Research

Staphylococcus aureus in oral infection

Submitted to the Committee of under Graduate Studies of the College of Dentistry / University of AL-Qadisiyah in Partial Fulfillment of the Requirements for the Degree of Bachelor in Dentistry (B.D.S).

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ABSTRACT

The aim of this study was to determine the prevalence of *Staphylococcus aureus* in the oral cavity of healthy adults and the factors that may influence the presence of the bacteria. A cross-sectional study was conducted on a number of selected healthy adults in a district in Malaysia, during which, information about their socio-demographic background and oral hygiene practices was obtained. Oral rinse samples of the respondents were also collected using phosphate buffered saline and the data obtained was subsequently analyzed using SPSS.* A total of 140 oral rinse samples were collected and the results of the analysis conducted showed a prevalence of approximately 40% of the *Staph. aureus* in the oral cavity of the participants. There was no significant association observed between both the socio- demographic factor and oral hygiene practices with the presence of *Staphylococcus aureus.* The use of prostheses was found to be a significant factor for a higher prevalence of *Staphylococcus aureus* in the oral cavity .The prevalence of *Staphylococcus aureus* in the oral cavity of healthy adults was high and the use of prostheses was a factor associated with the presence of the bacteria. This accentuates the importance of a good oral hygiene, as oral cavity can be the primary route for *Staphylococcus aureus* to cause potential systemic infections.

***SPSS. / Statistical Package for the Social Sciences**

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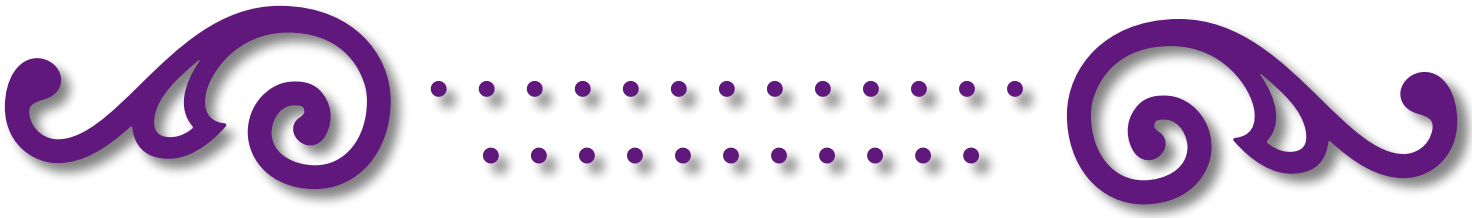
Chapter _one



1-Introduction

Staphylococcus sp. **represents one of the main groups of microorganism involved in human or animal infections. Penicillin and its derivatives, including methicillin, have been used for the treatments of infections caused by this microorganism; however, certain strains developed resistance known as Methicillin- Resistant Coagulase-Negative Staphylococci (MRS) and methicillin-resistant *S.aureus* (MRSA). This resistance to methicillin is determined by the mecA gene, which encodes the low affinity penicillin-binding protein PBP 2 [1]. The prevalence of MRSA/MRS strains in infections has increased worldwide, and an alternative treatment has been the use of na antibiotic glycopeptides such as vancomycin. However, it has emerged as another resistant profile of these microorganisms, the methicillin- resistant *S. aureus* with intermediate sensitivity to vancomycin (VISA). These bacteria may be disseminated to the community through colonized medical staff or discharged patients. The emergence and spread of MRSA in the community, independent of the healthcare setting and in the absence of typical risk factors for nosocomial MRSA infections, are matters of further concern [2]. An additional potential risk is born by emergence and spread and transferable antibiotic resistance genes coding, so far, unknown resistance mechanisms and accumulation of MRSA in animals, and transfer to humans, therefore, also has an impact on regulations of antibiotic usage in animals[3] . Dogs, cats, and horses have become an important part of most families; therefore, there are high chances of human colonization or infection with MRSA from these animals [4]. Therefore, there is a high chance of hospital colonization which may be from contact with an MRSA colonized patient or contaminated objects . Also, the areas contaminated in the hospital include medical instruments, beddings, clothing, furniture, toiletries, and the atmosphere [5] Since 2006,**

MRSA is widely disseminated among various livestock animals mainly as an asymptomatic nasal colonizer [6]. It can be introduced to hospitals and cause nosocomial infections such as postoperative surgical site infections, ventilator- associated pneumonia, septicemia, and infections after joint replacement. Because of its capacity to cause a variety of infections in humans, MRSA became a public health issue. Prevention of further dissemination of MRSA with a zoonotic potential needs concerted action of veterinary infection control specialists and clinicians[3-7] . but it is necessary to obtain information regarding the prevalence of MRSA infection before implementing strategies for infection control in veterinary medical practice. The data presented in the present study could provide some information regarding the transmission of MRSA/VISA in aveterinary hospital environment.

1.2 Shape:

S.aureus **is the most dangerous of all of the many common staphylococcal bacteria[8]. These gram-positive, sphere-shaped (coccal) bacteria (figure -1-)often cause skin infections but can cause pneumonia, heart valve infections, and bone infections.**

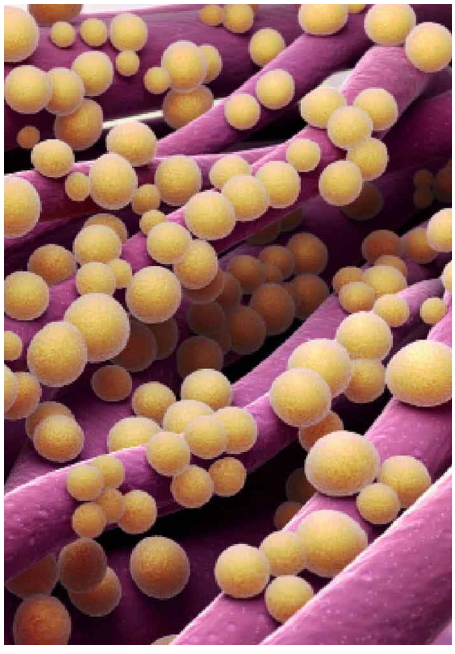
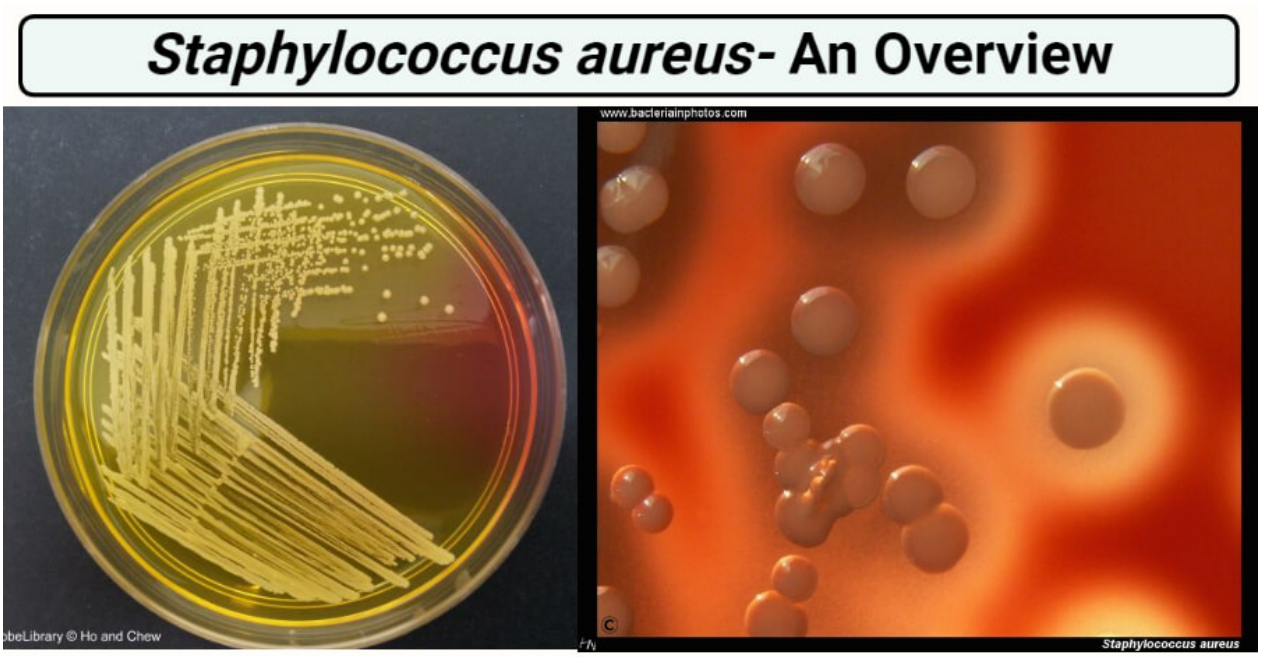


figure -1- shape of *s. aureus*

1.3 Morphology:

Microscopic morphology. S. aureus cells are Gram-positive and appear in spherical shape[9]. They are often in clusters resembling bunch of grapes when observed under light microscope after Gram staining.(figures -2-)



(figures -2-)

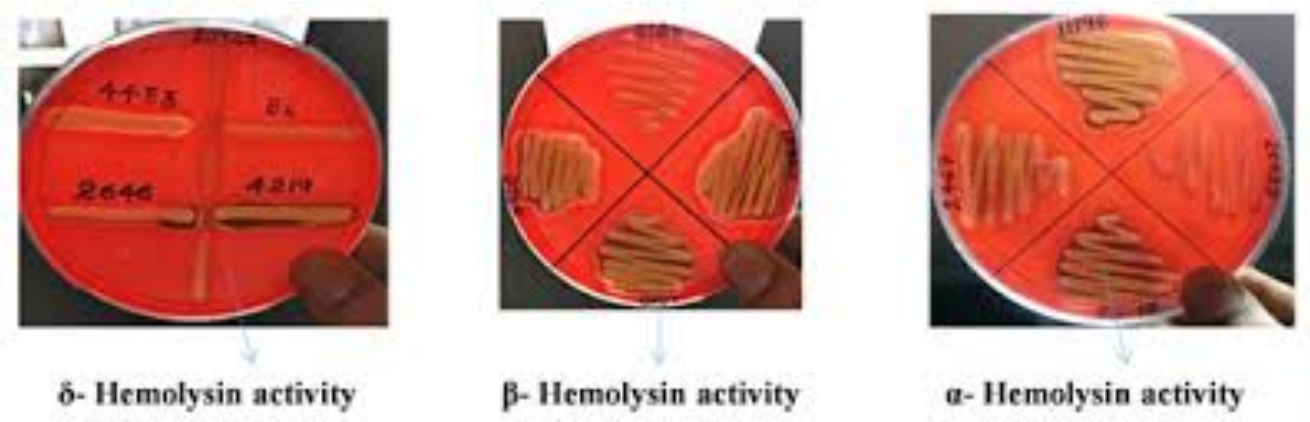
1.4 Symptoms:

Symptoms include redness, swelling, and pain at the site of infection[10].

- ***S. aureus* can also cause serious infections such as pneumonia (infection of the lungs) or bacteremia (bloodstream infection). ...**
- **If you suspect you may have an infection with S. aureus contact your health care provider.**

1.5 Hemolysis

Staphylococcus aureus **is a common pathogen causing both hospital and community-acquired infections[11]. Hemolysin is one of the important virulence factors for *S. aureus* and causes the typical β-hemolytic phenotype which is called complete hemolytic phenotype as well.(figures -3-)**



(figures -3-)A/no hemolysis, B/simi hemolysis, C/complete hemolysis

1.6 Virulence factor

1.6.1 Enzyme

S. aureus **produces various enzymes such as coagulase (bound and free coagulates) which clots plasma and coats the bacterial cell, probably to prevent phagocytosis. Hyaluronidase (also known as spreading factor) breaks down hyaluronic acid and helps in spreading it. *S. aureus* also produces deoxyribonuclease, which breaks down the DNA, lipase to digest lipids, staphylokinase to dissolve fibrin and aid in spread[12], and beta-lactamase for drug resistance.**

1.6.2 Toxins

Depending on the strain, *S. aureus* is capable of secreting several exotoxins, which can be categorized into three groups[13]. Many of these toxins are associated with specific diseases.

Superantigens

Antigens known as superantigens can induce toxic shock syndrome (TSS). [14]This group includes the toxins TSST-1, and enterotoxin type B, which causes TSS associated with tampon use. Toxic shock syndrome is characterized by fever, erythematous rash, low blood pressure, shock, multiple organ failure, and skin peeling. Lack of antibody to TSST-1 plays a part in the pathogenesis of TSS. Other strains of *S. aureus* can produce an enterotoxin that is the causative agent of a type of gastroenteritis[15]. This form of gastroenteritis is self-limiting, characterized by vomiting and diarrhea 1–6 hours after ingestion of the toxin, with recovery in 8 to 24 hours. Symptoms include nausea, vomiting, diarrhea, and major abdominal pain.

1.6.3 Exfoliative toxins

Exfoliative toxins are exotoxins implicated in the disease staphylococcal scalded skin syndrome (SSSS), [16]which occurs most commonly in infants and young children. It also may occur as epidemics in hospital nurseries. The protease activity of the exfoliative toxins causes peeling of the skin observed with SSSS.

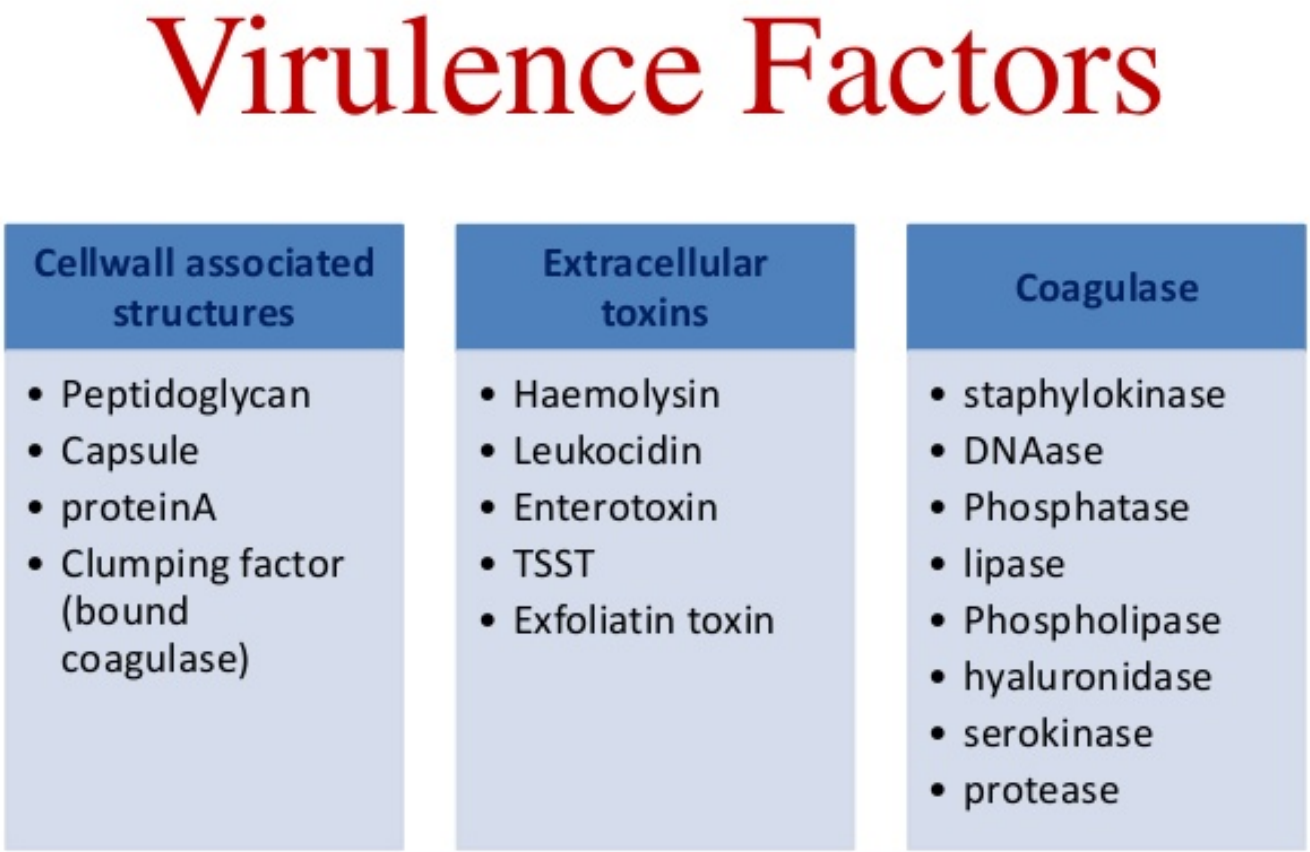
Other toxins

Staphylococcal toxins that act on cell membranes include alpha toxin, beta toxin,[17] delta toxin, and several bicomponent toxins. Strains of *S. aureus* can host phages, such as the prophage Φ-PVL that produces Panton-Valentine leukocidin (PVL), to increase virulence. The bicomponent toxin PVL is

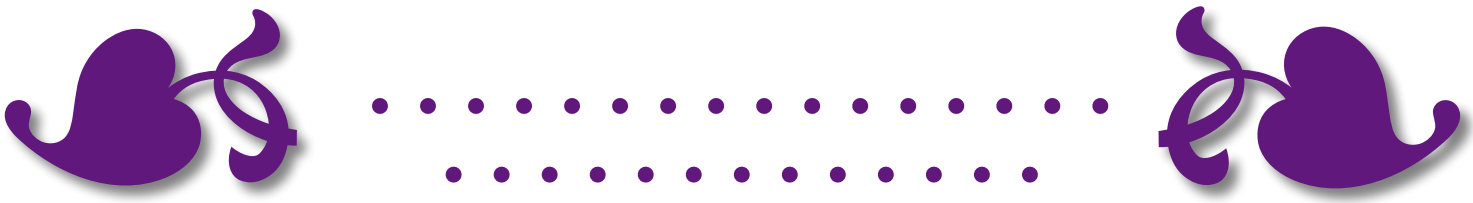
associated with severe necrotizing pneumonia in children. The genes encoding the components of PVL are encoded on a bacteriophage found in community-associated MRSA strains.

1.6.4 Small RNA

The list of small RNAs involved in the control of bacterial virulence in *S. aureus* is growing. [18]This can be facilitated by factors such as increased biofilm formation in the presence of increased levels of such small RNAs. For example, RNAIII, SprD,SprC,RsaE,SprA1,SSR42,ArtR, SprX, and tag4.



Chapter _two



2.1 Staphylococcus aureus in oral infection

Despite the extensive literature on *Staphylococcus aureus* and coagulase negative *staphylococci* ,CNS), relatively little attention has been paid to the oral cavity[19] as a reservoir for these organisms. The oral ¯ora contains F300 known species of bacteria in addition to numerous non-cultivable organisms which are being discovered as a result of molecular biological techniques. Whilst the importance of *staphylococci* as medical pathogens has been recognized for many years, the presence of *Staphylococcus* species as a component of the resident oral ¯ora is controversial but[20], surprisingly, there have been relatively few detailed studies of the distribution of *staphylococci* in the mouth. Some infections in the circum_oral region are caused, at least in part, by *S. aureus.* These include angular cheilitis, some endodontic infections, osteomyelitis of the jaw[21], parotitis [and, more recently recognized, a form of oral mucositis in elderly, highly dependent patients receiving parenteral nutrition Interestingly, there is now a growing body of evidence to suggest that *staphylococci* can be isolated frequently from the oral cavity of particular patient groups such as children ,the elderly and some groups with systemic disease such as the terminally ill, those with rheumatoid arthritis and patients with hematological malignancies. Of further concern is the observation that the oropharynx is frequently colonized with strains of methicillin-resistant *S. aureus* ,MRSA) which may prove difficult to eradicate. Therefore, it is apparent that the oral cavity may represent a hitherto poorly recognized reservoir of *staphylococci*, some of which may, under appropriate conditions, cause local or systemic infection[22]. There is also the potential for dissemination of oral *staphylococcal* strains to re-colonise other body sites or as a source of cross-infection to other patients or staff. The aim of this review is to examine the current knowledge of the ecology of *staphylococcal* species in the oral cavity and their impact on systemic health.

2.2 Causes of a staph infection in your mouth

Staphylococcus bacteria cause staph infections. [23]These bacteria commonly And her colonize the skin and nose. In fact, according to the CDC*, about 30 percentTrusted Source of people carry staph bacteria inside their nose.

Staph bacteria are also capable of colonizing the mouth. One study found that 94 percent of healthy adults carried some form of *Staphylococcus bacteria* in their mouth and 24 percent carried *S. aureus.[24]*

Another studyTrusted Source of 5,005 oral specimens from a diagnostic laboratory found that more than 1,000 of them were positive for *S. aureus.* This means the mouth could be a more significant reservoir for staph bacteria than previously believed.

.CDC /Centers for Disease Control and Prevention

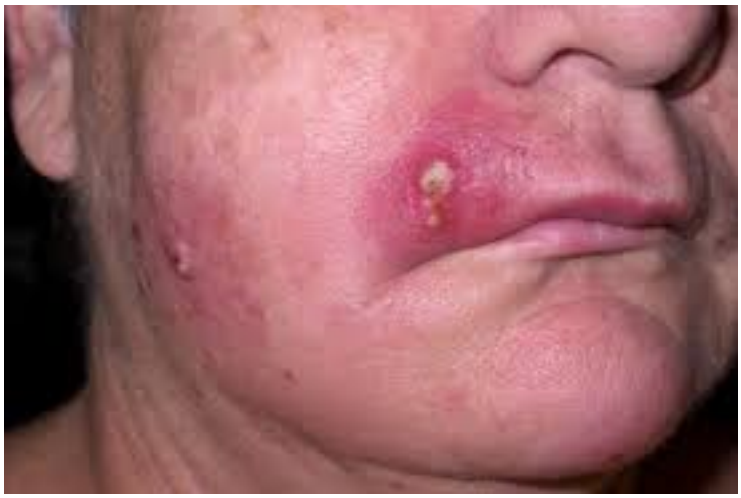
2.3 Symptoms of a staph infection in your mouth

The general symptoms of an oral staph infection can include:

1-Redness or swelling inside the mouth
2-Painful or burning sensation in the mouth
3-Inflammation at one or both corners of the mouth (angular cheilitis)

***S. aureus* bacteria have also been found in 0.7 to 15 percentTrusted Source of dental abscesses.[25]**
A dental abscess is a pocket of pus that develops around a tooth due to a bacterial infection. Symptoms can include:(figures _4_)

1-Pain, redness, and swelling around the affected tooth
2-Sensitivity to temperature or pressure
3-Fever
4-Swelling in your cheeks or face
5-Bad taste or bad smell in your mouth



(figures _4_)

2.4 Characterization of *staphylococcus aureus*

(*Staphylococcus Aureus*) is a Gram-positive, non-motile bacterium. They are so called (*staphylococci*) because they collect in the form of irregular balls that resemble a cluster of grapes when seen under a microscope.[26]

As for the golden designation, it appears in the form of yellow colonies when grown in blood agar, and can fully analyze red blood cells, and it is anaerobic optional (it can live in the presence or in the absence of oxygen). *S.aureus* usually lives naturally on human skin, in the nasal cavity or in the respiratory system. However, it can cause a range of diseases, from minor skin infections such as pimples, impetigo, boils, cellulitis, folliculitis, scalded skin syndrome and abscesses, in addition to life-threatening diseases such as pneumonia. Meningitis, osteomyelitis, and bacteremia (septicemia). [27]It is one of the most common causes of hospital acquired disease. *S. aureus* is an opportunistic pathogen responsible for many purulent infections in both humans and animals.

New antibiotic-resistant Staphylococcus aureus species have recently emerged, most notably methicillin-resistant Staphylococcus aureus.[28]

2.5.1 Culture

Aerobes and facultative anaerobes

Opt. Temp. For growth= 37°C

Opt. pH for growth= 7.5

On Nutrient agar

[29]golden yellow and opaque colonies with smooth glistening surface, 1-2 mm in diameter (figures _5_)

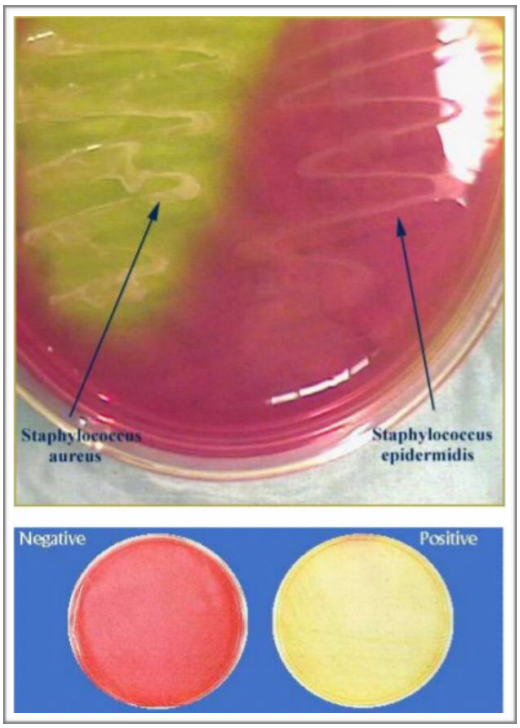


FIGURE 5/CULTURES COLONY

2.5.2 culture (....contd)

On Mannitol salt agar

S.aureus **ferments mannitol and appear as yellow colonies - MSA is a useful selective medium for recovering *S.aureus* from faecal specimens,[30] when investigating food poisoning**

(Figures _6_)

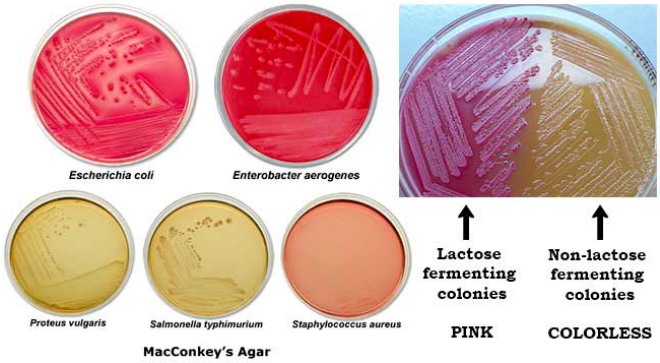


FIGURE 6/S.AUREUS IN MANNITOL AGAR

2.5.3 On Blood agar

golden yellow colonies, [31] surrounded by a clear zone of hemolysis (beta- hemolysis), esp. (figures 7)When incubated in sheep or rabbit blood agar in atmosphere of 20% CO2

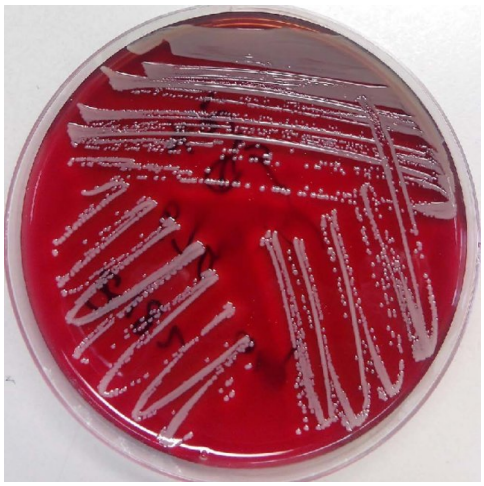


FIGURE 7/S.AUREUS ON BLOOD AGAR

- ### 2.5.4 On MacConkey agar

smaller colonies than those on NA(0.1-0.5 mm) [32]and are pink colored due to lactose fermentation(figures 8)

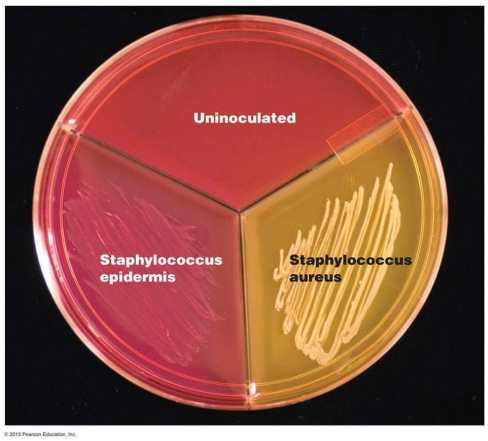


FIGURE 8/S.AUREUS IN MACCONKEY AGAR

2.6 Complications of a staph infection in your mouth

Although many staph infections can be easily treated, sometimes serious complications can occur.

2.6.1 Bacteremia

In some cases, *staph bacteria* can spread from the site of infection into the bloodstream. [33]This can lead to a serious condition called bacteremia. (figures 9)

Symptoms of bacteremia can include fever and low blood pressure. Untreated bacteremia can develop into septic shock

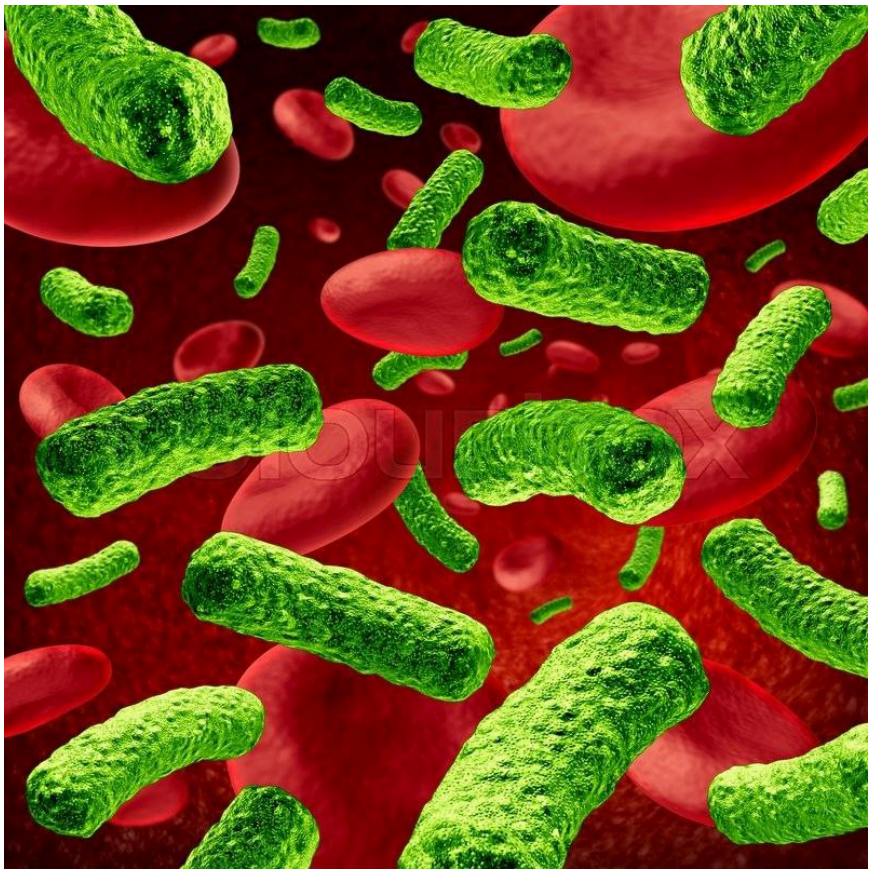


FIGURE 9 / BACTEREMIA

2.6.2 Toxic shock syndrome

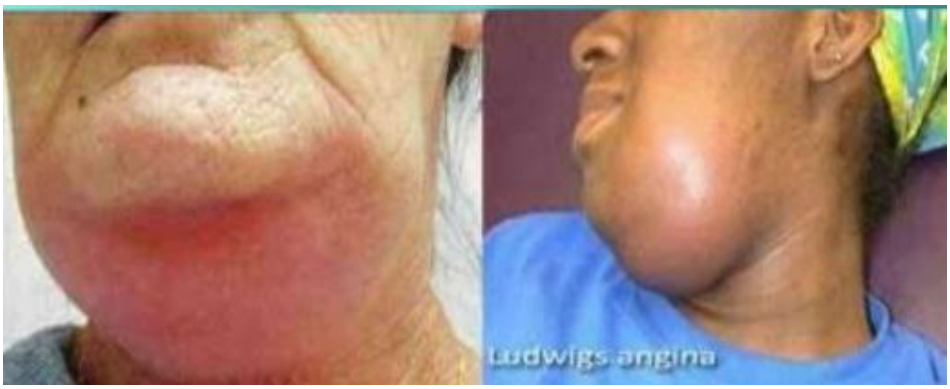
Another rare complication is toxic shock syndrome. It's caused by toxins produced by *staph bacteria* that have entered the blood.[34] Symptoms can include:

1-High fever
2-Nausea or vomiting
3-Diarrhea
4-Aches and pains
5-Rash that looks like a sunburn
6-Abdominal pain

2.6.3 Ludwig's angina

Ludwig's angina is a severe infection of the tissues of the bottom of the mouth and neck. [35]It can be a complication of dental infections or abscesses. Symptoms can include:

1-Pain in the affected area
2-Swelling of the tongue, jaw, or neck.(figures 10)
3-Difficulty with swallowing or breathing
4-Fever
5-Weakness or fatigue



(figures 10)shows swollen in neck

2.7 Is a staph infection in the mouth contagious?

The bacteria that cause a staph infection are contagious. That means that they can be spread from person to person.[36]

Someone with *staph bacteria* colonizing their mouth may spread it to other people by coughing or talking. Additionally, you may get it by coming into contact with a contaminated object or surface and touching your face or mouth.

Even if you're colonized with staph, it doesn't mean you'll get sick. *Staph bacteria* are opportunistic and often only cause infections under specific circumstances,[37] such as the presence of an open wound or an underlying health condition.

2.8 Risk factors for a staph infection in the mouth

Most people colonized with staph don't get sick. Staph is opportunistic. It typically takes advantage of a specific situation to cause infection.

You may be more likely to get an oral staph infection if you have[38]:

1-An open wound in your mouth
2-Had a recent oral procedure or surgery
3-Recently stayed in a hospital or other healthcare facility
4-An underlying health condition like cancer or diabetes
5-A compromised immune system
6-A medical device inserted, such as a breathing tube

2.9 Treating a staph infection in your mouth

Many staph infections respond well to antibiotic treatment. [39]If you're prescribed oral antibiotics (figures 11), be sure to take them as directed and finish the entire course to prevent a recurrence of your infection.

Some types of staph are resistant to many types of antibiotics. In these cases, you may need stronger antibiotics, some of which may need to be given via IV.

A doctor may perform antibiotic susceptibility testing [40]on a sample from your infection. This can help to better inform them on which types of antibiotics may be most effective.

In some cases, treatment with antibiotics may not be necessary. For example, if you have an abscess, the doctor may choose to make an incision and drain it.

FIGURES 11

2.10 Preventing staph infections

There are a few ways that you can help to prevent getting a staph infection in your mouth:

1-Keep your hands clean. Wash your hands frequently with soap and warm water. If this isn't available, use an alcohol-based hand sanitizer.
2-Practice good oral hygiene. [41]Taking care of your teeth and gums through brushing and flossing can help prevent things like dental abscesses.
3-Visit a dentist for regular teeth cleanings.
4-Don't share personal items like toothbrushes and eating utensils

2.11 Diagnosis

Diagnosis is based on performing tests with colonies. [42]Tests for clumping factor, coagulase, hemolysins and thermostable deoxyribonuclease are routinely used to identify *S aureus*. Commercial latex agglutination tests are available. Identification of *S epidermidis* is confirmed by commercial biotyping kits.

To diagnose a staph infection, your doctor will:

- **Perform a physical exam. During the exam, your doctor will closely [43] examine any skin lesions you may have.**
- **Collect a sample for testing. Most often, doctors diagnose staph infections by checking a tissue sample or nasal secretions for signs of the bacteria.**
- **Other tests. If you're diagnosed with a staph infection, your doctor may order an imaging test called an echocardiogram to check if the infection has affected your heart. Your doctor may order other imaging tests,**

- **depending on your symptoms and the exam results.**

2.12 Identification of Staphylococci in the Clinical laboratory Structure

Staphylococci **are Gram-positive cocci about 0.5 – 1.0 µm in diameter. They grow in clusters, pairs and occasionally in short chains. [44]The clusters arise because** ***staphylococci*** **divide in two planes. The configuration of the cocci helps to distinguish micrococci and** ***staphylococci*** **from** ***streptococci*****, which usually grow in chains. Observations must be made on cultures grown in broth, because** ***streptococci*** **grown on solid medium may appear as clumps(figures 12). Several fields should be examined before deciding whether clumps or chains are present.**

FIGURES 12/ DIFFERENT BETWEEN STAPHYLOCOCCUS AND STREPTOCOCCUS IN SOLIDS MEDIUM GROWTH

2.12.1 Catalase Test

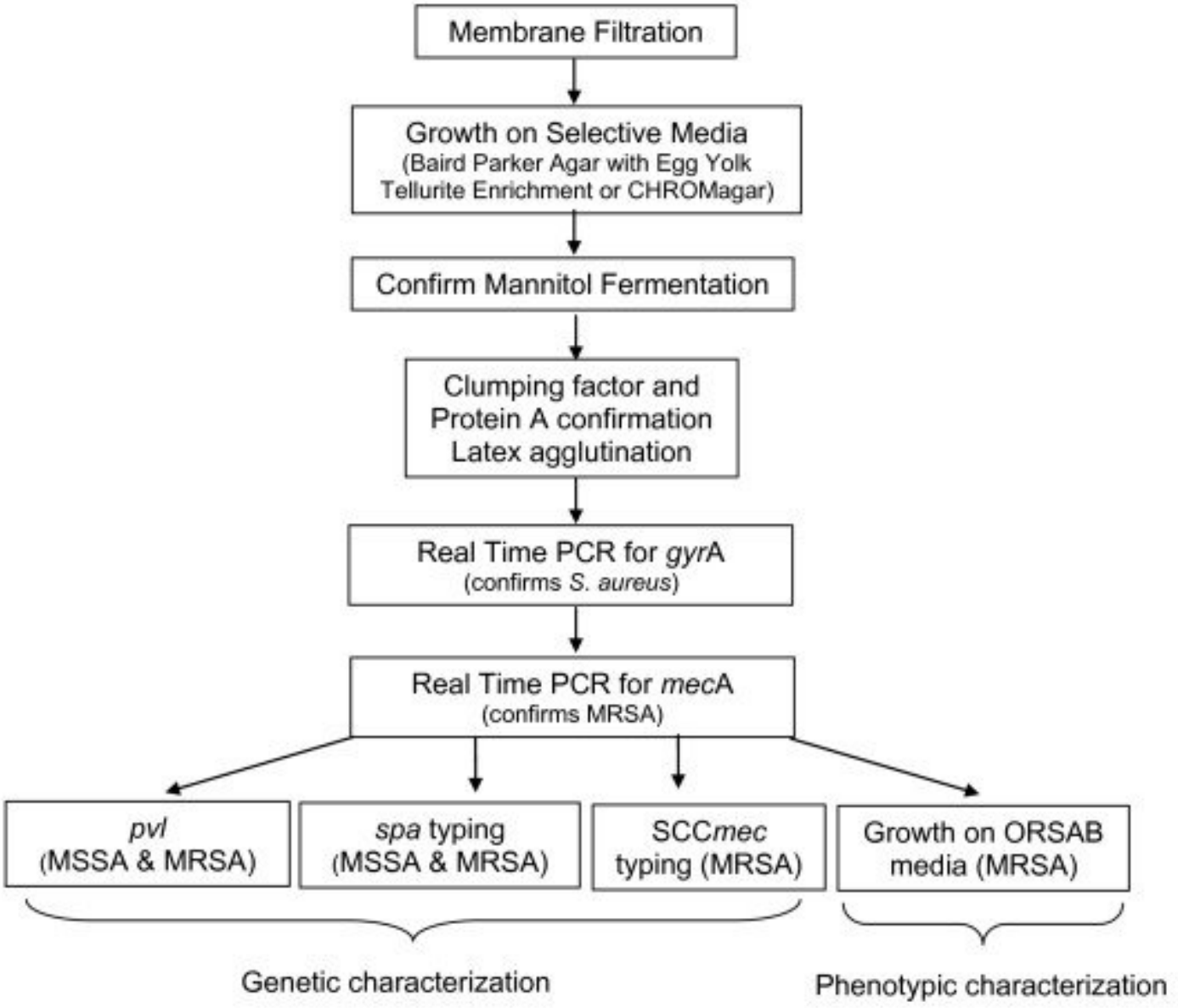
The catalase test is important in distinguishing streptococci (catalase-negative) staphylococci which are catalase positive(figures 13). The test is performed by flooding an agar slant or broth culture with several drops of 3% hydrogen peroxide. Catalase-positive cultures bubble at once. [45]The test should not be done on blood agar because blood itself will produce bubbles.

FIGURES 13/ S.AUREUS .CATALYSE TEST

2.12.2 Isolation and Identification

The presence of *staphylococci* in a lesion might first be suspected after examination of a direct Gram stain. [46]However, small numbers of bacteria in blood preclude microscopic examination and require culturing first.

The organism is isolated by streaking material from the clinical specimen (or from a blood culture) onto solid media such as blood agar, tryptic soy agar or heart infusion agar. Specimens likely to be contaminated with other



Figures 14 / Process Flow of Bacterial isolation and identification for S. aureus and MRSA.

microorganisms can be plated on mannitol salt agar containing 7.5% sodium chloride, which allows the halo-tolerant staphylococci to grow. Ideally a Gram stain of the colony should be performed and tests made for catalase and coagulase production, allowing the coagulase-positive *S aureus* to be identified quickly. Another very useful test for *S aureus* is the production of thermostable deoxyribonuclease. *S aureus* can be confirmed by testing colonies for

agglutination with latex particles coated with immunoglobulin G and fibrinogen which bind protein A and the clumping factor, respectively, on the bacterial cell surface. These are available from commercial suppliers [47](e.g., Staphaurex). The most recent latex test (Pastaurex) incorporates monoclonal antibodies to serotype 5 and 8 capsular polysaccharide in order to reduce the number of false negatives. (Some recent clinical isolates of *S aureus* lack production of coagulase and/or clumping factor, which can make identification difficult.)

The association of *S epidermidis* (and to a lesser extent of other coagulase-negative staphylococci) with nosocomial infections associated with indwelling devices means that isolation of these bacteria from blood is likely to be important and not due to chance contamination, particularly if successive blood cultures are positive. Nowadays, [48]identification of *S epidermidis* and other species of *Staphylococcus* is performed using commercial biotype identification kits, such as API Staph Ident, API Staph-Trac, Vitek GPI Card and Microscan Pos Combo. These comprise preformed strips containing test substrates.

(figures 14)

2.12.3 Confirmation of diagnosis

To confirm a diagnosis, the sample from the patient is placed onto a culture media. This could be a liquid or gel that provides sources of nutrition, carbon, energy and nitrogen for the bacteria to grow. For S. aureus, the medium used is suffused with blood and lactose. Also commonly used is the mannitol salt agar, which is a selective medium with 7–9% salt or sodium chloride that allows *S. aureus* to grow selectively. These media are placed on petri dishes and swabbed with the sample. [49]The dishes are then incubated overnight at 37 degrees Celsius. After a set period of time the typical golden colonies of *S. aureus* are seen. These are then stained with Gram stain for confirmation and also undergo specific characteristic tests like the catalase test or the coagulase test for diagnosis.

Rapid diagnostic tests

These help in detection of the bacteria in real-time. These techniques include Real-time PCR and Quantitative PCR and are increasingly being employed in clinical laboratories.

2.12.4 Identification of the bacteria

A small portion of the sample is swabbed onto a glass slide. This is then stained with Gram stain or dyes like crystal violet and basic fuschin and viewed under the microscope. *S. aureus* is Gram positive and stains blue or purple and appears as small round cocci or short chains and most commonly as grape-like clusters. [50]Since *S. aureus* may be normally present on skin and mucous membranes, this test is not always confirmatory

2.13 Treatment of staph.aureus

Treatment depend on which microorganism cause the infection ..if its bacteria cause the disease treatment with antibiotic(AB)[51]

(Ab) is medication that kill the bacteria and end the infection

Main antibiotic are

1-penicillin such as flucloxacillin and amoxicillin

2-cephalosporins such as cefalexin

3-tetracycine such doxycycline

4-cindamycin

2.13.1 Most common drugs used to treat staph.infection is

1- cefazoline..cephalexin... amoxicillin .

2-penicillin .

3-macrolides such as tetracycline and erythromycine and aminoglycosides

4-cephalosporin are most commonly used

5-vancomycine used commonly given IV

6-rifampicin sometimes used

DRUG (ORAL)	USUAL DOSE	ADJUSTED DOSE
Amoxicillin/clavulanate	875mg q12h	250mg 500mg q24h. On dialysis days, dose AD.
Azithromycin	500mg x1, then 250mg qd x4days	No adjustment
Cefdinir	300mg q12h	300mg qod. On dialysis days, give 300mg AD.
Cephalexin	500mg q8-12h	250mg q12-24h. On dialysis days, dose AD.
Ciprofloxacin	500mg q12h	250 q12h or 250-500mg q24h. Dose AD on dialysis days.
Clindamycin	150-300mg q8h-q12h	No adjustment
Dicloxacillin	150-450mg q6h	No adjustment
Levofloxacin	500mg q24h	500mg x1, then 250mg q48h. On dialysis days, dose AD.
Linezolid	600mg q12h	No adjustment
Minocycline	100mg q12h	No adjustment
Moxifloxacin	400mg q24h	No adjustment
TMP/SMX	1DS q12h	Avoid if possible. If unavoidable, give one DS q24h. On dialysis days, dose AD.

FIGURE 15 /ANTIBIOTIC CHOICE OF TREATMENT S.AUREUS INFECTION

Sometimes emergence of antibiotic resistent strains of *staph.bacteria* these types described as methicillin resistant *staph .aureus* (MRSA)

-MRSA resistant to penicillin because of production of enzyme called beta lactamase or pencillinase (figure 16)

In this case use antibiotic has stronger effect *than pencillin such as flucloxacillin*

-Staph aureus take 10_20 days to heal if left untreatment

-Sometimes staph aureus treat with surgical drainage of pus from infected site and no needs for antibiotics[52]

-when staph infection in the skin that normally shaving ..should stop the shaving until the infection clear up ..if they do have to shave the area .should use clean disposable razor .

-keep the area cover whenever possible to prevent spread the infection .don't touch the infected skin .

Antibiotic	MRSA (n = 10)	MSSA (n = 36)
Penicillin	100	97.2
Erythromycin	90.0	41.7
Clindamycin*	100	38.9
Tetracycline	90.0	41.7
SMX-TMP	60.0	13.9
Moxifloxacin	90.0	30.6
Vancomycin	0.0	0.0
Fusidic acid	10.0	5.6

*including inducible clindamycin resistance.
SMX-TMP: sulphamethoxazole-trimethoprim; MRSA: methicillin resistant Staphylococcus aureus; MSSA: methicillin susceptible Staphylococcus aureus.
doi:10.1371/journal.pone.0059775.t003

FIGURE 16/ ANTIBIOTIC RESISTANCE PATTERNS OF 46 S. AUREUS FROM BLOOD,

2.14 International research and studies

. Materials and Methods

2.14.1. Bacterial Strains and Phenotypic Identification

Samples were collected at the Veterinary Hospital of UFG (Jataí-GO) as follows: swabs of desks, surgical tables, hospitalization cages, stethoscopes, thermometers, and any instruments used in direct contact with animals. Swabs were wetted in Butterfield's solution containing 0.1% of Tween 20. [52]The collections were performed 9 times in periods not less than 30 days between each collection. The collected samples were cultured in nutrient broth, blood agar, and mannitol agar at 35°C for 24–48 hs. For the identification of *S. aureus*, the following tests were used: the Gram stain, catalase test, furazolidone and bacitracin sensitivity, coagulase test, DNAse test, mannitol fermentation, mannose fermentation, raffinose, sucrose, trehalose and xylose, maltose fermentation, and the VP test. Then, the pure cultures were stored with freezed glycerin at −80°C for further analysis.

2.14.2 Determination of the Antimicrobial Sensitivity of the Isolates

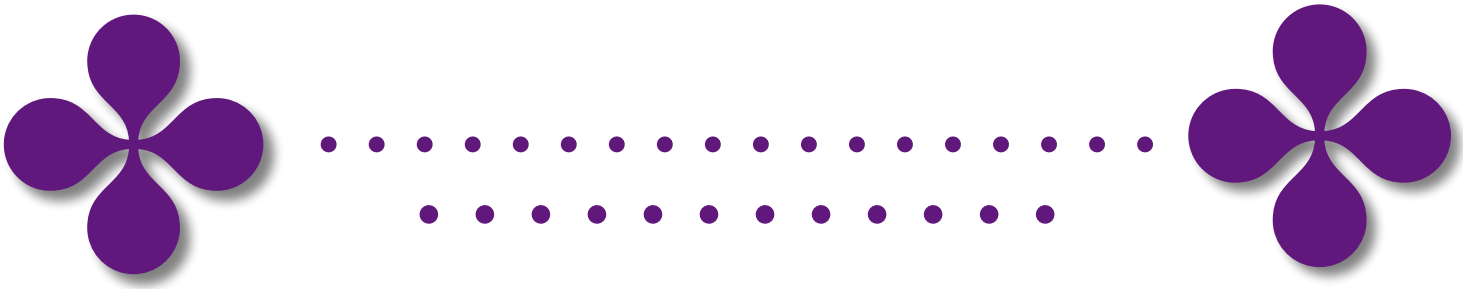
The methicillin-resistant *S. aureus* (MRSA) was investigated, and the method used was described by CLSI.*[53] The minimal inhibitory concentration (MIC) test for vancomycin, clindamycin, and gentamicin antimicrobials was performed using the concentration of 0.016– 256 µg/mL present in the strips (Liofilchem), whereas for ciprofloxacin, the concentration was 0.002–32 µg/mL and for sulfamethoxazole, the concentration was 0.064–1024 µg/mL. Control strains included *S. aureus* ATCC 29213.

CLSI*/The Clinical and Laboratory Standards Institute

2.14.3 mecA-PCR

Bacterial DNA was obtained as follows: the strains were cultured in BHI broth for 12 hours at 35°C and, then, centrifuged at 5000 rpm for 4 min. The supernatant was discarded, and the pellet washed 3 times with 200 µl of TE buffer. Subsequently, the pellet was resuspended in 100 µl of TE buffer, and the micro tubes were heated at 95°C in a water bath for 10 min and, then, centrifuged at 5000 rpm for 20 sec. The supernatant (100 µL) was transferred to a microtube, frozen at −20°C, and stocked. The detection of the mecA gene by the polymerase chain reaction (PCR) was performed according Gortel et al. [54] with modifications established by Neves et al.q[55] The primers used for the detection of a 533 bp fragment were 5′-AAA ATC GAT GGT AAA GGT TGG C 3′ and 5′ AGT TCT GCA GTA CCG GAT TTG C 3′. 2 µL of the DNA diluted previously (30 ng) ; 2.5 µl of 1x PCR buffer (50 mM KCl, 200 mM TRIS-HCl, pH 8.4); 2.5 U of Taq DNA polymerase; 0.2 mM dNTP; 1.5 mM MgCl2, 1 µg of each primer, and sterile milli water were used until the reaction volume was 20 µL. The samples were placed in a thermocycler with the following cycle: 2 min at 94°C; 1 min at 94°C; 2 min at 52°C; 2 min at 72°C; 39 cycles from step 2; 5 min at 72°C, and maintenance of the samples under refrigeration at 5°C. All products after the amplification process were analyzed on agarose gel with 1.5% ethidium bromide and processed at 65 V for 1 h 30 min.

Chapter _three



3.1 Discussion

Both in human and veterinary medicine, there is a concern with MRSA, which has been gradually increasing due to the difficulty of combating them, being a worldwide public health problem where most of the infections occur through some lineages with pathogenic potential. MRSA are becoming increasingly frequent in nosocomial infections, both in the human environment and in the veterinary environment. In addition, reports of animals and humans serving as reservoirs and transporting bacteria to both environments [56] have been reported. In our study, we found a prevalence of 1.6% for MRS and 0.64% for MRSA, and it is observed that depending on the study site and sample size, the global rates are extremely variable (0.03% to 80%) . Professionals who work in veterinary hospitals and, therefore, have frequent contact with animals should be trained on the risks of transmitting MRS and MRSA in the environment. In this work, two methicillin-resistant strains(15-MRSA and 24- MRS) were found in the surgical materials, and this evidences the importance of the correct autoclaving of this material because the possibility of transmission to animals (at a vulnerable time, such as surgery) is real and with disastrous consequences. In these clinics and hospitals, hygienic measures such as hand washing before and after contact with contaminated surfaces and avoiding close contact with the discharges from the nose, mouth, and wounds of infected humans and animals should be adopted. Our data demonstrated that MRSA and MRS strains may be circulating in the veterinary hospital setting, evidencing the significant contamination capacity of this microorganism, as well as its persistence in the environment. Knowing that the isolates of samples collected and found were cages, muzzles,

non autoclaved materials, desks, and stethoscopes; therefore, there is concern regarding nosocomial infections that have a connection with MRS and MRSA, since they are direct contact materials and between animals and the professionals who work there. In addition, the spatial distribution of MRSA may indicate interspecies transmission and colonization of difference populations. Although hospital cleaning can reduce MRSA/MRS contamination in the environment, in some cases, it does not eliminate it. Veterinarians should be encouraged to choose antibiotics for therapy according to antibiogram tests, as well as to wear masks and gloves when handling patients.In the case of vancomycin, which was first released in the 1950s, resistance was not reported until the mid 1990s, with the description of vancomycin intermediate-resistant *S.aureus* (VISA)[57]. Vancomycin is an antibiotic used for the treatment of Gram-positive bacterial infections. Traditionally, it has been used as a drug of last resort; however, clinical isolates of MRSA strains with decreased susceptibility to vancomycin, VISA, and more recently, with high- level vancomycin resistance (vancomycin- resistant *S. aureus* [VRSA]) have been described in the clinical literature [58]. In this study, an isolate (strain 18) was identified as methicillin-resistant S. aureus with intermediate sensitivity to vancomycin (VISA). An MRSA isolate with decreased susceptibility to vancomycin was first reported in Japan in 1997[59]. As in our study, the isolated strain in Japan had only a modestly increased minimum inhibitory concentration (MIC) value for vancomycin, in the range of 3– 8 µg/ml. However, there have been reports of VISA strains with a vancomycin MIC value greater than 100 µg/ml in the United States. Therefore, in the case of these strains producing an infection, the use of this antibiotic, the last available option, is therapeutically impossible. VISA isolates do not carry imported foreign genetic elements; rather, the increased vancomycin MIC values are related to mutations that appear in the invading pathogen during vancomycin therapy in vivo. VISA

began to be reported with increasing frequency among MRSA isolates identified all over the world[60].Resistance to the other antibiotics tested was detected mainly in strains 5, 10, and 14, which were resistant to antibiotics of different classes, being classified as SDR*. Resistance to drugs of different classes is worrying because it shows that bacteria are constantly passing through the process of genetic recombination and, consequently, they are acquiring endogenous genes, which, in the future, would allow the appearance of multi-drug- resistant, extensively-resistant, or even pan drug- resistant strains. High-level resistance to methicillin is caused by the mecA gene, which encodes an alternative penicillin-binding protein, PBP 2a [61] . Interestingly, of the four strains carrying the mecA gene, two (5 and 7) did not phenotypically express this resistance, and this may be due to differences in the regulation of gene expression in these cells or to a lower sensitivity of the test. Additionally, strain 5, which carries the mecA gene, has been shown to be resistant to clindamycin and gentamicin, so we have here a potentially multidrug-resistant strain because in the case of expression of this gene, this bacterium will be resistant to three different classes of antibiotics[62]. In addition, the wide distribution of microorganisms through the environment facilitates the possibility of colonization and/or infection of a wide variety of hosts (humans and animals), and this allows the contact of strains of different genetic profiles, an essential factor for genetic recombination between them. In this sense, these bacteria can acquire genetic material related to mechanisms of resistance to antibiotics, which may be carried by pathogenic or nonpathogenic strains.The clinical use of antimicrobials plays a role in the selection of resistant strains and is probably the main cause of resistance , especially in veterinary hospital where there is a higher concentration of antibiotic residues in the environment, as well as in the animal organism. As more bacterial strains become resistant to an increasing number of antibiotics, therapeutic options become

SDR*/ serine asparate repeat protein

increasingly limited and expensive and in some cases nonexistent. Effective interventions in the hospital serine aspirate repeat environment are, therefore, necessary to minimize the problem of microbial resistance, the control of antimicrobial use and the control and prevention of hospital infections being the main interventions that can be performed in this sense. However, the resistance of the bacterial species to antimicrobials is extremely variable among countries and, in this sense, it is necessary that actions to control this situation and the definition of microorganisms to be monitored are planned based on global and local epidemiological information.

For hospital infection, the hands and nostrils of colonized individuals are the major sources ofMRSA transmission. MRSA is released into the hospital environment either through aerosol, skin cells, or stools of an infected patient . Gehanno et al. found a similar strain of MRSA in patients of a hospital and the room atmosphere. Although hospital cleaning reduces MRSA contamination of the environment, in some cases, it does not eliminate it. The emergence of resistant microorganisms to the various classes of antimicrobials has been progressive in the last decades, constituting a threat to the world public health. The circulation of these strains (MRSA, MRS, and VISA) in the veterinary hospital environment is worrisome because the World Health Organization (WHO) classifies this profile as a high priority according to its list of pathogens according to the severity of the infections they cause and the number of antibiotics available for treatment. Our observations suggest the need for containment measures (good antisepsis practices) to avoid the possible transmission of resistant bacterial agents in the veterinary hospital environment.

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مقدم إلى لجنة الدراسات

العليا في كلية طب الأسنان / جامعة القادسية في

استيفاء جزئي لمتطلبات درجة البكالوريوس في طب

الأسنان (B.D.S).

إعداد :

نور فاضل

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نور ضياء

نور سلمان

إشراف // دكتوره رقيه حسين

ملخص

تعتبر(*S.aureus*)اختيارية لا هوائية ، وعادة ما تعيش بشكل طبيعي على جلد الإنسان أو في تجويف الأنف أو في الجهاز التنفسي ، وعادة ما تستعمر هذه البكتيريا الجلد والأنف

البكتيريا العنقودية قادرة أيضًا على استعمار الفم. ومع ذلك ، يمكن أن يسبب مجموعة من الأمراض ، من الالتهابات الجلدية البسيطة مثل(pimples, impetigo, boils, cellulitis, folliculitis, scalded skin syndrome and abscesses)، بالإضافة إلى الأمراض التي تهدد الحياة مثل الالتهاب الرئوي. التهاب السحايا والتهاب العظم والنقي وتجرثم الدم (تسمم الدم). إنه أحد الأسباب الأكثر شيوعًا للأمراض المكتسبة من المستشفيات

يعتمد تشخيص هذا النوع من البكتيريا على إجراء اختبارات على المستعمرات. تُستخدم اختبارات عامل التكتل ، والتجلط الدموي ، والهيموليزينات ، ونووكلياز الديوكسي ريبون المستقر حرارياً لتحديد(*S.aureus*). تتوفر اختبارات تراص اللاتكس التجارية. يتم تأكيد التعرف على *s.epidermidis* تجارياعن طريق مجموعات التنميط الحيوي .commercial biotyping kits.

ويعتمد علاج العدوى التي يسببها هذا النوع من البكتيريا على الكائنات الحية الدقيقة التي تسبب العدوى .. وإذا كانت البكتيريا المسببة للمرض فإن العلاج بالمضاد الحيوي (AB) هو الدواء الذي يقتل البكتيريا.

للوقاية من عدوى المكورات العنقودية ، هناك عدة طرق يمكنك من خلالها المساعدة في منع الإصابة بعدوى المكورات العنقودية في فمك:

حافظ على نظافة يديك. اغسل يديك باستمرار بالصابون والماء الدافئ. إذا لم يكن هذا متاحًا ، فاستخدم معقم اليدين المعتمد على الكحول. تدرب على نظافة الفم الجيدة. يمكن أن يساعد الاعتناء بأسنانك ولثتك من خلال التنظيف بالفرشاة والخيط في منع أشياء مثل خراجات الأسنان. قم بزيارة طبيب الأسنان لتنظيف الأسنان بانتظام. لا تشارك الأغراض الشخصية مثل فرش الأسنان وأدوات الأكل