Section: Microbiology

Original Research Article

PREVALENCE OF MECA- AND MECC-ASSOCIATED METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS ISOLATED FROM DIFFERENT CLINICAL SPECIMENS IN A TERTIARY CARE SETTING OF CENTRAL INDIA

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ABSTRACT

Background: Methicillin-resistant Staphylococcus aureus (MRSA) is a significant cause of healthcare and community-acquired infections. Resistance is predominantly mediated by the mecA gene, while the emergence of mecC-positive MRSA poses diagnostic and therapeutic challenges. This study aimed to determine the prevalence, molecular characteristics, and antimicrobial resistance patterns of mecA- and mecC-positive MRSA isolates from clinical specimens in a tertiary care hospital in Central India. **Materials and Methods:** A laboratory-based observational study was conducted from July to October 2024 at Chirayu Medical College & Hospital. Clinical specimens, including blood, pus, urine, respiratory samples, and body fluids, were processed for MRSA identification using standard microbiological techniques. Methicillin resistance was confirmed via cefoxitin disk diffusion, and antimicrobial susceptibility testing was performed using the Vitek 2 Compact system. Molecular detection of mecA and mecC genes was conducted via real-time PCR. **Result:** Of 1247 specimens, 40 (3.2%) were identified as MRSA, with the highest prevalence in pus (3.33%) and body fluids (3.42%). Molecular analysis showed mecA in 90% (36/40) and mecC in 10% (4/40) of isolates. All were resistant to cefoxitin (100%) with higher resistance rates in erythromycin (82.5%), ciprofloxacin (67.5%), clindamycin (57.5%), and cotrimoxazole (50%). All isolates were susceptible to vancomycin and linezolid. MecC-positive isolates showed slightly lower resistance rates than mecA-positive isolates. **Conclusion:** The predominance of mecA in MRSA and the presence of mecC highlight the need for advanced molecular diagnostics. Vancomycin and linezolid remain effective options. Enhanced surveillance is crucial to address emerging resistance patterns.

INTRODUCTION

Staphylococcus aureus is a Gram-positive pathogen responsible for a wide range of infections, extending from benign skin and soft tissue infections to invasive diseases such as bacteremia, pneumonia, endocarditis, and osteomyelitis. The emergence and global distribution of methicillin-resistant Staphylococcus aureus (MRSA) have posed noteworthy therapeutic and epidemiological challenges, principally in healthcare settings where multidrug-resistant strains contribute to increased morbidity, mortality, and healthcare costs.[1]

Methicillin resistance in S. aureus is primarily conferred by the mecA gene, located on a mobile genetic element termed as staphylococcal cassette chromosome mec (SCCmec). The mecA gene encodes an alternative penicillin-binding protein, PBP2a, resulting in reduced affinity for all β-lactam antibiotics, including penicillin's, cephalosporins, and carbapenems. This allows continued peptidoglycan synthesis and bacterial survival despite β-lactam exposure.[2,3] The integration of SCCmec into the S. aureus chromosome enables horizontal gene transfer, helping rapid clonal expansion of MRSA in both hospital-acquired and community-acquired settings.[4]

In 2011, a novel mecA homolog, mecC, was identified. Located on the SCCmec type XI element, mecC shares approximately 70% nucleotide identity

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with mecA and encodes PBP2c, another β-lactam-insensitive penicillin-binding protein. Like PBP2a, PBP2c confers resistance to β-lactams; however, its expression often escapes detection by conventional diagnostic modalities such as mecA-specific PCR assays and PBP2a latex agglutination tests, leading to potential misclassification of mecC-positive MRSA as methicillin-susceptible S. aureus (MSSA).[5,6] Moreover, mecC-positive MRSA isolates are frequently associated with zoonotic and environmental reservoirs, particularly in livestock, companion animals, and wastewater, underscoring the relevance of a One Health approach to antimicrobial resistance surveillance.[7]

Though mecA-mediated resistance is still predominant in Indian healthcare environment, there is a paucity of available epidemiological data reporting preponderance of mecC in clinical isolates that can be attributed to deficiency in molecular diagnostic capacity and under-reporting of mecC mediated resistance mechanism. This diagnostic blind spot may have significant implications for patient management, antimicrobial stewardship, and infection control policies.

On the basis of this background, the present study was planned to determine the prevalence of mecA and mecC genes among MRSA isolates recovered from diverse range of clinical specimens in a tertiary care hospital in Central India. Furthermore, the study aims to outline the antimicrobial resistance phenotypes associated with these genotypes and to explore their clinical implications, thereby enhancing the comprehensiveness of MRSA molecular epidemiology in the Indian subcontinent.

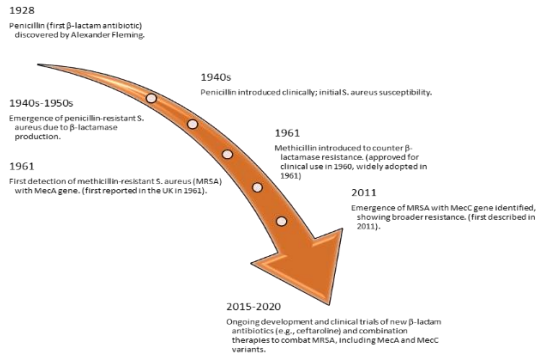

Figure 1: Timeline of β-Lactam Antibiotics and S. aureus Resistance (1928-2020); Source: Historical Record Analysis

MATERIALS AND METHODS

The observational study was done in the Department of Microbiology at Chirayu Medical College & Hospital, Bhopal, India, for over a period of four months, from July to October 2024. Various clinical specimens such as blood, pus, urine, body fluids, and respiratory samples were collected from both IPD and OPD patients attending various departments in the hospital.

Inclusion Criteria

- Samples from patients of all age groups and all genders that showed growth of MRSA.
- Only the first isolate from each patient was considered to avoid duplication.

Exclusion Criteria

- Samples that were culture-negative or has yielded organisms other than Staphylococcus aureus.
- Isolates found to be methicillin-sensitive S. aureus (MSSA).
- Repeat isolates from the same patient.
- Environmental and non-clinical swab samples.

Bacterial Identification and MRSA Detection

All specimens collected were sent immediately to the lab where the specimens were processed using standard laboratory methods. All clinical specimens were cultured on Blood Agar and MacConkey Agar and kept for Overnight incubation in Bacteriological incubator. After incubation the specimens showing growth of various isolates were studied and then colonies suggestive of S. aureus were further examined under the microscope post Gram staining to confirm the presence of Gram-positive cocci in clusters. Biochemical tests such as catalase and coagulase were used to differentiate S. aureus from other Staphylococcus species.

Diffusion Test using Cefoxitin Disc (30 µg) was used for detection of MRSA where a zone of inhibition ≤21 mm was interpreted as MRSA based on CLSI 2024 guidelines.

Antibiotic Susceptibility Testing

Antibiotic sensitivity testing was carried out using the Vitek 2 Compact system (bioMérieux), which provided minimum inhibitory concentrations (MICs) for key antibiotics including erythromycin, ciprofloxacin, clindamycin, vancomycin, and linezolid. The results helped in understanding resistance trends among MRSA strains. Where needed, the D-test was used to identify inducible clindamycin resistance.

Detection of mecA and mecC Genes

To confirm the genetic basis of methicillin resistance, DNA was extracted from the MRSA isolates using the HiPurA® Bacterial Genomic DNA Purification Kit (HiMedia Laboratories Private Limited, India). Detection of mecA and mecC genes was performed through real-time PCR using the Hi-PCR® MRSA Multiplex Probe Kit (HiMedia Laboratories Private Limited, India) on the ALTA RT 96 PCR platform (Athenese-Dx Private Limited, India). The PCR protocol included:

- Initial denaturation at 95°C for 10 minutes
- Followed by 45 cycles of:
  - Denaturation at 95°C for 5 seconds
  - Annealing/extension at 60°C for 30 seconds

The approach allowed us to distinguish between mecA- and mecC-positive MRSA strains with high specificity.

Target Genes & Rationale

- nuc – Encodes the thermostable nuclease; highly specific for S. aureus (Brakstad et al., 1992).

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- mecA – Encodes the PBP2a protein responsible for classical methicillin resistance.
- mecC – mecA homologue (formerly mecALGA251) associated with livestock- and community-acquired MRSA strains.

Primer Sequences (Commonly used)

Gene	Primer Name	Sequence (5′→3′)	Amplicon Size	Reference
nuc	nuc-F	GCGATTGATGGTGATACGGTT	279 bp	Brakstad et al., 1992.[8]
	nuc-R	AGCCAAGCCTTGACGAACTAAAGC		
mecA	mecA-F	AAAATCGATGGTAAAGGTTGGC	310 bp	Murakami et al., 1991.[9]
	mecA-R	AGTTCTGCAGTACCGGATTTGC		
mecC	mecC-F	GAAAAAAAGGCTTAGAACGCCTC	138 bp	García-Álvarez et al., 2011.[10]
	mecC-R	GAAGATCTTTTCCGTTTTCAGC		

Interpretation

- nuc only → MSSA (S. aureus, methicillin-susceptible)
- nuc + mecA → MRSA (classical mecA-positive)
- nuc + mecC → MRSA (mecC-positive)
- mecA/mecC without nuc → Coagulase-negative staphylococci carrying resistance genes

Data Analysis: Data collected from the study were entered and analyzed using IBM SPSS Statistics version 26.0. Descriptive statistics were used to summarize the prevalence of MRSA and the distribution of antibiotic resistance patterns. Statistical comparisons were performed using Chi-square and Fisher's exact tests based on summarized data. A p-value of <0.05 was considered statistically significant.

This study offers valuable insights into the current patterns of methicillin resistance in a tertiary care setting, with particular focus on the often-overlooked mecC-positive MRSA. The findings support the need for routine molecular screening and continued surveillance to guide better infection control and antibiotic stewardship strategies.

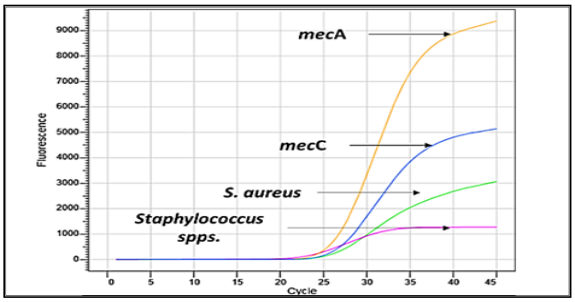

Figure 2: Image representing amplification plot of S. aureus, Staphylococcus spps., mecA and mecC using HiMedia's Hi-PCR® Methicillin Resistant Staphylococcus aureus (MRSA) Probe PCR Kit. As Shown in TD-MBPCR133.[14]

RESULTS

Among 1,247 clinical samples, 40 MRSA isolates were identified, with an overall prevalence of 3.2%. The mecA gene was predominant (90%), while mecC was less frequent (10%). MRSA was most common in pus samples (15 isolates), with body fluids showing the highest prevalence rate (3.42%). Resistance was universal to cefoxitin (100%) and high for erythromycin (82.5%), ciprofloxacin (67.5%), and clindamycin (57.5%), while vancomycin and linezolid were fully effective. MecA-positive isolates were detected across all specimens, most frequently in body fluids (100%), while mecC-positive isolates were rare, mainly in pus samples (50%). These findings highlight mecA dominance, significant resistance, and the efficacy of vancomycin and linezolid.

[Table 1] provides a comprehensive distribution of Methicillin-Resistant Staphylococcus aureus (MRSA) prevalence across various clinical specimens. The table lists the total number of samples tested for each specimen type, the number of MRSA isolates identified, and the prevalence percentage. The distribution of MRSA prevalence shows that the highest number of MRSA isolates were identified in pus samples (15 isolates), followed by blood (10 isolates), and respiratory samples (6 isolates). The overall MRSA prevalence across all specimen types is 3.2%, with body fluids having the highest individual prevalence rate at 3.42%. Statistical analysis using the Chi-square test showed no significant difference in MRSA prevalence across the various specimen types (blood, pus, urine, respiratory samples, and body fluids); $\chi^2 = 1.14$, degrees of freedom (df) = 4, $p = 0.88$. This indicates uniform distribution of MRSA among clinical samples during the study period.

Table 1: Prevalence of MRSA in Clinical Specimens

Specimen Type	Total Samples Tested	MRSA Isolates Identified	Prevalence (%)
Blood	300	10	3.33%
Pus	450	15	3.33%
Urine	200	5	2.5%
Respiratory Samples	180	6	3.33%
Body Fluids	117	4	3.42%
Total	1247	40	3.2%

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Table 2: Distribution of mecA and mecC Genes in MRSA Isolates

Gene Identified	Number of Isolates	Percentage (%)
mecA	36	90%
mecC	4	10%
Total	40	100%

[Table 2] displays the distribution of the mecA and mecC genes among the MRSA isolates identified. The table lists the number of MRSA isolates in which each gene was detected, along with the corresponding percentage of the total MRSA isolates. The data reveals that mecA was detected in the majority of the isolates (90%), while mecC was present in a smaller proportion (10%). This distribution highlights the prevalence of the mecA gene as the dominant mechanism of methicillin resistance in MRSA strains. The difference in distribution between mecA- and mecC-positive MRSA isolates was statistically significant, as assessed by the Chi-square goodness-of-fit test; $\chi^2 = 25.6$, df = 1, $p < 0.001$. This confirms that mecA is the predominant gene associated with methicillin resistance in the current clinical setting.

Table 3: Antibiotic Susceptibility Patterns of mecA- and mecC-Positive MRSA

Antibiotic	Resistant Isolates (mecA)	Resistant Isolates (mecC)	Total Resistance (%)
Cefoxitin	36	4	100%
Erythromycin	30	3	82.5%
Ciprofloxacin	25	2	67.5%
Clindamycin	20	3	57.5%
Cotrimoxazole	18	2	50%
Vancomycin	0	0	0%
Linezolid	0	0	0%

[Table 3] illustrates the antibiotic susceptibility patterns of mecA- and mecC-positive MRSA isolates. The table provides the number of resistant isolates for each antibiotic, segmented by the presence of the mecA and mecC genes, along with the total resistance percentage. Cefoxitin resistance is universal (100%) in both mecA- and mecC-positive isolates. High resistance rates are observed for Erythromycin (82.5%), Ciprofloxacin (67.5%), and Clindamycin (57.5%). Cotrimoxazole demonstrates moderate resistance (50%), with slightly lower resistance in mecC-positive isolates. Both Vancomycin and Linezolid remain fully effective, showing no resistance in any of the isolates. Comparative analysis of resistance patterns between mecA- and mecC-positive MRSA isolates was performed using Fisher's exact test (2-tailed) due to small sample size in the mecC group (n = 4). No statistically significant differences were found for any of the tested antibiotics (erythromycin, p = 1.00; ciprofloxacin, p = 1.00; clindamycin, p = 0.63; cotrimoxazole, p = 1.00), suggesting comparable resistance trends between the two gene variants.

Table 4: mecA-Positive MRSA Across Specimen Types

Specimen Type	mecA-Positive Isolates	Percentage of mecA (%)
Blood	9	90%
Pus	14	93.3%
Respiratory Samples	5	83.3%
Body Fluids	4	100%
Urine	4	80%

[Table 4] presents the distribution of mecA-positive MRSA isolates across various specimen types. The table indicates the number of mecA-positive isolates identified within each specimen type, along with the corresponding percentage of mecA-positive isolates. The data shows that mecA-positive isolates are prevalent across all specimen types, with the highest percentage found in Body Fluids (100%) and the lowest in Urine (80%). Overall, mecA-positive MRSA isolates demonstrate a high prevalence across diverse clinical samples. No formal statistical comparison was conducted for mecA distribution across specimen types as mecA was widely prevalent (>80%) across all groups, limiting variability required for meaningful significance testing. Descriptive data indicate high prevalence of mecA among isolates from body fluids (100%), pus (93.3%), and blood (90%).

Table 5: mecC-Positive MRSA Across Specimen Types

Specimen Type	mecC-Positive Isolates	Percentage of mecC (%)
Blood	1	10%
Pus	2	50%
Respiratory Samples	1	25%
Body Fluids	0	0%
Urine	0	0%

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[Table 5] outlines the distribution of mecC-positive MRSA isolates across various specimen types. The table shows the number of mecC-positive isolates identified within each specimen type, along with the corresponding percentage of mecC-positive isolates. The data indicates that mecC-positive isolates are relatively rare, with the highest percentage found in Pus samples (50%). No mecC-positive isolates were detected in Body Fluids and Urine specimens. The presence of mecC is less common compared to mecA, as reflected in the distribution. Fisher's exact test was used to assess the association between mecC positivity and specimen type due to the low number of mecC-positive isolates (n = 4). The test revealed a borderline statistically significant relationship ($p = 0.048$), with higher mecC detection in pus (50%) and respiratory samples (25%), suggesting a potential niche preference for these infection sites.

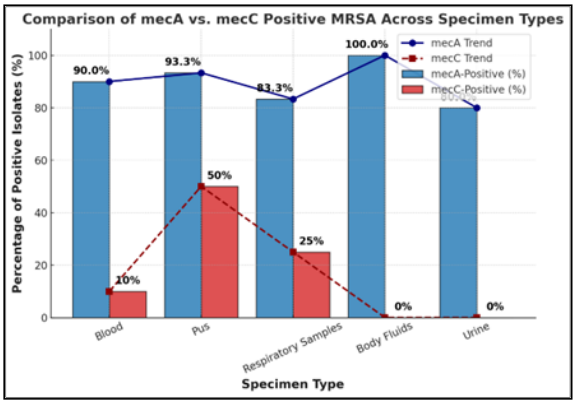

Figure 3: Distribution of mecA- and mecC-Positive MRSA Across Specimen Types. The bar chart represents the percentage of mecA-positive (blue) and mecC-positive (red) MRSA isolates detected in different specimen types. The overlaid line graph highlights trends, showing the dominance of mecA over mecC across clinical samples. This data underscores the higher prevalence of mecA-mediated resistance in MRSA strains, particularly in body fluids and pus samples.

DISCUSSION

In this study, the prevalence of MRSA in clinical specimens from a tertiary care hospital in Central India was found to be 3.2%. This is significantly lower than the 18–25% prevalence documented in previous Indian studies.[1,2] One likely explanation for this discrepancy could be the rigorous infection control policies and antimicrobial stewardship practices implemented at our institution. Although differences in MRSA detection rates were observed across specimen types, the variation was not statistically significant ($\chi^2 = 1.14$, $p = 0.88$), indicating a relatively uniform distribution of MRSA infections across clinical sample types.

Genotypic analysis showed that 90% of MRSA isolates were mecA-positive, while 10% harbored the mecC gene. This difference in gene prevalence was statistically significant ($\chi^2 = 25.6$, $p < 0.001$), confirming the predominance of mecA as the primary methicillin resistance determinant. This is consistent with global reports, where mecA is known to contribute to the majority of MRSA resistance, particularly in healthcare settings.[3] However, the identification of mecC-positive isolates in 10% of cases, including those from hospitalized patients, is noteworthy. While mecC-MRSA is often associated with livestock and community reservoirs,[4] its detection in our study population suggests a possible adaptation to the hospital environment, warranting further molecular epidemiological investigations.

Pus samples demonstrated the highest number of MRSA isolates (15 out of 450; 3.33%), reinforcing the clinical significance of MRSA in skin and soft tissue infections (SSTIs). This is in agreement with findings from Southeast Asia and Africa, where MRSA is routinely isolated from wound and pus cultures.[5,6] These infections can lead to complications such as abscesses, osteomyelitis, and bacteremia, highlighting the importance of early diagnosis and appropriate therapy.[8,9]

Antibiotic susceptibility testing revealed 100% resistance to cefoxitin, which phenotypically confirmed the methicillin-resistant status of all isolates. High resistance rates were also observed for erythromycin (82.5%), ciprofloxacin (67.5%), and clindamycin (57.5%), while moderate resistance to cotrimoxazole (50%) was seen. Encouragingly, no resistance to vancomycin or linezolid was observed. These results align with other Indian studies where glycopeptides and oxazolidinones remain effective treatment options against MRSA.[11] However, emerging reports from Europe and the United States have documented intermediate or resistant strains to vancomycin and linezolid, raising concern for the future efficacy of these last-resort drugs.[12]

Comparative analysis of resistance patterns between mecA- and mecC-positive MRSA isolates revealed no statistically significant differences (Fisher's exact test, $p > 0.05$ for all comparisons). For instance, erythromycin resistance was observed in 83.3% of mecA-positive and 75% of mecC-positive isolates ($p = 1.00$). These findings indicate that mecC-MRSA strains may pose similar therapeutic challenges as their mecA counterparts, and underscore the need for their routine molecular detection.

Analysis of mec gene distribution by specimen type provided additional insight. mecA-positive MRSA was prevalent across all sample types, with body fluids (100%), pus (93.3%), and blood (90%) showing the highest frequencies. Conversely, mecC-positive MRSA was most frequently detected in pus (50%) and respiratory samples (25%), with no detection in urine or body fluids. The association between mecC and certain specimen types showed a borderline statistical significance (Fisher's exact test, $p = 0.048$), suggesting a potential specimen-specific niche or tropism, as also documented in European studies.[13] The presence of mecC-MRSA in respiratory and soft tissue specimens supports earlier

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reports indicating its emergence in both hospital and community settings.

The detection of mecC in Indian hospital-acquired isolates is especially important, as current diagnostic protocols may fail to detect mecC-positive strains due to reliance on mecA-targeted assays.[4,14] This may lead to underreporting, misclassification, or delayed initiation of appropriate therapy. Integration of multiplex PCR kits or genotyping panels capable of detecting both mecA and mecC is therefore recommended.

In the broader Indian context, the lack of routine molecular surveillance, high population density, and widespread antimicrobial misuse continue to accelerate the spread of multidrug-resistant pathogens, including MRSA. There is an urgent need for enhanced diagnostic capability, clinician awareness, and One Health-based interventions that address both human and veterinary sources of resistant strains.[6,13]

In summary, the combined occurrence of mecA- and mecC-associated MRSA, high levels of multidrug resistance, and the presence of mecC in hospital-acquired isolates reflect a shifting epidemiological and resistance landscape for MRSA in Central India. These findings highlight the critical importance of molecular diagnostics, judicious antimicrobial use, and sustained infection control to mitigate the burden of MRSA in healthcare facilities.

Hypothesis Testing and Statistical Inference

To interrogate the study aims rigorously, three a priori hypotheses (H1a–H1c) were formulated and examined with appropriate inferential statistics ($\alpha = 0.05$). All computations were performed in IBM SPSS Statistics v26.0.

H1a – Gene specific prevalence

Observed frequencies (36 mecA, 4 mecC) deviated markedly from equiprobability. The goodness of fit test ($\chi^2 = 25.6$, $p < 0.001$) rejected the null hypothesis, confirming a nine-fold excess of mecA positive MRSA. The 95 % exact binomial confidence interval (CI) for mecA prevalence was 75.3–97.9 %, underscoring its predominance in the study population.

H1b – Comparative resistance phenotypes

Resistance proportions for erythromycin (83.3 % vs 75 %), ciprofloxacin (69.4 % vs 50 %), clindamycin (55.6 % vs 75 %), and cotrimoxazole (50 % vs 50 %) did not differ significantly between mecA and mecC groups (Fisher's $p \geq 0.63$). Because the mecC subset was small ($n = 4$), exact testing was mandatory; nevertheless, no antibiotic showed genotype linked divergence, indicating phenotypic equivalence in multidrug resistance.

H1c – Specimen niche association

Cross tabulation revealed that 50 % of mecC positive isolates originated from pus and 25 % from respiratory specimens, whereas none were recovered from urine or body fluids. Fisher's exact test produced a borderline significant association ($p = 0.048$), implying a potential ecological preference for superficial and respiratory niches. Given the limited cell counts, these data warrant confirmation in larger surveillance cohorts.

Interpretative Synopsis

Collectively, the inferential analyses validate the dominance of mecA as the chief methicillin resistance determinant, demonstrate phenotypic parity in antimicrobial resistance between mecA and mecC MRSA, and provide preliminary evidence that mecC strains may cluster in specific clinical specimens. These findings reinforce the necessity of dual target molecular diagnostics and specimen level surveillance to capture the full spectrum of MRSA genotypes circulating in Indian healthcare settings.

CONCLUSION

This study shows the co-occurrence of mecA- and mecC-associated MRSA in clinical specimens which brings to light the need for precise molecular diagnostics for effective management. To lessen the consequences of MRSA in healthcare facilities, effective antibiotic stewardship programs and diligent infection control are essential.

Further Recommendations: Developing strategies for the effective management of MRSA infections is largely dependent on understanding the local epidemiology. An integrated approach using sociodemographic and clinical data as one study showed can help track individual cases from notification to the hospital. Centralized databases can serve as a detailed resource while networking with multiple healthcare systems allows consolidation of data leading to focused MRSA studies. There is a gap in understanding the identified factors of hidden reservoirs of MRSA and understanding their implications is critical for mitigating its spread. Improved collaboration between the human and veterinary health worlds is necessary for the effective surveillance and control of MRSA in animals, which will, in turn, improve treatment outcomes for patients and slow the rate of antibiotic resistance.

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