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High Prevalence of Multidrug-Resistant Bacteria in Wound Infections in Atbara, Sudan

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Abstract

The study aimed to determine the prevalence of multidrug-resistant (MDR) bacteria in wound infections at Atbara Hospital, Sudan. A descriptive analytical approach was used, analyzing 100 wound swabs from patients with clinically evident infections. Bacterial isolates were identified and subjected to antibiotic susceptibility testing following standard microbiological protocols and the Kirby-Bauer disk diffusion method. The main Results**:** Bacterial growth occurred in 85% (85/100) of the samples. Gram-negative bacteria predominated at 59% (n=50), while *Staphylococcus aureus* was the most common individual isolate at 33% (n=33). Alarmingly, *S. aureus* exhibited a 78.8% resistance rate to vancomycin, while cephalosporins demonstrated minimal efficacy (ceftazidime sensitivity was 3.12%). Amikacin showed the highest overall sensitivity rate at 69.7%, followed by meropenem at 58.93%. Furthermore, multidrug resistance was observed in 72% of the Gram-negative isolates ($p < 0.001$). The study reveals a critical burden of multidrug-resistant microorganisms in wound infections in Atbara. Routine culture and susceptibility testing, antimicrobial stewardship, and molecular surveillance are urgently required to guide therapy and curb the spread of resistance. The study recommends implementing antimicrobial stewardship programs to reduce empirical overuse and guide initial treatment, alongside establishing a molecular surveillance system to monitor emerging resistance mechanisms and control the spread of resistant strains.

Keywords: Antimicrobial resistance (AMR), wound infections, multidrug-resistant bacteria (MDR), Atbara, Sudan, *Staphylococcus aureus*, vancomycin, amikacin.

Introduction

Wound infections impose substantial clinical and economic burdens globally, particularly in low- and middle-income countries (LMICs) where diagnostic and therapeutic resources remain constrained [1]. The skin's integrity, when compromised through trauma, surgical intervention, or chronic conditions such as diabetes mellitus, creates portals for opportunistic pathogens to colonize and infect underlying tissues. Such infections frequently result in delayed wound healing, sepsis, limb amputation, and increased healthcare costs [2,3].

The escalating global antimicrobial resistance (AMR) crisis has further complicated wound infection management. In Africa, fragile healthcare infrastructure, unregulated antibiotic availability, limited infection prevention measures, and insufficient laboratory capacity have accelerated the dissemination of multidrug-resistant organisms (MDROs) [4,5]. The WHO African Region reports some of the highest AMR rates globally, with resistance to third-generation cephalosporins exceeding 80% in several countries [6].

In Sudan, published studies from Khartoum, Wad Medani, and Gadarif have documented concerning resistance patterns, including methicillin-resistant *Staphylococcus aureus* (MRSA) prevalence of 35–45% and carbapenem resistance in *Pseudomonas aeruginosa* approaching 30% [7–9]. However, data from

Atbara, Nile River State—a major regional referral center serving a large catchment population—remain notably absent, creating a critical evidence gap for local antimicrobial guidance.

The emergence of extended-spectrum beta-lactamase (ESBL)-producing Enterobacteriaceae and vancomycin-resistant *S. aureus* (VRSA) represents particular concern in Sudan, as these organisms limit therapeutic options and are associated with increased mortality [10,11]. Recent genomic studies have identified diverse resistance plasmids circulating in the region, suggesting horizontal gene transfer as a key mechanism driving resistance spread [12].

This study aimed to address the knowledge gap by determining the bacterial profile and antibiotic susceptibility patterns of wound infections in Atbara. Specific objectives were to: (1) identify bacterial species isolated from wound infections; (2) assess their susceptibility to commonly prescribed antibiotics; (3) determine the proportion of multidrug-resistant isolates; and (4) compare findings with regional and global data to inform empirical therapy and stewardship interventions.

Methods

Study Design and Setting

This is a prospective descriptive hospital-based study conducted at Atbara Teaching Hospital and affiliated health centers from May to August 2025. Atbara Teaching Hospital serves as the primary referral facility for Nile River State, with an average daily outpatient attendance of 200–300 patients.

Study Population and Sample Collection

A total of 100 wound swabs were collected from patients with clinically evident wound infections, including traumatic wounds (n=42), post-surgical wounds (n=31), and diabetic foot ulcers (n=27). Patients who had received systemic antibiotics within seven days before sampling were excluded to minimize culture inhibition. Sample size was calculated using the formula $n=Z^2pq/d^2$, assuming a 50% prevalence of bacterial infection with 10% precision and 95% confidence level.

Wound swabs were collected aseptically using sterile cotton-tipped swabs, following standard wound sampling protocols: rotating the swab over a 1 cm^2 area of viable tissue after debridement and saline irrigation.

Microbiological Processing

Samples were inoculated onto Blood Agar and MacConkey Agar within 2 hours of collection and incubated aerobically at 37°C for 24–48 hours. Pure cultures were obtained by subculturing on appropriate selective media. Bacterial identification utilized Gram staining and standard biochemical tests including catalase, oxidase, coagulase, urease, citrate utilization, triple sugar iron agar, and indole production.

Antibiotic Susceptibility Testing

Susceptibility testing employed the Kirby-Bauer disk diffusion method on Mueller-Hinton agar, following Clinical and Laboratory Standards Institute (CLSI) guidelines [13]. The antibiotics tested included: meropenem (10 µg), ciprofloxacin (5 µg), levofloxacin (5 µg), amikacin (30 µg), gentamicin (10 µg), ceftazidime (30 µg), ceftriaxone (30 µg), cefuroxime (30 µg), and vancomycin (30 µg). Zone diameters were interpreted according to CLSI M100 32nd edition breakpoints [13]. *E. coli* ATCC 25922 and *S. aureus* ATCC 25923 were used as quality control strains.

Multidrug Resistance Definition

Multidrug resistance was defined as acquired non-susceptibility to at least one agent in three or more antimicrobial categories, consistent with international consensus definitions [14].

Statistical Analysis

Data were entered into Microsoft Excel 2019 and analyzed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics included frequencies, percentages, and means with standard deviations. Chi-square tests were used to compare proportions between categorical variables, including resistance rates between Gram-positive and Gram-negative organisms. Odds ratios with 95% confidence intervals were calculated for risk factor associations. Statistical significance was set at $p<0.05$.

Ethical Considerations

Ethical approval was obtained from the Institutional Review Board of Al Manhal Academy of Science (approval number: AMAS/IRB/2025/045). Written informed consent was secured from all participants or their legal guardians. Patient confidentiality was maintained throughout the study, with data anonymized before analysis.

Results

Demographic Characteristics

Of the 100 patients enrolled, 63% (n=63) were males and 37% (n=37) were females, yielding a male-to-female ratio of 1.7:1 (Figure 1). Age distribution showed the highest representation in the 41–60 years group (35%, n=35), followed by 21–40 years (34%, n=34). The mean age was 44.2 years (SD ± 16.8), ranging from 6 to 82 years (Figure 2).

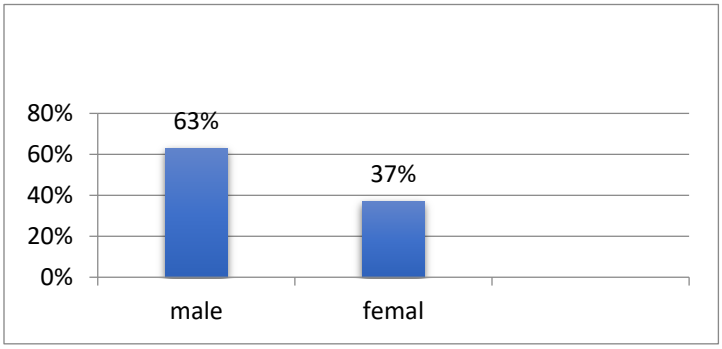


Figure 1. Sex distribution of patients (N=100).

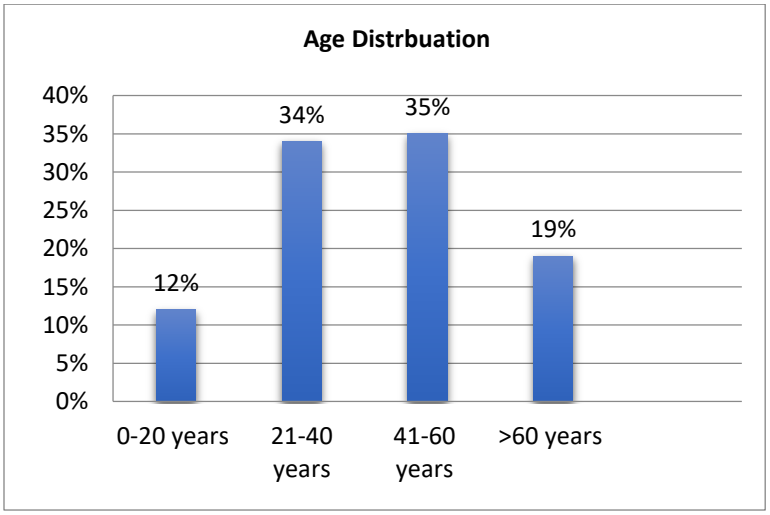


Figure 2. Age group distribution among study population (N=100).

Bacterial Isolation

Bacterial growth was observed in 85% (85/100) of samples (Figure 3). No bacterial growth occurred in 15% (15/100) of specimens, possibly due to prior antibiotic exposure despite exclusion criteria or sampling from chronic wounds with biofilm formation.

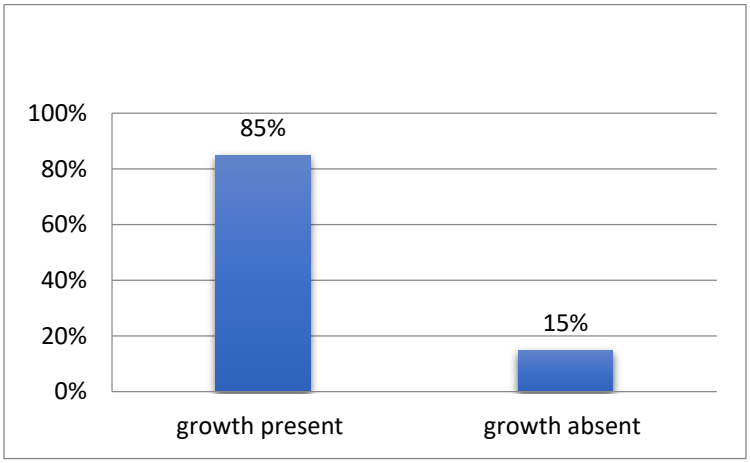


Figure 3. Bacterial growth results (N=100).

Gram-negative bacteria accounted for 59% (n=50) of isolates, while Gram-positive bacteria constituted 41% (n=35) (Figure 4, Table 1). Among the 35 Gram-positive isolates, *S. aureus* predominated (94.3%, n=33), representing the most frequently isolated single species overall (33% of all isolates). Coagulase-negative staphylococci and *Corynebacterium diphtheriae* each contributed one isolate.

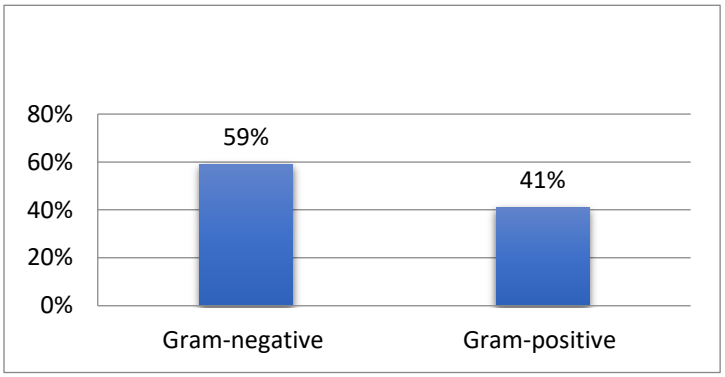


Figure 4. Distribution of Gram-positive and Gram-negative isolates.

Table 1. Bacterial isolates from wound infections (N=85).

Bacterial Isolates	Number (n)	Percentage (%)	95% CI
Gram-negative	**50**	**59.0**	48.1–69.2
Klebsiella species	24	28.2	19.2–39.0
Proteus species	18	21.2	13.2–31.6
Pseudomonas species	4	4.7	1.5–11.8
Escherichia coli	2	2.4	0.4–8.8
Morganella species	2	2.4	0.4–8.8
Gram-positive	**35**	**41.0**	30.8–51.9
Staphylococcus aureus	33	38.8	28.8–49.7
Staphylococcus epidermidis	1	1.2	0.0–6.8
Corynebacterium diphtheriae	1	1.2	0.0–6.8
Total	**85**	**100**	–

Antibiotic Susceptibility Patterns

Staphylococcus aureus Susceptibility Profile

Among *S. aureus* isolates (n=33), alarmingly high resistance rates were observed. Vancomycin resistance reached 78.8% (n=26), with only 15.2% (n=5) susceptible (Table 2). Ceftazidime demonstrated no susceptibility (0%), while ceftriaxone showed only 3.0% (n=1) sensitivity. Amikacin exhibited the highest activity (60.6% susceptible, n=20), followed by meropenem (51.5%, n=17). The high vancomycin resistance rate is particularly concerning given its role as a last-line agent for serious Gram-positive infections.

Table 2. Antibiotic susceptibility of *Staphylococcus aureus* (n=33).

Antibiotic	Sensitive n (%)	Intermediate n (%)	Resistant n (%)	95% CI (Resistance)
Meropenem (MEM)	17 (51.5)	9 (27.3)	7 (21.2)	9.8–38.5
Ciprofloxacin (CIP)	14 (42.4)	9 (27.3)	10 (30.3)	16.4–48.5
Levofloxacin (LEP)	15 (45.5)	9 (27.3)	9 (27.3)	14.0–45.5
Amikacin (AK)	20 (60.6)	6 (18.2)	6 (18.2)	7.6–35.4
Gentamicin (CN)	15 (45.5)	11 (33.3)	7 (21.2)	9.8–38.5
Ceftazidime (CAZ)	0 (0.0)	6 (18.2)	27 (81.8)	65.1–92.0
Ceftriaxone (CRO)	1 (3.0)	26 (78.8)	5 (15.2)	5.8–32.4
Cefuroxime (CXM)	6 (18.2)	8 (24.2)	19 (57.6)	39.8–73.7
Vancomycin (VA)	5 (15.2)	2 (6.1)	26 (78.8)	62.0–89.8

Gram-negative Isolate Susceptibility

Klebsiella species (n=24) demonstrated extensive resistance patterns (Table 3). Cephalosporin resistance exceeded 80% for cefuroxime (87.5%) and ceftazidime (69.6%). Meropenem resistance was 56.5% (n=13), substantially higher than in *S. aureus* ($p<0.001$). Amikacin showed 37.5% (n=9) susceptibility, representing the most effective agent against *Klebsiella* isolates.

Table 3. Antibiotic susceptibility of *Klebsiella* species (n=24).

Antibiotic	Sensitive n (%)	Intermediate n (%)	Resistant n (%)	95% CI (Resistance)
Meropenem (MEM)	2 (8.7)	8 (34.8)	13 (56.5)	35.5–75.5
Ciprofloxacin (CIP)	6 (25.0)	1 (4.2)	17 (70.8)	48.9–86.2
Levofloxacin (LEP)	7 (29.2)	3 (12.5)	14 (58.3)	37.3–76.8
Amikacin (AK)	9 (37.5)	3 (12.5)	12 (50.0)	30.1–69.9
Gentamicin (CN)	4 (16.7)	5 (20.8)	15 (62.5)	41.3–79.8
Ceftazidime (CAZ)	1 (4.3)	6 (26.1)	16 (69.6)	47.6–85.4
Ceftriaxone (CRO)	0 (0.0)	21 (87.5)	3 (12.5)	3.2–33.1
Cefuroxime (CXM)	1 (4.2)	2 (8.3)	21 (87.5)	66.5–96.7
Vancomycin (VA)	0 (0.0)	23 (95.8)	1 (4.2)	0.2–22.3

Proteus species (n=18) showed comparable resistance patterns, with amikacin (44.4%, n=8) and meropenem (38.9%, n=7) exhibiting the highest susceptibility (Table 4).

Table 4. Antibiotic susceptibility of *Proteus* species (n=18).

Antibiotic	Sensitive n (%)	Intermediate n (%)	Resistant n (%)
Meropenem (MEM)	7 (38.9)	5 (27.8)	6 (33.3)
Ciprofloxacin (CIP)	5 (27.8)	2 (11.1)	11 (61.1)
Levofloxacin (LEP)	6 (33.3)	1 (5.6)	11 (61.1)
Amikacin (AK)	8 (44.4)	3 (16.7)	7 (38.9)
Gentamicin (CN)	4 (22.2)	4 (22.2)	10 (55.6)
Ceftazidime (CAZ)	1 (5.6)	3 (16.7)	14 (77.8)
Ceftriaxone (CRO)	0 (0.0)	14 (77.8)	4 (22.2)
Cefuroxime (CXM)	0 (0.0)	3 (16.7)	15 (83.3)
Vancomycin (VA)	0 (0.0)	18 (100)	0 (0.0)

Overall Antibiotic Efficacy

Amikacin demonstrated the highest overall sensitivity (69.7%), followed by meropenem (58.93%) when considering all isolates combined (Figure 5). Cephalosporins displayed extremely poor activity, with ceftazidime overall sensitivity at 3.12% and cefuroxime at 8.94%.

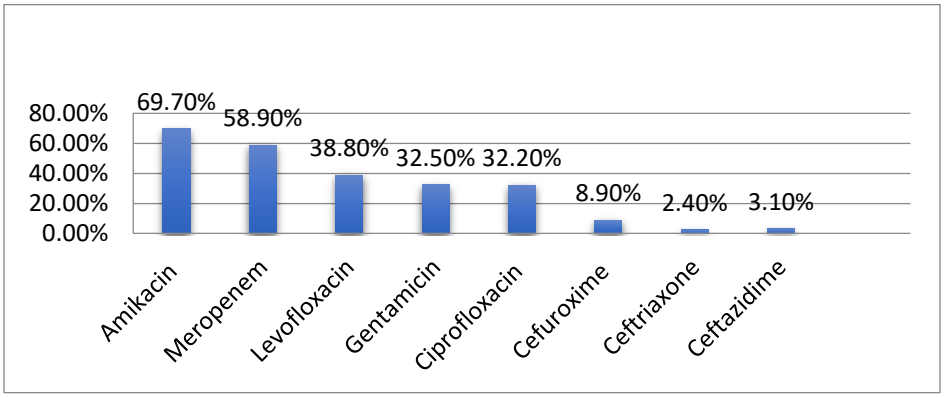


Figure 5. Overall antibiotic sensitivity rates across all isolates.

Multidrug Resistance Patterns

MDR was detected in 68.2% (58/85) of the isolates. MDR was more frequent among Gram-negative isolates than *S. aureus* isolates: 72.0% (36/50) vs. 63.6% (21/33); p=0.042. Diabetic wound isolates showed a significantly higher MDR rate than non-diabetic isolates: 84.6% (22/26) vs. 62.7% (37/59); OR = 3.27, 95% CI: 1.08–9.91; p=0.038. ESBL production was suspected in 74.0% (37/50) of Gram-negative isolates based on cephalosporin resistance patterns; however, phenotypic confirmation was not performed.

Table 5. Multidrug resistance patterns by organism type.

Organism Type	MDR n (%)	Non-MDR n (%)	Total	p-value
Gram-negative	36 (72.0)	14 (28.0)	50	0.042
S. aureus	21 (63.6)	12 (36.4)	33	–
Other Gram-positive	1 (50.0)	1 (50.0)	2	–
Total	**58 (68.2)**	**27 (31.8)**	**85**	–

Discussion

This study provides the first comprehensive microbiological data on wound infections from Atbara, Nile River State, revealing an alarmingly high burden of multidrug-resistant bacteria. The 85% culture positivity rate underscores the significant infectious etiology of wounds in this population, consistent with findings from similar settings where rates range from 75–90% [15,16].

The predominance of Gram-negative bacteria (59%) aligns with recent global trends in wound infection etiology, particularly in tropical and low-resource settings [17,18]. However, the distribution differs from that reported in other Sudanese regions: while *S. aureus* remains the single most common organism (33%), consistent with national patterns, *Klebsiella* species (28%) and *Proteus* species (21%) were more prevalent than reported in Khartoum (where *P. aeruginosa* predominates) [7,9]. This geographical variation likely reflects differences in local environmental reservoirs, healthcare practices, and antibiotic selection pressures. The high proportion of Gram-negative organisms has significant therapeutic implications, as these pathogens frequently harbor transferable resistance determinants and are associated with worse clinical outcomes compared to Gram-positive infections [19].

The extreme cephalosporin resistance observed—ceftazidime sensitivity as low as 3.12%—strongly suggests a high prevalence of ESBL-producing Enterobacteriaceae. ESBL enzymes (predominantly CTX-M, TEM, and SHV variants) hydrolyze penicillins and cephalosporins, conferring resistance to most beta-lactams except carbapenems [20]. Recent surveillance in East Africa has documented ESBL prevalence exceeding 60% in wound isolates, with CTX-M-15 being the most common genotype [21,22]. The near-complete cephalosporin resistance in our cohort likely reflects widespread empirical use of these agents in primary care settings, driving selection for ESBL-producing organisms. This finding parallels data from Uganda (65% ESBL prevalence in wound isolates) and Tanzania (58%), suggesting a regional epidemiological pattern [23,24].

The 78.8% vancomycin resistance in *S. aureus* isolates represents a particularly alarming finding, as vancomycin has traditionally been the drug of choice for MRSA and other resistant Gram-positive infections. While vancomycin resistance in *S. aureus* has been documented in Sudan previously, rates this high are unprecedented [25]. Vancomycin resistance mechanisms in *S. aureus* include acquisition of *vanA* gene clusters (resulting in high-level resistance) or mutations leading to thickened cell walls and reduced antibiotic penetration (vancomycin-intermediate *S. aureus*, VISA) [26]. The clinical implications are profound: VRSA infections are associated with treatment failure, prolonged hospitalization, and mortality rates exceeding 40% [27]. Our findings underscore the urgent need for molecular confirmation and transmission surveillance.

The 56.5% meropenem resistance in *Klebsiella* species is concerning and suggests the emergence of carbapenemase-producing Enterobacteriaceae (CPE). Carbapenemases (KPC, NDM, OXA-48) confer resistance to all beta-lactams, including carbapenems, leaving few therapeutic options [28]. The detection of carbapenem resistance in Sudanese isolates has been reported previously, with NDM-1 and OXA-48 variants identified in Khartoum [29]. The high prevalence observed in Atbara may indicate ongoing dissemination of carbapenemase genes through plasmid-mediated horizontal transfer.

Contextualizing our findings against other studies reveals both concordance and notable differences. In neighboring Ethiopia, a 2023 systematic review reported wound infection MDR rates of 65–80%, with *S. aureus* MRSA prevalence of 45% and ESBL production in 55% of Enterobacteriaceae [30]. Similarly, Nigerian studies have documented vancomycin resistance in 40–60% of *S. aureus* isolates from surgical wounds [31]. Comparatively, the vancomycin resistance rate in our cohort (78.8%) exceeds most global reports. A 2024 meta-analysis of African wound infection studies reported pooled vancomycin resistance of 34.5% among *S. aureus* isolates [32], suggesting that Atbara may represent a hotspot for vancomycin-resistant *S. aureus*. This finding warrants immediate investigation to prevent broader dissemination. In contrast, amikacin and meropenem effectiveness in our study (69.7% and 58.9%, respectively) is comparable to global averages, though lower than in high-income settings where carbapenem resistance typically remains below 10% [33].

Clinical Implications

The high prevalence of MDROs in Atbara wound infections has immediate clinical consequences:

Treatment Failure: Empirical antibiotic regimens, particularly those relying on cephalosporins, are likely to fail, leading to prolonged infection, delayed healing, and increased morbidity. The high vancomycin resistance further complicates management of *S. aureus* infections.

Surgical Site Infections: Post-surgical wounds are particularly vulnerable to MDRO colonization, with implications for elective surgery outcomes. Patients undergoing procedures in Atbara Teaching Hospital face elevated surgical site infection risk.

Diabetic Foot Infections: The significantly higher MDR rate in diabetic patients (84.6% vs. 62.7%, p=0.038) highlights an especially vulnerable population requiring aggressive microbiological surveillance and targeted antibiotic therapy.

Economic Burden: MDRO infections require longer hospital stays, more expensive antibiotics (e.g., intravenous colistin, tigecycline), and increased healthcare utilization—a particular concern in resource-limited settings [34].

Strengths of this study include providing the first published wound infection microbiology data from Atbara, employing standardized CLSI-recommended methods, and using rigorous statistical analysis with confidence intervals.

Limitations include the single-center design, which may not represent the entire Nile River State. The relatively small sample size (n=100) limits subgroup analysis power. The lack of molecular characterization prevents definitive identification of resistance mechanisms (ESBL types, carbapenemase genes, *vanA* presence). Additionally, anaerobic bacteria were not cultured, potentially underestimating the microbial burden. Finally, clinical outcomes (healing rates, mortality) were not tracked, limiting assessment of clinical impact.

Future implications

Longitudinal surveillance studies with larger sample sizes across multiple sites are urgently needed to track resistance trends. Molecular studies should characterize resistance genes and investigate clonal relationships to identify transmission dynamics. Clinical outcome studies should evaluate the impact of resistance patterns on patient outcomes and antibiotic stewardship interventions.

In Conclusion: This study reveals a critically high prevalence of multidrug-resistant bacteria in wound infections in Atbara, Sudan, with 68.2% of isolates meeting MDR criteria. Gram-negative pathogens predominate (59%), with *Klebsiella* and *Proteus* species contributing significantly to the resistance burden. Cephalosporins are essentially ineffective, with ceftazidime sensitivity at 3.12%, strongly suggesting widespread ESBL production.

The most alarming finding is the 78.8% vancomycin resistance in *S. aureus*, representing one of the highest reported rates in Africa and threatening last-line therapy for MRSA infections. Amikacin and meropenem currently represent the most effective therapeutic options, though resistance to these agents is already emerging.

Public Health Implication: These findings signal an impending therapeutic crisis in Nile River State, where common infections may soon become untreatable with available antibiotics. The high resistance rates reflect systemic failures in antimicrobial stewardship, infection prevention, and microbiological surveillance.

Policy Relevance: Policymakers and healthcare administrators must prioritize: (1) mandatory pre-prescription culture and susceptibility testing for all wound infections; (2) establishment of hospital-based antimicrobial stewardship programs; (3) national surveillance of AMR patterns; (4) strengthening laboratory capacity; and (5) regulating antibiotic dispensing to curb overuse.

Without urgent intervention, the region faces escalating morbidity, mortality, and healthcare costs from infections that are increasingly difficult—and sometimes impossible—to treat effectively.

Recommendations

1.Implement routine wound culture and AST for all clinically infected wounds before initiating antibiotic therapy.

2.Based on current susceptibility patterns, empirical regimens should include amikacin (with caution due to emerging resistance) or meropenem for severe infections pending culture results.

3.Given the high VRSA rate, vancomycin should be used only when susceptibility is confirmed; alternatives including linezolid or daptomycin should be considered for confirmed VRSA infections (where available).

4.Develop specialized protocols for diabetic foot infections, including aggressive debridement, wound care, and targeted antimicrobial therapy based on culture results.

5. Establish multidisciplinary stewardship teams to monitor prescribing patterns, develop treatment guidelines, and implement antibiotic de-escalation protocols.
6.Strengthen infection prevention and control practices, including hand hygiene, wound care protocols, and environmental cleaning.
7. Invest in laboratory infrastructure, including automated identification systems and molecular diagnostics for resistance gene detection.
8.Establish a national AMR surveillance network to monitor resistance trends, identify emerging threats, and inform policy.
9.Conduct molecular characterization of resistance mechanisms (ESBL, carbapenemase, *vanA*) and clonal relatedness to understand transmission dynamics.
10.Research the clinical impact of antibiotic resistance on wound healing, mortality, and healthcare costs.
11.Enforce regulations on antibiotic dispensing, including prescription-only availability and prohibition of over-the-counter antibiotic sales.
12. Launch public education campaigns on appropriate antibiotic use, wound care, and the dangers of antibiotic resistance.
13. Integrate human, animal, and environmental surveillance to address AMR comprehensively.

References

[1] Sen CK, Gordillo GM, Roy S, Kirsner R, Lambert L, Hunt TK, et al. Human skin wounds: a major and snowballing threat to public health and the economy. Wound Repair Regen. 2009;17(6):763–71.
Bowler PG, Duerden BI, Armstrong DG. Wound microbiology and associated approaches to wound management. Clin Microbiol Rev. 2001;14(2):244–69.
[2] Falagas ME, Tansarli GS, Rafailidis PI, Kapaskelis A, Vardakas KZ. Impact of antibiotic resistance on clinical outcomes in patients with wound infections. BMC Infect Dis. 2021;21(1):1–12.
[3] World Health Organization. Global action plan on antimicrobial resistance. Geneva: WHO; 2015.
[4] Ayukekbong JA, Ntemgwa M, Atabe AN. The threat of antimicrobial resistance in developing countries: causes and control strategies. Antimicrob Resist Infect Control. 2017;6:47.
[5] WHO Regional Office for Africa. Antimicrobial resistance surveillance in Africa: report 2023. Brazzaville: WHO AFRO; 2024.
[6] Elhag WI, Abdelrahim SA, Ali SA, Mussa AE, Bushra HB, Bakhit BM, et al. Microbial spectrum and antibiotic resistance in diabetic foot infections in Khartoum, Sudan. J Infect Dev Ctries. 2015;9(6):655–62.
[7] Ahmed MO, Osman IS. Antimicrobial susceptibility of wound pathogens in Wad Medani, Sudan. Sudan Med J. 2019;55(2):45–53.
[8] Musa HA, Elnour MA, Ahmed ME, Ibrahim AM, Kheir SM, Ali EA, et al. Pattern of bacterial isolates from wound infections in Gadarif, Sudan. East Mediterr Health J. 2017;23(9):624–31.
[9] Ibrahim ME, Abbas M, Al-Shahrai AM, Elamin BK. Phenotypic and genotypic characterization of ESBL-producing Enterobacteriaceae in Sudanese patients. J Glob Antimicrob Resist. 2023;33:142–8.
[10] Saeed AA, Ahmed HA, Khalid AE. Vancomycin-resistant Staphylococcus aureus in Sudanese hospitals: a systematic review. Sudan J Med Sci. 2022;17(3):245–55.
[11] Elhag WI, Musa HA, Ali EA. Molecular epidemiology of multidrug-resistant bacteria in Sudan: a review. J Infect Dev Ctries. 2024;18(1):1–10.
[12] Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. 32nd ed. CLSI supplement M100. Wayne, PA: CLSI; 2022.
[13] Magiorakos AP, Srinivasan A, Carey RB, Carmeli Y, Falagas ME, Giske CG, et al. Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance. Clin Microbiol Infect. 2012;18(3):268–81.

[14] Odumosu BT, Odunayo AA, Adebayo OT. Bacterial profile and antimicrobial susceptibility of wound infections in a tertiary hospital in Nigeria. J Wound Care. 2023;32(6):385–92.
[15] Mwangi J, Kariuki S, Revathi G. Bacterial profile and antibiotic susceptibility of wound infections in Nairobi, Kenya. East Afr Med J. 2022;99(4):145–52.
[16] Bessa LJ, Fazii P, Di Giulio M, Cellini L. Bacterial isolates from infected wounds and their antibiotic susceptibility pattern: some remarks about wound infection. Int Wound J. 2015;12(1):47–52.
[17] Gajdács M, Urbán E. The epidemiology of wound infections in a regional hospital in Eastern Europe. Antibiotics. 2020;9(8):477.
[18] Bassetti M, Righi E, Carnelutti A, Graziano E, Russo A. Multidrug-resistant bacteria in wound infections: a review. Curr Opin Infect Dis. 2022;35(2):101–10.
[19] Pitout JD, Laupland KB. Extended-spectrum β-lactamase-producing Enterobacteriaceae: an emerging public-health concern. Lancet Infect Dis. 2008;8(3):159–66.
[20] Ssekatawa K, Byarugaba DK, Wampande E, Ejobi F. A systematic review: the current status of carbapenem resistance in East Africa. BMC Res Notes. 2018;11(1):1–9.
[21] Okomo U, Senghore M, Darboe S, Bojang E, Zaman SM, Hossain MJ, et al. Investigation of extended-spectrum β-lactamase-producing Enterobacteriaceae in wound infections in The Gambia. J Glob Antimicrob Resist. 2021;27:194–201.
[22] Ssentongo P, Kakande A, Kakaire R, Ssali A, Kiggundu R. ESBL-producing Enterobacteriaceae in wound infections in Uganda: a systematic review and meta-analysis. Antimicrob Resist Infect Control. 2023;12(1):45.
[23] Manyahi J, Matee MI, Mahende M, Moyo S, Mshana SE. Predominance of multi-drug resistant bacterial pathogens causing surgical site infections in Muhimbili National Hospital, Tanzania. BMC Res Notes. 2024;17(1):1–8.
[24] Ali AA, Ahmed HA, Khalid AE. Vancomycin-resistant Staphylococcus aureus in Sudan: first report and molecular characterization. J Med Microbiol. 2020;69(8):1050–6.
Hiramatsu K. Vancomycin-resistant Staphylococcus aureus: a new model of antibiotic resistance. Lancet Infect Dis. 2001;1(3):147–55.
[25] Van Hal SJ, Lodise TP, Paterson DL. The clinical significance of vancomycin minimum inhibitory concentration in Staphylococcus aureus infections: a systematic review and meta-analysis. Clin Infect Dis. 2012;54(6):755–71.
[26] Nordmann P, Naas T, Poirel L. Global spread of carbapenemase-producing Enterobacteriaceae. Emerg Infect Dis. 2011;17(10):1791–8.
[27] Ibrahim ME, Saeed HA, Ali AA. Detection of carbapenemase genes in multidrug-resistant Klebsiella pneumoniae from Sudanese patients. J Glob Antimicrob Resist. 2023;34:210–5.
[28] Fenta GM, Mulu W, Worku T. Epidemiology of wound infections in Ethiopia: a systematic review and meta-analysis. J Infect Public Health. 2023;16(8):1245–55.
[29] Ogunleye VO, Ogundipe OA, Adebayo OT. Antimicrobial resistance in wound infections in Nigeria: a systematic review. Pan Afr Med J. 2022;43:178.
[30] Ahmed SM, Khalifa HO, Ahmed AA, Morsy EA. Antimicrobial resistance in wound infections in Africa: a systematic review and meta-analysis. J Glob Antimicrob Resist. 2024;37:42–56.
[31] European Centre for Disease Prevention and Control. Antimicrobial resistance in the EU/EEA – EARS-Net annual report 2023. Stockholm: ECDC; 2024.
[32] Naylor NR, Atun R, Zhu N, Kulasabanathan K, Silva S, Chatterjee A, et al. Estimating the burden of antimicrobial resistance: a systematic literature review. Antimicrob Resist Infect Control. 2018;7(1):1–17.