Original Article

Association of Antimicrobial Resistance Patterns with Oxidative Stress Markers in Hospital-Acquired Infections

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Abstract

Background: Hospital-acquired infections (HAIs) are a major cause of morbidity and mortality, and the rise of antimicrobial resistance (AMR) has complicated treatment. Oxidative stress, resulting from an imbalance between reactive oxygen species and antioxidant defences, plays a role in infection-related tissue damage. Limited data exist on the link between AMR profiles and oxidative stress in HAI patients. This is study to evaluate the association between antimicrobial resistance patterns of bacterial isolates from HAIs and oxidative stress biomarkers. **Materials and Methods:** A cross-sectional study was conducted in a tertiary care hospital from May 2023 to April 2024. Patients meeting CDC criteria for HAIs were enrolled. Clinical specimens were processed by standard microbiological methods, and antimicrobial susceptibility testing was performed according to CLSI guidelines. Isolates were classified as multidrug-resistant (MDR), extensively drug-resistant (XDR), or pan-drug-resistant (PDR). Venous blood samples were analysed for malondialdehyde (MDA), reduced glutathione (GSH), superoxide dismutase (SOD), and catalase using spectrophotometric assays. Data were analysed using SPSS v26.0, with $p < 0.05$ considered significant. **Results:** Of 212 patients (mean age 52.6 ± 16.4 years; 57.5% male), the predominant isolates were Klebsiella pneumoniae (28.3%), Escherichia coli (22.6%), Pseudomonas aeruginosa (15.1%), and Staphylococcus aureus (14.2%). MDR, XDR, and PDR strains comprised 46.7%, 21.2%, and 3.3% of isolates, respectively. Mean MDA levels were significantly higher in MDR (6.82 ± 1.14 nmol/mL) and XDR/PDR infections (7.45 ± 1.08 nmol/mL) compared to susceptible strains (4.96 ± 0.87 nmol/mL, $p < 0.001$). Antioxidant levels were lower in resistant infections: GSH (MDR: 2.81 ± 0.53 µmol/L; XDR/PDR: 2.43 ± 0.49 µmol/L vs. susceptible: 3.66 ± 0.61 µmol/L), SOD (MDR: 6.92 ± 1.28 U/mL; XDR/PDR: 6.14 ± 1.16 U/mL vs. susceptible: 8.53 ± 1.31 U/mL), and catalase (MDR: 34.5 ± 4.9 kU/L; XDR/PDR: 31.8 ± 4.2 kU/L vs. susceptible: 39.6 ± 5.1 kU/L) ($p < 0.001$ for all). MDR and XDR status independently predicted elevated MDA and reduced antioxidant activity after adjusting for confounders ($p < 0.01$). **Conclusion:** AMR in HAIs is associated with increased oxidative damage and reduced antioxidant defences. Oxidative stress markers may serve as adjunctive indicators of infection severity and prognosis in hospital settings.

Keywords: Hospital-acquired infections, antimicrobial resistance, oxidative stress, multidrug resistance, malondialdehyde.

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Introduction

Hospital-acquired infections (HAIs), also referred to as nosocomial infections, are infections that develop 48 hours or more after hospital admission, or within 30 days following a surgical procedure, and were neither present nor incubating at the time of admission.[1] They represent one of the most common adverse events in healthcare delivery, affecting millions of patients each year worldwide. The World Health Organization (WHO) reports that, in low- and middle-income countries, the prevalence of HAIs can be as high as 15 per 100 hospitalized patients.[2] These infections contribute to significant morbidity, extended hospital stays, higher treatment costs, and increased mortality, particularly among immunocompromised and critically ill patients.[3]

Common clinical syndromes of HAIs include bloodstream infections, ventilator-associated pneumonia, urinary tract infections, and surgical site infections. The causative organisms are often opportunistic pathogens capable of surviving in the hospital environment and colonizing medical devices.[4] Over recent decades, there has been a marked increase in the occurrence of antimicrobial resistance (AMR) among these pathogens. Gram-negative bacilli such as Klebsiella pneumoniae, Escherichia coli, Acinetobacter baumannii, and Pseudomonas aeruginosa, along with Gram-positive organisms such as methicillin-resistant Staphylococcus aureus (MRSA) and vancomycin-resistant Enterococcus (VRE), are frequently implicated. [5,6]

The emergence of multidrug-resistant (MDR), extensively drug-resistant (XDR), and pan-drug-resistant (PDR) strains has severely restricted therapeutic options, often necessitating the use of last-line antibiotics, which are costly and potentially more toxic.[7] AMR is associated not only with delayed

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initiation of effective therapy but also with worse clinical outcomes, including higher mortality and increased risk of complications.[8]

Infections also initiate a cascade of inflammatory and immune responses in the host, one of which is the generation of reactive oxygen species (ROS). ROS play a dual role: at physiological levels, they participate in signaling and microbial killing; at excessive levels, they contribute to oxidative stress, leading to damage of lipids, proteins, and nucleic acids.[9] Oxidative stress is counteracted by endogenous antioxidant defense mechanisms, including enzymatic antioxidants like superoxide dismutase (SOD) and catalase, and non-enzymatic antioxidants such as reduced glutathione (GSH).[10]

Lipid peroxidation, reflected by increased levels of malondialdehyde (MDA), is a well-recognized marker of oxidative damage.[11] Several studies have shown that infections, particularly those caused by resistant bacteria, can amplify oxidative stress due to prolonged inflammation, increased pathogen burden, and persistent immune activation.[12,13] It has also been postulated that certain bacterial virulence factors and biofilm formation in resistant strains may exacerbate oxidative injury.[14]

Despite the clinical importance of both AMR and oxidative stress in HAIs, the relationship between these two factors has not been well characterized, especially in the Indian tertiary care setting. Establishing such an association could have multiple benefits: oxidative stress markers might serve as adjunctive indicators of infection severity, provide prognostic information, and potentially guide adjunctive antioxidant therapies.

Therefore, the present study was undertaken to investigate the association between antimicrobial resistance patterns of bacterial isolates from patients with HAIs and selected oxidative stress biomarkers, namely MDA, GSH, SOD, and catalase, in a tertiary care hospital setting.

Materials and Methods

Study Design and Setting: The present study was designed as a hospital-based, cross-sectional observational investigation aimed at assessing the relationship between AMR patterns and oxidative stress markers in patients with HAIs. The study was carried out in the Department of Microbiology in collaboration with the Department of Biochemistry at Mahatma Gandhi Memorial (MGM) Hospital, Warangal, Telangana, India. It caters to a wide spectrum of patients from various socioeconomic backgrounds, making it an ideal setting to study hospital-based infections and their biochemical implications. The study period extended over 12 months, from May 2023 to April 2024.

Study Population: The study population comprised patients admitted to different clinical departments of MGM Hospital, including General Medicine, General Surgery, Orthopaedics, Paediatrics, and various Intensive Care Units (ICUs), who developed signs and symptoms suggestive of infection 48 hours or more after hospital admission. This time frame was chosen in accordance with the Centers for Disease Control and Prevention (CDC) definition of HAIs to exclude infections incubating before admission. The study targeted both medical and surgical inpatients to obtain a representative sample of nosocomial infections in a tertiary care setting.

Inclusion and Exclusion Criteria: Patients aged 18 years and above, fulfilling the CDC/NHSN diagnostic criteria for HAIs, and yielding bacterial growth on culture from relevant clinical specimens were considered eligible. Informed written consent was obtained from all participants or their legally authorized representatives. Patients were excluded if they had evidence of infection at admission, chronic inflammatory or autoimmune disorders, malignancies, ongoing antioxidant supplementation, or if their cultures yielded exclusively fungal or viral pathogens without bacterial co-isolation. This careful selection ensured that the study population was homogenous with respect to the main objective and reduced confounding from pre-existing oxidative stress.

Sample Size Determination: The sample size was calculated using prior literature reports indicating a moderate correlation ($r \approx 0.3$) between AMR status and oxidative stress parameters. Assuming a power of 80% and a significance level of 5%, the minimum required sample size was 200. Considering potential dropouts and culture-negative cases, the recruitment target was set at 220 patients, ultimately yielding a final analyzable sample of 212 patients.

Specimen Collection and Microbiological Processing: Clinical specimens were collected under strict aseptic precautions to avoid contamination. Blood samples were obtained for culture in cases of suspected bacteremia and processed using the BacT/ALERT 3D automated blood culture system (bioMérieux). Urine samples were collected midstream in sterile containers and inoculated on cystine lactose electrolyte-deficient (CLED) agar. Pus and wound swabs, as well as respiratory samples such as endotracheal aspirates and bronchoalveolar lavage, were inoculated on blood agar, MacConkey agar, and chocolate agar, and incubated aerobically at 37°C for 18–24 hours. Isolates were identified using standard biochemical methods including catalase, coagulase, oxidase, indole, citrate utilization, urease, and triple sugar iron tests, supplemented with Gram staining for morphological confirmation.

Antimicrobial Susceptibility Testing: Antimicrobial susceptibility was assessed by the Kirby–Bauer disk diffusion method on Mueller–Hinton agar following the Clinical and Laboratory Standards Institute (CLSI) 2023 guidelines. The antimicrobial panel was selected according to the specimen type and organism isolated, covering multiple antibiotic classes. MDR was defined as non-susceptibility to at least one agent in three or more antimicrobial categories; XDR was defined as non-susceptibility to at least one agent in all but two or fewer categories; and PDR was defined as non-susceptibility to all agents in all antimicrobial categories. Reference strains (*Escherichia coli* ATCC 25922, *Staphylococcus aureus* ATCC 25923, and *Pseudomonas aeruginosa* ATCC 27853) were used for quality control.

Grouping of Patients: Based on the antimicrobial susceptibility profiles, patients were stratified into three groups

for comparative analysis: Group 1 included infections caused by fully susceptible bacterial isolates; Group 2 included those caused by MDR strains; and Group 3 comprised infections due to XDR or PDR isolates.

Biochemical Analysis of Oxidative Stress Markers: From each patient, 5 mL of venous blood was collected in plain vacutainer tubes prior to the initiation of definitive antimicrobial therapy. Serum was separated by centrifugation at 3,000 rpm for 10 minutes and stored at –80°C until biochemical analysis. Oxidative stress markers were assessed using standard spectrophotometric assays. Lipid peroxidation was measured as MDA using the thiobarbituric acid reactive substances (TBARS) method described by Ohkawa et al. (1979).[15] GSH levels were determined using Ellman's reagent (DTNB) method.[16] SOD activity was measured by the method of Marklund and Marklund et al. (1974).[17] which involves inhibition of pyrogallol auto-oxidation. Catalase activity was determined following the method of Aebi et al. (1984).[18] based on the rate of decomposition of hydrogen peroxide monitored at 240 nm. All measurements were performed in duplicate using a Shimadzu UV–Visible spectrophotometer (UV-1800 model) in the Department of Biochemistry.

Quality Assurance: Strict adherence to standard operating procedures was maintained throughout the microbiological and biochemical analyses. All reagents were prepared fresh, and equipment calibration was performed regularly. Positive and negative controls were run for all assays. Duplicate analyses were conducted to ensure reproducibility, and inter-assay variation was kept below 5%.

Data Collection and Statistical Analysis: Demographic variables, clinical history, antimicrobial susceptibility profiles, and oxidative stress parameters were recorded in a structured proforma. Statistical analysis was conducted using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Comparisons between the three study groups were made using one-way ANOVA with post-hoc Tukey testing for normally distributed data, and the Kruskal–Walli's test for skewed distributions. The Chi-square test was applied for categorical data. Correlation between AMR status and oxidative stress parameters was assessed using Pearson or Spearman correlation coefficients as appropriate. Multiple linear regression analysis was employed to identify independent predictors of oxidative stress after adjusting for age, sex, and comorbidities. A p-value of less than 0.05 was considered statistically significant.

Results

Demographic and Clinical Profile

A total of 212 patients meeting the inclusion criteria were enrolled during the study period. The mean age of the participants was 52.6 ± 16.4 years, with the age range spanning from 19 to 86 years. Males accounted for 122 cases (57.5%) and females for 90 cases (42.5%), yielding a male-to-female ratio of 1.35:1. Most cases were admitted to the medical wards and ICUs (46.2%), followed by surgical wards (28.3%), orthopedic wards (15.1%), and pediatric ICU (10.4%). Common underlying comorbidities included type 2 diabetes mellitus (34.4%), hypertension (29.2%), and chronic kidney disease (11.8%).

Table 1: Demographic and Clinical Characteristics of the Study Population (n=212)

Parameter	Value
Mean age (years)	52.6 ± 16.4
Age range (years)	19–86
Male, n (%)	122 (57.5)
Female, n (%)	90 (42.5)
Ward/ICU distribution, n (%)	Medicine & ICU: 98 (46.2) Surgery: 60 (28.3) Orthopaedics: 32 (15.1) Paediatrics ICU: 22 (10.4)
Common comorbidities, n (%)	Diabetes: 73 (34.4) Hypertension: 62 (29.2) CKD: 25 (11.8)

Microbiological Profile of Isolates

The predominant pathogens isolated were Klebsiella pneumoniae (28.3%), Escherichia coli (22.6%), Pseudomonas aeruginosa (15.1%), and Staphylococcus aureus (14.2%). Other isolates included Acinetobacter baumannii (8.5%), Enterococcus faecalis (6.1%), and miscellaneous Gram-negative bacilli (5.2%). Among Gram-negative isolates (n=162), carbapenem resistance was observed in 39.5%. Among S. aureus isolates, methicillin resistance was detected in 58.6% of cases.

Table 2: Distribution of Bacterial Pathogens (n=212)

Organism	n (%)
Klebsiella pneumoniae	60 (28.3)
Escherichia coli	48 (22.6)
Pseudomonas aeruginosa	32 (15.1)
Staphylococcus aureus	30 (14.2)
Acinetobacter baumannii	18 (8.5)
Enterococcus faecalis	13 (6.1)
Others	11 (5.2)

Antimicrobial Resistance Patterns

Based on susceptibility testing, 99 isolates (46.7%) were classified as multidrug-resistant (MDR), 45 isolates (21.2%) as extensively drug-resistant (XDR), and 7 isolates (3.3%) as pan-drug-resistant (PDR). The remaining 61 isolates (28.8%) were fully susceptible to tested antimicrobials.

Carbapenem resistance was most common in *K. pneumoniae* (45.0%) and *A. baumannii* (61.1%), while aminoglycoside resistance was highest in *P. aeruginosa* (46.8%). Vancomycin resistance was not detected in *Enterococcus* isolates, but high-level gentamicin resistance was observed in 38.5%.

Oxidative Stress Marker Levels

The oxidative stress biomarker analysis demonstrated a distinct and progressive pattern across the antimicrobial resistance groups. MDA levels, an indicator of lipid peroxidation, were lowest in the susceptible group (4.96 ± 0.87 nmol/mL), increased in MDR infections (6.82 ± 1.14 nmol/mL), and reached the highest values in XDR/PDR infections (7.45 ± 1.08 nmol/mL), with statistically significant differences between all groups ($p < 0.001$, ANOVA with Tukey post-hoc test). In contrast, GSH levels exhibited an inverse relationship, being highest in susceptible infections (3.66 ± 0.61 µmol/L), moderately reduced in MDR cases (2.81 ± 0.53 µmol/L), and lowest in XDR/PDR infections (2.43 ± 0.49 µmol/L), with $p < 0.001$. Similarly, SOD activity declined progressively from 8.53 ± 1.31 U/mL in susceptible isolates to 6.92 ± 1.28 U/mL in MDR and 6.14 ± 1.16 U/mL in XDR/PDR infections, all comparisons being statistically significant ($p < 0.001$). Catalase activity followed the same downward trend, measuring 39.6 ± 5.1 kU/L in susceptible isolates, 34.5 ± 4.9 kU/L in MDR, and 31.8 ± 4.2 kU/L in XDR/PDR infections ($p < 0.001$). Overall, these findings indicate a consistent pattern of heightened oxidative damage accompanied by a progressive decline in antioxidant defences with increasing severity of antimicrobial resistance.

Table 3: Comparison of Oxidative Stress Markers across AMR Groups

Parameter	Susceptible (n=61)	MDR (n=99)	XDR/PDR (n=52)	*p*-value
MDA (nmol/mL)	4.96 ± 0.87	6.82 ± 1.14	7.45 ± 1.08	<0.001
GSH (µmol/L)	3.66 ± 0.61	2.81 ± 0.53	2.43 ± 0.49	<0.001
SOD (U/mL)	8.53 ± 1.31	6.92 ± 1.28	6.14 ± 1.16	<0.001
Catalase (kU/L)	39.6 ± 5.1	34.5 ± 4.9	31.8 ± 4.2	<0.001

Correlation Analysis

Pearson's correlation analysis further reinforced the observed relationship between antimicrobial resistance and oxidative stress parameters. A strong positive correlation was found between the AMR category and MDA levels ($r = 0.68$, $p < 0.001$), indicating that as the degree of resistance increased from susceptible to MDR and XDR/PDR strains, there was a corresponding and substantial rise in lipid peroxidation, reflecting greater oxidative damage to cellular membranes. In contrast, significant negative correlations were noted between AMR category and each of the antioxidant defence markers. Reduced GSH demonstrated a negative correlation coefficient of –0.61 ($p < 0.001$), suggesting that higher resistance levels were associated with a pronounced depletion of this crucial non-enzymatic antioxidant. Similarly, SOD activity showed a negative correlation of –0.58 ($p < 0.001$), indicating diminished capacity to neutralise superoxide radicals in patients with more resistant infections. Catalase activity, responsible for detoxifying hydrogen peroxide, also exhibited a negative correlation of –0.56 ($p < 0.001$) with AMR category, reflecting a reduced ability to counteract oxidative stress in the presence of highly resistant organisms (Figure 1). Collectively, these correlations provide strong statistical evidence of an inverse relationship between the severity of antimicrobial resistance and the host's antioxidant defence mechanisms, alongside a direct association with oxidative damage, thereby highlighting the potential role of oxidative stress profiling as an adjunctive marker of infection severity in hospital-acquired infections.

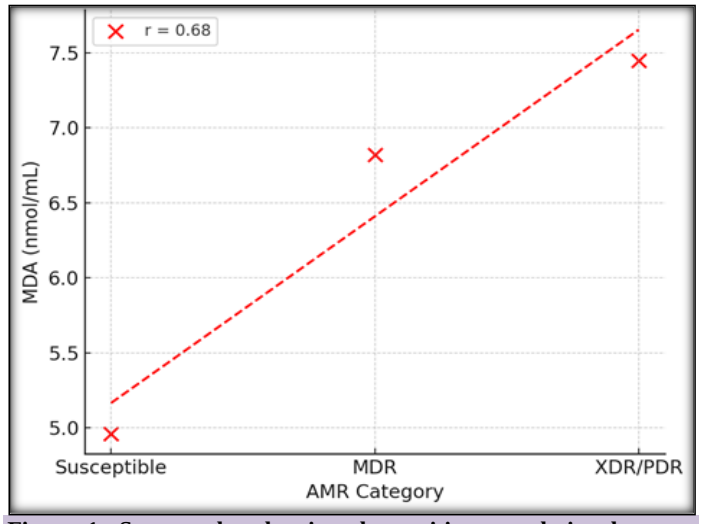

Figure 1: Scatter plot showing the positive correlation between antimicrobial resistance (AMR) category and serum malondialdehyde (MDA) levels in patients with hospital-acquired infections. AMR categories were coded as 1 = susceptible, 2 = multidrug-resistant (MDR), and 3 = extensively/pan-drug-resistant (XDR/PDR)

Multivariate Regression Analysis

Multiple linear regression analysis was performed to determine whether antimicrobial resistance status could independently predict oxidative stress levels after controlling for potential confounding variables such as age, sex, diabetes mellitus, and ICU admission. The model revealed that both MDR and XDR/PDR infection status were significant independent predictors of oxidative imbalance. Specifically, MDR and XDR/PDR status were strongly associated with elevated MDA levels ($\beta = 0.42$, $p < 0.001$), indicating that the presence of higher antimicrobial resistance directly contributed to increased lipid peroxidation irrespective of patient demographics or comorbidities. Similarly, these resistance categories independently predicted reduced levels of GSH ($\beta = –0.39$, $p <$

0.001), suggesting a marked depletion of antioxidant reserves in patients harbouring resistant pathogens. In addition to AMR status, ICU admission emerged as an independent predictor of heightened oxidative stress, likely reflecting the combined impact of severe illness, invasive procedures, and prolonged hospitalisation. Diabetes mellitus also independently correlated with higher oxidative stress markers, consistent with the pro-oxidative metabolic environment observed in hyperglycaemia. These findings underscore that the oxidative stress burden in hospital-acquired infections is not solely a by-product of infection severity but is significantly influenced by the resistance phenotype of the causative pathogen and key clinical factors such as critical care admission and underlying metabolic disease.

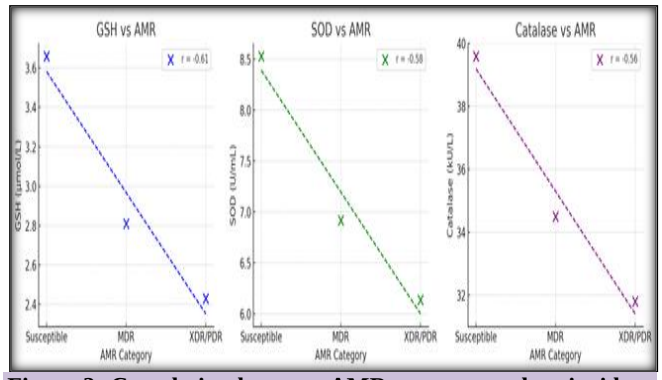

Figure 2: Correlation between AMR category and antioxidant defence parameters in patients with hospital-acquired infections. AMR categories were coded as 1 = susceptible, 2 = MDR, and 3 = XDR/PDR. (A) Reduced glutathione (GSH) levels showed a significant negative correlation with AMR severity (r = –0.61, p < 0.001), indicating progressive depletion with higher resistance. (B) Superoxide dismutase (SOD) activity demonstrated a significant inverse relationship with AMR category (r = –0.58, p < 0.001), reflecting reduced enzymatic antioxidant capacity in resistant infections. (C) Catalase activity also declined significantly with increasing resistance (r = –0.56, p < 0.001), further supporting the pattern of diminished antioxidant defence in higher resistance categories

Discussion

This study examined the relationship between AMR patterns and oxidative stress profiles in patients with HAIs at a tertiary care hospital. The findings revealed a progressive increase in oxidative damage, indicated by higher MDA levels, alongside a corresponding reduction in antioxidant defences, namely GSH, SOD, and catalase, as AMR severity increased from susceptible strains to MDR and XDR/PDR organisms. These associations remained significant even after adjusting for potential confounders such as age, sex, diabetes mellitus, and ICU admission, indicating that AMR status is an independent predictor of oxidative imbalance in patients with HAIs.

The predominance of Gram-negative bacilli, particularly Klebsiella pneumoniae and Escherichia coli, in this study is consistent with both global and Indian surveillance reports, where these pathogens are major contributors to HAIs.[2,3] The high prevalence of MDR (46.7%) and XDR/PDR (24.5%) isolates parallels reports from other tertiary care centres in India, where resistant Gram-negative isolates frequently exceed 40%.[19] ICU patients in our cohort demonstrated significantly higher AMR rates, consistent with findings that critical care environments promote resistance due to invasive procedures, high antibiotic pressure, and prolonged stays.[20]

The observed stepwise increase in MDA levels from susceptible to XDR/PDR groups aligns with evidence that prolonged infection duration and delayed effective treatment in resistant infections lead to sustained ROS generation. [12,21] MDA is a widely accepted marker of lipid peroxidation and membrane damage.[11]

Similarly, the inverse trend between AMR severity and antioxidant defences (GSH, SOD, catalase) matches previous studies showing that persistent ROS production depletes GSH and inactivates antioxidant enzymes through oxidative modification. [22,23] This depletion compromises the host's ability to counter oxidative injury, potentially worsening tissue damage in resistant infections.

Several mechanisms may explain these associations. Resistant infections are often more prolonged, allowing persistent ROS production by immune cells. Many resistant bacteria possess additional virulence factors, including biofilm-forming capacity, which prolong infection and inflammation.[14] Delays in initiating appropriate therapy in resistant infections also enable unchecked bacterial proliferation, further intensifying oxidative stress.[24] Additionally, pathogen-derived toxins and enzymes may directly stimulate oxidative bursts in host immune cells, overwhelming antioxidant defences.[22]

The findings of this study have significant clinical relevance. Firstly, oxidative stress biomarkers, particularly MDA, may serve as adjunctive indicators of infection severity and prognosis in patients with HAIs, especially where rapid microbiological confirmation is not available. Secondly, the consistent depletion of antioxidants in resistant infections raises the possibility of using antioxidant supplementation (e.g., N-acetylcysteine, vitamin E, or selenium) as an adjunctive therapy to reduce tissue damage in severe infections. Although antioxidant therapy should never replace antimicrobial treatment, it could be integrated into a multimodal approach to improve outcomes in critically ill patients. [25,26]

Additionally, the independent association of ICU admission and diabetes mellitus with higher oxidative stress in our cohort underscores the need for closer monitoring and targeted interventions in these high-risk populations. ICU settings are characterized by invasive devices, high pathogen exposure, and systemic inflammation, while diabetes is a well-known pro-oxidative state that predisposes patients to severe and recurrent infections. [27,28]

Strengths and limitations

This study has several strengths. It utilized both microbiological and biochemical analyses in the same patient cohort, enabling direct correlation between pathogen resistance phenotype and host oxidative stress profile. The use of established biochemical assays and adjustment for major confounders adds robustness to the findings.

However, certain limitations must be acknowledged. Being a

single-center study, the results may not be generalizable to other regions or healthcare settings. The cross-sectional design limits causal inference between AMR and oxidative stress, although the strength and consistency of associations suggest a plausible link. Furthermore, while key oxidative stress markers were measured, additional parameters such as total antioxidant capacity and inflammatory cytokines could provide a more comprehensive understanding of host response.

Future perspectives

Future research should focus on longitudinal designs to track oxidative stress changes over the course of resistant infections and recovery. Multi-Centre studies with larger and more diverse populations will help validate these findings. It would also be valuable to investigate whether adjunctive antioxidant therapy in AMR-associated HAIs can reduce oxidative injury and improve patient outcomes. Integrating oxidative stress measurement into clinical risk scoring systems could help clinicians stratify patients for more aggressive monitoring and tailored therapeutic interventions.

Conclusion

This study demonstrates a clear and significant association between AMR severity and oxidative stress status in patients with HAIs. Infections caused by MDR and XDR/PDR pathogens were characterised by markedly elevated MDA levels, indicating increased lipid peroxidation, along with significant depletion of key antioxidant defences, including GSH, SOD, and catalase. These alterations persisted even after adjusting for major confounding factors such as age, sex, diabetes mellitus, and ICU admission, suggesting that AMR itself is an independent contributor to oxidative imbalance in HAI patients.

The progressive oxidative deterioration observed from susceptible to MDR and XDR/PDR groups highlights the potential role of oxidative stress profiling as an adjunctive marker for assessing infection severity and prognosis. Additionally, the identification of ICU admission and diabetes as independent predictors of oxidative stress underscores the need for targeted monitoring and supportive measures in these high-risk subgroups.

Overall, our findings provide new insights into the interplay between pathogen resistance patterns and host oxidative responses, supporting the rationale for further research into adjunctive therapeutic strategies, including antioxidant supplementation, in the management of resistant HAIs.

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Conflicts of interest

There are no conflicts of interest.

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