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Case Report

Management of MRSA patients on the dental chair

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ABSTRACT

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a type of *staphylococcus* bacteria that is resistant to certain antibiotics, including methicillin, penicillin and amoxicillin. For decades, MRSA primarily has affected people who are immunocompromised, such as patients in hospitals and long-term care facilities. Recently, it has been detected in otherwise healthy people. These infections typically show up as skin infections, like abscesses or boils. Less often, these infections can be more severe, causing pneumonia, sepsis or other potentially life-threatening infections. Most of the MRSA infections are of skin origin in the community. Its main mode of transmission is through the hands i.e., of the health care workers. So, hand washing is the most crucial factor in preventing the spread of infection. In a treatment area, the dental chair including the seat and arm rest, floor beneath the chair, sink, towel dispenser, counter top, and suction chamber remain the sources of infection. These usually are not directly contacted with the patient. Other routes of transmission of MRSA include body fluid exposure to non-intact skin of health care personnel, mucous membranes, or through the sharp or percutaneous injuries. In dentistry, MRSA is known to colonize the saliva and so considered as potentially infected material and often contains blood. The present case report is to create awareness about MRSA transmission, as well as infection prevention and control measures for dental practitioners.

Keywords: Immunocompromised, MRSA, *Staphylococcus aureus*

INTRODUCTION

Use of antibiotics in dentistry for prophylactic and therapeutic purposes has been a protocol and the use of broad spectrum antibiotics alone or in combination has increased alarmingly leading to development of resistance to various bacterial infections caused by new strains like methicillin resistant *Staphylococcus aureus* (MRSA), vancomycin resistant *Staphylococcus aureus* (VRSA) and vancomycin-intermediate *Staphylococcus aureus* (VISA). Prophylactic prescription of antibiotics by the dentist is playing a significant role in the emergence of resistant microbial strain.[1]

Epidemiology

Staphylococcus aureus is a major cause of serious hospital-acquired infections worldwide. Between January 1999 and December 2002, 50,759 nosocomial isolates were collected from 495 hospitals in 26 countries.

The prevalence of MRSA varied from < 1% in Northern Europe to > 40% in southern and Western Europe. During this time, the incidence of MRSA increased significantly in many countries, including Belgium, Germany, Ireland, and the UK, highlighting the need for better infection control procedures.[2]

Mechanism of β-lactam resistance in MRSA

The cell wall of gram-positive bacteria is composed of a single, extensively cross-linked peptidoglycan macromolecule, which confers strength and rigidity, maintaining the bacterial shape and protecting against osmotic forces. Structurally, peptidoglycan is composed of polysaccharide chains of alternating N-acetylglucosamine and N-acetylmuramic acid sugar residues, which are cross-linked by peptide bridges. Its synthesis involves three distinct phases.[3] The first phase, which occurs in the cytoplasm, involves sequentially adding amino acids (L-alanine, D-glutamine, L-lysine, and a dimer of D-alanine) to a uridine-di-phosphate (UDP)-linked N-acetylmuramic acid molecule. In the second phase, this sugar pentapeptide is transferred from the UDP molecule to a lipid carrier (bactoprenol), which transports it across the cytoplasmic membrane, where a further N-acetylglucosamine residue is linked to the N-acetylmuramic acid and, in staphylococci, the e-amino group of the lysine residue is substituted by pentaglycine. During the third phase, which occurs at the external surface of the cytoplasmic membrane, the resulting disaccharide-pentapeptide is linked onto an existing polysaccharide chain in a reaction termed trans glycosylation. Cross-linking of the polysaccharide chains then follows, with attachment via their peptide substituents. This step is catalyzed by multiple D-alanyl-D-alanine transpeptidases. They, along with other penicillin susceptible (but non-critical) enzymes, are referred to as penicillin-binding proteins (PBPs) and are the key targets of β -lactam action. β-Lactams owe their ability to inhibit these enzymes to a conformational resemblance to D-alanyl-D-alanine; in consequence, they irreversibly inhibit the D-alanyl-D-alanine transpeptidases by covalent acylation of an active site serine. *S. aureus* has three essential PBPs with transpeptidase activity, PBP1, PBP2 and PBP3, and all remain, unaltered, in MRSA. Rather, the resistance of MRSA to β -lactams is mediated by a supplementary peptidoglycan transpeptidase PBP, PBP2α (also known as PBP2a), which continues to function when the normal PBPs have been inactivated by β -lactams.[4] MRSA was first formally identified in the United States at Boston city hospital in 1968.[5]

Table 1: *S. Aureus* resistance: timeline for development of MRSA.[1]

History	
1941	Introduction of penicillin into treatment of infectious diseases.
1944	*S. aureus* penicillin-resistant.
1960	New penicillin-resistant drugs used to fight staph infections (i.e., Methicillin).
1968-1970	MRSA causing severe outbreaks in Europe and USA.
1968	Vancomycin introduced into MRSA therapy.
1996	*S. aureus* strain with intermediate vancomycin resistance reported in Japan.

Mode of transmission

Active MRSA infections primarily involve patients who are debilitated and therefore medically compromised. Bradleys conducted a study and noted that infections at various sites: skin 43%, urinary tract 21%, lungs 19%, blood 10%, bone, heart and others 7%.[6] More commonly, MRSA colonizes people who remain asymptomatic and become carriers of the organism. In these cases, MRSA can be cultured from their nares, perineum, rectum, and/or saliva.[7] Healthy young workers and dental personnel can become passive MRSA carriers.

This may occur transiently if they carry MRSA from one patient to another on their skin, hands, unchanged gloves, or under their fingernails.[8] One of the least common mechanisms of secondary transmission is via environmental reservoirs (counter tops, instruments, etc).

The most common route of primary transmission is by direct skin/hand/finger contact by the contaminated hands of staff and/or visitor.[6] Body fluid like saliva, blood, exposure to non-intact skin of the mucous membrane, or through the sharp or percutaneous injuries. Indirect contact by contact with contaminated equipment or surfaces in the treatment area, the dental chair including the seat and the arm rest, floor beneath the chair, sink, and towel dispenser, counter top and suction chamber remains the source of infection.[9] Unfortunately, MRSA can sometimes survive on instruments and object surfaces for two or three days and on hands for up to three hours.[10]

MRSA haw been found and isolated in 1.6-2% of tongue swabs from healthy children and 0.7% of their oral rinses, as well as 10% of oral swabs from healthy denture-wearing adults.[11] MRSA organisms are sometimes very difficult to eradicate from dentures, requiring heat sterilization, relining, or fabrication of new prostheses.[5]

Risk for MRSA infection

MRSA has a significant morbidity and mortality rate among patients who are elderly and whose health is compromised. It is much less common and seldom fatal for younger patients.[6] Fortunately, MRSA colonization is uncommon in the normal healthy host and carriage of MRSA may be a marker of increased debility.[6] It is suggested that health care workers who are found to carry MRSA organisms should limit or eliminate direct patient contact until the MRSA colonization has been resolved.[8] Medical and exposure risks for MRSA infections include:

- History of MRSA infection or colonization
- History in the past year of healthcare contact (hospitalization, long-term care facility, dialysis, surgery, permanent catheters or devices through the skin)
- Recent and/or frequent antibiotic use
- Close contact with someone known to be infected or colonized with MRSA

- Recurrent skin disease
- Injection drug use
- Crowded living conditions (e.g., homeless shelters)
- Incarceration
- Sports participants who have skin-to-skin contact, skin damage, shared clothing or equipment.

Most invasive MRSA infections are healthcare-associated which means they occur among people currently or recently in a healthcare facility including hospitals, dialysis centres, and nursing homes. These cases typically have medical risk factors for MRSA. Community-associated infections occur in previously healthy individuals without medical risk factors.[12]

Clinical features

Clinical manifestations of MRSA include abscess or invasion by lymphatic, blood, and major organs. These lesions range from a simple abrasion to a large draining abscess. Even a most common and benign abrasion can turn as a source for a huge, disseminated, and devastating MRSA infection that can be systemic in nature and may not respond to multiple antibiotics in combination.

Carbuncles, painful lesions that can cause fever, are increased in WBC counts with an ineffective drainage site because of infection due to MRSA. It serves as a reservoir for recolonization and cross-infection between different body sites, different patients, and health care associates. The most frequent symptoms associated are erythema, inflammation and swelling, pain, or burning sensation of mucosa. Persistent MRSA infection can result in a more severe form of illness called "no menstrual toxic shock syndrome" and "scalded skin syndrome". These can present with hypotension, erythema, fever, and multi-organ dysfunction. Nonmenstrual toxic shock syndrome commonly occurs in newborn and post-operative patients. These lesions usually start as a superficial pustule, rupture, and form a yellow honey to brown red crust. These lesions spread and transform into vesicles and bullae.

Multi system dysfunction includes gastrointestinal disturbances like vomiting or diarrhoea; musculoskeletal disturbances like myalgias; hyperemic mucous membranes; increased blood urea and creatinine levels in renal system with pyuria; hepatic disturbances like increased bilirubin, aspartate and alanine transferase levels; neurologic findings like changes in mental status. Improving hygiene levels and preventing postoperative cross infection helps in preventing this type of infection in children. Major organ failures are considered as a final phase in MRSA toxic shock syndrome with systemic invasion.[1]

Guidelines for the laboratory diagnosis and susceptibility testing of methicillin-resistant staphylococcus aureus (MRSA)[13]

Recommendations are given for the identification of *S. aureus* and for suitable methods of susceptibility testing and screening for MRSA and for *S. aureus* with reduced susceptibility to glycopeptides.

These guidelines indicate what tests should be used but not when the tests are applicable, as aspects of this are dealt with in guidelines on control of MRSA. There are currently several developments in screening media and molecular methods.

Table 2: Screening media and molecular methods for *S. aureus*.

Identification of *S. Aureus* includes following tests	Methicillin (oxacillin) susceptibility or sensitivity test includes	Molecular methods includes
• Tube coagulase test • Slide coagulase test • Latex agglutination tests • DNAse and heat-stable nuclease tests • Commercial biochemical tests • Molecular tests	• Dilution methods • E-test methods • Breakpoint methods • Agar screening method • Disc diffusion • Latex agglutination • Automated methods • Quenching fluorescence method	• Direct identification of MRSA in blood cultures • Identification of MRSA in endotracheal aspirates and another clinical sample

CASE REPORT

A 15 years old male patient suffering from chronic kidney disease undergoing regular dialysis reported to the out-patient department of Medicine with positive MRSA report, who also required a dental treatment. His culture report revealed he was resistant to Penicillin, Ciprofloxacin and Cephalexin. On intra oral examination did not show any significant lesions and revealed inflamed gingiva with bleeding on probing due to the

negligence of oral hygiene practice. Complete oral prophylaxis was done following strict CDC protocol which includes:[14]

Table 3: Centers for disease control and prevention protocol.

Protocols for disease control and prevention
Administrative support
Education
Judicious use of antimicrobial agents
Multidrug- resistant organism surveillance, including the detection of emergency pathogens by clinical culturing
Infection control precautions (standard control precautions) • Universal surveillance • Contact precautions • Hand hygiene • Environmental control • Cultural transformation (safety culture)
Environmental measures
Decolonization

This case report is an attempt to increase the awareness among dental professionals regarding judicial use of antibiotics, chain of infection and potential modes of transmission in the dental office with an emphasis on standard precautions for all patient encounters keeping in view the raising morbidity due to these infections.

Table 4: Microbiological investigation report.

Investigation reports	
Culture: *Staphylococcus aureus* grown after 24 hours of incubation (MRSA)	
Sensitivity test for:	
	Sensitive (S)/Resistant(R)
Co-trimoxazole	S
Tetracycline	S
Vancomycin	S
Erythromycin	S
Clindamycin	S
Linezolid	S
Cefoxitin	R
Penicillin	R
Ciprofloxacin	R

DISCUSSION

Use of antibiotics in dentistry is a common scenario for treatment and prophylactic measures.

Maximized use of these antibiotics augmented the outbreak of resistant bacterial infections caused by new strains like methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin resistant *Staphylococcus aureus* (VRSA). *S Aureus* is a part of normal oral flora in the nose, throat and oral cavity MRSA is most frequently transmitted via the transiently contaminated hands of healthcare professionals, including dental health care professionals.[15,16] Thus, direct or indirect contact with a patient or the environment are the main routes of MRSA transmission.[15,17] De Carvalho MJ et al have indicated that MRSA can be isolated from saliva and dental plaque.[18] MRSA transmission via a droplet or an airborne route in dental settings is theoretically possible, but no investigators have reported actual transmissions. However, dental healthcare professionals should be aware of the possibility of transmission through droplets or airborne measures because study done by Petti S et al have reported that the dental health care environment can be contaminated by microbial aerosol and splashes from oral cavity produced by dental devices.[19] There are no published guidelines on MRSA transmission control that are specifically targeted to clinicians in dental health care settings. Instead, the standard precautions recommended by the centers for disease control and prevention (CDC) are generally adequate for preventing the transmission of MRSA in outpatient dental clinics.[17] These include the principle that blood, body fluids, secretions, excretions (except sweat), skin that is not intact, and mucous membranes may contain transmissible infectious agents. These standard precautions consist of general practice protocols such as hand hygiene; use of personal protective equipment (PPE) appropriate handling of contaminated equipment, materials, and surfaces; safe handling of sharps; safe injection practices; and respiratory hygiene and cough etiquette.[20] Therefore, it is important for dental healthcare professionals to be aware of spread of infection and control measures as for caring for patients with uncontrolled wound drainage, dental healthcare professionals should refer to the CDC's contact precautions in addition to the standard precautions.[17] These additional measures include applying PPE when entering a patient's room, placing patients in an isolated area and limiting the patient transport thereby minimizing or avoiding the environmental contamination.[17]

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