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ORIGINAL RESEARCH

Characterization of Bacterial Spectrum and Antimicrobial Resistance Patterns in Wound Infections from a Chinese Hospital

Hazrat Bilal[1], Muhammad Nadeem Khan[2], Muhammad Shafiq[3], Umar Farooq[4], Sabir Khan[4]

[1]Jiangxi Key Laboratory of Oncology (2024SSY06041), Jiangxi Cancer Hospital & Institute, the Second Affiliated Hospital of Nanchang Medical College, Nanchang, Jiangxi, 330029, People's Republic of China; [2]Department of Cell Biology & Genetics, Shantou University Medical College, Shantou, People's Republic of China; [3]Research Institute of Clinical Pharmacy, Shantou University Medical College, Shantou, People's Republic of China; [4]Jinfeng Laboratory, Chongqing, People's Republic of China

Correspondence: Sabir Khan; Umar Farooq, Email sabir_khan182@yahoo.com; umarfarooq@jflab.ac.cn

Background: Wound infections caused by antimicrobial-resistant bacteria challenge treatment and infection control, particularly in healthcare settings of underdeveloped regions, such as Shantou.

Methods: This retrospective study analyzed bacterial spectrum and antimicrobial resistance patterns of wound isolates collected from January 2015 to December 2023 at the Skin and Venereal Disease Prevention and Control Hospital, Shantou, China. Patient data (type, age, sex, infection site) were extracted independently by two researchers. Bacteria were identified using the MALDI Biotyper System or VITEK 2 COMPACT System. Data were analyzed with GraphPad Prism 8.0.2, using the chi-square test for comparisons; $p < 0.05$ was considered statistically significant.

Results: A total of 790 wound specimens were processed using standard microbiological techniques. *Staphylococcus aureus* was the most prevalent pathogen (39.4%), followed by *Klebsiella pneumoniae* (12.4%), *Pseudomonas aeruginosa* (6.89%), *Staphylococcus haemolyticus* (6.21%), *Staphylococcus epidermidis* (5.88%) and *Escherichia coli* (5.21%). Of these, male patients accounted for 363 of 420 (86.4%) cases, more than females, who accounted for 232 of 370 (62.7%). The highest incidence occurred in the 2–17-year age group (25.0%), followed by those aged 51–60 years (14.9%). Extended-spectrum β-lactamase (ESBL) production was noted in 41.9% of *E. coli* and 29.7% of *K. pneumoniae*. Among the Gram-positive multidrug-resistant (MDR) isolates, *S. haemolyticus* (91.8%), *S. epidermidis* (91.4%), and *S. aureus* (73.9%) were the most resistant. Methicillin-resistant *Staphylococcus aureus* (MRSA) accounted for 68.5% of *S. aureus*, with high resistance to penicillin (97.3%) and erythromycin (73.5%). MR-CoNS, mainly *S. epidermidis*, was resistant to erythromycin (88.5%) and oxacillin (77.1%).

Conclusion: The high prevalence of MRSA (68.5%) and MDR *S. aureus* (73.6%) may reflect widespread antibiotic misuse, emphasizing the need for culture-guided therapy and robust antibiotic stewardship.

Keywords: bacterial spectrum, wound infections, antimicrobial resistance trends, extended-spectrum β-lactamase, ESBL, methicillin-resistant *Staphylococcus aureus*, MRSA

Introduction

Wound infections occur due to complex interactions between the host immune system, potential pathogens, and external environmental factors. They can vary significantly in severity, ranging from mild, self-healing, to potentially fatal conditions. The site of the wound determines the capacity of bacterial pathogens to invade the tissue.[1] The most frequently isolated bacterial pathogens from wound infections include *S. aureus, S. epidermidis, S. haemolyticus, Acinetobacter* spp., *E. coli, Klebsiella* spp., *Enterobacter* spp., *Citrobacter* spp., and anaerobes, such as *Clostridium* spp. and *Peptostreptococcus* spp.[2,3] Studies suggest that hospital-acquired wound infections are the main cause of morbidity, with 0–80%, due to patients' vulnerability, hazardous microbes, and suboptimal healthcare practices in clinical settings.[2–5] These infections are typically treated using topical, oral, or intravenous antibiotics. However, the

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misuse and overuse of these drugs have led to the emergence of drug-resistant strains, posing significant challenges to global health, particularly in the underdeveloped regions of China, such as Shantou.

Among the resistant strains related to wound infections, Methicillin-resistant *Staphylococcus aureus* (MRSA) is a highly reported isolate.[6,7] MRSA significantly affects large surface areas of the skin or deeper soft tissues, causing various conditions, including abscesses, cellulitis, burns, and deep ulcers.[8,9] Enterobacteriaceae producing extended-spectrum β-lactamases (ESBL) are commonly involved in wound infections. In addition to ESBL-, carbapenemase-, and plasmid-mediated AmpC enzyme-producing strains, other types of resistant bacteria have also been reported in wound infections.[10] Rising antibiotic resistance can worsen infections, delay wound healing, prolong hospital stays, and increase healthcare costs.[11,12]

Data on the prevalence of ESBL-producing bacteria as a cause of wound infections in China are scarce,[13] creating a significant gap in understanding antimicrobial resistance (AMR) epidemiology, particularly in densely populated regions, such as Shantou, which face various healthcare challenges. Although our previous study analyzed microbial species and biomarker associations for wound infections using data from 2022 to 2024,[14] it primarily focused on biomarker correlations and included fungal pathogens, without examining longitudinal trends. In contrast, the present 9-year study (2015–2023) investigates bacterial spectrum and antimicrobial resistance patterns of MRSA, MRCoN, multidrug-resistant (MDR) bacteria, and ESBL-producing gram-negative strains, including inpatient–outpatient comparisons. This broader analysis addresses existing gaps and provides evidence-based insights for optimizing treatment strategies for wound infections in eastern Guangdong and similar settings.

Methods

Study Design

This retrospective study was conducted at Skin and Venereal Disease Prevention and Control Hospital in Shantou City, Guangdong, China. The hospital in question is the only tertiary care center for skin and venereal diseases in eastern Guangdong, providing healthcare services to 4,574,000 individuals. The study period was from January 2015 to December 2023.

Study Population and Data Collection

The source population consisted of all patients presenting with clinical signs of wound infection, such as redness, discomfort, swelling, persistent discharge, and foul odor, from which samples were sent to the hospital laboratory for microbiological culture. Patients included in the study were those presenting with clinical signs of wound infection and with complete clinical records available in the hospital electronic database. Patients were excluded if they had received antibiotics within 30 days prior to sample collection, had polymicrobial infections or infections caused by non-bacterial pathogens (eg, fungi), or if duplicate samples or repeated admissions were present; in such cases, only the first episode of infection per patient was analyzed. A total of 790 specimens, including 435 (55.0%) pus samples and 355 (44.9%) fine needle aspirates, were obtained.[15,16] The study received ethical approval from the hospital, which adhered to the standards of the Helsinki Declaration (Letter number: 2022–101). Data on patient type, age, sex, and infection site were collected from the hospital's electronic records and extracted from an Excel file (2021) by two researchers, SK and HB, independently. Subsequently, the files underwent cross-checking and comparison to remove any possible bias. Patients with incomplete clinical records, prior antibiotic use within 30 days, or polymicrobial infections were excluded from this study. Moreover, specimens cultured with non-bacterial pathogens were excluded to ensure clarity when assigning infections to MRSA or ESBL-producing Enterobacteriaceae. All eligible patients with wound infections were included to minimize selection bias. Duplicates or repeated admissions were excluded by analyzing only the first episode of infection per patient.

Routine Laboratory Protocol

Sample Collection

Pus specimens were collected from the wounds of patients in hospital wards using sterile cotton swabs and fine-needle syringes (FNSs).[17] Each sample was subsequently labeled with the patient's details, collection method, and date and time of collection. Patient information, including the infection site, signs and symptoms, underlying conditions, and previous antibiotic treatment, was recorded. The wound infection area was washed with sterile normal saline. A sterile cotton swab was then gently rolled over the wound surface to collect the samples. The pus-filled swabs were stored in sterile test containers with lids and labeled with relevant details. FNSs were used to obtain pus samples from deep wounds. Specimens were collected from wounds on various body parts, including the legs, hands, back, abdomen, feet, breasts, chest, head, and neck. The needles were appropriately labeled, capped, and promptly sent to the laboratory for fine-needle aspiration cytology.

Sample Processing, Bacterial Culturing, and Bacteria Identification

Depending on the type and location of the wound, each specimen was visually examined for consistency, color, turbidity, and the presence of blood.[16–18] Pus specimens were inoculated onto blood, chocolate, MacConkey, and nutrient agar plates manufactured by Zhuhai Deere Bioengineering Co., Ltd., for the isolation of bacterial species, following standard clinical laboratory guidelines.[19] The isolated bacteria were identified using the MALDI Biotyper System (Bruker Daltonics GmbH, Bremen, Germany) or the VITEK 2 COMPACT System (bioMérieux, Marcy L'étoile, France), according to the manufacturer's instructions.

Analysis of Antimicrobial Susceptibility Patterns

Antimicrobial susceptibility testing was performed using the Vitek 2 system (bioMérieux, Marcy l' Étoile, France) according to the manufacturer's instructions. Antibiotics, including β-lactams, aminoglycosides, macrolides, tetracyclines, fluoroquinolones, glycopeptides, oxazolidinones, lincosamides, streptogramins, rifamycins, nitrofurans, sulfonamides, and β-lactam/β-lactamase inhibitor combinations, were used against both gram-positive and gram-negative isolates. All antibiotics, media, and reagents used in this study were purchased from Zhuhai Deere Bioengineering Co., Ltd. (Zhuhai, Guangdong, China). The data results were analyzed as "susceptible", "resistant", or "intermediate" according to the guidelines set by the Clinical and Laboratory Standards Institute (CLSI M100-S25, 2024).[20] Bacteria were classified as MDR if they exhibited resistance to at least one antibiotic in three or more classes.

MRSA and ESBL Producers' Confirmation

Methicillin-resistant bacteria were suspected based on the MIC results and were confirmed using cefoxitin disc diffusion tests.[21] ESBL-producing bacteria were detected among Enterobacteriaceae isolates using antibiotic discs containing 30 μg of cefotaxime, ceftazidime, ceftriaxone, and aztreonam.[20] Bacterial isolates with ceftazidime zone diameter <22 mm and cefotaxime zone diameter <27 mm were considered potential producers of ESBL according to standard guidelines. Enterobacteriaceae suspected of producing ESBL were confirmed using the double-disc synergy test (DDST).[22]

Quality Control and Statistical Analysis

A sterility check was performed on all the prepared biochemical and streaking media. The reference strains *E. coli* ATCC 25922 and *S. aureus* ATCC 25923 were used as quality controls for AST and biochemical tests. Similarly, *E. coli* ATCC 25922 was used as a negative control for the screening and phenotypic confirmation of DDST assays against ESBL-producing gram-negative bacilli.

GraphPad Prism version 8.0.2 was used for data analysis. Frequencies were computed for categorical variables. The chi-square test was used for comparative analysis, and a *p*-value of less than 0.05 was considered statistically significant.

Results

Distribution of Bacterial Species

A total of 790 pus samples from patients with clinical wound infections were collected and examined over a 9-year study period. Of the collected samples, 187 (23.6%) were from the legs, 173 (21.8%) from the arms, 132 (16.7%) from the torso, 103 (13.0%) from generalised/widespread regions, 94 (11.8%) from the face, 48 (6.07%) from the feet, 34 (4.30%) from the genital area, and 19 (2.40%) from the nails. Of these, 595 (75.3%) bacterial isolates were identified, of which 385 (64.7%) were gram-positive and 210 (35.2%) gram-negative.

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Among the 595 bacterial isolates, *S. aureus* was the predominant (n = 235, 39.4%), followed by *K. pneumoniae* (n = 74, 12.4%), *P. aeruginosa* (n = 41, 6.89%), *S. haemolyticus* (n = 37, 6.21%), *S. epidermidis* (n = 35, 5.88%), and *E. coli* (n = 31, 5.21%). The overall distribution of bacterial species is presented in Table 1. The predominant bacterial species were more frequently isolated from inpatients (n = 352, 59.1%) than outpatients (n = 243, 40.8%).

Clinical Characteristics of Patients Associated with Wound Infections

Among 420 male patients, 363 showed aerobic bacterial growth, whereas among 370 female patients, only 232 showed positive cultures. The highest incidence of wound infection was reported in patients aged 2–17 years, with 149 (25.0%) cases, followed by those aged 51–60-year age group, with 89 cases (14.9%). The incidence of bacterial infections increased over time (Table 1; Figure 1). The incidence of *P. aeruginosa* infection rose from 0.05 per 1,000 patients in 2015 to 0.10 per 1,000 patients in 2023. The annual incidence of wound infections is shown in Figures 2 and 3. Among gram-negative bacteria, *K. pneumoniae* and *E. coli* were more prevalent in 2019, whereas, among gram-positive bacteria, *S. aureus* was common in 2017. Of the 595 patients, 352 (59.1%) were inpatients in different wards and 243 (40.8%) were outpatients (Table 2).

Distribution of Skin Diseases

Among the skin infections, eczema was the most frequent (n = 125, 28.7%), followed by herpes zoster (n = 31, 7.12%), urethritis (n = 17, 3.90%), pemphigus erythematosus (n = 12, 2.75%), pemphigus vulgaris (n = 11, 2.52%), acne (n = 9, 2.06%), and bullous pemphigoid (n = 9, 2.06%).

The bacterial species identified in the eczema group included *S. aureus* (n = 48, 41.0%), *K. pneumoniae* (n = 21, 32.3%), *P. aeruginosa* (n = 11, 28.2%), *E. coli* (n = 8, 28.5%), *A. baumannii* (n = 7, 35.0%), *S. haemolyticus* (n = 5, 17.8%), *E. cloacae* (n = 6, 27.2%), and *S. epidermidis* (n = 4, 14.2%). Herpes zoster was associated with *E. cloacae* (n = 6, 27.2%), *E. faecalis* (n = 2, 12.5%), *S. aureus* (n = 4, 3.41%), *K. pneumoniae* (n = 4, 6.15%), *E. coli* (n = 3, 10.7%), *A. baumannii* (n = 2, 10.0%), *S. epidermidis* (n = 2, 7.14%), *S. haemolyticus* (n = 1, 3.57%), and *P. aeruginosa* (n = 1, 2.56%). Bacterial species associated with various skin infections are listed in Table S1.

Antibacterial Susceptibility of Gram-Positive Bacteria

The antibacterial resistance of the gram-positive bacteria is shown in Figure 4. *E. faecalis* isolates were 100% susceptible to ampicillin, moxifloxacin, penicillin, tigecycline, vancomycin, linezolid, and piracillin/tazobactam. In contrast, 94.4% and 75.0% of *E. faecalis* isolates were resistant to tetracycline and clindamycin, respectively. All staphylococcal isolates, including *S. aureus, S. epidermidis*, and *S. haemolyticus*, were 100% susceptible to quinupristin, linezolid, tigecycline, and vancomycin. In contrast, *S. aureus* showed 97.3% resistance to penicillin. *S. epidermidis* and *S. haemolyticus* exhibited 88.5% and 91.8% resistance, respectively, to erythromycin. Of the total isolates, 68.5% were MRSA strains and 97.3% were resistant to penicillin, followed by erythromycin (73.5%), clindamycin (70.3%), cefoxitin (55.5%), and oxacillin (53.2%). In 80.2% of the methicillin-resistant coagulase-negative staphylococci (MRCoNS), *S. epidermidis* was 88.5% resistant to erythromycin, followed by oxacillin (77.1%), clindamycin (68.5%), trimethoprim/sulfamethoxazole (62.8%), and ciprofloxacin (45.7%). *S. haemolyticus* was 94.5% resistant to penicillin, followed by erythromycin (91.8%), oxacillin (83.3%), ciprofloxacin (70.2%), and clindamycin (64.8%) (Table S2). *S. haemolyticus* constituted the highest proportion (91.8%) of gram-positive MDR isolates, followed by *S. epidermidis* (91.4%) and *S. aureus* (73.6%). Gram-positive MDR species in wound infections compared to non-MDR wound infections were statistically significant ($p = 0.05$) (Figure 5).

Antibacterial Susceptibility of Gram-Negative Bacteria

The antibacterial resistance of the gram-negative bacteria is shown in Figure 6. Among the bacterial isolates, *A. baumannii* demonstrated 100% susceptibility to ciprofloxacin, gentamicin, tobramycin, and imipenem, and 100% resistance to ampicillin. *E. coli* showed 100% susceptibility to amikacin, aureomycin, ertapenem, and imipenem. However, ceftriaxone and cefazolin showed susceptibility rates of 44.8% (13 of 29), and 6.25% (1 of 16). All the

Table 1 Distribution of Clinical Isolates from Wound Infections Based on Age Groups, Gender, and Occurrence in Inpatients per 1,000 Patients per Year

	Total n (%) 595 (100)	***A. baumannii* 20 (3.36)**	***E. coli* 31 (5.21)**	***E. cloacae* 23 (3.86)**	***E. faecalis* 18 (3.02)**	***K. pneumoniae* 74 (12.4)**	***P. aeruginosa* 41 (6.89)**	***S. aureus* 235 (39.4)**	***S. epidermidis* 35 (5.88)**	***S. haemolyticus* 37 (6.21)**	**Others 81 (13.6)**
Age in years											
0–1	21 (3.52)	2 (9.52)	–			1 (4.76)	–	6 (28.5)		1 (4.76)	11 (52.3)
2–17	149 (25.0)	1 (0.67)	3 (2.01)	1 (0.67)	2 (1.34)	6 (4.02)	4 (2.68)	120 (80.5)	8 (5.36)	–	4 (2.68)
18–30	61 (10.2)	3 (4.91)	3 (4.91)	1 (1.63)	–	5 (8.19)	2 (3.27)	26 (42.6)	9 (14.7)	5 (8.19)	21 (34.4)
31–40	39 (6.55)	1 (2.56)	4 (10.2)	2 (5.12)	1 (2.56)	3 (7.69)	2 (5.12)	18 (46.1)	2 (5.12)	1 (2.56)	5 (12.8)
41–50	63 (10.5)	1 (1.58)	3 (4.76)	2 (3.17)	1 (1.58)	12 (19.0)	3 (4.76)	23 (36.5)	4 (6.34)	7 (11.1)	56 (88.8)
51–60	89 (14.9)	3 (3.37)	7 (7.86)	5 (5.61)	3 (3.37)	17 (19.1)	8 (8.89)	19 (21.3)	5 (5.61)	9 (10.1)	13 (14.6)
61–70	88 (14.7)	7 (7.95)	5 (5.68)	6 (6.81)	8 (9.09)	15 (17.0)	11 (12.5)	5 (5.68)	4 (4.54)	8 (9.09)	19 (21.5)
71–80	65 (10.9)	1 (1.53)	4 (6.15)	6 (9.23)	3 (4.61)	10 (15.3)	9 (13.8)	18 (27.6)	3 (4.61)	4 (6.15)	7 (10.7)
Above 80	20 (3.36)	1 (5.0)	2 (10.0)	–	–	5 (25.0)	2 (10.0)	–	–	2 (10.0)	12 (60.0)
Gender											
Male	363 (65.6)	16	17	10	17	47	21	133	25	8	69
Female	232 (60.4)	4	14	13	1	27	20	101	10	29	13
Ratio	1.56:1.0	4.0:1.0	1.21:1.0	0.76:1.0	17.0:1.0	1.74:1.0	1.05:1.0	1.3:1.0	2.5:1.0	0.27:1.0	5.3:1.0
Incidence/1,000 patients											
2015	0.66	0.03	0.05	0.01	0.03	0.08	0.05	0.10	0.08	0.07	0.16
2016	0.78	0.01	0.03	0.03	0.02	0.12	0.03	0.33	0.05	0.06	0.10
2017	0.84	0.02	0.02	0.02	0.02	0.06	0.05	0.46	0.03	0.07	0.09

(*Continued*)

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Table 1 (Continued).

	Total n (%) 595 (100)	***A. baumannii* 20 (3.36)**	***E. coli* 31 (5.21)**	***E. cloacae* 23 (3.86)**	***E. faecalis* 18 (3.02)**	***K. pneumoniae* 74 (12.4)**	***P. aeruginosa* 41 (6.89)**	***S. aureus* 235 (39.4)**	***S. epidermidis* 35 (5.88)**	***S. haemolyticus* 37 (6.21)**	**Others 81 (13.6)**
2018	0.71	0.03	0.02	0.03	0.01	0.11	0.01	0.34	0.02	0.01	0.12
2019	0.92	0.06	0.08	0.03	0.01	0.13	0.05	0.35	0.07	0.03	0.11
2020	0.44	0.05	0.04	0.03	–	0.07	0.05	0.13	0.02	0.01	0.04
2021	0.53	–	0.02	0.03	0.03	0.07	0.03	0.23	0.02	0.04	0.06
2022	0.51	–	0.02	0.03	0.03	0.07	0.04	0.19	0.02	0.06	0.05
2023	0.57	–	0.03	0.02	0.03	0.03	0.10	0.21	0.04	0.02	0.09
Means	0.6622	0.033	0.034	0.025	0.022	0.082	0.045	2.35	0.038	0.041	0.091

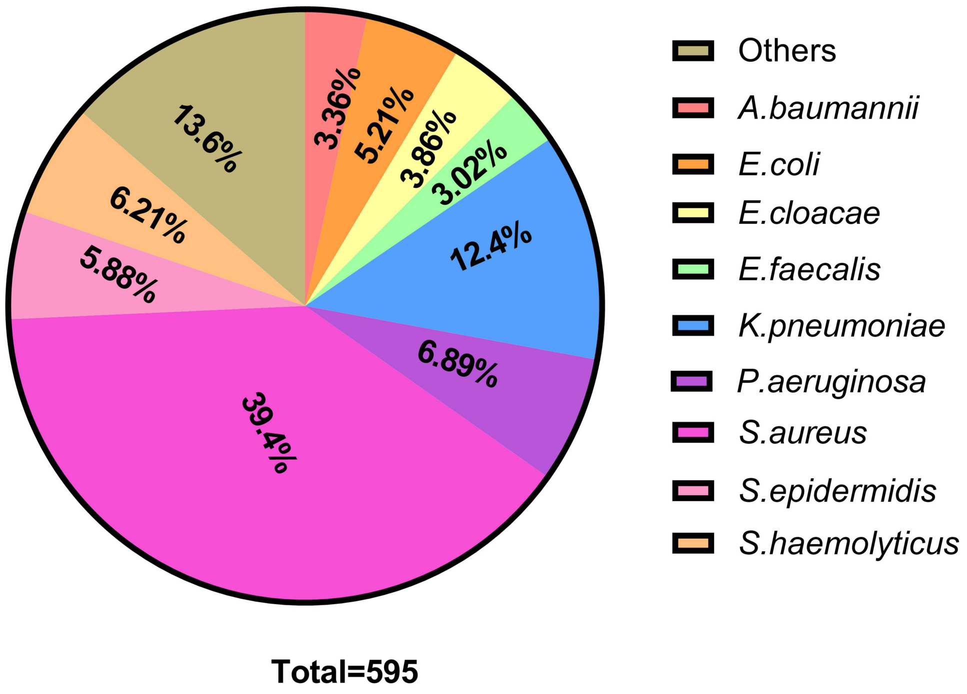

Figure 1 Distribution of bacteria causing wound infections.
Abbreviations: A, Acinetobacter; E. coli, Escherichia coli; E. faecalis, Enterococcus faecalis; K, Klebsiella; P, Pseudomonas; S, Staphylococcus.

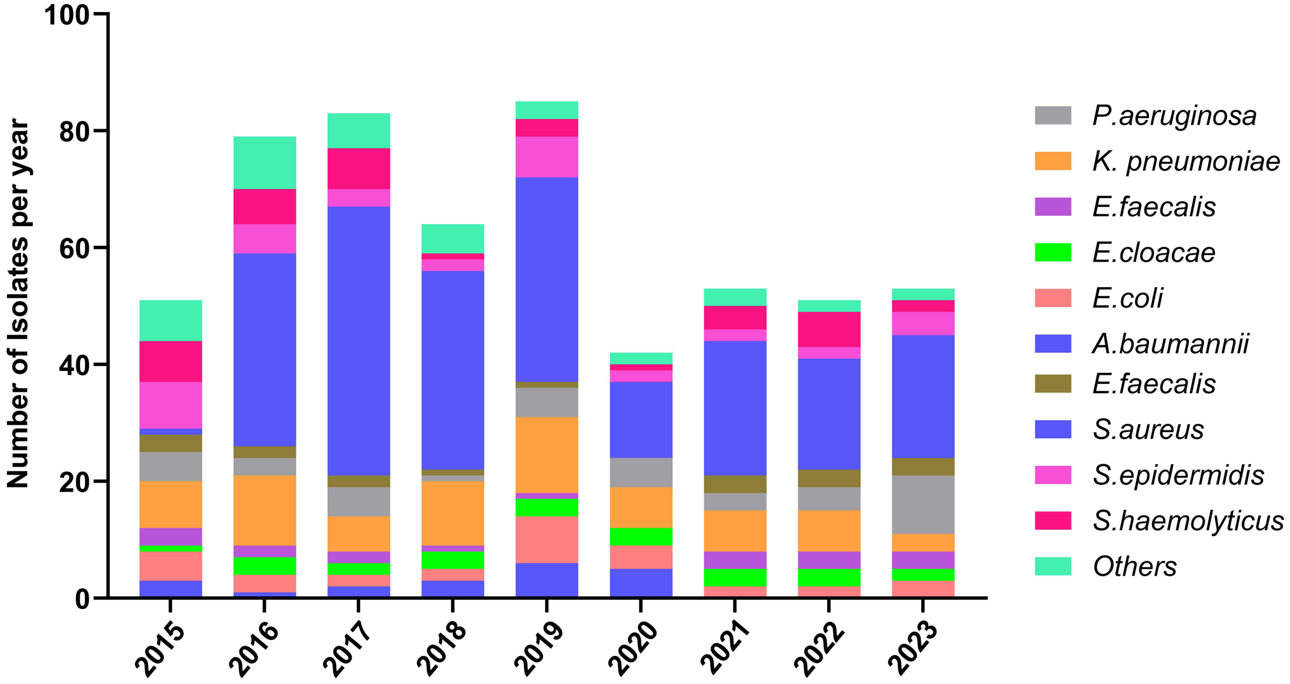

Figure 2 Prevalence of different bacteria causing wound infections over the years.
Abbreviations: A, Acinetobacter; E. coli, Escherichia coli; E. faecalis, Enterococcus faecalis; K, Klebsiella; P, Pseudomonas; S, Staphylococcus.

E. cloacae isolates were 100% susceptible to amikacin, ampicillin/sulbactam, ertapenem, gentamicin, ceftazidime, cefepime, vancomycin, and piperacillin/tazobactam. Nitrofurantoin showed the lowest susceptibility with a rate of 69.5% (16 of 23). Similarly, *K. pneumoniae* showed 97.1% susceptibility to cefepime and piperacillin/tazobactam,

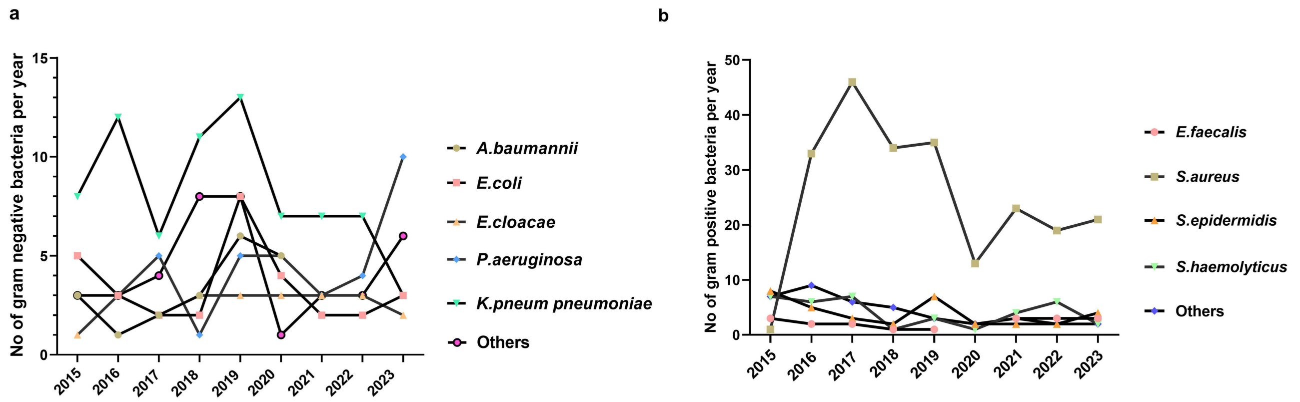

Figure 3 Trends of bacterial species causing wound infections over years. (**a**) Number of gram-negative bacteria causing wound infections per year. (**b**). Number of gram-positive bacteria causing wound infections per years.

with 40.5% showing intermediate resistance, and 28.3% showing resistance to nitrofurantoin. *P. aeruginosa* is 100% susceptible to aztreonam. In contrast, 100% of *P. aeruginosa* isolates were resistant to ampicillin/sulbactam, ampicillin, and trimethoprim/sulfamethoxazole. Of the 31 E. coli isolates, 13 (41.9%) were ESBL positive. Similarly, 22 isolates of *K. pneumoniae* (29.7%) and one isolate of *K. oxytoca* (16.6%) were positive for ESBL (Table S3). Among the gram-

Table 2 Comparative Analysis of Bacterial Isolates Among Inpatients and Outpatients Attending the Skin and Venereal Diseases Prevention and Control Hospital of Shantou City

Bacterial Isolates	Inpatient n (%)	Outpatient n (%)	Frequency n (%)	*P* Value
A. baumannii	2 (0.56)	18 (7.40)	20 (3.36)	0.001
A. lwoffii	1 (0.28)		1 (0.16)	–
Burkholderia cepacia	1 (0.28)		1 (0.16)	–
Citrobacter koseri		1 (0.41)	1 (0.16)	–
Chryseobacterium indologenes	1 (0.28)	–	1 (0.16)	–
Citrobacter freundii	1 (0.28)		1 (0.16)	–
E. coli	19 (5.39)	12 (4.93)	31 (5.21)	0.08
Elizabethkingia meningoseptica	–	1 (0.41)	1 (0.16)	–
A. *aerogenes*	1 (0.28)	1 (0.41)	2 (0.33)	0.99
E. cloacae	3 (0.85)	20 (8.23)	23 (3.86)	0.001
E. faecalis	14 (3.97)	4 (1.64)	18 (3.02)	0.001
E. faecium	–	1 (0.41)	1 (0.16)	–
Entero. gallinarum	–	2 (0.82)	2 (0.33)	–
K. oxytoca	4 (1.13)	3 (1.23)	7 (1.17)	0.59
K. ozaenae	1 (0.28)	–	1 (0.16)	–
A. *pneumoniae*	59 (16.7)	15 (6.17)	74 (12.4)	0.001
Morg. morganii	6 (1.70)	1 (0.41)	7 (1.17)	0.008

(*Continued*)

Table 2 (Continued).

Bacterial Isolates	**Inpatient n (%)**	**Outpatient n (%)**	**Frequency n (%)**	***P* Value**
Proteus mirabilis	1 (0.28)		1 (0.16)	–
P. aeruginosa	38 (10.7)	3 (1.23)	41 (6.89)	0.001
Ps. alcaligenes		1 (0.41)	1 (0.16)	–
Raou.ornithinolytica	1 (0.28)	–	1 (0.16)	–
Ps. fluorescens	2 (0.56)	1 (0.41)	3 (0.50)	0.41
Pantoea.agglomerans	1 (0.28)		1 (0.16)	–
Ser. marcescens	6 (1.70)	1 (0.41)	7 (1.17)	0.008
Sphingomonas paucimobilis	1 (0.28)	1 (0.41)	2 (0.33)	0.99
S. aureus	119 (33.8)	116 (47.7)	235 (39.4)	0.78
S. sciuri	1 (0.28)	–	1 (0.16)	–
S. cohnii. ssp. urealyticus	1 (0.28)	–	1 (0.16)	–
S. capitis	2 (0.56)	1 (0.41)	3 (0.50)	0.41
S. epidermidis	24 (6.81)	11 (4.52)	35 (5.88)	0.002
S. haemolyticus	23 (6.53)	14 (5.76)	37 (6.21)	0.06
S. hominis	6 (1.70)	2 (0.82)	8 (1.34)	0.05
S. ludgunensis	4 (1.13)	1 (0.41)	5 (0.84)	0.06
S. warneri	3 (0.85)	–	3 (0.50)	–
S. lentus	6 (1.70)	1 (0.41)	7(1.17)	0.008
S. agalactiae	–	10 (4.11)	10 (1.68)	–
Aeromonas hydrophila	–	1 (0.41)	1 (0.16)	–
Total	352 (59.1)	243 (40.8)	595 (100)	–

negative MDR isolates, *E. coli* was predominant (74.1%), followed by *E. faecalis* (61.1%), *K. pneumoniae* (45.9%), and *P. aeruginosa* (34.1%). The isolates with the lowest MDR rates included *A. baumannii* (25.0%) and *E. cloacae* (8.69%). Gram-negative MDR species in wound infections compared to non-MDR wound infections were statistically significant ($p = 0.05$) (Figure 5).

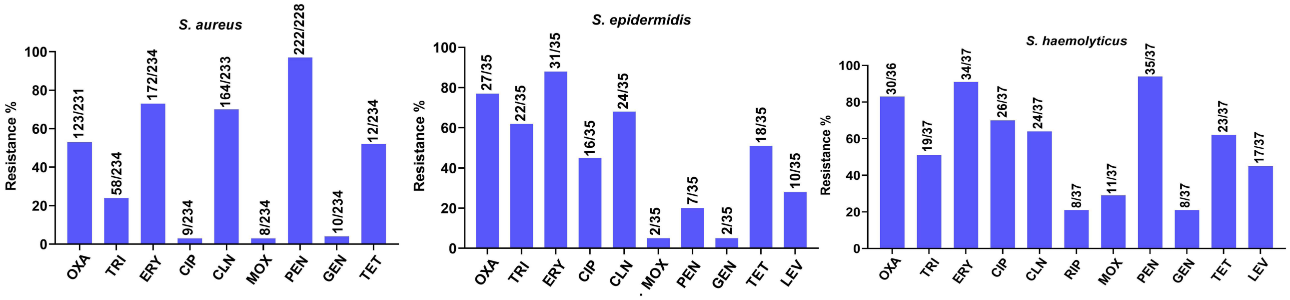

Figure 4 Antimicrobial resistance profiles of gram-positive bacterial isolates. The figure was drawn using GraphPad Prism version 8.0.2.

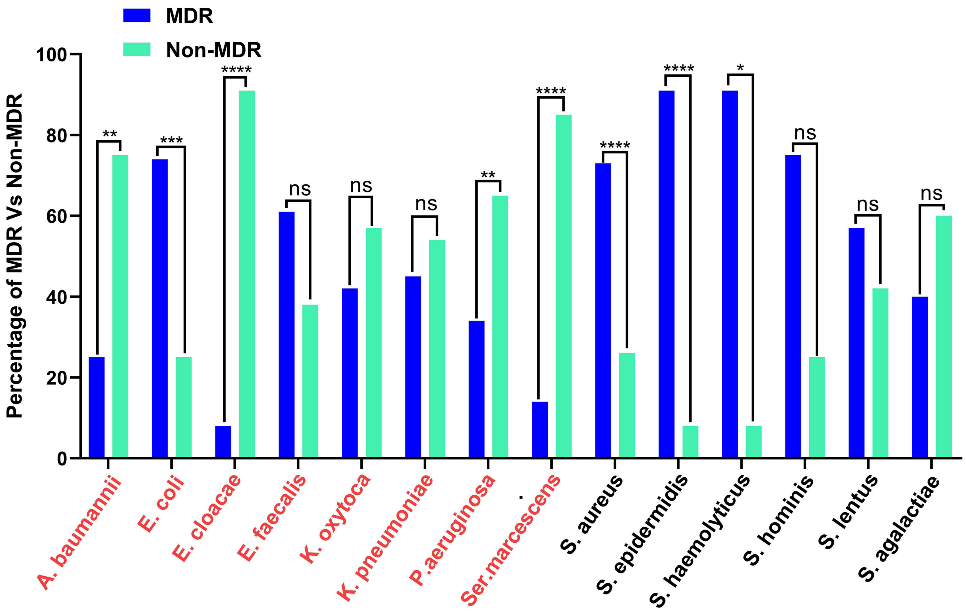

Figure 5 Comparison of multiple drug-resistant bacterial species with non-multiple drug-resistant species by using the Chi-squre test. The red species names indicate the Gram-negative bacteria, while black indicates Gram-positive bacteria. The compared values are shown. *, *P,* 0.05; **, *P,* 0.01; ***, *P,* 0.001; ****, *P,* 0.0001.
Abbreviation: ns, not significant.

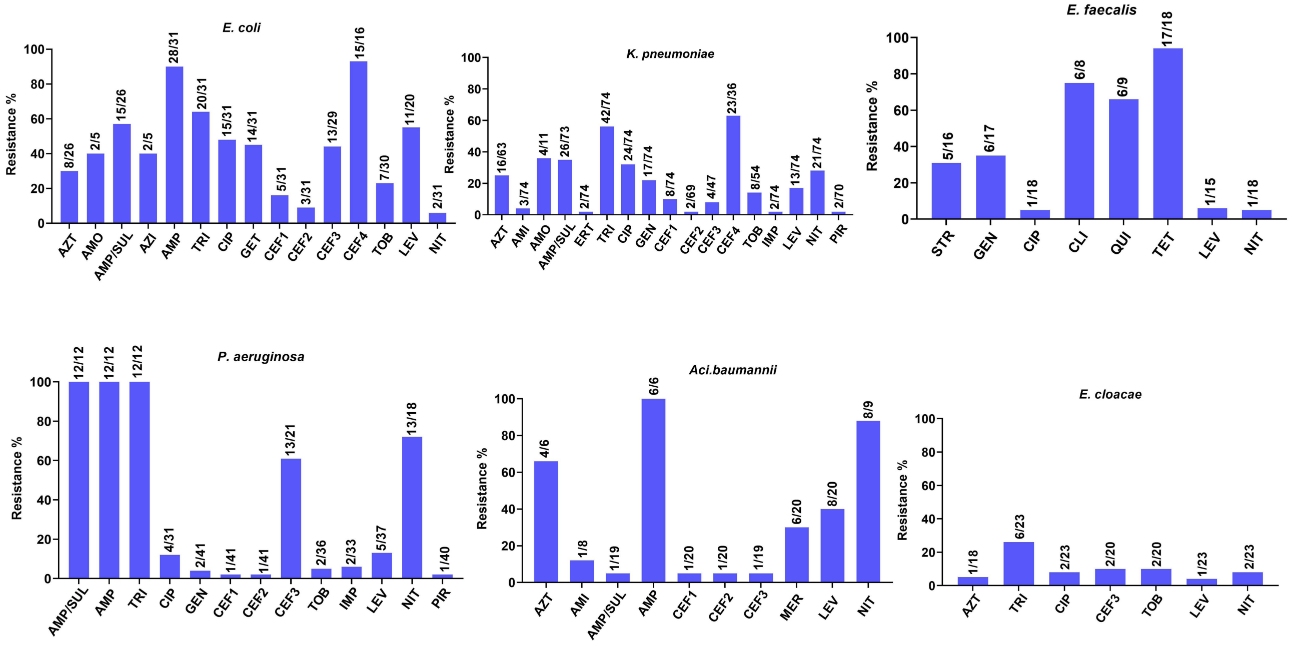

Figure 6 Antimicrobial resistance profiles of gram-negative bacterial isolates. The figure was drawn using GraphPad Prism version 8.0.2.

Discussion

This 9-year study identified various types of aerobic bacteria that cause wound infections using standard microbiological methods. Of the 595 bacteria-positive samples analyzed, 352 (59.1%) were from inpatients and 243 (40.8%) were from outpatients. The findings were nearly identical to those of a previously published study, in which 60.5% of the participants were inpatients and 39.5% were outpatients.[17] Wound infections have been reported to be high in inpatients, which may be due to prolonged hospital stay, surgical procedures, immunocompromised status, invasive procedures, and hospital-acquired infections.[23] The type and prevalence of bacteria associated with wound infections depend on various factors, including the type of wound, patient's immune status, risk of hospital-acquired infections, antibiotic use, geographic location, and hygiene practices.[24–26]

Among the age groups, the incidence of wound infection was highest in the 2–17-year-old age group, with 149 cases (25.0%). The increased incidence in the 2–17-year age group may be attributed to the development of the immune system.[27] Wound infections in male patients accounted for 363 of 420 (86.4%) cases, more than in females, 232 of 370 (62.7%). The high proportion of infections in the male population is consistent with previous studies in China.[28,29] This high rate may be due to increased socioeconomic activities and greater exposure of the male population compared to females.[30–32]

Among the bacterial isolates, the predominant species was *S. aureus* (n = 235, 39.4%), followed by *K. pneumoniae* (n = 74, 12.4%), *P. aeruginosa* (n = 41, 6.89%), *S. haemolyticus* (n = 37, 6.21%), *S. epidermidis* (n = 35, 5.88%), and *E. coli* (n = 31, 5.21%). The large proportion of *S. aureus*, reported in previous studies from China (29.2%), Nepal (50.0%), and Ethiopia (26.2%), differs from that in Sweden, where the predominant species was *S. pseudintermedius* (46.0%).[17,33,34] These differences may be due to variations in study populations, geographic distribution of bacterial species, or differences in infection sources. *S. aureus* is part of the normal flora of the human skin.[35] It is an opportunistic pathogen that causes wound infections owing to compromised host immunity or disruption of the skin barrier.[35] Moreover, *S. aureus* can colonize the skin, particularly in patients with compromised skin conditions such as atopic dermatitis.[36] Notably, *S. aureus* forms biofilms primarily in wound infections, thereby causing drug resistance by providing a protective environment for the bacteria and making them less susceptible to antibiotics.[37–39] All *S. aureus* isolates were 100% susceptible to vancomycin, which is consistent with the results of a previous study.[33] Similarly, other studies have reported that vancomycin resistance is typically <3% globally.[40] However, the growing trend of vancomycin resistance driven by the plasmid-mediated vanA gene is concerning, and careful use of this critical drug is essential.[41] High susceptibility to vancomycin is a vital treatment option; however, the high rates of resistance to other antibiotics necessitate urgent action to address antimicrobial resistance. In addition to vancomycin, other antibiotics, such as linezolid, tigecycline, and teicoplanin, are considered the most effective drugs in most studies, making them critical alternatives.[42] Among the staphylococcal isolates, 68.5% were MRSA strains, and 73.6% were MDR *S. aureus* strains. Our findings were higher than those of previous studies that reported MRSA strains ranging from 35.4% to 40.0%.[33,43] The high prevalence of MRSA and MDR *S. aureus* in our study may contribute to the widespread misuse or overuse of antibiotics in the region, which could lead to the selection of resistant strains. The results align with a previously published study from Nepal, which reported a 60.6% rate of cefoxitin-resistant MRSA.[17] The high resistance rates observed in our study emphasize the urgent need for antimicrobial resistance control programs and measures to manage the spread of resistant strains.

Among *K. pneumoniae* isolates, 32.4% were susceptible to ciprofloxacin. In a previous study, 35.7% of *K. pneumoniae* isolates were susceptible to ciprofloxacin.[44] Similarly, an analysis from China reported that ciprofloxacin resistance in *K. pneumoniae* has increased from 18.6% in 2006 to 44.61% in 2020.[45] Ciprofloxacin is a fluoroquinolone antibiotic used against *K. pneumoniae* to treat infections; however, the identified low susceptibility rate shows the growing challenge of antibiotic resistance in this pathogen. The high resistance of *K. pneumoniae* to ciprofloxacin is primarily due to mutations in *gyrA* and *parC*, as well as the horizontal transfer of plasmid-mediated *qnr* genes.[46] Additionally, efflux pump systems, such as *acrAB-tolC* and expelled ciprofloxacin from the cell, further contribute to resistance.[47] All *P. aeruginosa* were 100% resistant to ampicillin and 61.9% to ceftriaxone. It was previously reported that, among *P. aeruginosa* isolates, 34.8% were resistant to ampicillin and 27.3% were resistant to ceftriaxone.[33] The high resistance of *P. aeruginosa* may be due to intrinsic resistance mechanisms, such as low outer membrane permeability and the production of beta-lactamases and efflux pumps (eg, MexXY) that actively expel antibiotics from the cell.[48] Moreover, the potential for horizontal gene transfer (HGT) of resistance genes such as *blaCTX-M, blaTET*, and *blaSHV* among bacterial species has contributed to the increase in resistance.[49] Molecular characterization of resistance mechanisms in our locality is necessary to understand the genetic basis of resistance. *P. aeruginosa* caused wound infections increased from 0.05 cases per 1,000 patients in 2015 to 0.10 cases per 1,000 patients in 2023. Thus, wound infections caused by *P.* aeruginosa are a serious concern for the local population and may exacerbate the burden on the healthcare system through prolonged hospital stays, increased treatment costs, and the need for specialized therapies. The World Health Organization (WHO) 2024 bacterial pathogen list recently classified *P. aeruginosa* as a high-priority pathogen, emphasizing the urgent need for new antibiotics and enhanced infection control strategies to combat its spread.[50]

In the 80.2% MRCoNS strains, *S. epidermidis* was 100% resistant to penicillin, followed by erythromycin (88.5%), oxacillin (77.1%), clindamycin (68.5%), and ciprofloxacin (45.7%). Resistivities of penicillin (100%), oxacillin (100%), erythromycin (80.0%), clindamycin (80.0%), and levofloxacin (32.3%) against *S. epidermidis* have been reported previously.[51] The widespread resistance to beta-lactams may be due to the presence of the *mecA* gene in MRCoNS, which interferes with methicillin resistance by altering penicillin-binding proteins (PBPs).[52] The high resistance to erythromycin and clindamycin may contribute to the clonal dissemination of strains carrying *erm* genes, which alters macrolide-lincosamide-streptogramin B (MLSB) resistance.[53] Among the MDR strains, 91.8% and 91.4% were identified as *S. haemolyticus* and *S. epidermidis*, respectively. Several studies have been published on MDR bacterial strains, with a 10–59% occurrence rate in wound infections.[51,54–56] These differences may be due to regional antibiotic prescription practices, prolonged hospital stays, enhanced exposure to nosocomial pathogens, or lack of robust antimicrobial stewardship programs in our hospital setting.

All *A. baumannii* isolates were resistant to multiple drugs in the current study. Over the past decade, numerous nosocomial outbreaks caused by *Acinetobacter* species have been reported worldwide due to their ability to persist on surfaces, resist disinfectants, and acquire resistance genes through mobile genetic elements. Considering this emerging species, health care officials require special attention to mitigate this hazard.[57] *A. baumannii* showed lower resistance to amikacin (12.5%) and meropenem (30.0%). The lowest resistivity of amikacin (13.3%) and meropenem (26.7%) against *A. baumannii* has been reported previously.[33] However, due to the worldwide occurrence of plasmid-mediated carbapenem-resistant genes, such as blaKPC-2, blaNDM-1, blaIMP, and blaVIM, careful use of meropenem and imipenem is required.[58,59]

Similarly, 44.8% of *E. coli* isolates were susceptible to ceftriaxone, and 6.25% were susceptible to cefazolin, indicating high rates of resistance to these third- and first-generation cephalosporins. In a previous study, 40.6% and 43.5% of *E. coli* isolates were reported to be resistant to ceftriaxone and cefazolin, respectively.[33] Resistance is possibly due to the widespread prevalence of ESBLs (eg, *blaCTX-M* and *blaTEM*) and AmpC beta-lactamases, which hydrolyze cephalosporins and are often plasmid-mediated, facilitating horizontal gene transfer in the clinical setting.[60,61] *E. coli* plays a significant role in nosocomial infections, as demonstrated by its high rate of antibiotic resistance.[62] All *E. faecalis* isolates were 100% susceptible to vancomycin and 94.4% were resistant to tetracycline. Similarly, Guan et al reported 100% vancomycin susceptibility and 79.2% tetracycline resistance in *E. faecalis*.[33] This shows that the resistance of *E. faecalis* to tetracycline was high in the Shantou region, which might be due to the misuse and easy availability of this drug.

In total, 74.1% of *E. coli*, 61.1% of *E. faecalis, and* 8.69% of *E. cloacae* isolates were identified as MDR strains. *E. coli* MDR strains were predominant among gram-negative bacteria. This predominantly aligns with *E. coli*'s ability to acquire resistance genes using plasmids, integrons, and transposons.[63] In the current study, 41.9% of *E. coli* and 29.7% of *K. pneumoniae* isolates were ESBL-producing. A previous study in Ethiopia reported that 13.51% of *E. coli* and 16.55% of *K. pneumoniae* isolates produced ESBL. Another study reported that 25% of *E. coli* and 40% of *K. pneumoniae* isolates from Nepal were ESBL producers.[17,64] In Iraq, it was reported that 87.6% of isolates were ESBL-positive.[65]

Wound infections are a significant public health challenge, particularly in developing countries.[2] Wound infections, which are common nosocomial conditions, account for up to 80% of deaths in high populations, including burn victims, immunocompromised patients, diabetic ulcers, infection type, and pathogen virulence.[5] Antimicrobial resistance has become an increasingly serious problem. Therefore, modern infection control and preventive methods cannot completely prevent wound infections.[66] Antimicrobial resistance patterns can shift rapidly, as demonstrated by the present results compared to previous studies. Antimicrobial resistance can develop rapidly among pathogens owing to multiple factors, including overuse and underuse of antibiotics. MRSA and ESBL-producing bacteria pose significant threats to wound management and control.[29,33,67,68]

This retrospective study has some limitations that warrant consideration. This study was conducted at a single hospital; therefore, the results may not be applicable to all patients with wound infections or to other healthcare settings.

The distribution and prevalence of wound infections vary greatly, depending on the institution. Variations in patient demographics, local antibiotic prescription practices, and hospital-specific infection control protocols can significantly influence the pathogen distribution and resistance patterns.[69,70] Furthermore, potential limitations include the retrospective design, exclusion of polymicrobial infections, and possible misclassification of commensal CoNS. Future studies should consider prospective designs, include molecular characterization of isolates, and assess broader epidemiological factors influencing antimicrobial resistance. However, the study findings help develop strategies for improving the hospital's management of wound infections in our locality and in similar settings. The absence of molecular characterization restricts our study from tracking transmission pathways or confirming clonal outbreaks. Similarly, biofilm-forming capacity, an important virulence factor in chronic wounds, was not observed, leaving gaps in our understanding of treatment failure.

Conclusion

This study identified *S. aureus, K. pneumoniae, P. aeruginosa, S. haemolyticus, S. epidermidis*, and *E. coli* as the predominant pathogens in wound infections. MDR strains were highly prevalent, including 91.8% of *S. haemolyticus*, 91.4% of *S. epidermidis*, and 74.1% of *E. coli*, while ESBL production was noted in 41.9% of E. coli and 29.7% of *K. pneumoniae*. These findings highlight the serious threat posed by MDR and ESBL-producing bacteria to wound management, particularly in developing regions. Culture-guided therapy is essential to prevent overuse of broad-spectrum antibiotics, reserving vancomycin for MRSA and carbapenems for ESBL producers. Implementation of MRSA decolonization protocols, antimicrobial stewardship programs, and regional resistance surveillance systems is critical. Rapid molecular diagnostics and targeted interventions are urgently needed to limit the spread of resistant strains and ensure effective wound infection management.

Institutional Review Board Statement

This study was approved by the Medical Ethical Review Committee for conducting scientific research and technical projects at the Second Affiliated Hospital of Shantou University Medical College, China, which adhered to the standards of the declaration of Helsinki (Letter No. 2022-101).

Patient Consent Form

The data were obtained from the hospital record as a secondary source; no patient image or figure is involved. Therefore, informed consent was waived by the Medical Ethics Review Committee for conducting scientific research and technical projects at the Second Affiliated Hospital of Shantou University Medical College, China.

Data Sharing Statement

All data are presented in the manuscript; any raw data are available upon request from the corresponding author (email: sabir_khan182@yahoo.com).

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest in this work.

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