GSC Advanced Research and Reviews

eISSN: 2582-4597 CODEN (USA): GARRC2

Cross Ref DOI: 10.30574/gscarr

Journal homepage: https://gsconlinepress.com/journals/gscarr/

(RESEARCH ARTICLE)

Check for updates

# Current epidemiology and antimicrobial resistance phenotype profile of *Klebsiella pneumoniae* at the Avicenna Military Hospital of Marrakech

Hakim Mechbal *, Yousra Boughalem, Youssef El Kamouni, Lamiae Arsalane and Said Zouhair

*Department of Bacteriology and Virology, Avicenna Military Hospital, Faculty of Medicine and Pharmacy, Cadi Ayyad University, Marrakech, Morocco.*

GSC Advanced Research and Reviews, 2025, 25(01), 046-050

Publication history: Received on 01 July 2025; revised on 01 October 2025; accepted on 03 October 2025

Article DOI: https://doi.org/10.30574/gscarr.2025.25.1.0235

## Abstract

*Klebsiella Pneumoniae* is a gram-negative bacillus found ubiquitously in nature and colonizing nasopharynx and gastrointestinal tract in humans. It has been confirmed as an important cause of urinary tract, respiratory tract and soft tissues infections, as well as an important cause of bacteriemia. This bacterium has developed multiple mechanisms of resistance to antimicrobials resulting in different phenotypes that can differ slightly or more substantially from one geographical location to another. This study analyzes the epidemiological characteristics of *KP*, and the different antimicrobial resistance phenotypes found in the Avicenna Military Hospital of Marrakech, Morocco.

**Keywords:** *Klebsiella*; Epidemiology; Antimicrobials; Resistance; ESBL

## 1. Introduction

*Klebsiella pneumoniae (KP)* is a gram-negative bacillus belonging to the *Enterobacteriaceae* group. It is found ubiquitously in the environment and in humans, it can be found as a saprophytical bacteria in the gastrointestinal tract and in the nasopharynx [1,2]. *Klebsiella pneumoniae* was known as an important cause of infection in immunodepressed, diabetic and alcoholic patients, but in recent years it has become a cause of severe community-acquired and a major cause of nosocomial infections [2]. This bacterium is usually isolated in urinary tract infections, pneumonia, bacteriemia and soft tissues infections such as hepatic abscess [1,3], and usually found in urinary samples, hemocultures and respiratory tract samples [1,4,5]. A relevant fact concluded is its importance as a carrier of antimicrobial resistance between the same species and other gram-negative bacterium species [6]. This fact and the increasing antimicrobial resistance that has acquired made it a challenge for the control of the spreading of multiresistant bacterium, and for this reason, the management and treatment of infections caused by *KP* must be done meticulously to prevent further spreading and additional acquisition of antimicrobial resistances [3,7].

This article has two main objectives: to carry an epidemiological study of infections caused by *Klebsiella Pneumoniae*, and the description of antimicrobial resistance phenotypes found in patients with these infections in the Avicenna Military Hospital of Marrakech.

## 2. Materials and methods

We are facing a retrospective and descriptive study of the epidemiological aspects of infections caused by *Klebsiella Pneumoniae* and characterization of antimicrobial resistance phenotype profiles of this bacterial species.

* Corresponding author: Hakim Mechbal

The study has been done in the Laboratory of Bacteriology of the Avicenna Military Hospital of Marrakech and the identification and antibiograms have been obtained using the automated system BD Phoenix M50™ .

The study includes 262 samples obtained from males and females of different ages, from different departments, and outpatients of the Avicenna Military Hospital of Marrakech, from January 2022 until March 2025.

The statistical analysis has been elaborated using Microsoft Excell v 2506.

This study has been conducted following the Avicenna Military Hospital of Marrakech's ethical guidelines and directives, and in accordance with the Law of Data Protection.

## 3. Results

*Klebsiella Pneumoniae* has been isolated in different samples from 59% males (n= 154) and 41% females (n= 108).

Patients whose samples were analyzed came from the following departments: 43% Outpatients (n= 155), 27.1% Intensive Care Unit (n= 71), 7.6% Emergency Unit (n= 20), 4.6% Urology department (n= 12), 3.1% Internal Medicine department (n= 8), 2.7% General Surgery department (n= 7), 1.9% Nephrology department (n= 5), 1.9% Clinic Hematology department (n= 5), 1.1% Cardiovascular Surgery department (n= 3), and 4.1% from other departments (n= 11).

As for the type of samples, 64.9% (n= 170) were urinary samples.19.9% (n= 52) were respiratory samples, from which 5.3% (n= 14) were sputum, 4.6% (n= 12) were Bronchial Aspiration, 9.5% (n= 25) were Bronchoalveolar Lavage (BAL) and 0.4% (n=1) were Protected Specimen Brush (PSB). 4.9% (n= 13) of the samples were Pus of different sources, 2.7% (n=7) were catheters, 2.3% (n= 6) were blood cultures and 0.4% (n=2) were other samples.

The percentages of antimicrobial resistance of *KP* are shown in the following figure

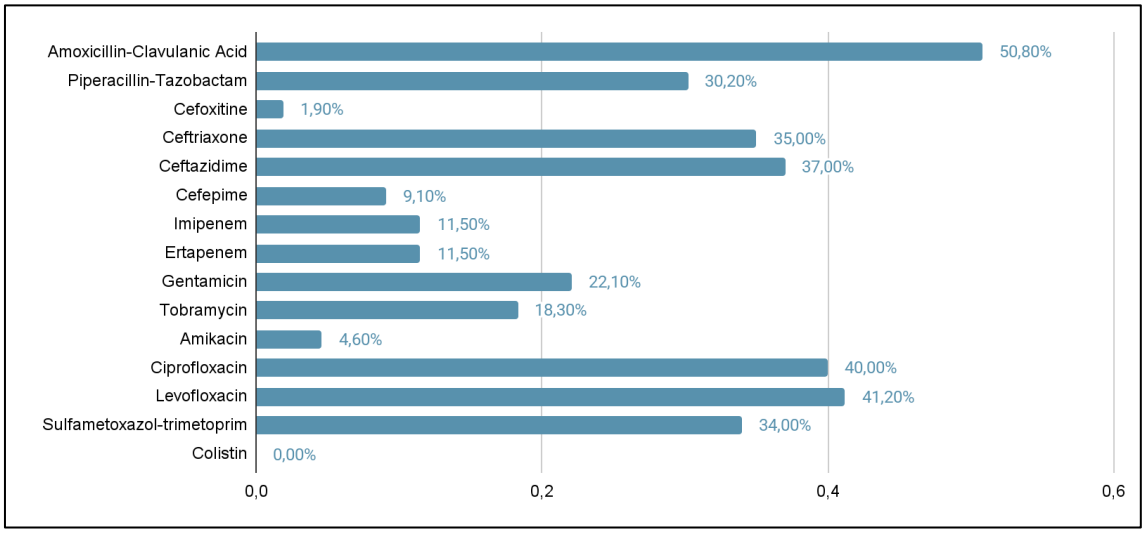


**Figure 1** Percentage of resistance to antimicrobials of *KP*

The phenotype profiles found in this study are 37% wild-type strains (n=97), 31.7% ESBL producer strains (n=31.7) and 11.5% Carbapenem-resistant strains (n=30).

## 4. Discussion

The results found in this study show the epidemiological and antimicrobial resistance characteristics of *Klebsiella Pneumoniae* at the Avicenna Military Hospital of Marrakech.

In our study, *KP* infections were not just a challenge in immunodepressed and patients with other comorbidities anymore, they rather became a cause of community-acquired infections as reflected by the 43% of samples obtained by outpatients, coinciding with the conclusion of other studies [9, 10].

The Intensive Care Unit was the main intra-hospital department where *KP* was isolated. This can be explained by the frequency of findings of this bacterium in respiratory devices such as mechanical ventilation devices as concluded by many studies [11, 16, 17] and in urethral catheters [12, 13]. The Emergency Unit followed the ICU as the second intra-hospital department affected by these infections and this fact can be explained as previously. Urology was the third, and the frequent finding of *KP* coincides with the high frequency of its findings in urine samples, and may be due to the proximity of the urinary tract to the main colonization site of this bacterium which is the gastrointestinal mucosa [1, 3], entering the urinary tract through the urethra and specially with procedures as urethral catheterization [12, 13] if the rules of asepsis are not respected carefully.

As for the type of samples where *KP* was isolated, it was found that more than 60% were urinary samples, which confirms this site as the most common site of infection by this bacterium, as exposed by Podshun and Ullman [1]. The next group was the respiratory tract samples as sputum, bronchial aspiration, BAL and PSB and coincide with other studies that confirm the nasopharynx as an important colonization site, in addition to the use of colonized respiratory devices that can be a cause of infection by *KP*. This bacterium has been also found in pus of different tissues and locations and is in accordance with conclusions from other studies that establish *KP* as a common cause of soft tissues infections [1, 3]. Finally, blood cultures and catheter samples were not spared. Both types of samples were also found infected by *KP*, which can be explained by the pathogenicity and virulence of this bacterium, being an important cause of bacteriemia, and its capacity to produce biofilms in devices such as catheters of different types [14, 15].

*Klebsiella Pneumoniae* is naturally resistant to aminopenicillins, carboxypenicillins and ureidopenicillins, remaining sensitive to their combination with beta-lactamase inhibitors such as Clavulanic Acid and Tazobactam [8]. However, it was found that half of the isolated strains developed a resistance to these combinations, being the Amoxicillin-Clavulanic Acid the most affected from resistance acquisition. Piperacillin-Tazobactam was affected as well and remains the most "effective" in vitro against *KP* from the group of Penicillins and inhibitors of beta-lactamases.

As for the cephalosporines, a third of the strains developed resistance to 3rd generation cephalosporines such as Ceftriaxone and Ceftazidime, a lower proportion in comparison with the systematic review and meta-analysis published by Masoumeh Beig et al. [7] and can be understood as a difference between strains belonging to different geographical locations. The cephalosporines less affected by the acquisition of resistance were Cefoxitine (2nd generation cephalosporine) and Cefepime (4th generation), due to unclear mechanisms that must be analyzed in further studies.

Carbapenems were less affected by the development of resistance. Only 11% of the strains of *KP* were resistant to Imipenem and Ertapenem. The fact that slightly more strains were resistant to this group than to Cefoxitin suggests the possibility that the mechanism of resistance may be the production of ESBL that spares this antimicrobial, in addition to another mechanism of decreased permeability or efflux for carbapenems and not necessarily the production of carbapenemases.

*Klebsiella Pneumoniae* strains that were isolated showed a considerable resistance to Aminosides as well as some differences between them. Nevertheless, the rate of resistance was found to be lower than expected if we take as reference the publication from of Masoumeh Beig et al. [7]. A similar profile of resistance was found for Gentamicin and Tobramycin, with around 20% of the strains being resistant. However, Amikacin was the least affected, with a resistance of less than 5%.

Fluroquinolones were found to be very affected by the development of resistance by *KP*, with almost no difference between Ciprofloxacin and Levofloxacin and a rate of resistance of around 40%, a very similar result to the previously cited review [7].

Sulfametozaxol-trimethoprim was also found to be affected by the development of resistance, with around 30% of the strains resistant to it and with an unclear mechanism that could be analyzed in further studies with the help of biomolecular and gene testing.

Colistin is the antimicrobial that wasn't affected, with none of the strain resistant to it, showing why it is used as the last resort for the treatment of multi-resistant *KP* in Morocco. However, this fact shouldn't encourage its use indiscriminately as it could cause the selection of mutant and resistant strains and their spreading, causing a major problem for the treatment and control of multi-resistant strains.

Finally, it was found in this study that 31.7% of the strains that were isolated were ESBL producers, 11.5% were carbapenem-resistant and 37% were wild-type strains. The remaining strains developed casual antimicrobials resistance but weren't categorized in any of the previous profiles. This fact confirms the capacity of acquisition of antimicrobial resistance for different groups and through different mechanisms that can be analyzed in further studies.

## 5. Conclusion

*Klebsiella Pneumoniae* has become a cause of community acquired infections. The hospital departments where *KP* has mostly been isolated are the ICU, Emergency Unit and the Urology department. The most frequent samples where this bacterium was isolated were urinary samples, respiratory samples, pus from different sources, blood cultures and catheters. The antimicrobial resistance phenotype profiles that were found were: 37% wild-type strains, 31.7% ESBL producers' strains and 11.5% carbapenem-resistant strains. The antimicrobials most affected by the development of resistance were Amoxicillin-Clavulanic acid, Piperacillin-Tazobactam, 3rd generation cephalosporines, Fluroquinolones and Sulfamethoxazole-trimethoprim. The antimicrobial that was spared by the development of resistance was Colistin with none of the strains being resistant to it. Mechanisms of resistance acquisition will be analyzed with biomolecular and gene testing in further studies.

## Compliance with ethical standards

### *Disclosure of conflict of interest*

The authors declare that there are no conflicts of interest.

### *Funding information*

The author(s) received no specific grant from any funding agency.

### *Statement of informed consent*

Informed consent was obtained from all individual participants included in the study.

## References

[1] Podschun R, Ullmann U. Klebsiella spp. as nosocomial pathogens: epidemiology, taxonomy, typing methods, and pathogenicity factors. Clin Microbiol Rev. 1998 Oct;11(4):589–603. doi: 10.1128/CMR.11.4.589. PMID: 9767057; PMCID: PMC88898.

[2] Wang G, Zhao G, Chao X, Xie L, Wang H. The characteristic of virulence, biofilm and antibiotic resistance of Klebsiella pneumoniae. Int J Environ Res Public Health. 2020 Aug 28;17(17):6278. doi: 10.3390/ijerph17176278. PMID: 32872324; PMCID: PMC7503635.

[3] García-Cobos S, Oteo-Iglesias J, Pérez-Vázquez M. Hypervirulent Klebsiella pneumoniae: epidemiology outside Asian countries, antibiotic resistance association, methods of detection and clinical management. Enferm Infecc Microbiol Clin (Engl Ed). 2025 Feb;43(2):102–9. doi: 10.1016/j.eimce.2024.12.008. PMID: 39914938.

[4] Ullmann U. Bakterielle Infektionserreger bei hospitalisierten Patienten [Bacterial infection agents in hospitalized patients]. Zentralbl Bakteriol Mikrobiol Hyg B Umwelthyg Krankenhaushyg Arbeitshyg Prav Med. 1986 Dec;183(2–3):103–13. German. PMID: 3107262.

[5] Orrett FA. Nosocomial infections in an intensive care unit in a private hospital. West Indian Med J. 2002 Mar;51(1):21–4. PMID: 12089869.

[6] Wyres KL, Holt KE. Klebsiella pneumoniae as a key trafficker of drug resistance genes from environmental to clinically important bacteria. Curr Opin Microbiol. 2018 Oct;45:131–9. doi: 10.1016/j.mib.2018.04.004. Epub 2018 May 1. PMID: 29723841.

[7] Beig M, Aghamohammad S, Majidzadeh N, Asforooshani MK, Rezaie N, Abed S, Khiavi EHG, Sholeh M. Antibiotic resistance rates in hypervirulent Klebsiella pneumoniae strains: a systematic review and meta-analysis. J Glob Antimicrob Resist. 2024 Sep;38:376–88. doi: 10.1016/j.jgar.2024.06.018. Epub 2024 Jul 26. PMID: 39069234.

[8] CASFM/EUCAST. Recommandations 2024 V1.0 – Juin. Paris: Société Française de Microbiologie; 2024. Available from: https://www.sfm-microbiologie.org/wp-content/uploads/2024/06/CASFM2024_V1.0.pdf

[9] Martin RM, Bachman MA. Colonization, infection, and the accessory genome of Klebsiella pneumoniae. Front Cell Infect Microbiol. 2018 Jan 22;8:4. doi: 10.3389/fcimb.2018.00004. PMID: 29404282; PMCID: PMC5786545.

[10] Wyres KL, Lam MMC, Holt KE. Population genomics of Klebsiella pneumoniae. Nat Rev Microbiol. 2020 Jun;18(6):344–59. doi: 10.1038/s41579-019-0315-1. Epub 2020 Feb 13. PMID: 32055025.

[11] Thurlow CJ, Prabaker K, Lin MY, Lolans K, Weinstein RA, Hayden MK; Centers for Disease Control and Prevention Epicenters Program. Anatomic sites of patient colonization and environmental contamination with Klebsiella pneumoniae carbapenemase-producing Enterobacteriaceae at long-term acute care hospitals. Infect Control Hosp Epidemiol. 2013 Jan;34(1):56–61. doi: 10.1086/668783. Epub 2012 Nov 19. PMID: 23221193; PMCID: PMC8376186.

[12] Montgomerie JZ, Gilmore DS, Graham IE, Schick DG, Ashley MA, Morrow JW, Bruce SK. Klebsiella pneumoniae colonization in patients with spinal cord injury. Diagn Microbiol Infect Dis. 1987 Aug;7(4):229–35. doi: 10.1016/0732-8893(87)90137-4. PMID: 3315404.

[13] Clegg S, Murphy CN. Epidemiology and virulence of Klebsiella pneumoniae. Microbiol Spectr. 2016 Feb;4(1). doi: 10.1128/microbiolspec.UTI-0005-2012. PMID: 26999397.

[14] Schroll C, Barken KB, Krogfelt KA, Struve C. Role of type 1 and type 3 fimbriae in Klebsiella pneumoniae biofilm formation. BMC Microbiol. 2010 Jun 23;10:179. doi: 10.1186/1471-2180-10-179. PMID: 20573190; PMCID: PMC2911432.

[15] Magill SS, Edwards JR, Bamberg W, Beldavs ZG, Dumyati G, Kainer MA, et al. Multistate point-prevalence survey of health care-associated infections. N Engl J Med. 2014 Mar 27;370(13):1198–208. doi: 10.1056/NEJMoa1306801. Erratum in: N Engl J Med. 2022 Jun 16;386(24):2348. doi: 10.1056/NEJMx210023. PMID: 24670166; PMCID: PMC4648343.

[16] Kalanuria AA, Ziai W, Mirski M. Ventilator-associated pneumonia in the ICU. Crit Care. 2014 Mar 18;18(2):208. doi: 10.1186/cc13775. Erratum in: Crit Care. 2016 Jan 28;20:29. doi: 10.1186/s13054-016-1206-8. PMID: 25029020; PMCID: PMC4056625.

[17] Selina F, Rahman M, Hossain M, Uddin MM, Ahmed M, Rahman A, et al. Organisms associated with ventilator-associated pneumonia in intensive care units. J Bangladesh Soc Anaesthesiol. 2009;22(2):72–7.