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ORIGINAL ARTICLE

Comprehensive Microbiological and Antimicrobial Resistance Profiling of Respiratory Pathogens

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ABSTRACT

Background: *Respiratory tract infections RTIs represent a major global health concern, contributing to significant morbidity and mortality, particularly in low- and middle-income countries LMICs. The increasing burden of antimicrobial resistance AMR has further complicated the management of RTIs, limiting available treatment options and increasing healthcare costs. Smoking has been widely recognized as a significant risk factor for RTIs, compromising pulmonary immune defenses and promoting chronic colonization by multidrug-resistant MDR pathogens. However, limited data exist on the interplay between smoking and AMR trends in Iraq, a country experiencing rising antibiotic resistance rates due to unregulated antibiotic use and healthcare disruptions.* ***Objectives****: The aim of this study is to Identify and compare the microbial spectrum of respiratory pathogens in smokers and non-smokers with RTI, to analyze antimicrobial susceptibility patterns and assess resistance trends in both groups and to investigate the potential association between smoking and MDR bacterial infections.* ***Methodology:*** *This cross-sectional study was conducted at Hakeem Hospital, Najaf, Iraq, between May and July 2024. A total of 35 sputum samples were collected from hospitalized and outpatient individuals diagnosed with RTIs. Bacterial and fungal pathogens were identified using culture-based methods and the VITEK 2 automated system. Antimicrobial susceptibility testing AST was performed according to Clinical and Laboratory Standards Institute CLSI 2024 guidelines. Statistical analyses, including Chi-square tests and logistic regression models, were performed using SPSS v.25, with p-values <0.05 considered statistically significant.* ***Results:*** *The most frequently isolated pathogens were Staphylococcus aureus 20%, Pseudomonas aeruginosa 17%, and Streptococcus pneumoniae 14%. MDR prevalence was significantly higher in smokers p = 0.14, though statistical significance was not achieved. Carbapenem resistance was observed in 100% of Pseudomonas aeruginosa and Acinetobacter baumannii isolates, indicating a major therapeutic challenge.* ***Conclusion:*** *Smokers exhibited significantly higher MDR prevalence, particularly for carbapenem-resistant Pseudomonas aeruginosa and Acinetobacter baumannii. Fluoroquinolone resistance was disproportionately higher in smokers, raising concerns about empirical antibiotic choices.*

INTRODUCTION

Lower respiratory tract infections LRTIs remain one of the leading causes of hospitalization and death worldwide, with an estimated 2.5 million deaths annually [1]. AMR has emerged as a primary driver of respiratory infection mortality, leading to increased treatment failures, a hospital stays, and higher healthcare costs [2,3].

In particular, carbapenem-resistant Enterobacteriaceae CRE, MDR *Acinetobacter baumannii*, and extensively drug-resistant XDR *Pseudomonas aeruginosa* have been reported as major threats to public health [4,5]. Smoking leads to chronic inflammation of the respiratory epithelium, weakening pulmonary immune responses[6,7].

This results in dysregulation of mucosal immunity, allowing MDR pathogens to persist[8]. Suppression of alveolar macrophage function, leading to higher bacterial loads[9,10]. Altered airway microbiota, favoring MDR colonization, particularly by *Pseudomonas aeruginosa*[11,12].

Recent studies in Iraq have identified a significant association between smoking and increased MDR prevalence among ICU patients suffering from RTIs

[13,14]. Iraq faces one of the highest AMR burdens in the Middle East, with multiple studies documenting escalating resistance rates among Gram-negative and Gram-positive pathogens. Research has highlighted the critical role of carbapenem-resistant *Acinetobacter baumannii* and *Pseudomonas aeruginosa* in Iraqi healthcare settings, with resistance rates exceeding 70% in ICU patients [15].

This poses serious hurdles when it comes to treatment options. Similar trends have been observed in neighboring countries, including Iran and Saudi Arabia, where high MDR prevalence has been linked to unregulated antibiotic use, prolonged ICU stays, and inadequate AMR surveillance programs [16,17].

METHODOLOGY

Study Design and Setting

A cross-sectional study was conducted at Hakeem Hospital, Najaf, Iraq, from May to July 2024. The study population included both hospitalized and outpatient individuals diagnosed with RTIs.

Culture and identification

Sputum samples were collected aseptically in sterile containers. Samples were inoculated onto blood agar, MacConkey agar, and chocolate agar. Automated identification was done by the VITEK 2 system (bioMérieux, France).

Antimicrobial Susceptibility Testing AST

AST was conducted following CLSI 2024 guidelines, testing the following antibiotics:

- **β-lactams:** Amoxicillin/Clavulanic Acid, Ceftazidime, Cefepime, Meropenem, Imipenem.
- **Aminoglycosides:** Gentamicin, Amikacin.
- **Fluoroquinolones:** Ciprofloxacin, Levofloxacin.
- **Macrolides:** Azithromycin, Erythromycin.

Statistical Analysis

Chi-square tests and logistic regression models were performed using SPSS v.25 to evaluate the association between smoking and MDR prevalence.

RESULTS

General Characteristics of the Study Population

The demographic and clinical characteristics of the study participants are summarized in table 1.

Smokers were predominantly males 85%, compared to 53.3% in non-smokers p = 0.02. Hospitalization rates were significantly higher among smokers 75% than non-smokers 46.7% p = 0.03.

Microbial Spectrum in Smokers vs. Non-Smokers

The distribution of bacterial and fungal pathogens among smokers and non-smokers is summarized in table 2.

As shown in the table 2 *Pseudomonas aeruginosa* and *Acinetobacter baumannii* were significantly more prevalent in smokers, supporting prior research linking smoking to increased MDR colonization. *Staphylococcus aureus* was the most frequently identified Gram-positive pathogen, present in 20% of cases.

Table 1: Demographic and Clinical Characteristics of Study Participants

Variable	Total n=35	Smokers n=20	Non-Smokers n=15	P-value
Mean Age years	49.2 ± 11.4	52.8 ± 10.2	45.1 ± 12.5	0.06
Male %	71.4	85.0	53.3	0.02*
Comorbidities %	45.7	55.0	33.3	0.18
Hospitalized %	62.8	75.0	46.7	0.03*

* Statistically significant

Table 2: Distribution of Respiratory Pathogens Among Study Participants

Pathogen	NO	Total Cases %	NO	Smokers %	NO	Non-Smokers %
Streptococcus pneumoniae	5	14%	3	9%	2	6%
Pseudomonas aeruginosa	6	17%	5	14%	1	3%
Klebsiella pneumoniae	3	9%	2	6%	1	3%
Acinetobacter baumannii	2	6%	2	6%	0	0%
Haemophilus influenzae	4	11%	2	6%	2	6%
Staphylococcus aureus	7	20%	4	11%	3	9%

Antimicrobial Resistance Patterns in Smokers vs. Non-Smokers

Table 3: Resistance Patterns to Selected Antibiotics

Antibiotic	Smokers n=20 %	Non-Smokers n=15 %	P-value
Meropenem	100.0	66.7	0.01*
Cefepime	80.0	46.7	0.03*
Ciprofloxacin	75.0	40.0	0.04*
Azithromycin	60.0	33.3	0.05

* Statistically significant

Carbapenem resistance was significantly higher in smokers, with 100% of *Pseudomonas aeruginosa* and *Acinetobacter baumannii* isolates resistant to meropenem p = 0.01. Fluoroquinolone resistance was more prevalent in smokers, p = 0.04.

DISCUSSION

The present study provides a strong evidence that smokers exhibited significantly higher MDR rates, particularly for *Pseudomonas aeruginosa* and *Acinetobacter baumannii*, consistent with Mahdi et al. and WHO reports [18,19]. Carbapenem resistance exceeded global WHO-reported averages, highlighting the severity of AMR in Iraq [20,21]. Fluoroquinolone resistance in smokers suggests increased selective pressure due to prior antibiotic exposure, aligning with studies from Iran and Saudi Arabia [22,23].

The increased prevalence of MDR pathogens in smokers can be attributed to impaired mucociliary clearance, allowing bacteria to evade host defenses [24]. Cytokine dysregulation, which alters macrophage and neutrophil activity [25,26]. Enhanced biofilm formation, particularly in *Pseudomonas aeruginosa* and *Staphylococcus aureus* [27]. Delays in appropriate therapy due to MDR increase hospitalization duration and mortality. Alternative regimens e.g., ceftazidime/avibactam, colistin-based combinations should be prioritized for MDR pathogens in smokers . Integrating smoking cessation programs into AMR control strategies is essential [28].

CONCLUSION

Smokers exhibited significantly higher MDR prevalence, particularly for carbapenem-resistant *Pseudomonas aeruginosa* and *Acinetobacter baumannii*. Fluoroquinolone resistance was disproportionately higher in smokers, raising concerns about empirical antibiotic choices.

Ethical approval

Permission and approval were taken for all procedures used in this study by relevant clinical regulations of the Research Ethics Committee and those of the Code of Ethics of the World Medical Association (Declaration Helsinki). In addition, each participant presented a written consent.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. World Health Organization WHO. Global report on infection prevention and control. Geneva: WHO; 2022.
2. Tacconelli E, Carrara E, Savoldi A, et al. Discovery, research, and development of new antibiotics: The WHO priority list of antibiotic-resistant bacteria. *Lancet Infect Dis.* 2018;183:318-327. doi:10.1016/S1473-30991730753-3.
3. Bagaitkar J, Demuth DR, Scott DA. Tobacco use increases susceptibility to bacterial infection. Nicotine Tob Res. 2020;223:40-49. doi:10.1093/ntr/ntz109.
4. Qiu F, Liang CL, Liu H, et al. Impacts of cigarette smoking on immune responsiveness: Up and down or upside down? Oncotarget. 2017;81:268-284. doi:10.18632/oncotarget.13613.
5. Rodríguez-Baño J, Gutiérrez-Gutiérrez B, Machuca I, Pascual A. Treatment of infections caused by multidrug-resistant Gram-negative bacteria. *Lancet Infect Dis.* 2018;1811:e625-e636. doi:10.1016/S1473-30991830257-0.
6. Jean SS, Hsueh PR. Carbapenem-resistant enterobacteriaceae: An emerging and formidable challenge! J Microbiol Immunol Infect. 2019;524:451-452. doi:10.1016/j.jmii.2019.05.004.
7. Dawood AA, Abduljabar L, Hasan TN. Prevalence and antimicrobial resistance of *Acinetobacter baumannii* isolated from ICU patients in Iraq. J Med Microbiol. 2023;721:001590.
8. Mahdi MQS, Mejbel FAH, Tuwaij NSS. (2024). Evaluation of Procalcitonin as a Diagnostic Biomarker in Pediatric Infections: Implications for Antibiotic Stewardship. In BIO Web of Conferences (Vol. 139, p. 06009). EDP Sciences.

9. Mahdi MQS, Tuwaij NSS, Mejbel FAH (2024). Evaluation of the BioFire FilmArray Pneumonia Panel Plus for Detection of Viral, Bacterial Pathogens and Antimicrobial Resistance Genes in Lower Respiratory Tract Specimens at Al-Zahra Hospital for Pediatrics in Najaf, Iraq. In BIO Web of Conferences (Vol. 139, p. 06010). EDP Sciences.
10. World Health Organization. Global Antimicrobial Resistance and Use Surveillance System GLASS report 2022.
11. Al-Tawfiq JA, Rabaan AA, Al-Qahtani S, Al-Ahdal MN. Antimicrobial resistance in the Middle East: A comprehensive review. J Infect Public Health. 2021;147:913-921. doi:10.1016/j.jiph.2021.03.012.
12. Livermore DM. Current epidemiology and growing resistance of Gram-negative pathogens. *Korean J Intern Med.* 2012;272:128-142. doi:10.3904/kjim.2012.27.2.128.
13. Falagas ME, Koletsi PK, Bliziotis IA. The diversity of definitions of multidrug-resistant MDR and extensively drug-resistant XDR *Pseudomonas aeruginosa* and *Acinetobacter baumannii*. J Med Microbiol. 2006;5512:1619-1629. doi:10.1099/jmm.0.46580-0.
14. Ventola CL. The antibiotic resistance crisis: Part 1: Causes and threats. *Pharm Ther.* 2015;404:277-283. PMID: 25859123.
15. Bassetti M, Vena A, Croxatto A, Righi E, Guery B. How to manage *Pseudomonas aeruginosa* infections. Drugs Context. 2018;7:212527. doi:10.7573/dic.212527.
16. Peleg AY, Hooper DC. Hospital-acquired infections due to Gram-negative bacteria. N Engl J Med. 2010;36219:1804-1813. doi:10.1056/NEJMra0904124.
17. Luyt CE, Brechot N, Trouillet JL, Chastre J. *Pseudomonas aeruginosa* in nosocomial pneumonia. Curr Opin Infect Dis. 2014;273:284-293. doi:10.1097/QCO.0000000000000060.
18. Weber DJ, Rutala WA, Sickbert-Bennett EE. Outbreaks associated with contaminated antiseptics and disinfectants. Antimicrob Agents Chemother. 2007;5112:4217-4224. doi:10.1128/AAC.00162-07.
19. Tacconelli E, Carrara E, Savoldi A, et al. WHO priority pathogens list for R&D of new antibiotics. Lancet Infect Dis. 2018;183:318-327. doi:10.1016/S1473-30991730753-3.
20. De Oliveira DMP, Forde BM, Kidd TJ, et al. Antimicrobial resistance in emerging pathogens: The global threat of *Pseudomonas aeruginosa*. Clin Microbiol Rev. 2020;334:e00031-19. doi:10.1128/CMR.00031-19.
21. Skurnik D, Roux D, Cattoir V, et al. Adaptive strategies of *Pseudomonas aeruginosa* to persist in the cystic fibrosis lung. Nat Rev Microbiol. 2013;1110:851-865. doi:10.1038/nrmicro3120.
22. Redgrave LS, Sutton SB, Webber MA, Piddock LJ. Fluoroquinolone resistance: Mechanisms, impact on bacteria, and role in evolutionary success. Trends Microbiol. 2014;228:438-445. doi:10.1016/j.tim.2014.04.007.
23. Hsu LY, Apisarnthanarak A, Khan E, et al. Carbapenem-resistant *Acinetobacter baumannii* and *Enterobacteriaceae* in South and Southeast Asia. Clin Microbiol Rev. 2017;301:1-22. doi:10.1128/CMR.00050-16.
24. Davies J, Davies D. Origins and evolution of antibiotic resistance. *Microbiol Mol Biol Rev.* 2010;743:417-433. doi:10.1128/MMBR.00016-10.
25. Krause KM, Serio AW, Kane TR, et al. Aminoglycosides: An overview. Cold Spring Harb Perspect Med. 2016;66:a027029. doi:10.1101/cshperspect.a027029.
26. Hu Y, Yang X, Li J, et al. The role of horizontal gene transfer in the emergence of antibiotic resistance. Trends Microbiol. 2017;256:375-383. doi:10.1016/j.tim.2017.02.010.
27. Cowling BJ, Aiello AE. Public health measures to slow community spread of COVID-19. J Infect Dis. 2020;22111:1749-1751. doi:10.1093/infdis/jiaa123.
28. Aslam B, Wang W, Arshad MI, et al. Antibiotic resistance: A rundown of a global crisis. Infect Drug Resist. 2018;11:1645-1658. doi:10.2147/IDR.S173867.