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The growing threat of antibiotic resistance in wound infections: evidence from tertiary care in Pakistan

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Abstract: The present study analyzed 361 non-duplicated wound swab samples from 187 males and 174 females, ranging in age from 0 to 100 years with a mean age of 37.1±1.9 years, and to determine the prevalence of bacterial wound infections and the diversity of antibacterial susceptibility patterns of the isolated bacteria to detect the presence of unique/rare resistance types. Of these, 53.46% (193) were found to have wound infections. Most of the infected patients fell in the age group II (21-40 years). A total of 14 bacterial species were identified, with *Staphylococcus aureus* and *Escherichia coli* being the most common Gram-positive and Gram-negative bacteria, respectively. Linezolid and vancomycin were the most effective antibiotics against the isolated Gram-positive bacteria, while most Gram-negative bacteria were sensitive against colistin and polymyxin-B. Based on antibiotic resistance, 129 types of resistance were detected. Multi-resistance was detected in 157 (81.3%) bacterial strains, while 162 strains had a multi-antibiotic resistance index (MAR) of 0.2. Simpson and Shannon diversity indices indicated high bacterial diversity in the wound samples. The study provides valuable insight into the prevalence of bacterial infections in wounds and that antibiotic resistance patterns can be useful in guiding the development of effective treatment strategies.

Keywords: wound infections; antibacterial susceptibility; multi-antibiotic resistance (MAR) index; resistance patterns; diversity indices

INTRODUCTION

A wound can be defined as any injury to the outermost layer of skin that results in a loss of protective function, integrity, and continuity of the underlying epithelium with or without involvement of connective tissue [1]. Wound infection is characterized by the presence and proliferation of microbes on the wound surface that, because of their virulence and pathogenicity, outpace the host's immune response, thus allowing the microbes to invade and damage the wound and delay healing, leading to prolonged hospitalization, limb amputation, sepsis, and, in severe cases, eventual death [2-4].

The most common pathogens found in infected wounds include *S. aureus*, *Streptococcus pyogenes*, *Enterococci*, *Escherichia coli*, *Klebsiella pneumonia*, *Proteus* spp., and *Pseudomonas aeruginosa* [5,6]. The bacteriological profile of infected wounds remains almost the same, but a diversity of antibiotic resistance has been observed due to the unnecessary and indiscriminate use of antibiotics and their inadequate dosing, leading to an increase in the emergence of multidrug-resistant, extensively drug-resistant, and PAN drug-resistant strains of a variety of pathogens [7,8]. The multi-antibiotic index (MAR index) is an efficient, economical and time-saving technique to estimate the nonsensical and frequent misuse or contamination of antibiotics, whereas the diversity of resistance among the multidrug-resistant (MDR), extensively drug-resistant (XDR) and pandrug-resistant (PDR) bacterial strains can be assessed by reporting the patterns of the antibiotic resistance of the isolated strains [9].

Antibiotic resistance (ABR) is globally emerging as one of the biggest and most life-threatening challenges of the 21st century, limiting the number of antibiotic options available for the treatment of bacterial infections, which, for many years, were previously treatable with conventional antibiotics [10]. The rapid

emergence of MDR and XDR strains of bacteria in the past few years has not only prolonged the treatment course but has also made the management of wound infections more tedious and complicated, emphasizing the need for the collection of data regarding resistant strains, their antibacterial susceptibility patterns on a larger scale and strictly implementing antimicrobial stewardship guidelines to limit this unseen threat to healthcare [11].

Prior to prescribing antimicrobial therapy, it is important to identify the pathogens responsible for infection and test their antibacterial susceptibility for proper management of the patients [12]. Antibiotics are of great value in prophylaxis and treatment of infections if used wisely [13]. The purpose of the present study was to characterize the bacteriological profile of infected wounds and evaluate their antibiotic susceptibility profiles, calculate the multi-antibiotic resistance index (MAR), and determine the diversity of antibiotic resistance patterns.

MATERIALS AND METHODS

Ethics statement

The research was conducted in accordance with the principles of the Declaration of Helsinki and in compliance with local regulatory requirements. All patients included in the study were informed that their identity and the use of their data for research purposes would not be disclosed. The study was approved by the institutional review board (IRB) of The University of Lahore on 10-12-2021 under Approval No. IRB-UG-23711.

Study population and sample size

The wounds of diverse etiologies, location and duration with clinical signs and symptoms of the infection were considered in the study prior to applying any antibiotic therapy. The patients diagnosed with known fungal or parasitic infections were excluded. A total of 361 wound samples were collected from different tertiary care hospitals in Lahore, Pakistan, including the Mayo Hospital, the University of Lahore Teaching Hospital, the General Hospital and the Services Hospital.

The sample size was calculated using the following equation as described previously [14].

$$n = Z\alpha^{2} pq/d^{2}$$

where p is the prevalence of wound infection (0.815) [11] and d (0.05) is the maximum tolerable error.

Sample collection

Only one wound sample was collected from each patient by the Levine technique [15]. After cleaning the wound surfaces with sterilized saline, a sterilized cotton swab (Nuova Aptaca SRL, Canelli, Italy) was used to collect the samples by moving the applicator 1 cm2 from the center to the edges of the wound beds with appropriate pressure in a zigzag pattern. The swab samples were sent within an hour to the microbiology laboratory in a transport medium (Stuart's medium) for further processing.

Culturing and identification of bacterial isolates

The samples were cultured on different agar media, including blood agar, chocolate agar and MacConkey's agar (Bio Lab, Hungary), and incubated for 24 h at 37°C. The bacterial isolates were characterized based on their morphological and biochemical features. The API 20E and API 20 NE identification systems (Biomerieux, France) were used for Enterobacteriaceae and non-Enterobacteriaceae, respectively, to evaluate their biochemical profiles.

Molecular detection of the most prevalent bacteria

The most prevalent Gram-positive (*S. aureus*) and Gram-negative (*E. coli*) bacteria were detected at the molecular level by targeting Sau-02 and uidA genes, respectively, as described previously [16,17]. Bacterial genomic DNA was extracted for PCR amplification using the QIAamp DNA mini kit (Qiagen, Germany) as per the manufacturer's instructions. A primer set Sau-02-F (5'-GTAAAAAGACGACATGCAGGAA-3') and Sau-02-R (5'-CCATCATTTCAAAACTTTGACA-3') was used to amplify the S. aureus-specific Sau-02 gene, whereas uidA-F (5'-TGGTAATTACCGACGAAAACGGC-3') and uidA-R (5'-ACGCGTGGTTACAGTCTTGCG-3') were used to amplify the E. coli-specific uidA gene. S. aureus ATCC 43300 and E. coli ATCC 43890 were used as positive controls. A DNA ladder of 1 kb (Thermo Fisher Scientific-US) was used for the estimation of amplicon size. Amplified products were resolved on

1% agarose and recorded after staining with ethidium bromide.

Antibiotic susceptibility profiling

Antibiotic susceptibility of isolated bacteria was determined by the standard disk diffusion method as described previously [18]. *E. coli* (ATCC no. 25922) and *S. aureus* (ATCC no. 25923) strains were used as controls and the results were interpreted as per CLSI guidelines. MDR pathogens were defined as isolates that were capable of being resistant to at least one antibiotic from three or more classes of antimicrobial agents used. Non-susceptibility to all the categories of antibiotics used, except two or fewer classes of antibiotics, were referred to as XDR. The pathogens that resisted all the antibiotic classes were referred to as PDR [19].

Calculation of the multi-antibiotic resistance (MAR) index

The MAR index was calculated and interpreted by dividing the number of antibiotics to which an isolate was resistant by the total number of antibiotics tested [20].

Calculation of diversity indices

The diversity on the basis of unique resistance profiles was calculated for all the isolated bacterial populations using the Shannon-Wiener diversity index

$$[H = -\sum_{i=1}^{S}(Pi)(logPi)]$$

and Simpson's diversity index

$$[1 - D = -\sum_{i=1}^{S}(Pi^2)],$$

where *S* and *Pi* represent the total number of unique resistance profiles and number of organisms with a unique profile (n)/total number of organisms in the community (N), respectively.

Statistical analysis

Data were analyzed using the Statistical Package for Social Sciences (SPSS) version 25 (IBM, USA). Continuous variables were presented by mean±SD, while categorical variables were calculated as frequency and percentage. Categorical variables were compared using the chi-square test at a 95% confidence interval. A two-tailed p value of less than 0.05 was designated as statistically significant. R package (3.5.1) was used to construct clustered heat maps. The clustering was done based on Dice similarity of 1% band tolerance and UPGMA (unweighted pair group method using average) linkages. Diversity indices were calculated using PAST (V4.03).

RESULTS

Non-duplicated wound swabs were obtained from 361 patients, 187 (52%) men and 174 (48%) women. The age range of the patients was 0-100 years with a mean age of 37.1±1.9 years. Among 361 patients, only 193 (53.46 %) were found to have wound infections. Based on the age difference, the patients were divided into five age groups (Supplementary Fig. S1). Most patients (n=83, 43%) with infected wounds fell into the age group II, while the fewest were in age group I (n=7, 3.6%).

A total of 14 bacterial species, 5 (35.7 %) Gram-positive and 9 (64.3%) Gram-negative, were detected (Supplementary Fig. S2). Among Gram-positive bacteria, *S. aureus* was the most common bacterium (n=65, 69.9%), followed by MRSA (n=11, 11.8%), *S. epidermidis* (n=8, 8.6%), *Streptococcus* spp. (n=5, 5.4%) and *Enterococcus* spp. (n=4, 4.3%). The presence of MRSA is problematic as it causes chronic infection and bacteremia. Among the Gram-negative bacteria, *E. coli* was the most dominant pathogen (n=29, 29%), followed by *P. aeruginosa* (n=28, 28%), *Klebsiella* spp. (n=13, 13%), *Acinetobacter* spp. (n=12, 12%), *Proteus* spp. (n=7, 7%), *Enterobacter* spp. (n=4, 4%), *Citrobacter* spp. (n=3, 3%), *S. marcescens* (n=3, 3 %) and *M. morganii* (n=1, 1%). A single bacterial species was found infecting the wounds in 99.5% (n=192) of patients, while coinfection with *Enterobacter* and *Citrobacter* was observed in 0.5% (n=1) of patients.

The prevalence of bacterial infections in wounds was also assessed in terms of patient sex and age. Out of 193 total patients with infected wounds, 142 were males and 51 were females. All the bacterial species were found to be more prevalent in male patients, except *Enterococcus* spp. and *S. epidermidis* (Supplementary Table S1). *S. aureus* was the most prevalent in males

Table 1. Antibiotic sensitivity patterns against Gram-positive bacteria.

Antibiotics	*S. aureus*	*Streptococcus* spp.	*S. epidermidis*	MRSA	*Enterococcus* spp.
Methicillin	65 (100)	---	---	0 (0)	---
Penicillin	1 (1.6)	3 (60)	0 (0)	0 (0)	2 (100)
Amoxicillin	1 (9.1)	---	0 (0)	---	2 (100)
Ampicillin	0 (0)	2 (100)	0 (0)	---	4 (100)
Cloxacillin	6 (54.5)	---	1 (25)	---	---
Oxacillin	---	---	0 (0)	---	---
Augmentin	7 (53.8)	---	0 (0)	---	2 (100)
Ceftriaxone	6 (54.5)	2 (50)	1 (25)	---	---
Cefoxitin	1 (16.6)	0 (0)	---	---	---
Cefuroxime	5 (50)	---	1 (33.3)	---	---
Cefotaxime	6 (54.5)	---	1 (25)	---	---
Cephradine	6 (60)	---	1 (25)	---	---
Imipenem	6 (54.5)	---	1 (33.3)	---	---
Meropenem	5 (50)	---	1 (50)	---	---
Amikacin	37 (69.8)	---	4 (100)	7 (100)	---
Gentamicin	38 (60.3)	---	3 (50)	9 (81.8)	---
Co-trimoxazole	15 (23.4)	---	4 (57.1)	4 (36.3)	---
Tetracycline	19 (39.5)	0 (0)	---	---	---
Doxycycline	12 (66.6)	---	5 (71.4)	5 (62.5)	0 (0)
Ciprofloxacin	12 (19.0)	2 (100)	2 (28.5)	2 (18.1)	1 (25)
Levofloxacin	---	2 (66.6)	---	---	---
Erythromycin	22 (34.3)	1 (20)	2 (25)	7 (63.6)	0 (0)
Clarithromycin	3 (42.8)	---	0 (0)	---	---
Vancomycin	63 (96.9)	5 (100)	5 (100)	8 (100)	4 (100)
Teicoplanin	35 (53.8)	2 (100)	5 (100)	4 (100)	2 (100)
Tigecycline	32 (71.1)	2 (100)	---	---	---
Linezolid	64 (98.4)	2 (100)	8 (100)	9 (100)	4 (100)
Chloramphenicol	53 (91.4)	3 (60)	4 (100)	---	---
Clindamycin	36 (58.1)	2 (50)	3 (37.5)	7 (77.7)	---
Rifampicin	52 (89.6)	2 (100)	3 (75)	---	---
Fusidic-acid	42 (66.6)	---	1 (12.5)	7 (77.7)	0 (0)

n (%)

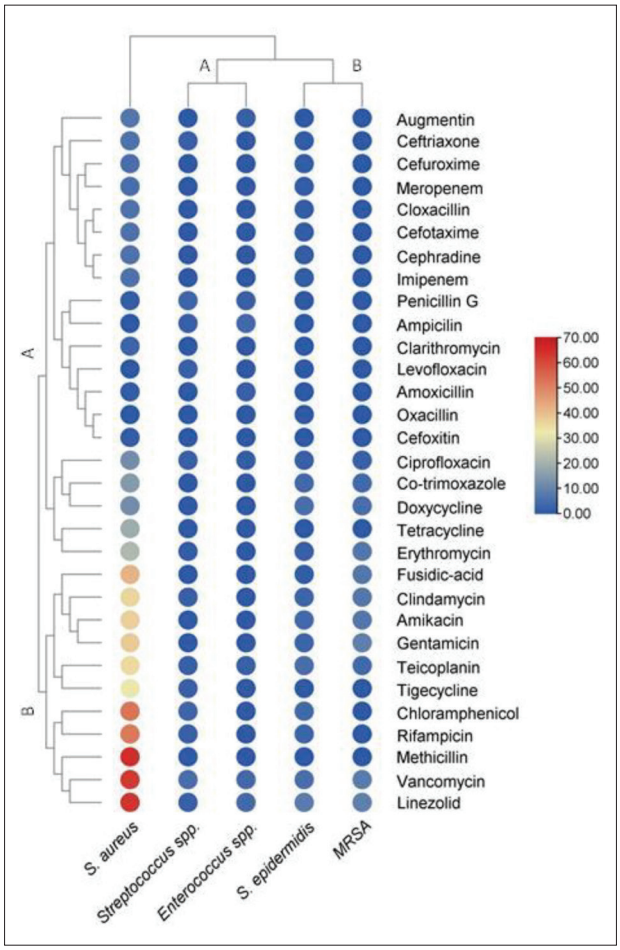

Fig. 1. Clustered heat map exhibiting antibiotic susceptibility profiles of Gram-positive bacteria. The color of the bubbles represents the number of susceptible bacteria.

(n=50, 35.2%), whereas *M. morganii* was the bacterium least responsible for infections in males, and in females, *Citrobacter* and *Enterobacter* species were among the least isolated bacteria.

Antibacterial susceptibility patterns

S. aureus exhibited 100% resistance against ampicillin, followed by penicillin G (98%) and ciprofloxacin (80%). *Streptococcus* species demonstrated the highest resistance against tetracycline (100%), cefoxitin (100%) and erythromycin (80%), whereas *S. epidermidis* was found to be resistant to penicillin antibiotics and clarithromycin, followed by fusidic acid (87.7%), cefotaxime (75%), cephradine (75%) and ceftriaxone (75%). MRSA exhibited a high resistance rate against methicillin (100%), ciprofloxacin (81.8%) and co-trimoxazole (63.6%). All *Enterococcus* spp. were resistant to doxycycline, erythromycin and fusidic acid. Linezolid and vancomycin were found to be the most sensitive antibiotics (0% resistance) against isolated Gram-positive bacteria (Table 1). Moreover, clustering analysis was performed to evaluate the effectiveness of each antibiotic against multiple bacteria as well as the susceptibility pattern of all the tested antibiotics. Clustering analysis of Gram-positive bacteria based on their susceptibility to multiple antibiotics showed that *S. aureus* was present as an out-group, while all the other bacteria were grouped into two main clusters (A and B). Cluster A was a monofolium containing MRSA, while cluster B grouped *S. epidermidis*, *Streptococcus* spp., and *Enterococcus* spp. (Fig. 1). The results revealed that the

Table 2. Antibiotic susceptibility profile of Gram-negative bacteria.

Antibiotics	*Acinetobacter*	*E. coli*	*P. aeruginosa*	*Klebsiella Spp.*	*Citrobacter Spp.*	*M. morgani*	*S. Marcescens*	*Proteus Spp.*	*Enterobacter Spp.*
Amoxicillin	0 (0)	0 (0)	---	0 (0)	0 (0)	---	0 (0)	0 (0)	0 (0)
Ampicillin	0 (0)	0 (0)	---	0 (0)	0 (0)	---	---	0 (0)	0 (0)
Augmentin	0 (0)	0 (0)	---	0 (0)	---	---	---	0 (0)	0 (0)
Piperacillin/tazobactam	2 (18.1)	13 (44.8)	19 (70.3)	6 (50)	2 (100)	1 (100)	3 (100)	6 (85.7)	0 (0)
Ceftriaxone	0 (0)	1 (3.5)	0 (0)	3 (23.1)	0 (0)	1 (100)	3 (100)	1 (14.2)	0 (0)
Ceftazidime	0 (0)	1 (6.2)	17 (62.9)	3 (30)	0 (0)	---	1 (100)	1 (20)	0 (0)
Cefuroxime	0 (0)	1 (7.14)	---	3 (30)	0 (0)	---	0 (0)	1 (16.6)	0 (0)
Cefotaxime	---	0 (0)	---	---	---	1 (100)	3 (100)	---	---
Cefixime	0 (0)	1 (6.6)	---	2 (22.2)	0 (0)	---	1 (100)	0 (0)	0 (0)
Cefepime	0 (0)	3 (20)	12 (50)	2 (22.2)	1 (50)	---	1 (100)	3 (60)	0 (0)
Cefoperazone+Sulbactam	0 (0)	---	---	1 (50)	---	---	---	---	---
Imipenem	2 (16.6)	26 (89.6)	19 (70.1)	10 (76.9)	2 (100)	---	3 (100)	6 (100)	2 (66.6)
Meropenem	2 (16.6)	21 (72.4)	18 (66.6)	8 (61.9)	1 (100)	1 (100)	3 (100)	7 (100)	0 (0)
Ertapenem	1 (12.5)	8 (53.3)	---	5 (62.5)	1 (100)	---	---	5 (100)	0 (0)
Doripenem	1 (12.5)	10 (66.6)	7 (63.6)	6 (75)	2 (100)	---	---	5 (100)	0 (0)
Amikacin	3 (25)	25 (86.2)	17 (62.9)	8 (61.5)	1 (50)	1 (100)	3 (100)	7 (100)	0 (0)
Gentamicin	1 (8.3)	19 (65.5)	14 (51.8)	8 (61.5)	0 (0)	0 (0)	3 (100)	4 (57.1)	0 (0)
Tobramycin	2 (40)	5 (38.46)	11 (73.33)	1 (20)	0 (0)	---	2 (100)	1 (33.3)	0 (0)
Co-trimoxazole	2 (16.6)	3 (10.71)	0 (0)	3 (25)	2 (100)	0 (0)	1 (33.3)	1 (14.2)	0 (0)
Tetracycline	1 (12.5)	3 (20)	---	2 (25)	2 (100)	---	---	0 (0)	0 (0)
Minocycline	4 (44.4)	5 (35.7)	---	4 (50)	2 (100)	---	---	0 (0)	0 (0)
Doxycycline	---	1 (20)	---	---	---	0 (0)	1 (50)	---	0 (0)
Ciprofloxacin	1 (8.33)	4 (13.7)	16 (61.5)	3 (25)	0 (0)	1 (100)	3 (100)	2 (28.5)	0 (0)
Levofloxacin	2 (22.2)	2 (10)	8 (47.0)	4 (40)	1 (50)	---	3 (100)	2 (40)	0 (0)
Norfloxacin	1 (12.5)	2 (13.3)	7 (63.6)	2 (25)	0 (0)	---	---	1 (20)	0 (0)
Colistin	11 (100)	15 (100)	15 (78.94)	11 (100)	2 (100)	---	---	0 (0)	3 (100)
Polymyxin-B	8 (100)	15 (100)	12 (80)	8 (100)	2 (100)	---	---	0 (0)	2 (100)
Aztreonam	---	---	1 (33.3)	---	---	---	---	---	---
Tigecycline	3 (37.5)	15 (100)	---	7 (77.7)	2 (100)	---	---	4 (80)	1 (50)
Chloramphenicol	3 (37.5)	13 (86.6)	---	7 (77.7)	1 (50)	---	---	2 (40)	2 (100)
Rifampicin	---	---	0 (0)	---	---	---	---	---	---

n (%)

susceptibility patterns of *S. epidermidis*, *Streptococcus* spp. and *Enterococcus* spp. were comparable as they shared the same cluster, while *S. aureus* showed a very different pattern. The clustering of antibiotics based on their effectiveness against Gram-positive bacteria grouped the antibiotics into two large clusters (A and B). Cluster A was found to be smaller, harboring 10 antibiotics and had two subclusters. Chloramphenicol, rifampicin, vancomycin and linezolid were found to share the same subcluster, while carbapenems were in different large clusters exhibiting very different effectiveness against the bacteria.

An increasing pattern of resistance of Gram-negative bacteria to the penicillin and cephalosporin classes of antibiotics has been observed. Ampicillin, amoxicillin, augmentin, cefixime, cefuroxime, ceftriaxone, ceftazidime and cefepime were found to be least effective, as most bacteria were found to be resistant to them. One case of *M. morganii* was reported that had 100% resistance to co-trimoxazole, gentamicin and doxycycline. Most Gram-negative bacteria were sensitive to colistin and polymyxin-B, except for *P. aeruginosa* and *Proteus* spp. *M. morganii* showed 100% sensitivity to amikacin, ceftriaxone, ciprofloxacin, meropenem, and piperacillin+tazobactam (Table 2). Cluster analysis revealed two large clusters (A and B); cluster B was a difolium harboring *E. coli* and *P. aeruginosa* and had similar susceptibility to the antibiotics tested, whereas cluster A was larger and consisted of two subclusters. Antibiotic grouping revealed two large clusters and 3 complex small clusters with multiple subclusters

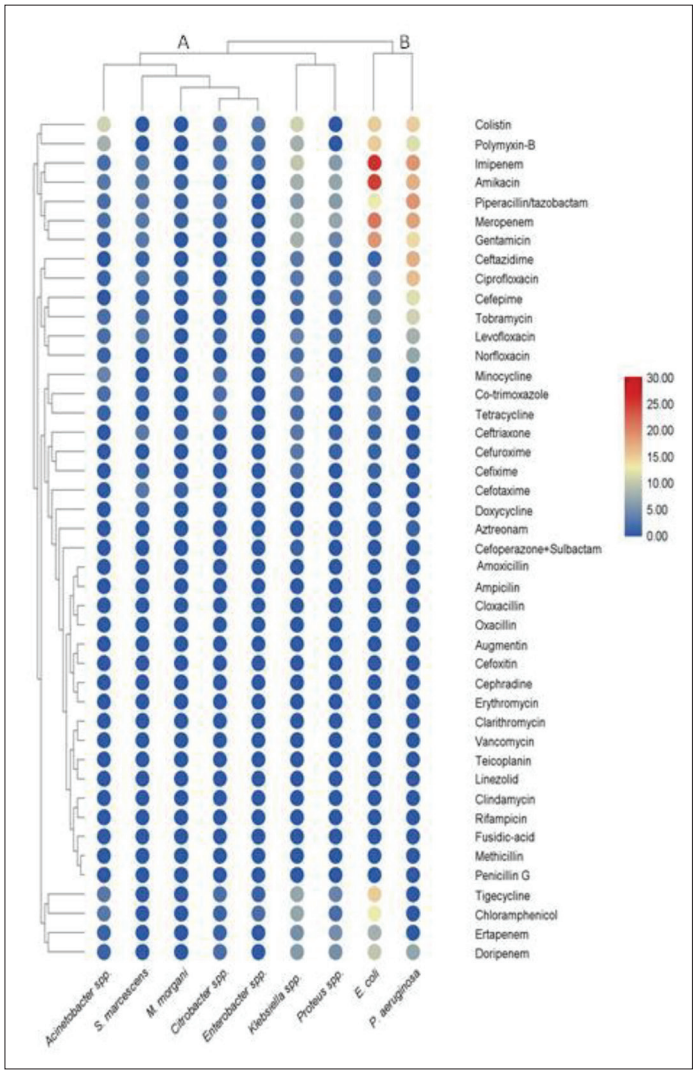

Fig. 2. Clustered heat map demonstrating antibiotic susceptibility profiles of Gram-negative bacteria. The color of the bubbles represents the number of susceptible bacteria.

(Fig. 2). The efficacy of tigecycline, chloramphenicol, ertapenem and doripenem was quite similar, sharing the same subcluster, but different from colistin and polymyxin-B, which were in different clusters.

Antibiotic resistance patterns of isolated pathogens

Based on antibiotic resistance against all the commonly used antibiotic classes, 129 different resistance patterns (R1-R129) were observed (Supplementary Table S2). The resistance pattern R64 was highly repetitive, as shown by 4 strains of *E. coli*, 2 strains of *Klebsiella* spp. and 2 strains of *Enterobacter* spp., followed by a resistant pattern R68 displayed by 3 strains of *E. coli* and one strain of *Klebsiella* spp. and *Proteus* spp. each (Supplementary Fig. S3).

Diversity indices of the isolated bacteria

Overall, 14 different bacterial species were obtained from wounds on different parts of the body. The calculated values for richness, and the Shannon and Simpson diversity indices for *E. coli* were 19, 2.81 and 0.93, respectively, which were the highest among the other isolated pathogens (Table 3).

Table 3. Diversity index values for the isolated bacteria.

Pathogens	Simpson (1-D)	Shannon (H)	Evenness (E)	Richness (S)
Acinetobacter spp.	0.86	2.10	0.91	9.0
E. coli	0.93	2.81	0.87	19.0
Klebsiella spp.	0.91	2.46	0.97	12.0
Citrobacter spp.	0.50	0.69	1.00	2.0
P. aeruginosa	0.81	1.83	0.89	7.0
Streptococcus spp.	0.80	1.61	1.00	5.0
Proteus spp.	0.83	1.79	1.00	6.0
Enterobacter spp.	0.44	0.64	0.94	2.0
Enterococcus spp.	0.63	1.04	0.94	3.0
Citrobacter / *Enterobacter* spp.	0.50	0.69	1.00	2.0
M. morganii	0.00	0.00	1.00	1.0
MRSA	0.86	2.04	0.96	8.0
S. marcescens	0.50	0.69	1.00	2.0
S. epidermidis	0.88	2.08	1.00	8.0

Distribution of MDR, XDR and PDR among isolated pathogens

The prevalence of MDR was 66.8% (n=129, followed by XDR (n=26, 13.5%) and PDR (n=2, 1%). Of the MDR, *S. aureus* (n=64, 49.6%) was more prevalent, followed by *E. coli* (n=24, 18.6%) and *Klebsiella spp.* (n=8, 6.2%) (Table 4). Out of 12 *Acinetobacter spp.* isolates, 8 (66.6%) were found to have extended drug-resistant (XDR). *P. aeruginosa* was the only bacterium whose 2 (7.1%) strains were found to be resistant to all the tested classes of antibiotics, whereas all the strains (n=3) of *S. marcescens* were non-MDR (Table 4). This differential distribution of the bacterial isolates into three categories was found to be statistically significant (P=<0.01). The prevalence of MDR in patients with respect to age and gender was evaluated, revealing that male patients were more prone to infection by MDR (n=93, 48.2%) and XDR pathogens (n= 26, 13.5%), and that most MDR

(n=56, 43.4%) and XDR (n=14, 53.8%) caused infection in the age group 21-40 years (Table S3).

Evaluation of the MAR index

The MAR index for isolated pathogens ranged from 0 to 1 with an average MAR index of 0.47. The cutoff value for the MAR index was 0.2. All the isolates of *Citrobacter* spp., *Enterobacter spp.* and *S. epidermidis* exhibited a MAR index that was greater than 0.2, followed by *S. aureus* (n=61, 93.8%) and *Acinetobacter spp.* (n=11, 91.7%) (Table 4).

DISCUSSION

Infected wounds are among the most important factors that can lead to morbidity or mortality, contributing to longer duration and higher total cost of care [21]. Prior to the use of antibiotic therapy, determination of the microbial flora in wounds and their current resistance profile along with resistance types is critical. The current study was conducted to evaluate the bacteriological profile of infected wounds and also to assess the antibacterial susceptibility of the isolated bacteria, to estimate the prevalence of MDR, XDR and PDR, to calculate the MAR index and to investigate the diversity of resistance patterns that contribute to raising awareness of this threat to healthcare.

In the current study, it was observed that male patients and patients aged 21-40 years were more prone to wound infections. Several other studies also reported that most culture-positive wound specimens were found in males and individuals aged 20-40 years [22,23], which may be due to the fact that males and individuals in this age group actively participate in social and physical activities and are more exposed to the external environment [13].

The current study revealed that Gram-negative pathogens were the dominant bacterial isolates affecting 51.8% of the total wound infections analyzed. *E. coli* was the most prevalent pathogen among Gram-negative bacteria, infecting 29% of the patients with infected wounds, whereas overall *S. aureus* was the dominant bacterial isolate infecting 33.6% of the total samples. *P. aeruginosa* and *Acinetobacter* spp. were found to be the 3rd and 4th most common pathogens, infecting 14.5% and 11.4% of wounds, respectively. These results are in great agreement with several other studies, which reported the high prevalence (77%) of Gram-negative bacteria in wound infections [24], with *S. aureus* as the most dominant [25] and *E. coli* [26], *P. aeruginosa* and *Acinetobacter* spp. [13] as the 2nd, 3rd and 4th most prevalent bacteria. The high prevalence of these microbial pathogens is attributed to their ability to grow well in moist conditions and persist in hospital settings [27]. The prevalence pattern may vary in different geographical locations, which can be attributed to differential clinical practices around the world.

Like studies, the antibiogram analysis indicated linezolid, teicoplanin, chloramphenicol and vancomycin as the drugs of choice against Gram-positive bacteria [28]. In contrast to the current study, 81% of the *S. aureus*-infected patients were sensitive to cefoxitin, followed by gentamicin (76%) and erythromycin (72%), whereas cefoxitin (16.6%), gentamicin (60.3%) and erythromycin (34.3%) were found to be less effective, which can be

Table 4. Distribution of MDR, XDR and PDR among the isolated pathogens and their MAR index.

Isolated Bacteria	Non MDR	MDR	XDR	PDR	MAR Index > 0.2
Acinetobacter spp.	1 (8.3)	3 (25)	8 (66.7)	0 (0)	11 (91.7)
Citrobacter spp.	1 (33.3)	2 (66.7)	0 (0)	0 (0)	2 (100)
Enterobacter spp.	0 (0)	2 (50)	2 (50)	0 (0)	3 (100)
E. coli	2 (6.9)	24 (82.8)	3 (10.3)	0 (0)	28 (96.6)
Klebsiella spp.	1 (7.7)	8 (61.5)	4 (30.8)	0 (0)	12 (92.3)
MRSA	4 (36.4)	7 (63.6)	0 (0)	0 (0)	10 (90.9)
M. morganii	0 (0)	1 (100)	0 (0)	0 (0)	1 (100)
Proteus spp.	1 (14.3)	4 (57.1)	2 (28.6)	0 (0)	6 (85.7)
P. aeruginosa	17 (60.7)	3 (10.7)	6 (21.4)	2 (7.1)	13 (46.4)
S. marcescens	3 (100)	0 (0)	0 (0)	0 (0)	0 (0)
S. aureus	1 (1.5)	64 (98.5)	0 (0)	0 (0)	61 (93.8)
S. epidermidis	1 (12.5)	7 (87.5)	0 (0)	0 (0)	8 (100)
Streptococcus spp.	1 (20)	3 (60)	1 (20)	0 (0)	4 (80)
Enterococcus spp.	3 (75)	1 (25)	0 (0)	0 (0)	3 (75)

n (%)

attributed to their differential use [5]. The increased resistance against these antibiotics over the last few years is the result of the irrational/indiscriminate use of antibiotics in our society. The antibacterial susceptibility pattern against Gram-negative bacteria revealed imipenem, meropenem, amikacin and tigecycline as the most effective, which is in agreement with several other studies [29]. Colistin, tigecycline and polymyxin-B are considered as the last hope against pathogenic microbes, but the current study revealed resistance against these drugs in various microbes, including *Proteus* spp. *Acinetobacter* spp., *Klebsiella* spp., *Proteus* spp. and *Enterobacter* spp. isolates, thus minimizing the available treatment options against these pathogens.

Furthermore, the MAR index of 162 (83.94%) isolates was above 0.2, which is comparable to the study conducted in Nigeria by Aisha Mohammed [30] where a MAR index of 90.3% of *S. aureus* isolates was above 0.2, followed by *E. coli* (88%), *Citrobacter* (85%) and *P. aeruginosa* (60%). A MAR index greater than 0.2% indicates the frequent use of antibiotics and reflects increased contamination [31]. The treatment of infected wounds becomes more challenging when bacteria develop resistance to multiple antibiotics. The emergence of MDR poses a serious threat to infected wound healing as we are left with very limited treatment options. The high rate of MDR in wound infections has been reported in several studies [32]. In a previous study, the percentage of MDR species of *S. aureus, Klebsiella* and *P. aeruginosa* was 81%, 88% and 84%, respectively, whereas in the present study *S. aureus* (98.5%) was the most prevalent MDR species, followed by *S. epidermidis* (87.5%) and *E. coli* (82.8%). In our study, 61.5% of *Klebsiella* spp. were MDR. In contrast to the present study, a study from Bangladesh described *Proteus* spp. (75.9%) as the most prominent MDR isolate, followed by *P. aeruginosa* (72.5%), *S. aureus* (68.3%) and *Klebsiella* (59.1%) [33]. The current investigation clearly showed an increasing trend in MDR strains over time, which is quite alarming and poses a serious threat to infected-wound healing. The high rate of MDR in wound infection has been reported in several studies [32].

The evaluation of antibiotic resistance patterns for all isolated bacteria revealed that. *S. aureus* was the most diverse, with 53 resistance types, followed by *E. coli* and *Klebsiella* spp. These findings are comparable to the study conducted by Raed Ennab [34], where *S. aureus*, *E. coli* and *Klebsiella* were among those bacteria that demonstrated diverse resistance patterns. The wide variety of resistance patterns indicates the uncertainty in the use of antibiotics for the treatment of infected wounds, as one and the same pathogen shows different resistances. After the success of diversity measures in ecology, there is a need to apply diversity matrices to quantify the uncertainty regarding the effectiveness of an antibiotic against a specific bacterial isolate. Previously, diversity matrices have been applied to measure molecular or species diversity of antibiotic-resistant bacteria [35], but data on the use of diversity matrices to quantify the antibiotic resistance diversity are scarce.

There are a variety of diversity indices, but there are no clear guidelines on how to select the indices that best serve the purpose. However, the Simpson and Shannon-Weiner diversity indices have been used extensively to measure microbial diversity [36]. In the current study, the Simpson and Shannon-Weiner diversity indices were applied to the antibiotic resistance data because these indices give more weight to unique resistance types. The results show that *E. coli* strains, followed by *Klebsiella* spp. strains, were more diverse in terms of resistance types. The results clearly show the danger of broad, unwise and uncontrolled use of antibiotics without knowledge of the susceptibility profile of bacterial species.

CONCLUSIONS

Our study describes the prevalence and antibiotic resistance patterns of bacteria involved in wound infections in this geographic region of the world. We believe this will be very helpful and invaluable to clinicians and health care workers in their efforts to manage wound infections. The study found that *S. aureus* was the most common bacterial species in wound infections, followed by *E. coli*, *P. aeruginosa* and *Acinetobacter* spp. and that linezolid, teicoplanin, chloramphenicol and vancomycin were the most effective, while ampicillin, amoxicillin and augmentin were the least effective and should not be used to treat wound infections. The presence of XDR and PDR along with very different types of resistance (129) points to the need to characterize the microbial pathogens in infected wounds and analyze their antibiotic susceptibility profiles before implementing antibiotic therapy aimed at reducing the emergence and spread of antibiotic resistance.

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SUPPLEMENTARY MATERIAL

Supplementary Table S1. Distribution of bacterial species in male and female populations.

Micro-organisms	Male	Female
Staphylococcus aureus	50	15
Acinetobacter Species	10	2
Escherichia coli	16	13
Pseudomonas Aeruginosa	21	7
Klebsiella species	11	2
Citrobacter species	2	0
Streptococcus species	3	2
Proteus Species	6	1
Enterobacter species	3	0
Enterococcus species	2	2
Staphylococcus epidermidis	4	4
Citrobacter /Enterobacter species	1	1
Morganella morganii	1	0
Methicillin resistant staph aureus	10	1
Serratia marcescens	2	1

Supplementary Table S2. Antibiotic resistance patterns.

Groups	Antibiotic Classes
R1	Penicillins, Fluoroquinolones, Tetracyclines, Sulfonamides, Aminoglycosides.
R2	Fluoroquinolones, Penicillins, Steroid Antibacterials (Fusidic Acid), Macrolides, Glycopeptides (Teicoplanin).
R3	Tetracyclines, Penicillins, Sulfonamides, Glycylcyclines (Tigecycline), Glycopeptides (Teicoplanin).
R4	Penicillins, Fluoroquinolones, Sulfonamides, Aminoglycosides, Glycopeptides (Teicoplanin).
R5	Fluoroquinolones, Penicillins, Sulfonamides, Aminoglycosides, Clindamycin (Lincosamides), Macrolides, Glycopeptides (Teicoplanin).
R6	Penicillins, Fluoroquinolones, Tetracyclines, Sulfonamides, Aminoglycosides, Glycylcyclines (Tigecycline), Clindamycin (Lincosamide), Macrolides.
R7	Fluoroquinolones, Penicillins, Sulfonamides, Aminoglycosides, Clindamycin (Lincosamides), Macrolides.
R8	Fluoroquinolones, Penicillins, Aminoglycosides, Steroid Antibacterials (Fusidic Acid), Glycopeptides (Teicoplanin), Cephalosporins.
R9	Penicillins, Fluoroquinolones, Tetracyclines, Aminoglycosides, Glycylcyclines (Tigecycline), Steroid Antibacterials (Fusidic Acid), Rifamycins (Rifampicin).
R10	Tetracyclines, Penicillins, Sulfonamides, Aminoglycosides, Clindamycin (Lincosamides), Macrolides, Glycopeptides (Teicoplanin), Cephalosporins.
R11	Penicillins, Fluoroquinolones, Tetracyclines, Sulfonamides, Aminoglycosides, Glycylcyclines (Tigecycline), Steroid Antibacterials (Fusidic Acid), Clindamycin (Lincosamides), Glycopeptides (Vancomycin), Macrolides, Glycopeptides (Teicoplanin).
R12	Penicillins, Fluoroquinolones, Tetracyclines, Sulfonamides, Aminoglycosides, Macrolides, Glycopeptides (Teicoplanin).
R13	Penicillins, Fluoroquinolones, Tetracyclines, Sulfonamides, Aminoglycosides, Glycylcyclines (Tigecycline), Clindamycin (Lincosamides), Macrolides, Glycopeptides (Teicoplanin).
R14	Penicillins, Fluoroquinolones, Tetracyclines, Sulfonamides, Aminoglycosides, Clindamycin (Lincosamide), Macrolides.
R15	Penicillin, Fluoroquinolones, Aminoglycosides, Macrolides.
R16	Tetracycline, Penicillins, Sulfonamides, Glycopeptides (Teicoplanin).
R17	Penicillins, Tetracyclines, Sulfonamides, Glycylcyclines (Tigecycline), Clindamycin (Lincosamides), Macrolides, Glycopeptides (Teicoplanin).
R18	Fluoroquinolones, Penicillins, Glycylcyclines (Tigecycline).
R19	Fluoroquinolones, Tetracyclines, Penicillins, Sulfonamides, Clindamycin (Lincosamides), Glycopeptides (Teicoplanin).
R20	Fluoroquinolones, Penicillins, Sulfonamides, Oxazolidinones (linezolid), Steroid Antibacterials (Fusidic Acid), Rifamycins (Rifampicin), Amphenicols (Chloramphenicol), Glycopeptides (Vancomycin), Macrolides, Glycopeptides (Teicoplanin), Cephalosporins.

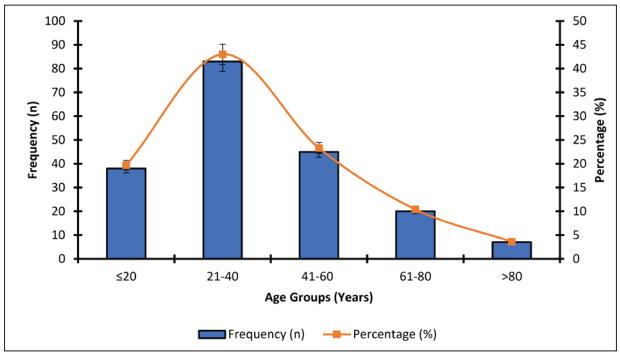
R21	Fluoroquinolones, Tetracyclines, Penicillins, Sulfonamides, Rifamycins (Rifampicin), Glycopeptides (Teicoplanin), Cephalosporins.
R22	Penicillins, Fluoroquinolones, Sulfonamides, Aminoglycosides, Glycylcyclines (Tigecycline), Steroid Antibacterials (Fusidic Acid), Clindamycin (Lincosamides), Macrolides, Glycopeptides (Teicoplanin).
R23	Fluoroquinolones, Penicillins, Sulfonamides, Aminoglycosides, Macrolides, Glycopeptides (Teicoplanin), Cephalosporins.
R24	Penicillins, Fluoroquinolones, Glycylcyclines (Tigecycline), Clindamycin (Lincosamides), Amphenicols (Chloramphenicol), Macrolides.
R25	Penicillins, Fluoroquinolones, Sulfonamides, Aminoglycosides, Macrolides, Glycopeptides (Teicoplanin).
R26	Penicillins, Sulfonamides.
R27	Penicillins, Fluoroquinolones, Tetracyclines, Sulfonamides, Rifamycins (Rifampicin), Macrolides, Glycopeptides (Teicoplanin).
R28	Penicillins, Fluoroquinolones, Macrolides.
R29	Penicillins, Fluoroquinolones, Tetracyclines, Aminoglycosides, Glycylcyclines (Tigecycline)s, Glycopeptides (Teicoplanin).
R30	Penicillins, Fluoroquinolones, Tetracyclines, Aminoglycosides, Clindamycin (Lincosamides), Macrolides.
R31	Tetracyclines, Penicillins, Sulfonamides, Aminoglycosides, Macrolides.
R32	Penicillins, Fluoroquinolones, Sulfonamides, Macrolides.
R33	Penicillins, Tetracyclines, Sulfonamides, Aminoglycosides.
R34	Penicillins, Sulfonamides, Aminoglycosides, Steroid Antibacterials (Fusidic Acid), Macrolides, Glycopeptides (Teicoplanin).
R35	Penicillins, Fluoroquinolones, Tetracyclines, Aminoglycosides, Glycopeptides (Teicoplanin).
R36	Tetracyclines, Penicillins, Sulfonamides, Aminoglycosides, Clindamycin (Lincosamides), Macrolides, Glycopeptides (Teicoplanin).
R37	Fluoroquinolones, Tetracyclines, Penicillins, Clindamycin (Lincosamides), Macrolides, Glycopeptides (Teicoplanin).
R38	Penicillins, Fluoroquinolones, Tetracyclines, Sulfonamides, Aminoglycosides, Glycylcyclines (Tigecycline), Macrolides.
R39	Penicillins, Fluoroquinolones, Tetracyclines, Sulfonamides, Rifamycins (Rifampicin), Amphenicols (Chloramphenicol), Macrolides, Glycopeptides (Teicoplanin).
R40	Fluoroquinolones, Penicillins, Sulfonamides, Steroid Antibacterials (Fusidic Acid), Clindamycin (Lincosamides), Macrolides, Cephalosporins, Carbapenems.
R41	Fluoroquinolones, Penicillins, Steroid Antibacterials (Fusidic Acid).
R42	Fluoroquinolones, Penicillins, Sulfonamides, Steroid Antibacterials (Fusidic Acid).
R43	Fluoroquinolones, Penicillins, Sulfonamides, Aminoglycosides, Steroid Antibacterials (Fusidic Acid), Rifamycins (Rifampicin), Clindamycin (Lincosamides), Macrolides, Carbapenems, Cephalosporins.
R44	Fluoroquinolones, Penicillins, Sulfonamides, Steroid Antibacterials (Fusidic Acid), Clindamycin (Lincosamides), Cephalosporins, Carbapenems.
R45	Penicillins, Sulfonamides, Steroid Antibacterials (Fusidic Acid).
R46	Fluoroquinolones, Penicillins, Sulfonamides, Aminoglycosides, Clindamycin (Lincosamides), Macrolides, Cephalosporins, Carbapenems.
R47	Fluoroquinolones, Penicillins, Sulfonamides, Steroid Antibacterials (Fusidic Acid), Clindamycin (Lincosamides), Macrolides.
R48	Fluoroquinolones, Penicillins, Sulfonamides, Steroid Antibacterials (Fusidic Acid), Clindamycin, Amphenicols (Chloramphenicol), Macrolides.
R49	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Steroid Antibacterials (Fusidic Acid),
R50	Penicillins, Fluoroquinolones, Sulfonamides, Tetracyclines.
R51	Penicillins, Tetracyclines, Sulfonamides, Steroid Antibacterials (Fusidic Acid).
R52	Penicillins, Fluoroquinolones, Tetracyclines, Macrolides.
R53	Fluoroquinolones, Penicillins, Sulfonamides, Tetracyclines, Aminoglycosides, Steroid Antibacterials (Fusidic Acid), Macrolides.
R54	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Aminoglycosides, Glycylcyclines (Tigecycline), Amphenicols (Chloramphenicol), Cephalosporins, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R55	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Aminoglycosides, Amphenicols (Chloramphenicol), Cephalosporins, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R56	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Aminoglycosides, Cephalosporins, Carbapenems.
R57	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Aminoglycosides, Glycylcyclines (Tigecycline), Amphenicols (Chloramphenicol), Cephalosporins, Carbapenems.
R58	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Aminoglycosides, Glycylcyclines (Tigecycline), Cephalosporins, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R59	Fluoroquinolones, Penicillins, Tetracyclines, Aminoglycosides, Cephalosporins.
R60	Fluoroquinolones, Tetracyclines, Sulfonamides, Aminoglycosides, Cephalosporins, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).

R61	Fluoroquinolones, Sulfonamides, Aminoglycosides, Cephalosporins, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R62	Fluoroquinolones, Penicillins, Sulfonamides, Aminoglycosides, Cephalosporins, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R63	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Amphenicols (Chloramphenicol), Cephalosporins, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R64	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Aminoglycosides, Cephalosporins, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R65	Fluoroquinolones, Penicillins, Sulfonamides, Cephalosporins, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R66	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Amphenicols (Chloramphenicol), Cephalosporins.
R67	Penicillins, Tetracyclines.
R68	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Aminoglycosides, Cephalosporins.
R69	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Cephalosporins.
R70	Fluoroquinolones, Penicillins, Cephalosporins, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R71	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Aminoglycosides, Cephalosporins, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R72	Penicillins, Cephalosporins.
R73	Sulfonamides, Aminoglycosides, Cephalosporins.
R74	Fluoroquinolones, Penicillins, Sulfonamides, Cephalosporins, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R75	Fluoroquinolones, Sulfonamides, Aminoglycosides, Cephalosporins.
R76	Fluoroquinolones, Penicillins, Sulfonamides, Cephalosporins.
R77	Fluoroquinolones, Sulfonamides, Cephalosporins, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R78	Fluoroquinolones, Sulfonamides, Cephalosporins.
R79	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Cephalosporins, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R80	Fluoroquinolones, Penicillins, Tetracyclines, Cephalosporins, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R81	Sulfonamides, Cephalosporins.
R82	Sulfonamides.
R83	Cephalosporins, Polymyxins (Colistin & Polymyxin B).
R84	Fluoroquinolones, Aminoglycosides, Cephalosporins, Polymyxins (Colistin & Polymyxin B), Aztreonam.
R85	Fluoroquinolones, Sulfonamides, Aminoglycosides, Rifamycins (Rifampicin), Cephalosporins, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R86	Fluoroquinolones, Sulfonamides, Aminoglycosides, Carbapenems, Polymyxins (Colistin).
R87	Penicillins.
R88	Penicillins, Tetracyclines, Sulfonamides, Cephalosporins.
R89	Penicillins, Tetracyclines, Sulfonamides, Amphenicols (Chloramphenicol).
R90	Fluoroquinolones, Penicillins, Sulfonamides.
R91	Fluoroquinolones, Penicillins, Aminoglycosides, Cephalosporins, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R92	Fluoroquinolones, Penicillins, Tetracyclines, Cephalosporins.
R93	Fluoroquinolones, Penicillins, Aminoglycosides, Amphenicols (Chloramphenicol), Cephalosporins.
R94	Fluoroquinolones, Penicillins, Aminoglycosides, Cephalosporins.
R95	Aminoglycosides, Cephalosporins, Polymyxins (Colistin & Polymyxin B), Aztreonam.
R96	Cephalosporins.
R97	Aminoglycosides.
R98	Fluoroquinolones, Aminoglycosides, Cephalosporins, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R99	Fluoroquinolones, Cephalosporins.
R100	Fluoroquinolones.
R101	Aminoglycosides, Carbapenems, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R102	Penicillins, Fluoroquinolones, Tetracyclines, Clindamycin (Lincosamides), Amphenicols (Chloramphenicol), Macrolides, Cephalosporins.
R103	Tetracyclines, Clindamycin (Lincosamides), Macrolides.

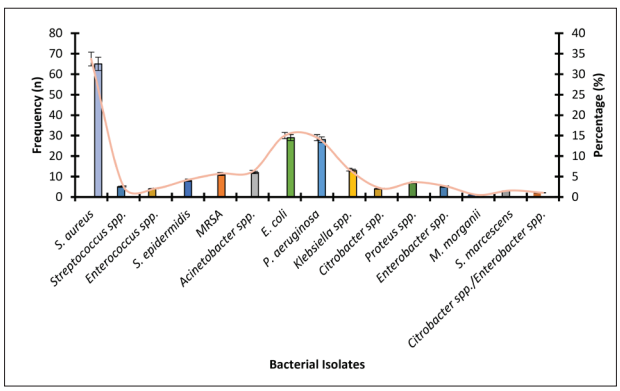
R104	Tetracyclines, Amphenicols (Chloramphenicol), Macrolides.
R105	Tetracyclines, Macrolides.
R106	Tetracyclines, Penicillins, Cephalosporins.
R107	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Aminoglycosides, Glycylcyclines (Tigecycline), Amphenicols (Chloramphenicol), Cephalosporins.
R108	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Amphenicols (Chloramphenicol), Cephalosporins, Polymyxins (Colistin & Polymyxin B).
R109	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Aminoglycosides, Amphenicols (Chloramphenicol), Cephalosporins.
R110	Fluoroquinolones, Steroid Antibacterials (Fusidic Acid), Macrolides.
R111	Steroid Antibacterials (Fusidic Acid), Macrolides.
R112	Fluoroquinolones, Tetracyclines.
R113	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Glycylcyclines (Tigecycline), Amphenicols (Chloramphenicol), Cephalosporins, Beta-lactamase-inhibitor (Piperacillin+Tazobactam).
R114	Sulfonamides, Tetracyclines, Aminoglycosides.
R115	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Steroid Antibacterials (Fusidic Acid), Clindamycin.
R116	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Macrolides, Cephalosporins.
R117	Fluoroquinolones, Sulfonamides, Macrolides.
R118	Fluoroquinolones, Penicillins, Aminoglycosides, Steroid Antibacterials (Fusidic Acid).
R119	Fluoroquinolones, Clindamycin (Lincosamides), Macrolides.
R120	Fluoroquinolones, Penicillins, Aminoglycosides, Macrolides.
R121	Penicillins, Sulfonamides, Cephalosporins.
R122	Tetracyclines, Sulfonamides.
R123	Fluoroquinolones, Penicillins, Tetracyclines, Sulfonamides, Aminoglycosides, Steroid Antibacterials (Fusidic Acid), Rifamycins (Rifampicin), Clindamycin (Lincosamides), Macrolides, Cephalosporins, Carbapenems,
R124	Penicillins, Steroid Antibacterials (Fusidic Acid), Clindamycin (Lincosamides).
R125	Penicillins, Sulfonamides, Steroid Antibacterials (Fusidic Acid), Clindamycin (Lincosamides), Macrolides, Cephalosporins.
R126	Fluoroquinolones, Penicillins, Sulfonamides, Aminoglycosides, Steroid Antibacterials (Fusidic Acid), Clindamycin (Lincosamides), Macrolides, Cephalosporins, Carbapenems.
R127	Fluoroquinolones, Penicillins, Tetracyclines, Steroid Antibacterials (Fusidic Acid), Clindamycin (Lincosamides)
R128	Penicillins, Steroid Antibacterials (Fusidic Acid), Macrolides.
R129	Fluoroquinolones, Penicillins, Steroid Antibacterials (Fusidic Acid), Macrolides.

Supplementary Table S3. Frequency of MDR, XDR and PDR bacteria in male and female populations of different age groups.

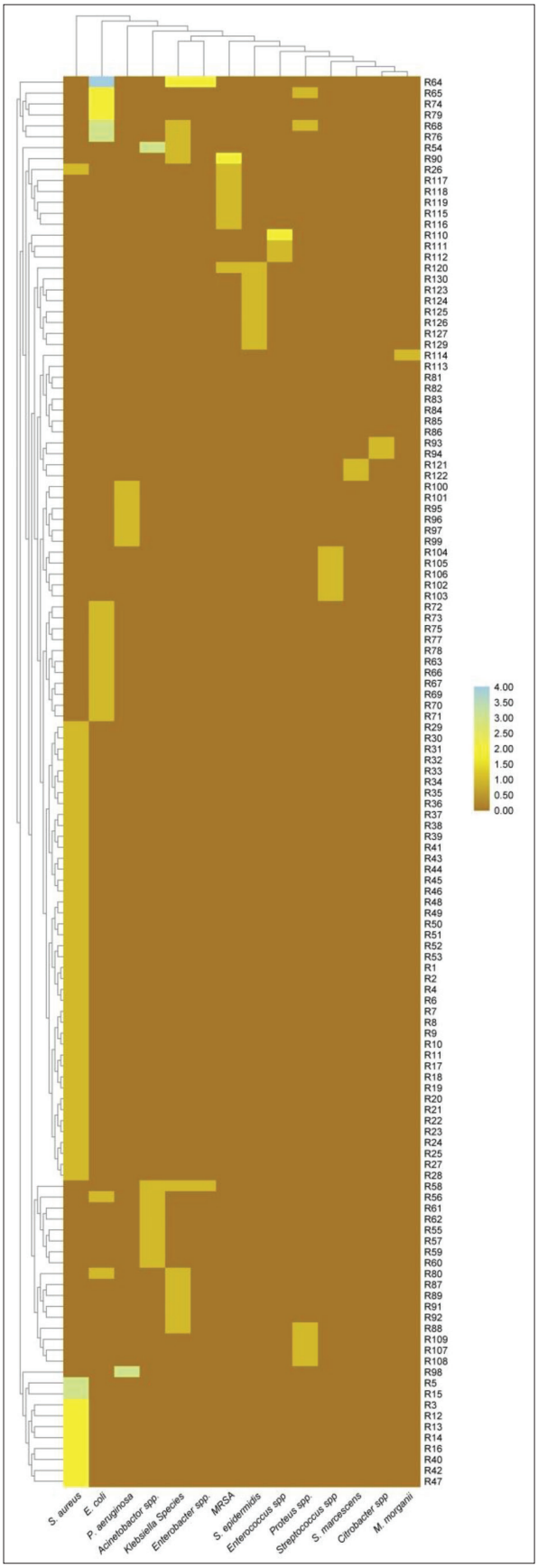
Gender	Non MDR	MDR	XDR	PDR	Total
Male	28	93	20	1	142
Female	8	36	6	1	51
Age Groups (years)					
0-20 years	2	30	5	1	38
21-40 years	12	56	14	1	83
41-60 years	13	26	6	0	45
61-80 years	7	12	1	0	20
81-100 years	2	5	0	0	7



Supplementary Fig. S1. Prevalence of wound infection in different age groups.



Supplementary Fig. S2. Prevalence of bacterial species in wound infections.



Supplementary Fig. S3. Heat map representation of the diversity in antibacterial resistance patterns of the isolated bacteria.