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Methicillin-Resistant *Staphylococcus aureus* [MRSA] Bacterial Infection Overview

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ABSTRACT

Methicillin-resistant Staphylococcus aureus (MRSA) is a significant public health concern due to its ability to cause severe infections and its resistance to multiple antibiotics. This abstract aim to provide an overview of MRSA, including its epidemiology, pathogenesis, clinical manifestations, diagnosis, treatment, and prevention strategies. MRSA is a strain of Staphylococcus aureus that has acquired resistance to the beta-lactam class of antibiotics, including methicillin, which traditionally served as an effective treatment against Staphylococcal infections. The emergence and spread of MRSA strains, both in healthcare settings (hospital-associated MRSA) and in the community (community-associated MRSA), have posed substantial challenges in the management of staphylococcal infections. The primary mode of transmission for MRSA is through direct contact with infected individuals or contaminated surfaces. Risk factors for MRSA acquisition include prolonged hospital stays, invasive procedures, immunosuppression, and close contact with carriers. MRSA infections can manifest as skin and soft tissue infections, bloodstream infections, pneumonia, or bone and joint infections, among others. In conclusion, MRSA poses a significant public health challenge worldwide. Understanding its epidemiology, pathogenesis, diagnosis, treatment, and prevention strategies is crucial for healthcare professionals to effectively manage MRSA infections, limit its spread, and develop strategies to combat antibiotic resistance.

1. INTRODUCTION

Methicillin-resistant Staphylococcus aureus (MRSA) is a significant public health issue that affects people all over the world, leading to high morbidity and mortality, as well as increased medical expenses. MRSA was first identified in clinical isolates from hospitalized patients in the 1960s, but it has since spread quickly throughout the community since the 1990s [5,4]. Over 18 000 deaths per year were attributed to the estimated 94 360 invasive MRSA infections that occurred in the United States in 2005. Methicillin-resistant S aureus (MRSA) frequency has grown during the past ten years, and hospital discharges linked to MRSA skin and soft tissue infections have tripled since 2004. MRSA infections are linked to lengthier hospital stays and a greater financial impact on society, with all inpatient days in 2003 costing an estimated US $14.5 billion. An example of the increased morbidity and mortality associated with MRSA can be seen when comparing the yearly infection rates and mortality rates in the United States for MRSA, AIDS, viral hepatitis, and tuberculosis. Methicillin-resistant S aureus is estimated to cause more infections than the other diseases combined and more deaths per year than AIDS [1].

It is now the primary cause of infective endocarditis (IE) in much of the world and is the second most frequent cause of hospital bloodstream infections. IE is still a dangerous and fatal condition despite recent improvements in detection and treatment. Left-sided IE still has a 30-day all-cause mortality rate of 23.9%, and right-sided IE has a mortality rate of 11.8%. The characteristic peripheral IE stigmata are frequently ambiguous or absent, especially in patients whose Staphylococcus aureus infection caused their IE. The high prevalence of individuals without clinical signs and the high mortality of untreated IE highlights the significance of a diagnostic approach sensitive enough to identify the condition. Transthoracic echocardiography (TTE) is advised for all patients with suspected IE, according to recent studies, the American College of Cardiology/American Heart Association guidelines for IE, and the European Society of Cardiology. Transesophageal echocardiography (TEE) should be performed first in adults with suspected IE, according to cost-effective calculations. Additionally, it has been suggested that all SAB patients should be viewed as having a high risk of developing IE and that they should all receive TTE/TEE evaluations. Due to the increased clinical suspicion of IE in patients with SAB, Habibetal recommended that a negative TTE in patients with SAB be followed by a TEE. Uncertainty exists regarding whether a TEE is necessary for all SAB patients. Recent research indicates

that additional effort is required to identify a subgroup of SAB patients who might only require TTE for their assessments of IE [6]. It has been suggested that patients with SAB who have low risk of IE and don't need a TEE test can be identified by the absence of specific clinical features. Our study's objective was to identify SAB patients with low risk of IE by applying streamlined prediction parameters that take into account prevalent IE risk factors [8]. Bacterial infections of PUs are one of the main reasons people with diabetes and obesity are admitted to hospitals. Osteomyelitis, sepsis, and amputation of diseased limbs are all possible outcomes of infections that can spread to underlying tissues and bones. According to estimates, 15% of patients who develop a diabetic foot ulcer in the United States will need to have the affected limb amputated. This is frequently because of a bacterial infection that prevents the production of granulation tissue and impairs wound healing [7]. According to recent estimates based on clinical data, illnesses brought on by microorganisms resistant to antibiotics are thought to be the cause of about 25,000 fatalities annually in the EU and 700,000 worldwide. The World Health Organisation (WHO) in particular is working very hard to put into effect a worldwide action plan to combat antimicrobial resistance by establishing strategic objectives like awareness, surveillance, and study of antimicrobial resistance. MRSA strains that cause human infections are classified into three types based on their origin: healthcare-associated (HA-MRSA), community-associated (CA-MRSA), and livestock-associated (LA-MRSA). MRSA strains that causes human infections are classified into three types based on their origin: healthcare-associated (HA-MRSA), community-associated (CA-MRSA), and livestock-associated (LA-MRSA). CA-MRSA strains typically attack seemingly healthy persons and differ significantly from HA-MRSA strains in terms of antibiotic resistance and the presence of the virulence component CA MRSA isolates, on the other hand, have become a significant cause of hospital epidemics. Finally, LA MRSA strains have been found in agricultural and companion animals, as well as food derived from animals such as pigs, poultry, and cattle [8].

A prior systematic study investigated the potential risk factors for MRSA at the time of hospitalization or intensive care unit admission. It included 29 research and discovered that past hospitalization, nursing home exposure, a history of exposure to healthcare-associated microorganisms, congestive heart failure, diabetes mellitus, lung disease, immunosuppression, and renal failure were all potential risk factors. A recent systematic analysis of nine high-quality studies (level A) found that antibiotic use and prior hospitalization were independent risk factors for MRSA colonisation in HIV patients [9].

2. MICROBIOLOGY

It is gram positive bacteria, non-motile and pus producing occur. Appearance 0.5 to 1.5 millimetre and they are clubbed together like grapes. More than 200 strains of auerus and possess several virulence factors that combined with increase antibiotic resistance contribute to its success as an infective agent.

3. DEVELOPMENT OF MRSA BACTERIA

MRSA development Alexander Fleming found in 1929 that a mould called Penicillin could release a chemical that killed bacteria, including certain staphylococci, and he named the filtrate of a broth of this culture penicillin. S aureus isolates became resistant to penicillin within a year of its introduction, and S aureus went on to develop resistance to other antibiotics. Only a decade after widespread prophylactic use of postoperative antibiotics became common, the first epidemics of nosocomial penicillin-resistant staphylococci were documented in Europe and North America in the 1950s. Penicillin-resistant strains were already deemed pandemic by the 1960s. Methicillin (also known as methicillin in other countries) was first used as an antibiotic against Staphylococcus aureus in 1959. By 1961, S aureus had developed methicillin resistance, which quickly spread throughout the world and is currently regarded endemic in most hospitals as HA-MRSA. As time progressed, S aureus not only evolved to become resistant to a variety of antibiotics, such as HA-MRSA, but also spread outside of healthcare institutions, causing infection in otherwise healthy individuals of the population as CA-MRSA [,6,10,11].

Methicillin-resistant S aureus are strains of S aureus that have evolved resistance to common antibiotics, even if they may be resistant to other recognized drugs in the penicillin and cephalosporin categories. Methicillin-resistant S aureus can survive in harsh settings for months and is thus spread from surfaces long after it has been deposited. Individuals with community-acquired MRSA and their close contacts are more prone to develop skin and soft tissue infections. CA-MRSA has recently become more common in people who are normally thought to be healthy (for example, athletes and soldiers). Because CA-MRSA and HA-MRSA are both common, it has been seen that the strains may coexist, with community strains imported into hospitals and HA-MRSA existing in the community. The distinction between HA-MRSA and CA-MRSA is thus blurring, and co-colonization in the hospital is predicted to become normal [6,12]. There is a higher likelihood of finding both HA-MRSA

and CAMRSA in clinics where postoperative patients interact with nonoperative patients, necessitating professional vigilance for both types and the adoption of appropriate infection control measures. Drug resistance in methicillin-resistant S aureus is still developing. Drugs including macrolides, lincosamides, fluoroquinolones, and aminoglycosides are effective against more than 50% of MRSA strains, and trimethoprim-sulfamethoxazole is effective against 30% of MRSA strains. One of the few drugs still in use to treat MRSA was vancomycin; however, vancomycin-resistant MRSA is now a reality. Methicillin-resistant S aureus is transmitted more easily in the community but has generally remained more susceptible to a broader range of antibiotics; multidrug resistance in CA-MRSA, nevertheless, has been detected. Furthermore, as community and hospital strains mix and patients and community members carry these strains into the hospital, there is growing concern that highly virulent community strains that infect healthy people will become less vulnerable to antibiotics[13].

4. ETIOLOGY

Teichoic acid on the cell wall mediates the initial bacterial adhesion to the host's epithelial cells during S. aureus colonisation, whereas microbial surface components recognising sticky matrix molecules play a part later on in the nasal colonization process. S. aureus clumping factor B (ClfB), one of these components, has been investigated in vitro and in volunteers[14]. Inoculated into the nose were a wild-type strain and its single locus ClfB knockout variation; the knockout version cleared considerably faster than the wild-type strain. ClfB-deficient strains, on the other hand, may interact with nasal cells, demonstrating that numerous independent microbial surface components contribute to colonisation. It should also be mentioned that this study only used one strain. In addition to host and pathogen factors, S. aureus interacts with other nasal-colonizing organisms (for example, Corynebacterium spp., Propionibacterium acnes, Staphylococcus lugdunensis, and Staphylococcus epidermidis). The presence of certain species in the nasal microbiota corresponds with the presence or absence of S. aureus, according to research [15]. The nasal microbiota species compete with one another in a variety of ways. For example, they struggle for adhesion sites and nutrients: the human nose has few nutrients. S. aureus can survive in lower-nutrient conditions than coagulase-negative staphylococci, probably due to changes in metabolism, and is hence better adapted to the human nose. However, there was no change in nutritional levels between carriers and non-carriers [16]. Antibiosis is another way microbiota

species compete; certain strains can create antimicrobial compounds that hinder their microbial competitors. For example, S. lugdunensis produces lugdunin, an antibacterial molecule that inhibits and destroys S. aureus (including MRSA) in vitro and in a mouse model, probably by causing fast breakdown of bacterial energy supplies. Nasal colonization with S. lugdunensis has been linked to a sixfold decreased incidence of S. aureus colonisation in humans. Although these findings are intriguing, they only explain a subset of carriage patterns, as S. lugdunensis colonisation has been observed in just 9.26% of the total population. Finally, S. aureus competes by inducing host defenses, which means that it causes the development of host antimicrobial proteins that are less toxic to S. aureus than to other commensal bacteria[17]. Many studies suggest the significance of these pathways in interactions between S. aureus and commensal microbiota, however, no one mechanism can explain all observed carriage patterns.

4.1. Virulence

S. aureus possesses a large arsenal of virulence factors (including adhesion, host-cell damaging, and immune modulatory molecules) that vary in their presence or specificity between clones, reflecting the wide range of illnesses that S. aureus can cause. Because many virulence genes are situated on mobile genetic components, their combination varies significantly between clones and even among closely related strains[18]. The potential link between various virulence factors and specific types or aggressiveness of S. aureus infections remains obscure, most likely because many of these factors have redundant, partially overlapping activities. Furthermore, because many virulence variables are human-specific, they cannot be studied in animal models [19]. This section focuses on the most common virulence mechanisms and invasion routes.

4.2. Initiation of infection

Staphylococcus aureus SSTIs are typically caused by bacterial translocation (most likely by hand contact) from the primary reservoir in the nose to open skin microlesions and wounds. Surface proteins from Staphylococcus aureus (such as fibronectin-binding protein A (FnBPA), FnBPB, clumping factor A (ClfA), ClfB, and collagen adhesin (Cna)) bind to extracellular matrix proteins, allowing the germs to cling to and grow on injured tissues S. aureus' ability to adhere to and form biofilms (sticky agglomerations of microorganisms embedded in an extracellular matrix; biofilms facilitate resistance to mechanical interference,

host defences, and antibiotic treatment) on artificial plastic or metal surfaces makes S. aureus a common cause of catheter-associated, joint-replacement-associated, or ventilator-associated infections. S. aureus manipulates the following inflow of polymorphonuclear leukocytes (PMNs), which affects local inflammation[19].

4.3. Abscess formation

By forming a fibrin pseudo-capsule around the bacteria and infiltrated PMNs, the S. aureus coagulase proteins stop additional leukocyte influx[20]. For example, S. aureus can prevent opsonization by creating a polysaccharide microcapsule and blocking the complement cascade. However, crucial MRSA clones like USA300 lack the microcapsule. Bacteria that are phagocytosed by PMNs can survive not only by counteracting PMN-killing mechanisms but also by gradually destroying them with the help of cytolytic toxins. For instance, many CA-MRSA clones produce pore-forming peptide (phenol soluble modulins; PSMs) and protein toxins (-toxin; also known as -hemolysin; and several bi-component leukocidins; PVL); which are host species-specific and bind to host leukocyte membranes, causing pores to form and lytic cell death; this increases bacterial virulence). S. aureus superantigen toxins, which bind to MHC class II antigen-presenting cells and activate a significant portion of T cells non-specifically, exacerbate the severe inflammation induced by activated or necrotic PMNs. This systemic hyper-inflammation is known as "cytokine storms"[21].

4.4. Systemic infection

Abscesses may rupture later on, releasing pus and live bacteria either onto the skin's surface to aid in the spread of pathogens or into the bloodstream to result in bacteremia. Endovascular S. aureus can stick to endothelial surfaces and platelets, which can lead to endocarditis, encourage the growth of metastatic abscesses, or cause the uptake of bacteria into endothelial cells, where they are difficult for antibiotics and host defence molecules to reach. If the endovascular spread of the bacteria is not controlled, the agglutinating activity of coagulases is thought to contribute to systemic blood coagulation, and massive release of microorganism-associated molecular pattern molecules combined with superantigen toxin-induced cytokine storms results in fulminant systemic inflammation, sepsis, and multi-organ failure[22].

5. RISK FACTOR:

MRSA infection in the inpatient setting is lower the immune system and it is primary risk factor. Most of risk infections are chronically ill ,burn survivor, organ transplant, cancer patient, steroids and IV drug user and diabetic patient aids .Other risk for MRSA infection is long stay in hospital, exposure to antibiotic and outpatient, skin to skin contact with patient, crowd are , athelet, and military or prison infants sharing kits[1,23,].

6. SIGN AND SYMPTOMS:

It appears as a bump or infected area on the skin that might be

- Warm to touch
- Painful
- Red
- Swallon
- Full of pus or other drainage
- Accompanied by a fever

7. DIAGNOSTIC TEST

MRSA-isolating microbiological samples can be roughly divided into clinical and screening samples. Individuals with symptoms or signs are given clinical samples (such as samples of purulent discharge, deep tissues, sputum, and blood) to look for active infection, whereas

screening samples (such as nasal, perineal, and throat swabs) are taken to look for asymptomatic colonization. MRSA can be detected directly from clinical or screening samples using a variety of phenotypic and non-phenotypic techniques, or it can be detected from presumed staphylococcal colonies isolated from clinical samples. In most cases, phenotypic techniques are preferred for clinical diagnoses.

7.1. Phenotypic methods

The disk diffusion method can be used to test for methicillin resistance in pure S. aureus cultures that were produced by plating clinical samples on appropriate culture conditions. Applying a cefoxitin disc to Mueller-Hinton agar or adding 6 micrograms of oxacillin and 4% NaCl to Mueller-Hinton agar (following the recommendations of the Clinical and Laboratory Standards Institute, or CLSI) is required for this procedure. Initially, oxacillin was used as the marker antibiotic to identify MRSA; however, CLSI now advises cefoxitin since it induces mecA and mecC more effectively than oxacillin and produces a phenotype that is easily recognisable. To avoid producing misleading negative results, the disk-diffusion method needs strict adherence to temperature (35 °C) and time (reading after 24 hours). This is because the resistant isolates develop more slowly because the mecA-expressed PBP2a is less effective in crosslinking the pentapeptide chains of the cell wall peptidoglycan during cell wall formation. This event creates a population of cells with varied levels of resistance, some of which are also phenotypically susceptible. The slower-growing MRSA subpopulation can reach detectable levels in a heteroresistant population thanks to the aforementioned susceptibility testing recommendations. Rarely, MRSA may present with phenotypic sensitivity to cefoxitin (and oxacillin) and require an overnight exposure to low concentrations of cefoxitin to exhibit resistance. In this case, the presence of inducible mecA should be considered. Methicillin resistance in S. aureus colonies and cultures can also be detected by means of an antigen–antibodybased latex agglutination test that detects PBP2a by using an anti-PBP2a antibody. Moreover, several automated instruments performing identification and antimicrobial susceptibility testing of staphylococci have shown high sensitivities and specificities for the MRSA strains tested. The development of bacteriophage-based assays has regained attention for the direct phenotypic detection of MRSA from positive blood cultures. The KeyPath MRSA/MSSA blood culture test (MicroPhage Inc., Longmont, Colorado, USA) is a non-genotypic, fast test for the detection of methicillin resistance directly from positive blood cultures that has been authorized by the

US FDA. With a turnaround time of 5 hours, the assay may identify the amplification of S. aureus-specific bacteriophages in the presence of methicillin. With a median turnaround time of 16.9 hours compared to 46.9 hours calculated for conventional tests for the identification of S. aureus and differentiating between MRSA and MSSA in positive blood cultures, a multi-center evaluation of this assay on 1,116 blood cultures showed 91.8% sensitivity, 98.3% specificity, 96.3% positive predictive value, and 96.1% negative predictive value[2].

7.2. Non-phenotypic methods

Matrix-assisted laser desorption-ionization time-of-flight mass spectrometry is one of the most promising non-genotypic techniques for direct identification of pathogens from positive blood cultures. The protein profile produced by mass spectrometry from a bacterial or fungal sample is compared to a library of profiles derived from numerous characterized microorganisms. However, because MALDI-TOF MS performance is heavily dependent on the purity and quantity of a bacterium, bacterial enrichment and purification methods are necessary from positive blood cultures, which include high concentrations of interfering non-microbial material [24]. A retrospective study of 227 cases of S. aureus bacteremia comparing turnaround time and therapy adjustment before and after the introduction of MALDI-TOF MS plus real-time PCR to detect mecA revealed a nearly 50% reduction in turnaround time of MRSA identification compared to S. aureus identification and -lactam susceptibility testing using conventional methods. Although the length of hospitalisation and rates of adequate empirical antibiotic therapy were comparable in the two groups, therapy optimisation happened more frequently in the MALDI-TOF MS group [25]. Current DNA-based methods for direct MRSA detection from clinical samples are multiplex real-time PCR assays to detect S. aureus and the presence of mecA and are well-validated assays. Results are obtained in approximately 1.5 hours. The FilmArray (Idaho Technology, Salt Lake City, Utah, USA) is a multiplex PCR-based system designed to detect 25 microorganisms (90–95% of the pathogens involved in blood cultures) along with mecA, as well as the presence of genes encoding resistance to vancomycin (A and B) and carbapenems (blaKPC)[26]. With an average turnaround time of 2.5 hours, this technique offers higher sensitivity than MALDI-TOF MS in detecting bacteria from blood culture bottles before positive. Since in vitro culture, the application of WGS to bacterial pathogens has been hailed as the single most significant innovation in diagnostic microbiology and monitoring. However, practical uses of WGS in diagnostic microbiology are still limited, owing to technological limits in getting

results promptly that can impact patient care, as well as the requirement for standardized protocols and automated data interpretation. With the introduction of the third generation of sequencers (such as the Oxford Nanopore MinION by Pacific BioSciences and Oxford Nanopore, Oxford, UK), longer reads (obtained sequence lengths) that can span repeat regions in the bacterial sequence and enable complete bacterial genome assembly, as well as increased portability of the machinery and a potential reduction in error rates, have resulted. The Oxford Nanopore MinION sequencer provides an important benefit in that sequencing data can be analyzed in real time, potentially leading to strain identification within 30 minutes and antibiotic resistance profile prediction within 10 hours after the start of a run, making this assay potentially useful for clinical diagnostics. The utility of WGS has been well demonstrated for studying antibiotic resistance and the population biology of MRSA[27] and has also led to many useful insights regarding the transmission of MRSA during hospital outbreaks and in community settings[2].

7.3. Screening methods

The effectiveness of screening measures is reviewed in the Prevention section below. Since the first MRSA chromogenic medium was introduced (that is, a medium containing synthetic chromogenic enzyme substrates; in the presence of the specific target enzyme, the chromogenic substrate is processed and results in a corresponding bacterial colony of a specific color, thereby enabling pathogen recognition), these media have undergone rapid improvements in terms of chromogen sensitivity and antibiotics used. [28,29]. Since their introduction in the 2000s, they have become the principal quick diagnostic assays used for active surveillance for MRSA colonisation as well as patient diagnoses. An external quality review conducted in 23 European countries and Israel in 2005 discovered that 88% of participating laboratories used a chromogenic medium alone to screen for MRSA. The use of chromogenic medium in conjunction with MALDI-TOF MS to identify the species of numerous colonies in Real-time PCR-based assays for MRSA screening from nasal swabs can reduce turnaround time to 1-2 hours, whereas chromogenic media-based tests can take 14-18 hours without confirmatory testing and thus may not always be useful to guide clinical decisions. An observational cohort study found that screening with a same-day commercial real-time PCR assay resulted in a significant reduction in MRSA transmission compared to screening with conventional culture (swabs incubated overnight in 7% NaCl and subcultured on mannitol salt agar with 2 milligrams per litre oxacillin for 48 hours): MRSA transmission

was 4.9 new acquisitions per 1,000 patient bed days with real-time PCR versus 13.9 (A patient bed day is a unit of time during which a patient occupies a bed and spends the night in a health-care institution; for example, 50 patients at a hospital over the course of one day would equal 50 patient bed days.) A large study in 13 ICUs in eight European countries, however, found no difference in the acquisition and transmission rates of multidrug-resistant bacteria (including MRSA, vancomycin-resistant enterococci, and highly resistant Enterobacteriaceae) using PCR-based tests versus chromogenic media. Similarly, a UK-based study comparing real-time PCR-based tests to slower laboratory-based methods (MRSA-selective broth and chromogenic medium) found a significant reduction in turnaround times (from 40.4 to 3.7 hours) but no effect on MRSA acquisition rates, casting doubt on the utility of the more expensive but faster PCR-based screening[2,29].

8. PREVENTION

- MRSA infection control interventions have been widely implemented in healthcare facilities. This control prevents the spread of infection among patients or other interactions. Several measures should be performed to prevent MRSA bacterial infection [1], and they are as follows:

8.1. Hand hygiene

Health care personnel can get MRSA on their hands via handling equipment that has been contaminated with MRSA or from coming into touch with patients who have the infection. MRSA can then be spread from one patient to another through this method. Using soap and water or alcohol-based hand rub, good hand hygiene seeks to reduce the spread of MRSA through this pathway. Even though the hand hygiene campaign was implemented alongside other national infection control measures, after accounting for all other interventions, higher purchases of alcohol hand rub during the campaign were found to be independently linked with a decrease in the prevalence of MRSA bacteremia [1,30].

8.2. Contact precautions and isolation

The risk of MRSA transmission via contaminated hands and clothing is decreased in many facilities by using contact precautions (disposable gowns and gloves) when caring for patients with MRSA colonization. Although the prior evidence supporting this intervention was of low quality, more solid data currently suggests that this practice is linked to a decrease in

MRSA acquisition. Patients with MRSA colonization should also generally be segregated in separate rooms. However, single-room isolation proved ineffective in preventing MRSA transmission in a prospective study conducted in an ICU setting where MRSA was endemic and hand hygiene compliance was low [1,2,].

8.3. Antibiotic stewardship program

Since May 2006, there have been some initiatives in place to promote antibiotic stewardship due to worries about the connection between antibiotic overuse and the emergence of multidrug-resistant organisms in the hospital. Aiming to reduce incorrect prophylactic antibiotic use during surgery, inappropriate antibiotic combinations, and inappropriate antibiotic use, associated bylaws were established in March 2007. Beginning in August 2008, the strategy was strengthened by computerised prescription restrictions on third-generation cephalosporin and aminoglycoside for surgical procedures. Starting in August 2009, improper antibiotic combinations, such as redundant antimicrobial coverage, were also prohibited by computerized prescription systems. A committee of infectious specialists agreed on exceptions and clinical indications. Every three months, the antibiotic stewardship committee examined the amount of antibiotic use. The responsible departments received the results by letter [5,31].

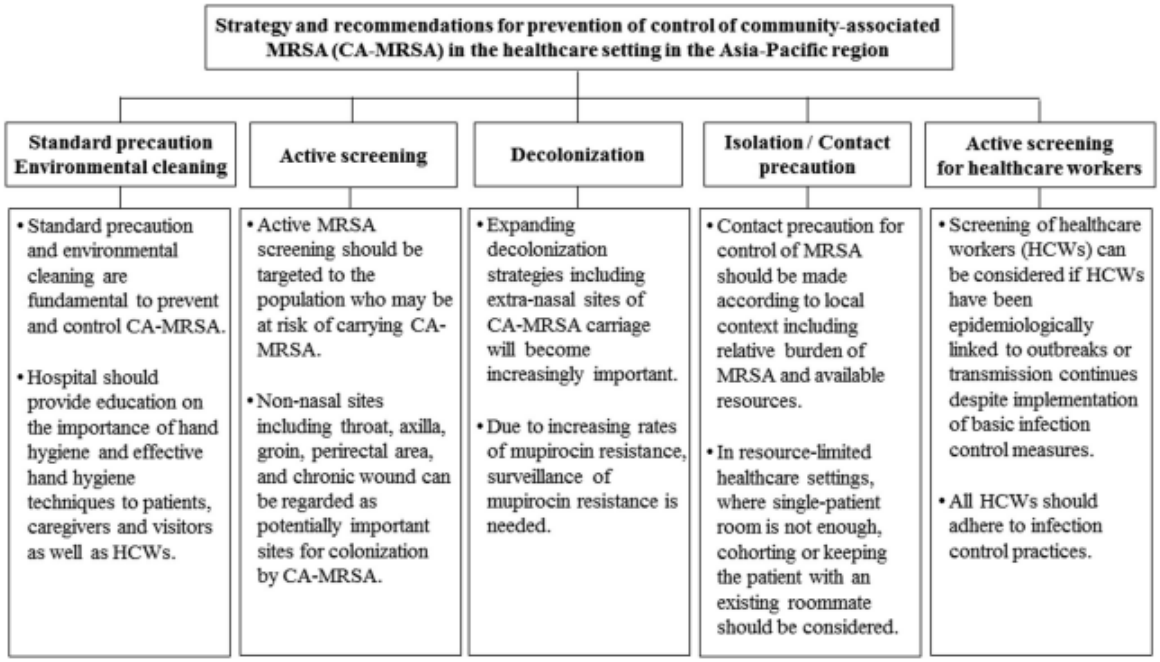
8.4. Prevention of health care

Preventing accidents for medical personnel When treating infected patients, standard measures are advised. As a result, medical professionals should use proper hand washing techniques and wear gloves when inspecting or treating body parts suspected to have cutaneous lesions. This includes washing hands properly, donning gowns, and protecting their eyes, mouths, and noses. Simple hand washing can significantly reduce the spread of illnesses [1]. For example, after establishing an alcohol-based gel hand sanitizer program for patients and their contacts, a research in Canada observed a 51% reduction in the incidence of HA-MRSA per 1000 hospital admissions, saving significant morbidity, mortality, and more than Can $858 000. Recently, US Veterans Affairs acute care hospitals reduced MRSA incidence across the country by implementing basic precautions, with an emphasis on hand hygiene and infection control [2]. The protocol's implementation resulted in a 45% drop in the frequency of MRSA infections linked to medical care in these healthcare settings. Exam or treatment tables, adjusting tables, plinths and equipment for physical therapy,

stethoscopes, blood pressure cuffs, and exercise mats are just a few examples of items frequently used in a manual practitioner's office that can quickly become contaminated with MRSA and stay that way for a while if not cleaned properly. Usage appropriate infection control procedures, such as disinfecting treatment tables, gym equipment, and mats with a wipe or cleaning agent after each usage[32].

8.5. Prevention of patient and athlete

Patients' and athletes' prevention Simple hygiene precautions are the cornerstone of MRSA infection prevention for healthy people who don't exhibit any symptoms or signs of infection. Hands should be properly washed with soap and warm water; if the hands are not obviously dirty, washing can be substituted with alcohol-based hand massages. People should refrain from sharing personal goods (towels, razors, washcloths, soiled clothes, used athletic equipment) and keep personal objects (clothes, bedding, towels, work/study space) clean to lower the chance of infection [1,2,32]. It's crucial to stop the spread of illnesses among individuals who already have them. Anyone helping to change a wound dressing should wash their hands first. Until they are healed, wounds should be bandaged, and any that contain pus should be covered with a clean, dry bandage to stop the spread of infection [1]. Bandages and wound dressings can be discarded with regular rubbish. If a person has a suspected MRSA skin infection, he or she should avoid self-treatment by popping, draining, or lancing the infection and instead seek the opinion of a health care expert. Because many sports entail close physical contact, including the use of communal equipment and bathing spaces, participants and athletic facilities can benefit from additional advice. It is especially critical to prohibit athletes with cutaneous MRSA infections from participating in close-contact sports until the wound can be completely covered or full recovery is demonstrated. Sharing clothing or protective gear, such as helmets and body armour, as well as bar soap, should be avoided among athletes. Wearing clean uniforms and changing clothes on a regular basis reduces infection risk and should be promoted. Athletes with known cutaneous MRSA infections should avoid using public pools, especially treatment whirlpools unless the pool water is changed regularly. Shared sporting equipment, such as weight machines and benches, should be disinfected on all surfaces where skin comes into contact with the equipment; floors, mats, and doors should also be cleaned on a regular basis [1,32].



Strategy and recommendations for prevention and control of community-associated methicillin-resistant Staphylococcus aureus (CA-MRSA) in the healthcare setting in the Asia-Pacific region. HCWs, healthcare workers.

9. TREATMENT

MRSA treatment is determined by the type of infection, its location, and its severity. When MRSA infection is detected, clinical practise guidelines for MRSA treatment recommend that proper medical care be initiated immediately [20]. The patient should avoid spreading the infection and not use wet compresses. Medical care utilising incision and drainage is the preferred treatment for skin abscesses. Using a disinfectant to cleanse the skin every day is a common component of CA-MRSA dermatological therapy. Antibiotics may be administered if nearby cellulitis is suspected. The selection of an antibiotic should be determined by the community's susceptibilities, but it typically starts with trimethoprim-sulfamethoxazole or, in the case of a patient who is sulfa allergic, doxycycline or minocycline. Cephalexin, dicloxacillin, or clindamycin typically offer further protection against methicillin-resistant S aureus and streptococci. If the first treatment for a methicillin-resistant S aureus infection is unsuccessful, multidrug therapy, such as the use of vancomycin in conjunction with one or more other antibiotics, may be necessary [1].

Although practically all -lactams are ineffective against common MRSA, particularly those linked to skin and soft tissue infections (SSTIs), -lactams continue to be a significant class of antibiotics for the treatment of S. aureus infections. Antibiotic selection is influenced by bacterial susceptibility, patient features, and the location of the infection. MRSA responds to specific medications from each antibiotic class, but due to MRSA prevalence in hospitals, antibiotic resistance, and disease load, it is often essential to treat persistent infections with last-line or novel antibiotics. The preferred antibiotic for the treatment of significant MRSA infections is vancomycin, but its effectiveness is limited by chronic or recurring bacteremia, nephrotoxicity, and the formation of non-susceptible strains. Alternative antibiotics, such as linezolid and daptomycin, are as effective as vancomycin. Currently, vancomycin, teicoplanin, arbekacin, and linezolid are used as therapeutic agents for the treatment of MRSA infections. Furthermore, daptomycin and tedizolid were approved for the treatment of MRSA infections in Japan in 2011 and 2018, respectively. Ceftaroline fossil, a novel cephalosporin antibiotic, has been licenced in Europe and the United States for the treatment of complex SSTIs and community-acquired (CA) pneumonia.[an antibiotic].Combinations of two primary active agents [Vancomycin and Linezolid] that act on the cell membrane via a complex process that results in cell membrane depolarization and permeabilization, ion leakage, and, eventually, cell death[6].

10. CONCLUSION

MRSA (Methicillin-resistant Staphylococcus aureus) is a type of bacteria that is resistant to many commonly used antibiotics, making it difficult to treat. Infection with MRSA can lead to a range of symptoms, from mild skin infections to severe and life-threatening conditions such as pneumonia, bloodstream infections, and surgical site infections.

In conclusion, MRSA bacterial infection presents significant challenges in healthcare settings and communities worldwide. It is a major concern due to its ability to resist multiple antibiotics, limiting treatment options and increasing the risk of complications. Prevention and control measures, including good hygiene practices, appropriate antibiotic use, and infection control protocols, are crucial to managing MRSA infections and preventing their spread.

Healthcare providers and individuals need to be vigilant and take necessary precautions to minimize the risk of MRSA transmission. Early identification, prompt treatment, and

adherence to infection control measures are essential in effectively managing MRSA infections and preventing their dissemination. Ongoing research and development of new antibiotics and alternative treatment strategies are also critical in combating this antibiotic-resistant pathogen.

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"One of the ways that we believe people express their appreciation to the rest of humanity is to make something wonderful and put it out there".

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