Pathogenic Cocci and Staph Infections

DHYG 405: Microbiology and Immunology

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CASE

David, a 50 year old male construction worker, came into your dental clinic for a new patient exam. During his medical history review, David reported undergoing prosthetic knee replacement surgery two weeks ago and has contracted septic arthritis. He presents with: boils on his arm, bright red palms, bradycardia, blisters on the chin, and recently had a fever.

Before his knee replacement surgery, he was advised to take antibiotics but does not remember the name. Differential diagnoses for David's current signs and symptoms include: bacteremia, chemical burns, impetigo, Kawasaki disease, carcinoma, and rheumatic fever.

As primary healthcare providers (PHCPs), we wanted to ensure his care is comprehensive, so we contacted his general practitioner regarding his current condition. It was revealed that David is infected with Methicillin-resistant Staphylococcus Aureus (MRSA).

CASE QUESTIONS

1) What type of staph infections can David present with?

Staphylococcal bacteria live in human flora of both healthy and ill individuals. They are commonly found on the anterior nares of the nose, skin, and mucosal membranes. Staphylococcus aureus (S.aureus) can cause serious infections when they invade deeper body tissues and the bloodstream.[1] Common staph infections include: skin infections, pneumonia, toxic shock syndrome (TSS), and bacteremia (Table 1).[2]

Table 1: Different Types of Staph Infections

Skin Infections[3,4] 1. Cellulitis: redness and swelling which is caused by bacterial entrance into an opening area of the skin 2. Impetigo: highly contagious yellowish-brown crusting of the skin presented as blisters and can be bullous or non-bullous	Pneumonia[5-7] 1. Hospital-acquired pneumonia: colonization of bacteria in the oropharynx and upper airways, especially in ill hospital patients. Can be caused by inhalation of contaminated aerosols, like influenza virus 2. Ventilator-associated pneumonia: diagnosed if a patient receiving mechanical ventilation shows signs and symptoms after 48 hours 3. Healthcare-associated pneumonia: occurs in hospital or nursing facilities and is more severe as it mainly affects individuals who are already ill (ex. Chronic obstructive pulmonary disease
Toxic Shock Syndrome[8] TSS is caused by the release of toxins from S. aureus. It begins with fever and malaise, gastrointestinal symptoms, confusion, and refractory hypotension. TSS is associated with diffused erythroderma which spreads	Bacteremia[9] The presence of bacteria in the bloodstream and can occur during tissue infections or after treatments. Bacteremia can cause infections, like endocarditis and septic shock.

to extremities, like the palms and soles, and presents either as a rash, vesicles, blisters, or bullae.	

Methicillin-Resistant Staphylococcus Aureus (MRSA)

Methicillin-sensitive staphylococcus aureus (MSSA), is a type of staph infection that can be treated with various antibiotics. Methicillin-resistant staphylococcus aureus (MRSA), is a staph infection that does not respond well to antibiotics. MRSA causes significantly more deaths annually than MSSA due to elevated virulence factors.[10]

MRSA primarily affects those who are already ill or immunocompromised, and may prolong hospitalization length and increase cost, as well as mortality.[11,12] It is more difficult to treat than ordinary staph infections as strains do not respond well to common antibiotics and are difficult to eradicate. Spread of MRSA is usually through skin-to-skin contact, or contact with contaminated objects. It is commonly spread through hands in healthcare or community settings, and does not spread through the air, like the common cold or flu, unless an individual presents with MRSA pneumonia and is coughing.

MRSA is encoded by the mecA gene, located on the staphylococcal cassette chromosomes and are capable of exchanging genetic material between staphylococcal bacteria.[11,12] Resistance to beta-lactam antibiotics occurs through mutation of penicillin-binding protein 2A (PBP-2A), which is an enzyme that catalyzes the production of peptidoglycan in the bacterial cell wall.[13,14] This results in S. aureus strains that continues to grow even with the presence of antibiotics. MRSA can be community-associated and healthcare-associated, found on skin and in soft tissue infections (SSTI), like cellulitis, necrotizing fasciitis, and ulcers.[12] It also causes bone and joint infections, like septic arthritis of native or prosthetic joints, which is what David presents with.

2) **What are the intraoral and extraoral manifestations of staph and cocci infections?**

Symptoms for staph and cocci infections can occur extra- or intra-orally. Extra-orally, the patient may present with boils, impetigo, cellulitis or scalded skin syndrome.[15-19] Boils are pus-filled lesions found in a hair follicle or oil gland, often located under the arms, around the groin or on the buttocks.[15] Impetigo is a superficial, contagious infection of the skin.[16] The patient may experience pain and the lesions will resemble blisters with exudate, eventually developing a yellow crust.[15,17] Dental professionals should be aware as facial lesions usually develop around the nose and mouth, and can spread onto previously damaged skin.[16,18] Two types exist: Bullous (blisters up to 2 centimetres often on children) or non-bullous types (vesicles with erythematous base often on adults).[16] Cellulitis is a deep skin infection, causing redness, swelling, sores or ulcers (lymphangitis) and exudate.[15,19] Of particular interest is the subtype erysipelas, which affects the upper dermis and superficial lymphatics. It usually affects the lower limbs and face (cheeks, eyelids, and bridge of the nose).[19] Lesions are painful,

well-circumscribed, swollen, and distinctly superficial. Its appearance may resemble lupus erythematosus, therefore, caution should be exercised during diagnosis. Finally, scalded skin syndrome occurs as a result of toxin build-up and commonly occurs in infants or children.[15] Symptoms include fever, rash and blisters, which break and result in exposed burned-looking surface. Intra-orally, several staph bacteria are found in the healthy oral cavity.[20] S. epidermis and S.aureus are two of the common species. Some species are more common in children and while others can be harboured in plaque from fissures.[21-23] Certain oral diseases are also associated with an increased amount of staph and cocci bacteria, including: jaw cysts, oral mucosal lesions, angular cheilitis, parotitis, osteomyelitis (jaw), endodontic infections, severe mucositis with systemic disease.[21,24,25] Furthermore, colonisation of TSS toxin-1 produces strains that cause mucosal damage, leading to haematological malignancies. However, there has been no significant difference detected for S.aureus amounts between gingivitis and other periodontal diseases. MRSA itself is not associated with specific oral lesions, as the mouth is not usually of concern. However, in known carriers, suspicion is high for unusual lesions.[26] Studies have shown that it is difficult to eradicate frequent MRSA from the oropharynx.[21] Moreover, extra-oral symptoms like cellulitis and erysipelas can result in local necrosis and abscess formation.[19] Failing dental implants and prostheses may also enable bacterial colonisation.[21]

3) How are staph bacteria managed in the dental setting? (ie. waterlines, PPE, disinfection and sterilization)

Clients and dental health care practitioners (DHCP) can be exposed to pathogenic cocci on environmental surfaces and dental unit waterlines (DUWL).[13,27] Due to the presence of staphylococcal bacteria present in clinical settings, up to 80% of healthcare professionals are carriers of S. aureus.[13] Universal precautions including risk assessments, hand hygiene, personal protective equipment (PPE) for both PHCP and clients, disinfection, sterilization, and maintenance protocols prevent direct contact from person-to-person and indirect transmission with fomites.[13,27] Chemical prevention includes disinfection, sterilization, and maintenance protocols. Low level disinfection kills live and vegetative bacteria, including S. aureus, fungi, and enveloped viruses.[27]

PPE serves as a physical barrier to shield clients, DHCP and bacteria-harbouring surfaces from transmission.[28] PPE can be donned by the DHCP or client. Moreover, hand hygiene has been determined to be the most efficient and important method at preventing transmission of microorganisms.[28] Low level disinfectants for hand hygiene such as a 70-90% alcohol-based hand sanitizer or with soap and water, when hands are visibly soiled, breaks the chain of infection. In a British dental practice, it was documented that an ungloved dentist transmitted MRSA from his fingernails to two of his patients.[28] In this case, following infection prevention and control guidelines for hand hygiene and PPE reduces the spread of S. aureus.[28] S. aureus is an aerobic and anaerobic bacteria, allowing it to survive on environmental surfaces and in DUWL. S. aureus can live on non-critical items for 12 days. These surfaces may come in contact with skin and are chemically treated with an intermediate-level disinfectant.[28,29] SciCan's broad spectrum disinfectant, OPT*IM*® 33TB is an active germicidal for S. aureus, among additional pathogens.[30] Furthermore, saliva containing S. aureus can be retracted into DUWLs,

which can transmit S. aureus to DHCP and clients through output water, exposing people to uncertain microbiological quality.[28,31] DUWLs supply water to hand-pieces, ultrasonic scalers, and air/water syringes, and carry extra water, saliva, and debris through suctions.[30] Flushing DUWLs reduced levels of microorganisms in output water. However it does not affect the microorganisms present in DUWL.[32] Backflow of output water into client's mouth can be prevented by using specially designed saliva ejectors.[30] The gold standard is to use an enzymatic disinfectant according to manufacturer directions to ensure good quality DUWL output by reducing the microorganisms that reside in the tubing.[32] The Centers for Disease Control and Prevention and Infection Prevention and Control guidelines further recommend flushing hand-pieces following each appointment further reducing the bacterial load from DUWL.[30,34] Lastly, sterilization involves the processing of critical and semi-critical items, killing all forms of pathogenic microorganisms.[30] Mechanical, chemical and biological indicators should be used according to manufacturer's directions to monitor the sterilization effectiveness.[30]

4) How does one test for staph infections?

There are two methods to test for staph infections.[32] First, a physical examinations then a medical examination. The physical examination includes medical history and social determinants of health and will help to understand the cause of disease.[32] The second part is medical exam, which includes sample collection for testing.[33] Samples can be blood, mucus, or urine, which are used for bacterial wound culture. Following the culture, a susceptible testing is completed to determine which antimicrobials inhibit the microbial growth causing the infection. This is performed on the bacteria/fungi causing disease to determine the potential effectiveness of specific antibiotics.[33] In some cases, this is performed to test if the bacteria developed resistance to the antibiotic. A faster alternative is called the Rapid Gene Test detects the mecA gene or associated protein: Penicillin-Binding Protein 2a(PBP2A).[33] This test may take two to five hours to complete, although faster results can lead to earlier treatment. This option is more expensive, thus it is rarely used.[33]

5) Which immune response is activated to combat staph and cocci bacteria? What antibiotics would be recommended?

The immunity arm responsible to combat against S. aureus is both innate and acquired immunity.[34,35] Control of the immune response relies on the innate immunity as IFN-γ prevents infection by this staphylococci. Ab-17A and IL-17A mediate the protection response.[34] Furthermore, B and T cells response influence this control and may influence host susceptibility to S. aureus. The adaptive immunity determines the outcomes of chronic persistent infections.[35] Staphylococci species are difficult to fully eradicate as humans are unable to develop robust immunity against this infection. This is due to the ever-changing cell wall composition of S. aureus, which limits the production of pro-inflammatory signals to activate polarization by Th17.[36] Currently, there is no vaccine to protect against S. aureus but there are antibiotics to eradicate MRSA. The common antibiotics to treat MRSA include vancomycin and rifampicin, as listed in the table below. There are other antibiotics as well but as previously mentioned, MRSA

is resistant to penicillin, among others, as it is an antibiotic one tend to overuse. MSSA can be treated through β-lactams and non-β-lactam antibiotics such as cephalosporins and co-trimoxazole. Attempts of complete oropharyngeal eradication is difficult to achieve. Successful eradication in the throat by MRSA is done by a combination of rifampicin, fusidic acid, and mupirocin. As well, 0.2% chlorhexidine (CHX) along with nasal mupirocin, CHX body washes, and normal measures for patient isolation have been used for eradication.[37] Please refer to Table 2 and 3 for common antibiotics used for MRSA and MSSA.[38,39]

Table 2: Common Antibiotics to Treat MRSA and MSSA

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

Table 3: Common Antibiotics to Treat MRSA and MSSA

	β-lactam antibiotics	Non-β-lactam antibiotics
Methicillin-susceptible staphylococcus aureus (MSSA)	Penicillins - Flucloxacillin - Dicloxacillin	Macrolides and azalides - Erythromycin - Clarithromycin - Roxithromycin - Azithromycin
	Penicillin/β-lactamase inhibitor combinations - Amoxicillin - Ticarcillin - Piperacillin	Lincosamides - Lincomycin - Clindamycin
	Cephalosporins - Penicillinase-resistant penicillins (Flucloxacillin, Dicloxacillin) - Cefazolin	Co-trimoxazole (trimethoprim/sulphamethoxazole)
	Carbapenems	Tetracyclines
		Aminoglycosides

Methicillin-resistant staphylococcus aureus (MRSA)	Vancomycin
	Teicoplanin
	Rifampicin
	Fusidic acid
	Fluoroquinolones

CONCLUSION

Infection control and prevention is of utmost importance when dealing with conditions similar to David's case. It is the duty of a dental hygienist to remain fully up-to-date with current research on infections and which necessary precautionary steps to take to prevent spread of illnesses to especially those who have weakened immune response. In terms of treating David in the clinic, after conducting assessments and ensuring that David has not develop pneumonia-MRSA or other contraindications, treatment with the necessary precautions would proceed as usual. Moreover, David may not be aware or may not link all of his conditions together, or how all of this may affect his oral health. Educating the client on his condition would be another role the dental hygienist fulfills.. This will benefit both parties to keep information as transparent as possible to prevent misconceptions or liability. Education also provides the client the opportunity to take more proactive steps to change their lifestyle as needed. Since David works a stressful occupation, he would benefit from relaxation techniques or mindfulness meditation. As PHCPs, they can inform him on the implications of his septic arthritis and taking antibiotics. To this end, this case study explored MRSA and its implications on oral and overall health, and what roles a dental hygienist can take beyond the clinical aspect.

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