Original Article

Burden of Multidrug Resistance in Wound Infections in Kashmir: Microbial Profile and Clinical Correlates

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Abstract

Background: Wound infections remain a major cause of morbidity, prolonged hospitalization, and increased healthcare costs. The rise of antimicrobial resistance among wound pathogens further complicates treatment and outcomes. Continuous surveillance of causative organisms and their resistance patterns is essential to guide appropriate empirical therapy and improve patient recovery. This prospective cross-sectional study was conducted at a tertiary care hospital to identify the predominant bacteria in wound infections, assess their antimicrobial resistance profiles, and correlate findings with clinical outcomes and patient factors. **Material and Methods:** Pus samples from wound infections were collected over a two-year period aseptically. A total of 496 samples were collected and processed as per standard microbiological protocols. **Results:** Among 496 patients, culture positivity was higher in males (80.8%) than females (72.6%) ($p = 0.037$). Of the 387 culture-positive samples, 68.7% were Gram-negative. *Staphylococcus aureus* (18.6%) and *Pseudomonas aeruginosa* (22.2%) were the most common Gram-positive and Gram-negative organisms, respectively. Gram-positive isolates were generally susceptible to vancomycin and linezolid; however, 69.44% of *S. aureus* were MRSA. Gram-negatives showed high resistance: *P. aeruginosa* (93% to amikacin, 86% to piperacillin-tazobactam), *Acinetobacter* (85.71% to piperacillin-tazobactam), and *Klebsiella* (69.4% to meropenem). Clinically, 64.9% of patients achieved complete healing, while 35.1% had delayed healing or complications. Wound sepsis occurred in 3.1%, with 1.6% mortality. Outcomes were worse in patients with diabetes and immunosuppression. Empiric antibiotics were appropriate in 62% of cases and associated with better healing (72.6% vs 24%) and fewer complications (27.4% vs 75.4%) ($p<0.01$). All deaths were observed in those initially on inappropriate therapy. **Conclusion:** Wound infections are becoming harder to treat because of rising drug resistance. Our study shows that timely and appropriate antibiotic use, guided by local resistance data, can make a real difference in recovery and survival. Strengthening stewardship and prevention practices is essential to improve patient outcomes and reduce complications.

Keywords: Wound infection, Antimicrobial resistance, Empirical therapy.

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Introduction

Wound infections present a major challenge in healthcare, leading to substantial morbidity, prolonged hospitalization, and increased healthcare costs worldwide.[1] Surgical site infections (SSIs) remain common, accounting for roughly 20% of all healthcare-associated infections and frequently causing delayed recovery and unplanned readmissions.[2] They also significantly heighten patient mortality risk; up to 20% of postoperative deaths have been linked to wound infections.[3] One important factor is the formation of bacterial biofilms in wounds, which are now recognized as major contributors to chronicity and delayed healing. The Kashmir Valley's distinct geographic and climatic conditions, along with limited medical resources, further complicate wound infection management. In our region, challenges such as the overuse or inappropriate use of antibiotics have led to the emergence of multidrug resistant organisms.[4,5] This situation is compounded by healthcare access issues and delayed patient presentations, resulting in complex infections. Notably, chronic illnesses like diabetes mellitus and immunosuppressive states predispose patients to more frequent and severe wound infections, often with poorer healing outcomes.[6]

A wide variety of bacteria can be involved in wound infections. Both Gram-positive organisms and Gram-negative rods are common pathogens. Polymicrobial infections can occur, especially in chronic wounds.[7] The improper or prolonged use of broad-spectrum antibiotics has led to pathogens developing resistance mechanisms making infections more difficult to treat.[5] Such trends underscore the need for periodic local surveillance of wound flora and their antibiograms to guide effective empirical therapy.[8]

The present study was undertaken to determine the prevalence of wound infection in a tertiary care hospital in Kashmir, to identify

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the predominant bacterial pathogens causing these infections, and to evaluate the antimicrobial resistance profiles of the isolates. Additionally, we sought to correlate the microbiological findings with clinical outcomes, specifically, wound healing, complications, and mortality, and to assess the impact of patient comorbidities and the efficacy of empirical versus targeted antibiotic therapy. The overarching goal is to generate evidence that can inform better clinical management and antibiotic stewardship in our region.

Materials and Methods

Study design and setting: A cross-sectional study was conducted in the Department of Microbiology at a tertiary care hospital in central Kashmir, India, over a 2-year period.

Inclusion criteria: The study population included patients of either gender, presenting with clinical signs of wound infection in inpatient wards or outpatient clinics. Wound infections were defined by the presence of purulent discharge, redness, swelling, or delayed healing of a wound.[9]

Exclusion criteria: Improperly labelled samples, repeat samples of the same patient

Sample collection: Pus or wound discharge samples were collected aseptically from each patient preferably prior to the initiation of antibiotic therapy or before administration of the scheduled dose. For each wound, two sterile cotton swab samples or aspirates were obtained after gentle cleaning of the wound surface with sterile normal saline. Samples were labeled with patient details, and the time and site of collection, promptly transported to the microbiology laboratory, or in appropriate transport media if delay was anticipated.

Microbiological processing: Samples were inoculated onto blood agar and MacConkey agar plates, and incubated at 37°C aerobically (with 5% CO_2 for blood agar) for 18–24 hours. Growth was inspected; organisms were identified by colony characteristics, Gram staining, and standard biochemical tests. If no growth was observed at 24 hours, plates were further incubated up to 48 hours before reporting as culture- negative.

Antibiotic susceptibility testing: All confirmed isolates were subjected to antimicrobial susceptibility testing by the Kirby-Bauer disk diffusion method on Mueller-Hinton agar following CLSI guidelines.[10] Zone diameters were measured and interpreted as Sensitive, Intermediate, or Resistant according to CLSI breakpoints. Quality control strains *E. coli* ATCC 25922 and *S. aureus* ATCC 25923 were used with each batch of tests to ensure accuracy.

Clinical data collection: In addition to microbiological analysis, relevant clinical information was recorded for each culture-positive patient using a standardized proforma. Data included age, gender, underlying comorbidities, and details of treatment given. The use of empirical antibiotics before culture results and any subsequent changes to therapy were noted. Patients were followed until discharge or outpatient follow-up (up to at least 30 days post-presentation) to assess outcomes. Wound healing was defined as complete wound closure (by secondary intention or successful surgical closure) without signs of infection by 30 days.[11] "Complications" were defined as any of: persistent infection requiring additional surgical intervention (debridement or reoperation), development of bacteremia/sepsis, as documented by a positive blood culture, or progression to chronic non-healing status at 30 days.[12] In-hospital mortality attributable to wound infection (death with active infection or sepsis) was recorded.

Data analysis: All data were entered into IBM SPSS Statistics (version 25). Categorical variables (proportion of culture-positive cases by gender, antibiotic susceptibility rates, healing vs. non-healing outcomes by subgroup) were summarized as frequencies and percentages. Continuous variables (e.g., age) were summarized as means ± standard deviation. Chi-square (χ^2) tests (or Fisher's exact test when expected frequencies were small) were used to evaluate associations between categorical variables, such as culture positivity and demographic factors, or outcome differences between patient subgroups (diabetic vs non-diabetic, appropriate vs inappropriate empirical therapy). A p value <0.05 was considered statistically significant.

Ethical considerations: The study was conducted on de-identified laboratory samples. Institutional ethical approval was obtained (IEC- 37 Dt 23/12/2023). Patient confidentiality was maintained throughout the study.

Results

Patient demographics and culture positivity: A total of 496 patients with clinical wound infections were included, comprising 328 males (66.1%) and 168 females (33.9%), with a male-to-female ratio of ~1.95:1. The patients ranged in age from 10 to 68 years (mean 38.0, SD 13.1). Of the 496 wound samples, 387 yielded positive cultures (overall wound infection culture positivity rate 78.3%), while 109 (21.97%) showed no bacterial growth. Males had a higher likelihood of culture-positive infection (265/328, 80.8%) than females (122/168, 72.6%), and this difference was statistically significant (p = 0.037). The age distribution of patients did not significantly affect culture positivity - 55.6% of infections occurred in patients ≤40 years and 44.4% in those >40, roughly mirroring the age makeup of the sample, with no significant association between age group and culture result (p = 0.333).

Departmental source of wound samples: The highest number of cases came from the Plastic Surgery department (109 cases, 21.97%). This was followed by the Outpatient department (77 cases, 15.52%), indicating many community-onset wound infections presenting to clinics. Other notable sources were General Surgery (70 cases, 14.1%), Orthopedics (54, 10.9%), and the Burn unit (50, 10.1%). The Medical ICU and Surgical ICU together contributed 28 cases (5.6%). The lowest was the Medical Observation Ward with only 1 case.

Microbiological profile of wound infection: Out of the 387 culture-positive specimens, a total of 410 bacterial isolates were obtained (some wounds yielded two organisms). Gram-negative bacteria accounted for the majority of isolates (266/410, 68.7%), while Gram-positive bacteria comprised 31.3% (121/410). *Staphylococcus aureus* was the most common gram-positive organism isolated, with 72 isolates constituting 18.6% of all cases and 59.5% of Gram-positive isolates. Coagulase-negative

staphylococci (CONS) were isolated in 19 cases (4.9%), and *Enterococcus* species in 25 cases (6.5%). *Streptococcus* spp. (mostly β-hemolytic streptococci) was less frequent (5 isolates, 1.3%). On the Gram-negative side, *Pseudomonas aeruginosa* predominated (86 isolates, 22.2% of total, 32.3% of Gram-negatives). Next were *Proteus* spp. (39 isolates, 10.1%), *Acinetobacter* spp. (42, 10.9%), *Klebsiella* spp. (36, 9.3%), and *E. coli* (34, 8.8%). Less frequent were *Enterobacter* spp. (15, 3.9%) and *Providencia* spp. (14, 3.6%).

Antibiotic susceptibility patterns: The antimicrobial resistance profile of the Gram-positive bacteria is summarized in Table 1. The resistance profiles of the major Gram-negative organisms are provided in [Table 2].

Table 1: Antimicrobial Resistance Profile of Gram-Positive Isolates (values represent number and (%) of isolates resistant to the antibiotic)

Antibiotics	*S. aureus*	*Streptococcus*	*Enterococcus*
Erythromycin	70 (97.22)	0	NT
Amoxicillin-Clavulanate	20(27.78)	NT	NT
Ampicillin	20(27.78)	NT	12(48)
Cefoxitin/Oxacillin	50(69.44)	NT	NT
Clindamycin	40(55.56)	0	NT
Levofloxacin	50(69.44)	0	12(48)
Linezolid	0	0	0
Tetracycline	10(13.88)	NT	2(8)
Vancomycin	0	0	4(16)
Total Isolates	**72**	**5**	**25**

Resistance rates of Gram-positive isolates to selected antibiotics. *S. aureus* includes MRSA screening by cefoxitin (69.44% were resistant, i.e., MRSA rate). Vancomycin and linezolid showed 0% resistance in staphylococci and streptococci; a few enterococci (16%) were resistant to vancomycin, indicating possible VRE. NT = not tested (certain antibiotics were not applicable or not tested for some organisms).

Table 2: Antimicrobial Resistance Profile of Major Gram-Negative Isolates (values are number (%) of isolates resistant)

Antibiotic	*Pseudomonas*	*E. coli*	*Klebsiella* spp.	*Acinetobacter* spp.	*Proteus* spp.	*Providencia* spp.	*Enterobacter* spp.
Amikacin	80 (93.02)	6 (17.65)	8 (22.22)	16 (38.10)	17 (43.59)	4 (28.57)	5 (33.33)
Ampicillin-sulbactam	NT	27 (79.41)	14 (38.89)	12 (28.57)	35 (89.74)	6 (42.86)	NT
Aztreonam	48 (55.81)	2 (5.88)	2 (5.56)	NT	2 (5.13)	6 (42.86)	2 (13.33)
Cefepime	28 (32.56)	18 (52.94)	16 (44.44)	28 (66.67)	12 (30.77)	5 (35.71)	4 (26.67)
Cefoperazone-sulbactam	60 (69.77)	NT	16 (44.44)	NT	14 (35.90)	6 (42.86)	2 (13.33)
Ceftazidime	70 (81.40)	10 (29.41)	9 (25.00)	12 (28.57)	34 (87.18)	8 (57.14)	4 (26.67)
Ceftriaxone	NT	13 (38.24)	4 (11.11)	4 (9.52)	30 (76.92)	9 (64.29)	NT
Cefuroxime	NT	NT	16 (44.44)	5 (11.90)	20 (51.28)	8 (57.14)	NT
Ciprofloxacin	57 (66.28)	18 (52.94)	19 (52.78)	28 (66.67)	7 (17.95)	6 (42.86)	2 (13.33)
Cotrimoxazole	NT	25 (73.53)	19 (52.78)	26 (61.90)	6 (15.38)	5 (35.71)	NT
Gentamicin	38 (44.19)	8 (23.53)	19 (52.78)	21 (50.00)	16 (41.03)	4 (28.57)	2 (13.33)
Levofloxacin	35 (40.70)	13 (38.24)	16 (44.44)	27 (64.29)	5 (12.82)	6 (42.86)	6 (40.00)
Meropenem	30 (34.88)	NT	25 (69.44)	17 (40.48)	14 (35.90)	2 (14.29)	5 (33.33)
Piperacillin-Tazobactam	74 (86.05)	NT	30 (83.33)	36 (85.71)	14 (35.90)	3 (21.43)	4 (26.67)
Tetracycline	NT	8 (23.53)	20 (55.56)	32 (76.19)	NT	NT	NT
Ticarcillin-Clavulanate	74 (86.05)	7 (20.59)	NT	12 (28.57)	15 (38.46)	7 (50.00)	NT
Tigecycline	NT	2 (5.88)	2 (5.56)	2 (4.76)	NT	NT	NT
Total	**86**	**34**	**36**	**42**	**39**	**14**	**15**

Antibiotic resistance profile of key Gram-negative organisms isolated. NT = not tested for that organism (due to intrinsic resistance or not part of panel).

Notable findings: *P. aeruginosa* shows very high resistance to AMK and β-lactams, but lower to cefepime and meropenem; *E. coli* and *Klebsiella* have high resistance to common drugs but low to TGC and AT; *Proteus* is highly resistant to β-lactams but mostly susceptible to fluoroquinolones; *Providencia* shows high cephalosporin resistance; *Enterobacter* has moderate resistance overall

Clinical outcomes and risk factor analysis: In addition to the microbiological findings, our study evaluated patient outcomes. Out of the 387 patients with confirmed wound infection, 252

patients (65.11%) achieved complete wound healing within one month of appropriate care. The remaining 135 patients (34.88%) had suboptimal outcomes: 112 (28.9%) experienced significantly delayed wound healing or required prolonged wound care beyond 30 days, and 23 patients (6.2%) developed major complications. Among these complications, 12 patients (3.1% of infected cases) progressed to systemic infection (sepsis), often necessitating intensive care, and 6 patients (1.6%) ultimately succumbed to infection-related causes (wound sepsis and its sequelae). All recorded mortalities occurred in patients with deep wound infections leading to bacteremia. Notably, all six fatalities were in patients whose initial empirical antibiotic therapy was ineffective against the infecting organism, resulting in uncontrolled infection until culture results guided changes.

We found strong associations between patient comorbidities and healing outcomes. Diabetic patients fared markedly worse: of 100 diabetic patients with wound infections, only 50 (50%) achieved complete healing by 30 days, compared to 202 of 287 patients without diabetes (70.4% healing rate). This difference is statistically significant ($\chi^2 = 14.1$, $p = 0.0002$) and indicates that diabetes roughly doubled the risk of non-healing or delayed healing in our cohort. Diabetics also had a higher incidence of complications (10 cases of wound-related sepsis occurred in diabetics vs 2 in non-diabetics). Immunosuppressed patients (including those on chronic steroids or with conditions like advanced chronic kidney disease) also had poorer outcomes: among 30 immunosuppressed individuals, only 12 (40%) healed fully by 30 days, versus 238 of 357 (67%) immunocompetent patients ($p = 0.016$). Immunocompromised status was associated with a higher rate of persistent infection and was present in 3 of the 6 fatal cases.

Empirical antibiotic therapy (prior to culture results) had been started in 342 of 387 culture-positive cases (88.4%), reflecting standard practice. We analyzed these cases based on whether the empiric regimen was appropriate (i.e., the isolated organism was susceptible in vitro to ≥1 of the antibiotics given). In 212 cases (62.0%), the empiric antibiotics were deemed appropriate; in the remaining 130 (38.0%), therapy was inadequate and had to be escalated or changed once sensitivity results became available. Patients who received appropriate empiric coverage had a significantly higher healing rate: 154/212 (72.6%) healed, compared to 98/130 (75.4%) of those with initially inappropriate therapy who failed to heal (only 32/130, 24.6%, healed by 30 days). In other words, the risk of poor outcome was much higher when initial treatment did not cover the pathogen (unhealed or complicated infection in 75% vs 27%, $p<0.001$). Furthermore, the progression to severe complications (especially sepsis) was predominantly seen in the inappropriate-therapy group (10 of 12 sepsis cases were from the 130 patients with inadequate initial antibiotics). As noted, all mortalities were in this group as well.

Discussion

This study provides an updated overview of the bacteriological profile and antibiotic resistance patterns of wound infections in a tertiary care hospital in the Kashmir Valley, and, importantly, correlates these microbiological findings with clinical outcomes. We observed a high culture positivity rate (78.3%) among suspected wound infections, similar to the rate of 82.2% reported by other studies.[13,14] Such high isolation rates reflect that most clinically diagnosed wound infections indeed have a bacterial etiology. Differences in isolation rates between studies may arise from variations in sample types and inclusion criteria. Nonetheless, our findings underscore that wound infections in our setting are usually bacteriologically confirmed, supporting the use of culture for guiding therapy.

We found that younger adults (≤40 years) constituted a slightly larger proportion of wound infection cases (55.6%) than older patients, and a majority of our patients were male. Other regional studies have shown comparable trends, attributing it to greater exposure to trauma and occupational injuries in that age group.[15-17] Our results align with these observations. Younger, more active individuals may be at increased risk of injuries and consequent wound contamination, whereas older patients might have fewer injuries but potentially more chronic wounds.[18] The male predominance in our study (nearly 2:1) is also commonly reported in wound infection literature,[19] possibly because males, particularly in this region, are more likely to be involved in outdoor work, manual labor, or conflict-related injuries and may also be less attentive to prompt wound care.

Department-wise, the highest number of wound infections originated from the Plastic Surgery and Burn units, followed by general surgical wards and outpatient clinics. Simachew et al. similarly documented that specialized surgical units had a high burden of wound infections.[20] This is expected, as such units manage complex wounds that often require repeated interventions and have higher chances of nosocomial infection. The substantial fraction of cases from the outpatient department (15.52%) indicates that community-acquired wound infections are also a major concern. This calls for robust outpatient antimicrobial stewardship and wound care follow-up, since these infections can harbor resistant organisms that were perhaps selected through prior antibiotic misuse in the community.

Our microbiological findings reinforce the need for broad initial coverage in wound infections. Gram-negative bacteria predominated (68.7%) over Gram-positives (~31%) in our study. Alharbi (2022) reported a comparable distribution (62% Gram-negative) in Saudi Arabia.[21] This trend may reflect the selection pressure of antibiotics and the hospital environment favoring Gram-negative opportunists in wounds.[22] *Pseudomonas aeruginosa* emerged as the most common single pathogen, consistent with its ubiquitous presence in hospital settings and propensity to infect burns and chronic wounds.[20] Interestingly, our proportion of *Pseudomonas* is somewhat lower than the 40% reported by Ahmed et al. possibly due to differences in wound types.[23] *Staphylococcus aureus* was the leading Gram-positive and remains a key pathogen, being part of normal skin flora and capable of infecting wounds readily.[24] The prevalence of *S. aureus* in wound infections is well documented across various studies. Mansoor et al. also found *S. aureus* to be the top Gram-positive isolate and *P. aeruginosa* the top Gram-negative, closely mirroring our results and lending credibility to the consistency of

these findings in this region.[22]

Another notable finding is the presence of *Enterococcus* spp. in our wound cultures. Enterococci are opportunistic pathogens that commonly inhabit the gastrointestinal tract and can colonize chronic wounds, especially in patients with prolonged hospital stays or broad-spectrum antibiotic use.[25] Our detection of *Enterococcus* in wound infections aligns with observations by other researchers that enterococci are frequent opportunists in chronic wounds and pressure ulcers. While not highly virulent, they can delay healing and contribute to poly-microbial infection burden, especially given their resistance against many antibiotics.[26,27]

The antibiotic resistance patterns observed in this study are alarming and carry important clinical implications. Among *S. aureus* isolates, the high MRSA rate (69.44%) means empirical therapy for wound infections should consider MRSA coverage [28]. Worth mentioning, all *S. aureus* in our series were sensitive to vancomycin and linezolid; no VISA, VRSA or LRSA were encountered. In contrast, 16% of Enterococcal isolates were VRE. However, the resistance of *S. aureus* to common oral agents like erythromycin and levofloxacin exceeds 60–90%, limiting outpatient oral therapy options. These patterns concur with regional data.[8,22] Enterococcus resistance to ampicillin (48%) is also significant, as ampicillin is the drug of choice for susceptible enterococcal infections.[29] Nearly half of our enterococci would require vancomycin or newer agents like linezolid or daptomycin for treatment. The emergence of vancomycin-resistant enterococci (VRE) in wound infections is a concern; we detected vancomycin resistance in a minority, but vigilance is needed to detect any high-level VRE.

Gram-negative resistance is perhaps an even greater challenge. The majority of *E. coli* and *Klebsiella* isolates in our study were resistant to β-lactam/β-lactamase inhibitor combinations and trimethoprim- sulfamethoxazole, and a substantial portion were resistant to carbapenem. These findings indicate a high prevalence of extended-spectrum β-lactamase and possibly carbapenemase-producing strains.[5] *Pseudomonas aeruginosa* and *Acinetobacter* spp. displayed multidrug resistance typical of nosocomial isolates.[22] The extremely high resistance to piperacillin-tazobactam (>85%) in *Pseudomonas* is notable; this drug can no longer be relied upon empirically for serious infections in our setting. We did find that cefepime and meropenem had substantially lower resistance rates in *Pseudomonas* (~33%), implying not all *Pseudomonas* are MDR; some remain susceptible to these antipseudomonal agents. Mansoor et al. similarly reported about one-third of *Pseudomonas* remained meropenem-sensitive, concordant with our findings.[22] The extremely high amikacin resistance in *Pseudomonas* (93%) is striking; this may be due to local aminoglycoside usage patterns or presence of aminoglycoside-modifying enzymes.[30] In contrast, *Proteus* species showed a high level of resistance to ampicillin-sulbactam but largely susceptible to fluoroquinolones – a pattern also noted by Ahmed et al.[23] This suggests that older drugs like cotrimoxazole and fluoroquinolones might still have a role for *Proteus* infections, whereas β-lactam options are limited.

Collectively, the resistance data mandate that empirical antibiotic choices for wound infections in our hospital cover MRSA and cover resistant Gram-negatives. A reasonable empiric regimen, based on our results, might be vancomycin plus a combination like meropenem and possibly an anti-pseudomonal quinolone or aminoglycoside. Each hospital must tailor such recommendations to its antibiogram.[31] Regular surveillance is crucial, as emphasized by our findings and those of others in similar regions.[32]

A novel aspect of our study is the integration of clinical outcomes with microbiological data. We found that approximately one-third of patients had delayed healing or complications, and the overall mortality attributable to wound infections was 1.6%. While on the surface this mortality seems low, it is significant considering these are localized infections in most cases – it underlines that wound infections can indeed be life-threatening, especially when they progress to sepsis in vulnerable patients. Our in-hospital mortality rate is comparable to that reported in some studies of surgical wound infections in developing countries. The five-year mortality of chronic diabetic foot infections is known to be extremely high (~50%), akin to many malignancies, highlighting the long-term risk these patients face.[33] We did not track long-term outcomes, but our short-term outcomes already show diabetes had a profound effect. Patients with diabetes in our cohort were significantly less likely to heal and more likely to suffer complications. This aligns with abundant literature that diabetes mellitus impairs wound healing by multiple mechanisms and predisposes to worse infection outcomes.[34] Our data, with only 50% healing in diabetics vs 70.4% in others at 30 days, reinforce that aggressive management of infections in diabetic patients is necessary. Immunocompromised patients also did worse, which is expected as their host defense is impaired. Immunosuppression is known to increase not only infection incidence but also the likelihood of wound healing failure and sepsis.[35] Strict infection prevention and perhaps prophylactic strategies might be warranted in such individuals to mitigate risk.[36]

One of the clearest messages from our study is the importance of appropriate empirical antibiotic therapy. We demonstrated a strong association between initial appropriate therapy and successful outcomes. In cases where the initial empiric antibiotic(s) covered the eventual pathogen, patients had significantly higher healing rates and lower complication rates than in cases where the pathogen was initially resistant and therapy had to be adjusted later. This finding is in concordance with multiple studies in serious infections. A large meta-analysis by Hung et al. in patients with bacteremia showed that inappropriate empirical antibiotics were associated with about twice the odds of mortality compared to appropriate therapy.[31] Our results in wound infection echo this; while wound infections are often localized, those with ineffective initial therapy can smolder, spread, and lead to systemic issues. The fact that all our infection-related deaths occurred in the context of initial inappropriate therapy highlights an opportunity for improvement: by using local resistance to inform empiric choices, we may prevent many of the complications. Of course, tailoring therapy once culture results are known is equally important to avoid unnecessary broad- spectrum use. Our findings strongly support the practice of obtaining wound cultures and adjusting antibiotics accordingly, as doing so

improved patient outcomes.

There are some limitations to our study. First, as a single-center study, our results may not be fully generalizable to other regions; however, they do reflect trends reported elsewhere and thus have broader relevance. Second, we focused on aerobic bacterial isolates; anaerobes and fungi were not specifically sought, which could miss their contribution in chronic wound infections. Third, the follow-up period was relatively short due to practical constraints, so we did not capture long-term outcomes like chronic wound recurrence or long-term mortality beyond discharge. In addition, some degree of selection bias may have influenced the findings, as only patients who presented to our center and underwent sampling were included. Despite these limitations, the study's strength lies in the comprehensive approach of linking microbiology with clinical context. It provides clinicians with a real-world perspective: knowing not just which antibiotics a bacterium is resistant to, but also what that means for the patient's healing and risk of complications.

Conclusion

Our research underlines the critical need for regular bacteriological surveillance of wound infections and for an individualized approach to patient care. The predominance of multidrug resistant Gram-negative bacteria in Kashmir's tertiary hospital wounds calls for prompt and potent empirical therapy, which should later be de-escalated based on culture results. Diabetic and immunosuppressed patients require closer monitoring and perhaps more aggressive or adjunctive therapies to improve outcomes. Additionally, infection control measures to prevent hospital-acquired wound infections are essential to curb the introduction and spread of resistant organisms. By addressing both the microbial and host factors, we can move towards reducing the burden of wound infections. In resource-limited settings like ours, these findings inform a pragmatic strategy: prioritize early culture and sensitivity testing, use broad empiric coverage when risk factors for resistance are present, and pay special attention to high-risk patient groups. This dual focus on microbiological intelligence and clinical vigilance will ultimately help improve healing rates and save lives.

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Conflicts of interest

There are no conflicts of interest.

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