-7.
22. Tawfick MM, Alshareef WA, Bendary HA, Elmahalawy H, Abdulall AK. The emergence of carbapenemasebla NDM genotype among carbapenem-resistant Enterobacteriaceae isolates from Egyptian cancer patients. Eur J Clin Microbiol Infect Dis. 2020;39(7):1251-9. https://doi.org/10.1007/ s10096-020-03839-2 PMid:32062725
23. Castanheira M, Davis AP, Mendes RE, Serio AW, Krause KM, Flamm RK. In Vitro Activity of Plazomicin against Gram-Negative and Gram-Positive Isolates Collected from U.S. Hospitals and Comparative Activities of Aminoglycosides against Carbapenem-Resistant Enterobacteriaceae and Isolates Carrying Carbapenemase Genes. Antimicrob Agents Chemother. 2018 Jul 27;62(8):e00313-18. doi: 10.1128/AAC.0031 3-18. PMID: 29866862; PMCID: PMC61 05795.
24. Hidalgo L, Hopkins KL, Gutierrez B, Ovejero CM, Shukla S, Douthwaite S, Prasad KN, Woodford N, Gonzalez-Zorn B. Association of the novel aminoglycoside resistance determinant RmtF with NDM carbapenemase in Enterobacteriaceae isolated in India and the UK. J AntimicrobChemother. 2013 Jul;68(7):1543-50. doi: 10.1093/jac/dkt078. Epub 2013 Apr 11. PMID: 23580560.
25. Taylor E, Sriskandan S, Woodford N, Hopkins KL. High prevalence of 16S rRNA methyltransferases among carbapenemase-producing Enterobacteriaceae in the UK and Ireland. International Journal of Antimicrobial Agents. 2018 Aug 1;52(2):278-82.
26. Pragasam AK, Jennifer SL, Solaimalai D, Muthuirulandi Sethuvel DP, Rachel T, Elangovan D, Vasudevan K, Gunasekaran K, Veeraraghavan B. Expected plazomicin susceptibility in India based on the prevailing aminoglycoside resistance mechanisms in Gram-negative organisms derived from whole-genome sequencing. Indian J Med Microbiol. 2020 Jul-Dec;38(3 & 4):313-318. doi: 10.41 03/ijmm.IJMM_20_384. PMID: 33154241.