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Multidrug Resistance Profiling of Some Bacterial Pathogens from Mobile Phones of Health Workers and Surfaces of Kwara State University (KWASU) Health Centre

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Abstract

Multidrug resistance (MDR) is a major public health challenge, and the direct and indirect transmission of these resistant pathogens can occur via healthcare centre surfaces and the phones of healthcare personnel. This study aimed to investigate the multidrug resistance profile of pathogenic bacteria present on the phones of health officers and surfaces of the KWASU health centre, Malete. A total of 40 swab samples were obtained from phones and different surfaces in the centre, and cultured on selective media to obtain Staphylococcus aureus, Pseudomonas aeruginosa, Klebsiella pneumoniae and Escherichia coli with a prevalence of 100%, 50%, 50% and 65% prevalence, respectively. Antibiotic susceptibility testing revealed widespread MDR among the isolates. Staphylococcus aureus exhibited 100% MDR, with complete resistance to pefloxacin, cefuroxime, amoxicillin, erythromycin, and azithromycin, and reduced susceptibility (15-24%) to gentamicin, ciprofloxacin, ceftriaxone (Rocephin), and levofloxacin. Klebsiella pneumoniae also showed 100% MDR, displaying total resistance to augmentin, pefloxacin, and ceftriaxone, and minimal susceptibility (0-24%) to other agents. Pseudomonas aeruginosa was completely resistant to augmentin, streptomycin, cefuroxime, ceporex, and ceftriaxone, indicating moderate to high MDR prevalence among its isolates. Escherichia coli demonstrated high MDR prevalence, with partial resistance to augmentin (92.3%) and cefuroxime (69.3%), and complete resistance to ceftazidime, ciprofloxacin, ceporex, ceftriaxone, and streptomycin. The detection of MDR pathogens on these phones and surfaces calls for stricter disinfection practices, phone hygiene and routine antimicrobial surveillance practices.

Keywords: antibiotic sensitivity, multi-drug resistance, hospital-acquired infections, mobile phones, healthcare centre surfaces.

INTRODUCTION

The emergence of bacterial pathogens' resistance to more than 3 classes of antibiotics in the health sector is a great challenge in public health (Salam *et al.*, 2023). Bacteria such as methicillin-resistant *Staphylococcus aureus* (MRSA), extended-spectrum beta-lactamase (ESBL)producing *Klebsiella pneumoniae* and *Escherichia coli*, carbapenem-resistant *Pseudomonas aeruginosa* and *Acinetobacter baumannii* are important examples of these multidrug-resistant pathogens (Bai *et al.*, 2024). They have been able to develop multidrug resistance via horizontal gene transfer, genetic and physiological mutations due to inappropriate use of antibacterial agents in the treatment of infections (Belay *et al.*, 2024). The exposure of patients and non-patients in the hospital environment to multidrug-resistant bacterial pathogens occurs via direct and indirect exposures to fomites such as medical devices,

surfaces of hospital furniture, like bed rails, door handles, tables and chairs, bed surfaces, drip stands and even phones of health care workers (Stephens *et al.*, 2019).

Mobile phones have become an indispensable tool for healthcare workers, used for communication, accessing medical information, and patient management. Unlike other medical equipment, which undergoes regular sterilisation, mobile phones are frequently handled but rarely disinfected, creating an environment conducive for microbial survival and transmission (Sharma *et al.*, 2022). Studies have shown that mobile phones are handled by healthcare workers multiple times per hour, often in between patient interactions, increasing the risk of cross-contamination (Ryabinina *et al.*, 2024). This frequent usage, coupled with environmental factors and poor hand hygiene compliance, contributes to the

spread of pathogens from mobile phones to healthcare workers' hands, hospital surfaces, and eventually to patients (Zenbaba *et al.*, 2023). Quite a number of studies have implicated phones of healthcare providers as an important vehicle for the transmission of nosocomial infections, reporting the isolation of *Staphylococcus aureus*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, *Escherichia coli*, etc, with a number of these phones' contaminants being resistant strains (Sapkota *et al.*, 2020 and Bissong and Moukou, 2022). A study in a teaching hospital's radiology department found that 73.3% of mobile phones were contaminated with bacteria such as *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Escherichia coli* before work began, and increased to 93.3% after patient contact (Eze *et al.*, 2022).

Frequently touched surfaces in the hospital have also been a means of transmitting infectious pathogens from the hospital environment to both healthy and unhealthy individuals visiting these health centres, thereby contributing to the spread of hospital-acquired infections (Cruz-López *et al.*, 2023). Various studies have highlighted the prevalence of multidrug-resistant bacterial pathogens in healthcare facilities, emphasising the role of contaminated surfaces in the spread of these pathogens (Chia *et al.*, 2020; Puzi *et al.*, 2022). Among the most frequently isolated multidrug organisms in healthcare settings are Methicillin-resistant *Staphylococcus aureus* (MRSA), which is associated with skin infections, pneumonia, and bloodstream infections, and Vancomycin-resistant *Enterococcus* (VRE), a leading cause of hospital-acquired bacteremia and urinary tract infections. Additionally, Carbapenem-resistant Enterobacteriaceae (CRE) and extended-spectrum beta-lactamase (ESBL)producing bacteria, including *Klebsiella pneumoniae* and *Escherichia coli*, have also been isolated (Adenipekun *et al.*, 2022). Other frequently isolated pathogens include *Pseudomonas aeruginosa*, which thrives in moist hospital environments and causes severe infections in immunocompromised patients, and *Acinetobacter baumannii*, notorious for its ability to survive on surfaces for prolonged periods and resist multiple antibiotics (Adenipekun *et al.*, 2022).

This study aimed at investigating the presence and resistance profile of multidrug-resistant resistant bacterial pathogens on the phones of healthcare workers and different surfaces at the KWASU health centre.

MATERIALS AND METHODS

Study Area and Sampling: The study was a cross-sectional design and performed at the Kwara State University (KWASU) health centre, Malete, located in the Moro Local Government Area of Kwara State, Nigeria (Figure 1). The health centre services both staff and students on campus and residents of the neighbouring community.

Sample Collection: Sterile swab sticks dipped in sterile nutrient broth were used aseptically to obtain samples from 15 mobile phones of health workers, 5 doorknobs, 5 drip stands, 5 bedside drawers, 5 wall surfaces and 5 bed rails from the Kwara State University (KWASU) health centre. The samples were transported immediately to the Department of Microbiology, KWASU laboratory for microbial analysis.

Isolation and Identification of Bacteria: Eosin Methylene Blue (EMB) agar, MacConkey agar, Mannitol Salt Agar (MSA), and Cetrimide agar were prepared according to the manufacturer's instructions, sterilised, and aseptically dispensed into sterile Petri dishes. After solidification, each agar plate was inoculated with the test samples using a sterile swab stick by streaking the surface of the medium. The inoculated plates were then incubated in an inverted position at 37°C for 24 hours to allow for microbial growth. The isolates obtained were identified based on their distinct morphological appearance on the selective mediaplates and reaction to confirmatory biochemical tests as described by Ferdaous *et al* (2021).

Antibiotic Sensitivity Testing and Determination of MDR: The Kirby-Bauer disc diffusion method, as described by Agbabiaka *et al.* (2020), was used to determine the resistance profile of the isolates on Mueller-Hinton agar. Conventional antibiotic discs commercially obtained from Optun Diagnostics Nig. LTD containing Ofloxacin 10µg, Augmentin 30µg; Ceftriaxone 30µg; Peflaxine 10µg; Ceftazidime 30µg; Gentamicin 10 µg; Ceporex 10µg; Ciprofloxacin 10µg; Streptomycin 30µg; Cefuroxime 30µg, Pefloxacin 10µg, Ampiclox 30µg, Zinnacef 20µg, Amoxicillin 30µg, Rocephin 25µg, Azithromycin 12µg, Levofloxacin 20µg and Erythromycin 10µg were used for this study. And the zones of inhibition around the discs after 24hours incubation were measured and compared to the CLSI standard (CLSI, 2024). Isolates that were resistant to at least one antibiotic in three or more classes of the

antibiotics tested were classified as MDR as described by Hassan *et al.* (2022).

RESULTS

All samples analysed were positive for bacterial growth, with *S. aureus* present in all samples, *E. coli* was observed in 65% of the samples, while both *P. aeruginosa* and *K. Pneumoniae* Were

found in 50% of all samples, though the latter was not found in samples from the bedside drawer (Table 1).

The isolates were identified based on their appearance on the selective media and their reaction to some confirmatory biochemical tests, as shown in Table 2.

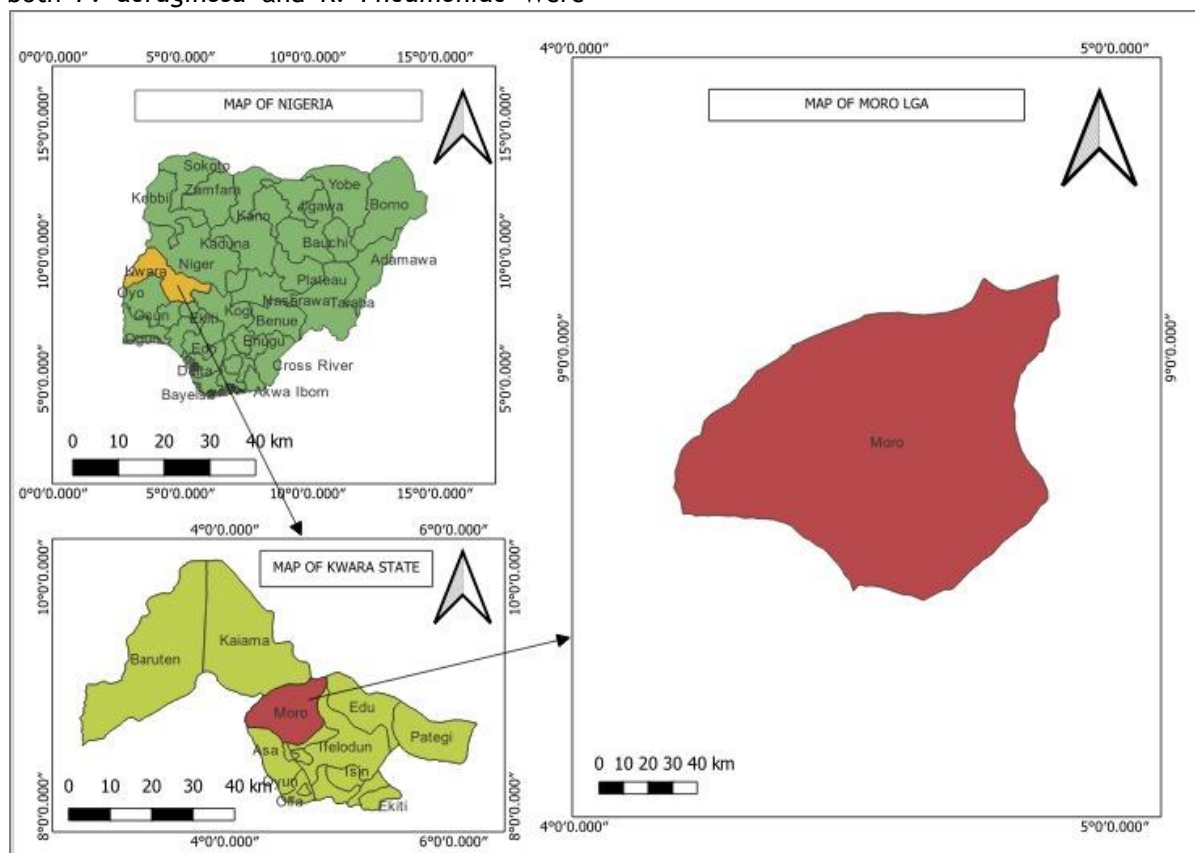


Figure 1: Map of Study Area
Coordinates: 8° 41' 59" N, 4° 28' 0" E

Table 1: Prevalence of Isolates from the Phones of Kwasu Health Workers and Healthcare Surfaces

Sample	Sample size	<i>S. aureus</i>	<i>K. pneumoniae</i>	<i>E. coli</i>	<i>P. aeruginosa</i>
Mobile phones	15	15	10	8	5
Doorknob	5	5	2	3	3
Drip stand	5	5	3	4	2
Bedside drawer	5	5	0	3	3
Bed rail	5	5	2	4	3
Wall	5	5	3	4	4
Total/Prevalence	40	40/100%	20/50%	26/65%	20/50%

The antibiotic susceptibility pattern *S. aureus* is shown in Table 3, where the isolates were completely resistant to pefloxacin, zinnacef, amoxicillin, erythromycin, azithromycin and cefuroxime, while showing a varying degree of susceptibility ranging between 15% to 24% to

gentamycin, ciprofloxacin, rocephin and levofloxacin. Table 4 shows the multidrug resistance pattern of *K. pneumoniae* to more than 3 of the tested antibiotics, with complete resistance to augmentin, peflacin and ceftriaxone; and a 0-24% resistance to the other

agents. *P. aeruginosa* was completely resistant to augmentin, streptomycin, cefuroxime, ceporex and ceftriaxone, while being inhibited by the other antibiotics as shown in Table 5. Unlike the other isolates that showed complete resistance to augmentin and cefuroxime, some

of the *E. coli* isolates showed a percentage resistance of 92.3% and 69.3% respectively, while showing total resistance to ceftazidime, ciprofloxacin, ceporex, ceftriaxone and streptomycin, as shown in Table 6.

Table 2: Morphological Characteristics and Biochemical Reaction of Isolates

	<i>S. aureus</i>	<i>K. pneumoniae</i>	<i>E. coli</i>	<i>P. aeruginosa</i>
Gram Reaction	+	-	-	-
Catalase	+	+	+	+
Oxidase	-	-	-	+
Coagulase	+	NA	NA	NA
Indole	NA	+	-	-
Methyl Red	NA	+	-	-
Voges-Proskauer	NA	-	+	-
Citrate	NA	-	+	+
Colonial Morphology				
<i>S. aureus</i>	Round, smooth yellow colonies on MSA			
<i>K. pneumoniae</i>	Large mucoid pink colonies on MacConkey agar			
<i>E. coli</i>	Dark blue-black colonies with greenish metallic sheen on EMB agar			
<i>P. aeruginosa</i>	Medium-sized light green colonies with uneven edges on centrimide agar			

Key; +: positive, -: negative, NA: Not applicable

Table 3: Antibiotic Susceptibility Pattern of *S. aureus* Isolated from Phones and Surfaces to Conventional Antibiotics

Antibiotics	Resistance Pattern	Mobile phones N=15(%)	Doorknob N=5(%)	Drip stand N=5(%)	Bedside drawer N=5(%)	Bed rail N=5(%)	Wall N=5(%)	Total N=40(%)
Pefloxacin	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	15 (100)	15 (100)	15 (100)	15 (100)	15 (100)	15 (100)	40 (100)
Zinnacef	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	15 (100)	15 (100)	15 (100)	15 (100)	15 (100)	15 (100)	40 (100)
Amoxicillin	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	15 (100)	15 (100)	15 (100)	15 (100)	15 (100)	15 (100)	40 (100)
Erythromycin	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	15 (100)	15 (100)	15 (100)	15 (100)	15 (100)	15 (100)	40 (100)
Gentamycin	S	5 (33.3)	3 (20)	2 (13.3)	5 (33.3)	5 (33.3)	0 (0)	20 (50)
	R	10 (66.7)	12 (80)	13 (86.7)	10 (66.7)	10 (66.7)	15 (100)	20 (50)
Ciprofloxacin	S	7 (46.7)	9 (60)	4 (26.7)	0 (0)	0 (0)	0 (0)	20 (50)
	R	8 (53.5)	6 (40)	11 (73.3)	15 (100)	15 (100)	15 (100)	20 (50)
Azithromycin	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	15 (100)	15 (100)	15 (100)	15 (100)	15 (100)	15 (100)	40 (100)
Rocephin	S	4 (26.7)	5 (33.3)	4 (26.7)	1 (6.6)	1 (6.6)	0 (0)	15 (37.5)
	R	11 (73.3)	10 (66.7)	11 (73.3)	14 (93.4)	14 (93.4)	15 (100)	25 (62.5)
Levofloxacin	S	6 (40)	3 (20)	3 (20)	6 (40)	6 (40)	0 (0)	24 (60)
	R	9 (60)	12 (80)	12 (80)	9 (60)	9 (60)	15 (100)	16 (40)
Cefuroxime	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	15 (100)	15 (100)	15 (100)	15 (100)	15 (100)	15 (100)	40 (100)

Key: S= susceptible (> 14mm); R= resistant (<13mm)

Table 4: Antibiotic Susceptibility Pattern of K. pneumoniae Isolated from Phones and Surfaces to Conventional Antibiotics

Antibiotics	Resistance Pattern	Mobile phones N=10(%)	Doorknob N=3(%)	Drip stand N=2(%)	Bedside drawer N=0(%)	Bed rail N=5(%)	Wall N=5(%)	Total N=40(%)
Ofloxacin	S	10 (100)	3(100)	2 (100)	-	5 (100)	5 (100)	25 (100)
	R	0 (0)	0 (0)	0 (0)	-	0 (0)	0 (0)	0 (0)
Augmentin	S	0 (0)	0 (0)	0 (0)	-	0 (0)	0 (0)	0 (0)
	R	10 (100)	3 (100)	2 (100)	-	5 (100)	5 (100)	25 (100)
Peflacine	S	10 (100)	3(100)	2 (100)	-	5 (100)	5 (100)	25 (100)
	R	0 (0)	0 (0)	0 (0)	-	0 (0)	0 (0)	0 (0)
Ceftazidime	S	0 (0)	0 (0)	0 (0)	-	0 (0)	0 (0)	0 (0)
	R	10 (100)	3 (100)	2 (100)	-	5 (100)	5 (100)	25 (100)
Gentamycin	S	9 (90)	3 (100)	2 (100)	-	2 (40)	4 (80)	20 (80)
	R	1 (10)	0 (0)	0 (0)	-	3 (60)	1 (20)	5 (20)
Ciprofloxacin	S	8 (80)	2 (66.7)	2 (100)	-	4 (80)	5 (100)	21 (84)
	R	2 (20)	1 (33.3)	0 (0)	-	1 (20)	0 (0)	4 (12)
Ceporex	S	0 (0)	0 (0)	0 (0)	-	0 (0)	0 (0)	0 (0)
	R	10 (100)	3 (100)	2 (100)	-	5 (100)	5 (100)	25 (100)
Ceftriaxone	S	10 (100)	3(100)	2 (100)	-	5 (100)	5 (100)	25 (100)
	R	0 (0)	0 (0)	0 (0)	-	0 (0)	0 (0)	0 (0)
Streptomycin	S	8 (80)	2 (66.7)	2 (100)	-	3 (60)	4 (80)	19 (76)
	R	2 (20)	1 (33.3)	0 (0)	-	2 (40)	1 (20)	6 (24)
Cefuroxime	S	0 (0)	0 (0)	0 (0)	-	0 (0)	0 (0)	0 (0)
	R	10 (100)	3 (100)	2 (100)	-	5 (100)	5 (100)	25 (100)

Key: S= susceptible (> 13mm); R= resistant (<12mm)

Table 5: Antibiotic Susceptibility Pattern of P. aeruginosa Isolated from Phones and Surfaces to Conventional Antibiotics

Antibiotics	Resistance Pattern	Mobile phones N=10(%)	Doorknob N=3(%)	Drip stand N=2(%)	Bedside drawer N=0(%)	Bed rail N=5(%)	Wall N=5(%)	Total N=40(%)
Ofloxacin	S	5 (100)	3(100)	2 (100)	3 (100)	3 (100)	4 (100)	20 (100)
	R	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Augmentin	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	5 (100)	3 (100)	2 (100)	3 (100)	3 (100)	4 (100)	20 (100)
Peflacine	S	5 (100)	3(100)	2 (100)	3 (100)	3 (100)	4 (100)	20 (100)
	R	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Ceftazidime	S	5 (100)	3(100)	2 (100)	3 (100)	3 (100)	4 (100)	20 (100)
	R	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Gentamycin	S	5 (100)	3 (100)	2 (100)	3 (100)	3 (100)	4 (100)	20 (100)
	R	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Ciprofloxacin	S	5 (100)	3 (100)	2 (100)	3 (100)	3 (100)	4 (100)	20 (100)
	R	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Ceporex	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	5 (100)	3 (100)	2 (100)	3 (100)	3 (100)	4 (100)	20 (100)
Ceftriaxone	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	5 (100)	3 (100)	2 (100)	3 (100)	3 (100)	4 (100)	20 (100)
Streptomycin	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	5 (100)	3 (100)	2 (100)	3 (100)	3 (100)	4 (100)	20 (100)
Cefuroxime	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	5 (100)	3 (100)	2 (100)	3 (100)	3 (100)	4 (100)	20 (100)

Key: S= susceptible (> 13mm); R= resistant (<12mm)

Table 6: Antibiotic Susceptibility Pattern of E. coli Isolated from Phones and Surfaces to Conventional Antibiotics

Antibiotics	Resistance Pattern	Mobile phones N=10(%)	Doorknob N=3(%)	Drip stand N=2(%)	Bedside drawer N=0(%)	Bed rail N=5(%)	Wall N=5(%)	Total N=40(%)
Ofloxacin	S	8 (100)	3(100)	4 (100)	3 (100)	4 (100)	4 (100)	26 (100)
	R	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Augmentin	S	2 (25)	0 (0)	1 (25)	0 (0)	0 (0)	0 (0)	2 (7.7)
	R	6 (75)	3(100)	3 (75)	3 (100)	4 (100)	4 (100)	24 (92.3)
Peflacin	S	8 (100)	3(100)	4 (100)	3 (100)	4 (100)	4 (100)	26 (100)
	R	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Ceftazidime	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	8 (100)	3(100)	4 (100)	3 (100)	4 (100)	4 (100)	26 (100)
Gentamycin	S	8 (100)	3(100)	4 (100)	3 (100)	4 (100)	4 (100)	26 (100)
	R	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Ciprofloxacin	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	8 (100)	3(100)	4 (100)	3 (100)	4 (100)	4 (100)	26 (100)
Ceporex	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	8 (100)	3(100)	4 (100)	3 (100)	4 (100)	4 (100)	26 (100)
Ceftriaxone	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	8 (100)	3(100)	4 (100)	3 (100)	4 (100)	4 (100)	26 (100)
Streptomycin	S	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	R	8 (100)	3(100)	4 (100)	3 (100)	4 (100)	4 (100)	26 (100)
Cefuroxime	S	3 (37.5)	0 (0)	2 (50)	0 (0)	1 (25)	2 (50)	8 (30.7)
	R	8 (62.5)	3(100)	4 (50)	3 (100)	4 (75)	2 (50)	18 (69.3)

Key: S= susceptible (> 13mm); R= resistant (<12mm)

DISCUSSION

Though with a differing level of prevalence, isolates are similar to the isolates obtained from mobile phones of health workers in a health facility in Libya by [Elbarghathi et al. \(2025\)](#) where 21.6% *P. aeruginosa*, 20.8% *E. coli*, 14.4% *K. pneumoniae* and 6.4% *S. aureus* were obtained, making *P. aeruginosa* the most prevalent and *S. aureus* the least prevalent, which is in contrast with the current findings. It is also in agreement with those obtained by [Kimwele et al. \(2024\)](#) from hospital surfaces in Kitui County, with *S. aureus* having the highest prevalence at 43% and *P. aeruginosa* having the lowest at 13%.

The high level of resistance exhibited by *S. aureus* to multiple antibiotics, including complete resistance to pefloxacin, zinnacef, amoxicillin, erythromycin, azithromycin, and cefuroxime, is consistent with previous studies reporting extensive resistance of *S. aureus* strains to beta-lactams and macrolides ([Fidelis et al., 2024](#) and [Rashed and Zaid, 2022](#)). This resistance is primarily due to genetic adaptations such as the presence of the *mecA* gene, which encodes an altered penicillin-binding protein (PBP2a) that reduces the

efficacy of beta-lactam antibiotics ([Rashed and Zaid, 2022](#)). The low susceptibility observed to gentamicin, ciprofloxacin, rocephin, and levofloxacin further supports growing concerns about the dwindling effectiveness of fluoroquinolones and aminoglycosides against *S. aureus* as reported by [Brdová et al. \(2024\)](#). This pattern of resistance across multiple antibiotic classes is characteristic of a multidrug-resistant (MDR) profile and signals a serious public health concern, particularly in healthcare settings where *S. aureus* is a leading cause of both community and hospital-acquired infections.

K. pneumoniae isolates displayed classic MDR characteristics, being completely resistant to augmentin, pefloxacin, and ceftriaxone, and minimally susceptible to other agents. This aligns with reports from similar Nigerian studies documenting extensive resistance in *K. pneumoniae* strains, particularly to cephalosporins and penicillin-beta-lactamase inhibitor combinations ([Ashefo et al., 2023](#)). The widespread resistance may reflect the organism's ability to produce extended-spectrum beta-lactamases (ESBLs), enzymes capable of hydrolysing a broad range of β -lactam antibiotics, including third-generation cephalosporins and β -lactam-inhibitor

combinations, thereby facilitating its survival in the presence of multiple antibiotics (Ezeh *et al.*, 2024). The presence of ESBLs, along with other resistance mechanisms such as efflux pumps and porin loss, enhances the organism's ability to evade multiple antimicrobial agents (Shah *et al.*, 2025).

P. aeruginosa also demonstrated notable resistance, with complete resistance to five antibiotics spanning across β -lactam inhibitors, aminoglycosides and cephalosporins. This pathogen is known for its intrinsic resistance mechanisms, including efflux pumps and low outer membrane permeability that enable it to resist a wide range of antibiotics, making infections difficult to treat. The synergy between these two resistance strategies is a significant factor in the organism's ability to survive in hostile environments, contributing to its status as a major nosocomial pathogen (Amisano *et al.*, 2025 and Yang *et al.*, 2024). However, the retained susceptibility to certain antibiotics in this study is encouraging and suggests the need for routine local antibiogram development to guide empirical therapy.

Unlike the other pathogens, *E. coli* isolates in this study exhibited partial resistance to augmentin and cefuroxime, alongside complete resistance to ceftazidime, ciprofloxacin, cephalexin, ceftriaxone, and streptomycin. This extensive resistance pattern, particularly to third-generation cephalosporins (ceftazidime, ceftriaxone), fluoroquinolones (ciprofloxacin), and aminoglycosides (streptomycin), suggests the emergence of a multidrug-resistant (MDR) phenotype. These findings are consistent with recent antimicrobial surveillance studies, which have reported increasing resistance in *E. coli*, especially in hospital and healthcare-associated environments where empirical antibiotic use and poor infection control practices are common (Ruiz-Lievano *et al.*, 2024 and White, 2021). The observed resistance to multiple β -lactams may be driven by extended-spectrum β -lactamase (ESBL) production, while resistance to ciprofloxacin often results from mutations in *gyrA* and *parC* genes (Naidoo, 2025). Such MDR strains significantly reduce available treatment options, heightening the risk of treatment failure and further transmission within clinical and community settings.

CONCLUSION

This study reports the occurrence of multidrug-resistant bacterial pathogens, namely *S. aureus*, *K. pneumoniae*, *P. aeruginosa*, and *E. coli*

mobile phones of healthcare workers and surfaces within the Kwara State University, Malete Health Centre. The prevalence and resistance patterns observed, especially to β -lactams, aminoglycosides, and fluoroquinolones, indicate the presence of clinically significant resistance. The likely involvement of mechanisms such as extended-spectrum β -lactamase (ESBL) production and reduced susceptibility to critical antibiotics highlights the need for regular antimicrobial resistance monitoring in the health centre and larger community. These findings support the implementation of appropriate hygiene measures, disinfection practices, and antibiotic stewardship to minimise the risk of multidrug-resistant pathogen transmission in healthcare environments.

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