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Methicillin Resistant Staphylococcus Infections: A Review

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Abstract: ***Staphylococcus is one of the most important pathogen gaining importance these days due to their role in healthcare as well as among community acquired infections. The organism's having various virulence factors associated with infections. The burden of infections is further complicated by the emergence of antimicrobial resistance among the pathogen. However antimicrobial resistance is another concern throughout the world. The emergence of methicillin resistance among staphylococcus aureus and coagulase negative staphylococcus makes the treatment options very limited. Appropriate antimicrobial stewardship programs must be conducted in order to combat with the antimicrobial resistance.***

Keywords: Staphylococcus, pathogen, infections, antimicrobial resistance, treatment options

1. Origin

Scottish surgeon Alexander Ogston (1844–1929) made the crucial discovery on what causes pus in 1880. Ogston was the one who translated Joseph Lister's antisepsis value into practise because of the high mortality rate and his refusal to accept death as a common surgical outcome (1827 - 1912).[1]

In 1882, Ogston's name was given to a group of micrococci known as "Staphylococci" which is Greek for "grape bunch". Two staphylococci strains were discovered by a German expert, Anton J. Rosenbach. He called them for the pigmented prevalence of their colonies as follows: strep throat in 1884 (1842 - 1923).[2]

Both HA and CA strains of S. aureus are now regarded as potentially harmful. Methicillin - resistant S. aureus (MRSA) was today on the rise in India, where methicillin - resistant S. aureus was first detected shortly after the country's founding in October 1960.3 MRSA is more common in the south of India, where it occurs at a rate of 50%, than in the west, where it occurs at a rate of 25%.[4]The advantages of S. aureus as a pathogen are due to a variety of potent virulence factors, including its ability to persist as a commensal and commonly gaining resistance to numerous virulence determinants and its many antimicrobial treatments.[5]

Antibiotics that are most frequently used to treat staphylococcal infections have seen an increase in resistance in India. antibiotics such semi - synthetic penicillin (methicillin, oxacillin, etc.), aminoglycosides, tetracyclines, and macrolides.[6]

In developing countries irrational antibiotic usage are the reasons for evolution of antibiotic resistance. The ingredients contributing to this are agriculture, animal husbandry, extensive surgical procedures and lack of barrier nursing practices.[7]

S. aureus

Rise in S. aureus antibiotic resistance, a key pathogen of concern (Lowy, 1998). Together with CoNS (e. g., S. epidermidis). In the growth of S. aureus, the cell wall is a strong, protective layer that is between 20 and 40 nm thick. The cell wall's primary component, peptidoglycan, accounts for up to 50% of the mass of the cell wall. Teichoic acids, which make up roughly 40% of the mass of the cell wall, are another component of the cell wall. They are a group of phosphate - containing polymers. The two are cell wall teichoic acid and lipoteichoic acid connected with cell membranes. Kinds of teichoic acids that are covalently attached to the peptidoglycan and introduced into the bacterial lipid membrane.[8]

Virulence factors

Staphylococcus become more drastic because of the virulence factors associated with different types of the toxic activities. Alpha toxin that is meditated as a major virulence factor of S. aureus in skin infections. Most of the *S. aureus* strains is secreted by the pore - forming toxin, which is water - soluble monomer that attack the RBC and consisting primarily of beta sheets. [9]LukS - PV and LukF - PV which are two different protein components based on their chromatography. are two highly effective cytotoxic components of Panton - Valentine leukocidin. The active toxin is associated with recurrent mucocutaneous, dermonecrotic disorders and chronic SSTI.[10]*S. aureus* has been generating various types of exoproteins contains taphylococcalenterotoxins (SEI, SEH, SECn, SEG, SEE, SED, SEB, and SEA), exfoliative toxins (ETB & ETA), andleukocidin.1[1]Hazardous Shock Syndrome Toxic shock syndrome has been brought on by toxin - 1 (TSST - 1). Necrotizing pneumonia, a more recent member of the SAg origins, is brought on by SEI - X.[12]Phenol - soluble modulins are also significantly influenced by S. aureus skin infection (PSMs). Modulins that were soluble in phenol were pore - producing toxins consists of a family of seven amphipathic - helical peptides. Almost all strains of S. aureus release modulins that are phenol soluble.

Ensure that they have the ability to lyse human neutrophils, osteoblasts that increase tissue toxicity, erythrocytes, and monocytes.[13]

Review of Studies in Developed/ Developing Countries

In 2019MRSA represented for 80.4% (201/250) of the total isolates, with males accounting for 56.2% (113/201) and females accounting for 43.8% (88/201). Males did not outnumber females in terms of carriage of acquired MRSA infections ($p > 0.05$). Pus swabs isolated MRSA at a high rate of 28.8% (58/201), vaginal swabs 25.4% (51/201), and urethral swabs 25.4% (51/59), whereas ear discharge had a lower rate of 0.5% (1/201). There was no statistically significant relationship between the incidence of Strains isolated and clinical specimens ($p > 0.05$). The incidence of MRSA isolates was distributed according to hospital ward, with outpatients accounting for 49.3% (99/201) of the isolates.[14]

Another study stated that the isolation rate of S. aureus from the 130 samples was 82 (63.1%). MRSA accounted for 59 (72% of the isolates), methicillin - intermediate S. aureus (MISA) for 7 (8.5%), and methicillin - sensitive S. aureus (MSSA) for 15 (19.5%). Antimicrobial susceptibility testing revealed that 13 (15.9%) of the isolates were resistant to vancomycin, 9 (11.0%) were resistant to erythromycin, and 1 (1.2%) were resistant to gentamicin.

MRSA isolates were also found to be resistant to the following antibiotics: oxacillin/erythromycin 5 (6.1%) and oxacillin/ vancomycin 9 (11%). Oxacillin/ vancomycin/ erythromycin 2 (2.4%) and oxacillin/gentamicin and erythromycin 1 (1.2%) resistance were observed in a few isolates.[16]

2. A Review of Studies in a Developing Nation: India

The author demonstrated that 92 cases of *S. aureus* were reported from an expected investigation. MRSA infections from Bangalore were 52.2 percent in HA infections and 47.5 percent in CA infections. The majority of samples were from SSTI, a severe source of S. aureus infections in both the community and hospitalised patients.

Earlier studies stated that 9.4% and 10.9 percent of CA - MRSA have been the subject of review studies in both southern and northern India; Further study in AP (India rural) 's area showed a prevalence rate of 64.7 percent in the community. As a result, India has a particularly high rate of community acquired diseases when compared to the other European continents.[17]

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MRSA was found in 37.5% of the 576 distinct isolates tested. Resistance to most antibiotics was predicted by methicillin resistance: erythromycin (90.9%), clindamycin (85.4% including inducible resistance), gentamicin, cipro - /levo - /moxi - foxacin, trimethoprim - sulfamethoxazole (58.3%), tetracycline, and rifampin. Daptomycin, linezolid, tigecycline, and (approximately) vancomycin all had nil resistance rates. MDR isolates were found in 48.5% of cases.[18]

2018

The study estimated the prevalence of MRSA and *S. aureus* in the surgical site diseases (SSI) in Mangalore, a health aware, coastal city of almost 1 million populations.

They could see that, 300 pus samples that has been received from the victims with the (SSI), is the maximum incidence of SSIs were observed in the orthopaedic cases was (52.3%), followed by (17.6%) and (30.1%) was the obstetrics & gynaecology cases and surgical has been observed respectively. Further, the author notes that the majority of the orthopaedics patients (98) that were involved in this research were handled for the open reduction of fractures. Surgical site infections (SSI) in orthopaedic surgeries, contamination from the external nature are one of the possible reasons for the highest rate of infections. Among them, Male victim (63%) was higher in number than the female victim (37%). That can be due to the multiplying mobility rate in the male population.[19]

Methicillin – Resistant Staphylococcus Aureus (MRSA)

British scientist Jevons looked into the identification of an MRSA strain in 1961, two years following the licensure of methicillin. The gene for penicillin - binding protein 2a or 2 (PBP2a or PBP2′) (mecA), which united SCCmec of methicillin - sensitive S. aureus strains, was the cause of this resistance.[20]

It has emerged in the previous ten years as a nosocomial infection trigger for a number of quickly progressing, sometimes fatal illnesses, including endocarditis, necrotizing fasciitis, osteomyelitis, severe sepsis and toxic noses like toxic shock syndrome.[21] haemodialysis, advanced age, Immunosuppression, peripheral malperfusion, prolonged clinic stays, residency in every lasting care service, insufficient of antimicrobial therapy, insulin - dependent diabetes, indwelling devices, and decubitus ulcers are just a little of the multifactorial field of self – sufficient exposure agent for MRSA that were reported in the literature.[22]

New genetically different MRSA strain reservoirs, such as livestock - acquired MRSA and community - associated MRSA (CA - MRSA), were founded. Healthcare - associated methicillin - resistant S. aureus, also known as HA - MRSA, was first identified as a concern in healthcare facilities.[23]The public health issue brought on by MRSA has been made worse by the quick emergence of novel genetic lineages, clonal complexes (CC), and species as well as resistance many different types of antibiotics, as well as linezolid (oxazolidinone), the only latest antibiotic group in about 20 years.[24]

Healthcare - associated methicillin - resistant S. aureus (HA - MRSA) is currently linked to important mortality and morbidity (longer hospital stays) and increasing the financial burden of the world's limited healthcare resources (MSSA).[25]

In 1961, two years after the registration of methicillin, British scientist Jevons researched the isolation of an MRSA variant.; this resistance was created by the target gene penicillin - binding protein 2a or 2 PBP2a or PBP2′) (mecA), which would then merge SCCmec of methicillin -

sensitive S. aureus isolates. MRSA was first classified as either hospital - acquired MRSA (HA - MRSA) or community - acquired MRSA (CA - MRSA).[26]

Conflict of interest

There is no conflict of interest.

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