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Original Research

The Role of Staphylococcus Aureus in Food Poisoning

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Abstract:

Staph. aureus is an opportunistic pathogen. It is the most Common in skin and soft tissue. This microbe can cause more diseases, for Example: burn inflammation and tonsillitis through the production of virulence factors that are acquired by some plamidic virulence genes.

Staphylococcus aureus (S. aureus) is a Gram-positive bacterium that is carried by about one third of the general population and is responsible for common and serious diseases. These diseases include food poisoning and toxic shock syndrome, which are caused by exotoxins produced by S. aureus. Of the more than 20 Staphylococcal enterotoxins, SEA and SEB are the best characterized and are also regarded as superantigens because of their ability to bind to class II MHC molecules on antigen presenting cells and stimulate large populations of T cells that share variable regions on the chain of the T cell receptor. The result of this massive T cell activation is a cytokine bolus leading to an acute toxic shock. These proteins are highly resistant to denaturation, which allows them to remain intact in contaminated food and trigger disease outbreaks. A recognized problem is the emergence of multi-drug resistant strains of S. aureus and these are a concern in the clinical setting as they are common cause of antibiotic-associated diarrhea in hospitalized patient

Keywords: Poisoning, Staphylococcus aureus, Food

Introduction:

Staphylococcus aureus (S. aureus) is a gram-positive bacterium and produces a wide variety of toxins that cause various diseases, which range from mild skin infections to systemic, life-threatening diseases [1]. The virulence factors produced by S. aureus include secreted proteins (e.g., superantigens (Sags), exfoliative toxins (ETs) and cytotoxins), enzymes (e.g., proteases, staphylokinase and coagulase), and surface proteins (e.g., protein A, fibronec- tins, collagen adhesion and clumping factors) . In this review toxins are defined as secreted proteins that directly or indirectly harm the host and cause diseases. Toxins secreted by S. aureus can be divided into three categories [2], clinically important two exotoxins and cell membrane-damaging cytotoxins; (i) Sags that interfere with receptor function, (ii) ETs that destroy epidermal barrier function, and (iii) cell membrane-damaging cytotoxins that act in a receptor-dependent or receptor-independent fashion. These toxins mainly modulate host immune response by interfering with the receptor functions of immune cells or facilitate tissue penetration by destroying intercellular junctions or host cell membranes [3]. Many virulence factors, including exotoxins, are expressed under the control of the accessory gene regulator-quorum sensing (agr-QS) system, which is closely associated with the pathogenesis and biofilm formation by S. aureus and with antibiotic resistance S. aureus rapidly acquires antibiotic resistance, and the emergence of multidrug-resistant strains is an enormous burden. It has been reported the annual death [4, 5] toll due to antibiotic-resistant infections has reached 10 million, and that in 2050 it will exceed the number of deaths attributed to cancer. Thus, the appropriate uses of antibiotics and alternative therapeutic approaches have become necessities [6, 7].

The study aimed to discuss food poisoning with same bacteria in the references through the following:

1-research epidemiology of S. aureus.

2-explain Mechanism of infection in these bacteria.

3- Investigating food poisoning caused by the same bacteria.

4- Investigating ways to avoid infection with these bacteria and avoid poisoning caused by them.

General pathogenesis and clinical diseases:

1. Pathogenesis

The process of S. aureus infections involves five stages. They are: colonization, local infection, systemic dissemination and/or sepsis, metastatic infections and toxinosis. The organism is in carrier state in the anterior nares and can remain so without causing infec- tions for weeks or months. The colonization proceeds to infection under certain predisposing factors such as prolonged hospitalization, immune suppression, surgeries, use of invasive medical devices and chronic metabolic diseases. Localized skin abscess develops when the organism is inoculated into the skin from a site of carriage. This can further spread and results In various clinical manifestations of localized infections such as carbuncle, cellulitis, impe- tigo bullosa or wound infection [8, 9]. The organism can enter into blood and spread systemically to different organs causing sepsis. This haematogenous spread may result in endocarditis, osteomyelitis, renal carbuncle, septic arthritis and epidural abscess. Without a blood stream infection, specific syndromes can occur due to extra cellular toxins of Staph. aureus. These are toxic shock syndrome, scalded skin syndrome and foot borne gastroenteritis.

2. Hospital and community infections

Staph. aureus causes wide range of infections in human. The clinical infections of S. aureus are classified into community and nosocomial categories based on origin of infecti on. These two types are distinct in clinical manifestations of the infections, antibiotic susceptibility and the genetic background of the infecting S. aureus strains. For decades, S. aureus has been predomi-nately a nosocomial pathogen and is a leading cause of mortality and morbidity in hospitals. However, the community S. aureus infections are in rise [10, 11]. The important

clinical S. aureus infec-tions are bacteraemia, infective endocarditis, skin and soft tissue infections, osteoarticular infections and pleuropulmonary infections. Other clinical infections are epidural abscess, meningitis, toxic shock syndrome and urinary tract infections.

3. Virulence factors

Staph. aureus possess battery of virulence factors. These factors enable the organism to be successful as pathogen that causes wide range of human and animal infections. Virulence factors help in attachment to host cells, breaking down the host immune shield, tissue invasion, causing sepsis and elicit toxin-mediated syndromes. This is the basis for persistent staphylococcal infections without strong host immune response [12] .

Epidemiology of infections:

1. Nasal carriage

S. aureus is a commensal and opportunistic pathogen. The anterior nares are the principal ecological niche, where the organism colonizes in humans. The nasal carriage of Staph. aureus increases the risk of infection especially in the hospital settings [13]. The average nasal car- riage of S. aureus could be at 30% of human population. Since, the nasal carriage increases the risk of development of surgical site, lower respiratory and blood stream infections in hospitals, efforts are made to eliminate the carriage using various strategies. Methods such as local application of antibiotics (eg. Mupirocin) or disinfectants, administration of systemic antibiotics and use of a harmless S. aureus strain (type 502A) which competes for the coloniza-tion of nares with existing one are employed to decolonize the S. aureus from nares.

2. Emergence and evolution of MRSA

The MRSA are those S. aureus strains carrying a mecA gene, which codes for additional peni-cillin-binding protein, PBP2a. The beta-lactam antibiotics exert their antibacterial activity by inactivation of penicillin-binding proteins (PBPs), which are essential enzymes for bacterial cell wall synthesis. However, these antibiotics have only a low affinity towards PBP2a, thus this enzyme evades [14, 15] from inactivation and carry out the role of essential PBPs resulting in cell wall synthesis and survival of bacteria even in presence of beta-lactam antibiotics. Due to the presence of mecA, MRSA are resistant to nearly all beta-lactam antibiotics.

3. Health care-associated MRSA (HA-MRSA)

Health care-associated MRSA (HA-MRSA) are those S. aureus isolates obtained from patients 2 or more days after hospitalization or with the MRSA risk factors (history of recent hospi-Talization, surgery, dialysis, or residence in a long-term care facility within 1 year before the MRSA-culture date or presence of a permanent indwelling catheter or percutaneous medical device (e.g. tracheostomy tube, gastrostomy tube or Foley catheter) at the time of culture or previous isolation of MRSA. Community-associated MRSA (CA-MRSA) are those S. aureus isolates obtained from patients within 2 days of hospitalization and without the MRSA risk factors [16, 17].

Risk factor:

People who are predisposed to staphylococcal infections include

- Neonates and breastfeeding mothers
- Patients with influenza, chronic bronchopulmonary disorders (eg, cystic fibrosis, emphysema), leukemia, tumors, chronic skin disorders, or diabetes mellitus
- Patients with a transplant, an implanted prosthesis, other foreign bodies, or an indwelling intravascular plastic catheter
- Patients receiving adrenal steroids, irradiation, immunosuppressants, or antitumor chemotherapy
- Injection drug users
- Patients who have chronic kidney disease and are being treated with dialysis
- Patients with surgical incisions, open wounds, or burns

Predisposed patients may acquire antibiotic-resistant staphylococci from other patients, health care personnel, or inanimate objects in health care settings. Transmission via the hands of personnel is the most common means of spread, but airborne spread can also occur.

Diseases caused by Staph. aureus

Staphylococci cause disease by

- Direct tissue invasion
- Sometimes exotoxin production

Direct tissue invasion is the most common mechanism for staphylococcal disease, including the following:

- Skin infections
- Pneumonia
- Endocarditis
- Osteomyelitis
- Infectious (septic) arthritis

Multiple exotoxins are sometimes produced by staphylococci. Some have local effects; others trigger cytokine release from certain T cells, causing serious systemic effects (eg, skin lesions, shock, organ failure, death). Panton-Valentine leukocidin (PVL) is a toxin produced by strains infected with a certain bacteriophage. PVL is typically present in strains of community-associated methicillin-resistant S. aureus (CA-MRSA) and has been thought [18] to mediate the ability to necrotize; however, this effect has not been verified.

Toxin-mediated staphylococcal diseases include the following:

- Toxic shock syndrome
- Staphylococcal scalded skin syndrome
- Staphylococcal food poisoning

1. Staphylococcal bacteremia:

Staph. aureus bacteremia, which frequently causes metastatic foci of infection, may occur with any localized S. aureus infection but is particularly common with infection related to intravascular catheters or other foreign bodies. It may also occur without any obvious primary site. S. epidermidis and other coagulase-negative staphylococci increasingly cause hospital-acquired bacteremia associated with intravascular catheters and other foreign bodies because they can form biofilms on these materials. Staphylococcal [19] bacteremia is an important cause of morbidity (especially prolongation of hospitalization) and mortality in debilitated patients.

2. Staphylococcal skin infections:

Skin infections are the most common form of staphylococcal disease. Superficial infections may be diffuse, with vesicular pustules and crusting (impetigo) or sometimes cellulitis, or focal with nodular [20] abscesses (furuncles and carbuncles). Deeper cutaneous abscesses are common. Severe necrotizing skin infections may occur.

Some Staphylococcal Skin Infections
Impetigo

In impetigo, clusters of vesicopustular or bullous lesions form, rupture, and develop a honey-colored crust.

Nonbullous Impetigo (Infant)

This photo shows clusters of vesicles and pustules with developing honey-colored crust on the nose.

Furuncle

Furuncles (boils) are tender nodules or pustules that involve a hair follicle and are caused by staphylococcal infection.

Staphylococcin wound and burn [21] infections, postoperative incision infections, and mastitis or breast abscess in breastfeeding mothers.

Staphylococcal neonatal infections

Neonatal infections usually appear within 6 weeks after birth and include

- Skin lesions with or without exfoliation
- Bacteremia
- Meningitis
- Pneumonia

Staphylococcal pneumonia

Pneumonia that occurs in a community setting is not common but may develop in patients who

- Have influenza
- Are receiving corticosteroids or immunosuppressants
- Have chronic bronchopulmonary or other high-risk diseases

Staphylococcal pneumonia may be a primary infection or result from hematogenous spread of S. aureus infection elsewhere in the body (eg, IV catheter infection, endocarditis, soft-tissue infection) or from injection drug use [22]. However, S. aureus is a common cause of hospital-acquired pneumonia, including ventilator-acquired pneumonia.

Staphylococcal pneumonia is occasionally characterized by formation of lung abscesses followed by rapid development of pneumatoceles and empyema. CA-MRSA often causes severe necrotizing pneumonia.

Staphylococcal endocarditis

Endocarditis can develop, particularly in IV drug abusers and patients with prosthetic heart valves. Because intravascular catheter use and implantation of cardiac devices have increased, S. aureus has become a leading cause of bacterial endocarditis.

S. aureus endocarditis is an acute febrile illness often accompanied by visceral abscesses, embolic phenomena, pericarditis [23] , subungual petechiae, subconjunctival hemorrhage, purpuric lesions, heart murmurs, perivalvular abscess, conduction defects, and heart failure secondary to cardiac valve damage.

Staphylococcal osteomyelitis

Osteomyelitis occurs more commonly in children, causing chills, fever, and pain over the involved bone. Subsequently, the overlying soft tissue becomes red and swollen. Articular infection may occur; it frequently results in effusion, suggesting septic arthritis rather than osteomyelitis. Most infections of the vertebrae and intervertebral disks in adults involve S. aureus.

Staphylococcal infectious arthritis

Joints typically become infected via hematogenous infection, but infection can also be caused by extension of a bone infection, trauma, or direct infection during joint surgery. Prosthetic joints are particularly prone to infection. Staphylococcal infection of a prosthetic joint in the months after implantation is usually acquired during surgery, whereas infections occurring more than 12 months after surgery are likely due to hematogenous spread [24] . However, infections still may be secondary to organisms that were inadvertently introduced at the time of implantation and remained dormant and then became clinically evident several months later.

Staphylococcal toxic shock syndrome:

Staphylococcal toxic shock syndrome may result from use of vaginal tampons or complicate any type of S. aureus infection (eg, postoperative wound infection, infection of a burn, skin infection). Although most cases have been due to methicillin-susceptible S. aureus (MSSA), cases due to MRSA are becoming more frequent.

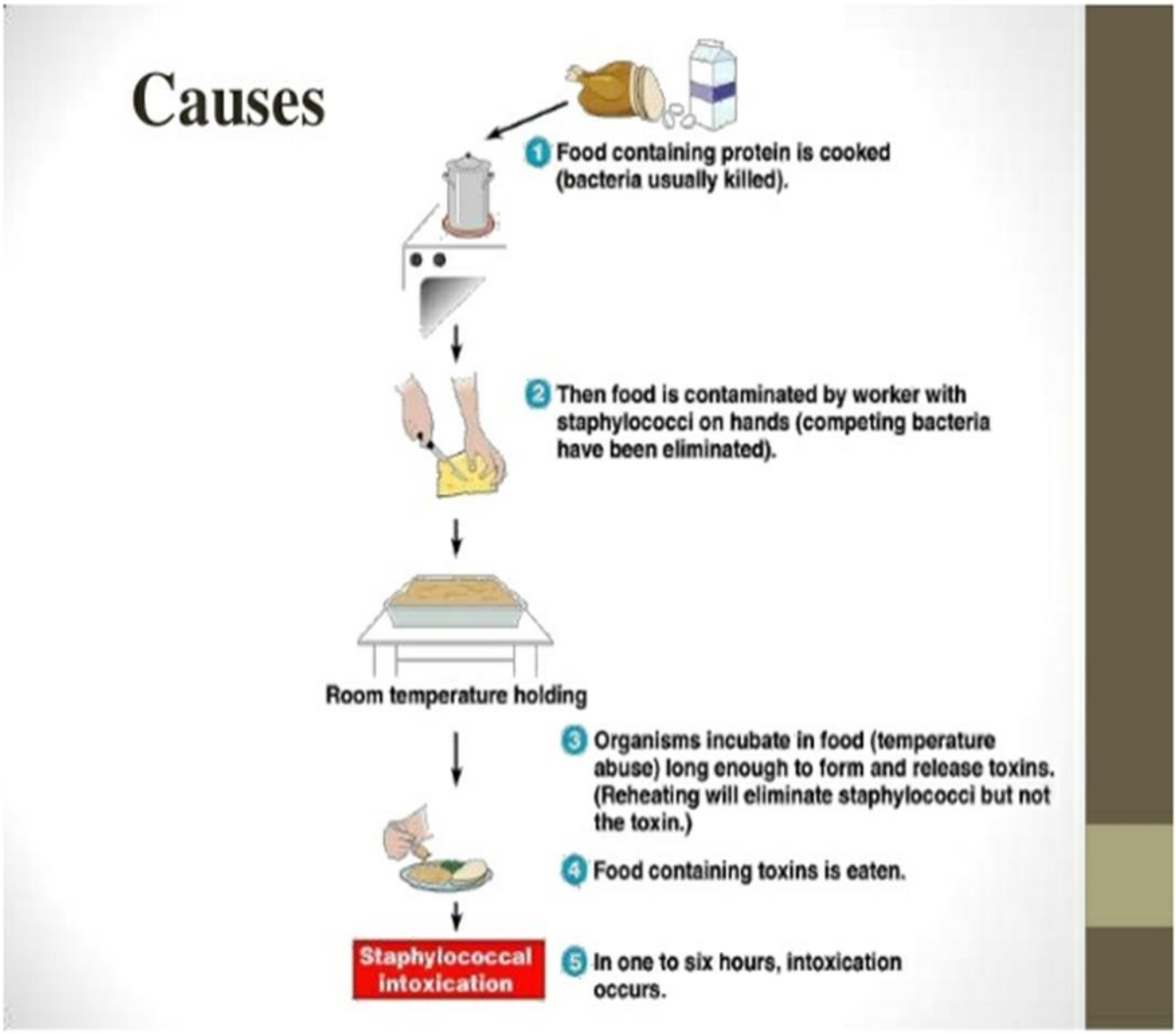
Staphylococcal scalded skin syndrome

Staphylococcal Scalded Skin Syndrome (Infant)

Staphylococcal scalded skin syndrome, which is caused by several toxins termed exfoliatins, is an exfoliative dermatitis of childhood characterized by large bullae and peeling of the upper layer of skin. Eventually, exfoliation occurs. Scalded skin syndrome most commonly occurs in infants and children < 5 years.

Staphylococcal food poisoning

Staphylococcal food poisoning is caused by ingesting a preformed heat-stable staphylococcal enterotoxin. Food can be contaminated by staphylococcal carriers or people with active skin infections. In food that is incompletely cooked or left at room temperature, staphylococci [24] reproduce and elaborate enterotoxin. Many foods can serve as growth media, and despite contamination, they have a normal taste and odor. Severe nausea and vomiting begin 2 to 8 hours after ingestion, typically followed by abdominal cramps and diarrhea. The attack is brief, often lasting < 12 hours



Food poisoning caused by Staphylococcus aureus:

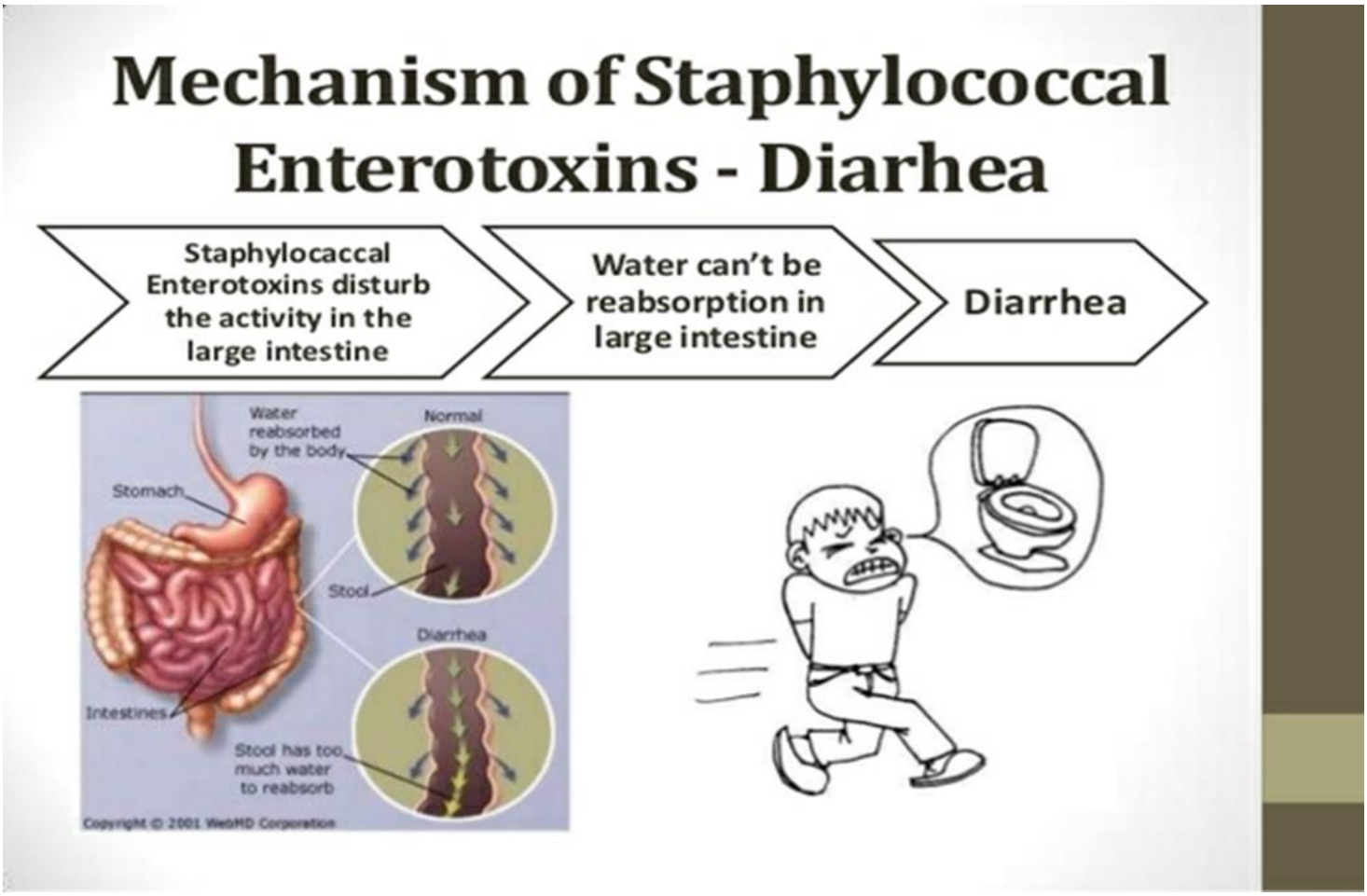
Staph food poisoning is a gastrointestinal illness caused by eating foods contaminated with toxins produced by the bacterium Staphylococcus aureus (Staph) bacteria.

About 25% of people and animals have Staph on their skin and in their nose. It usually does not cause illness in healthy people, but Staph has the ability to make toxins that can cause food poisoning [25] . People who carry Staph can contaminate food if they don't wash their hands before touching it. If food is contaminated with Staph, the bacteria can multiply in the food and produce toxins that can make people ill. Staph bacteria are killed by cooking, but the toxins are not destroyed and will still be able to cause illness.

Symptoms of Staph food poisoning:

Staph food poisoning is characterized by a sudden start of nausea, vomiting, and stomach cramps. Most people also have diarrhea.

Symptoms usually develop within 30 minutes to 8 hours after eating or drinking an item containing Staph toxin, and last no longer than 1 day. Severe illness is rare. The illness cannot be passed from one person to another.



Diagnosis of Staphylococcus aureus food poisoning:

In most cases, SFP does not require medical attention. It often clears up with rest and fluids. But contact your doctor if your illness lasts longer than three days, or if you are unable to drink enough fluids to prevent dehydration.Your doctor can diagnose SFP with a physical examination and a review of your symptoms. They may also ask questions about recent activities and things you have eaten [26, 27] . If symptoms are severe, your doctor may order blood tests or a stool culture.These tests can help

determine if the S. aureus bacterium is present, and may also help your doctor rule out other potential causes.

Prevention:

To prevent food poisoning and the spread of bacteria, take the following precautions:

- Avoid unpasteurized milk
- Wash hands and fingernails thoroughly before cooking, eating, or serving food
- Maintain clean and sanitary surfaces for food preparation
- Store hot foods at temperatures over 140˚F (60˚C) and cold foods under 40˚F (4˚C)
- Do not prepare food for others if you have wounds or sores on your hands or wrists

Foods that require a lot of handling and are stored at room temperature are often involved with SPF. These include:

- Sandwiches
- Puddings
- Cold salads, such as tuna, chicken, macaroni, or ham salad
- Sliced deli meats
- Cream-filled pastries

Staph food poisoning treated:

The most important treatment is drinking plenty of fluids. Your healthcare provider may give you medicine to decrease vomiting and nausea. People with severe illness may require intravenous fluids. Antibiotics are not useful in treating this illness because the toxin is not affected by antibiotics.

Conclusion:

Staph. aureus produces toxins that cause a wide variety of diseases in humans, for example, SEs cause SFP, and TSST-1 causes TSS, ETs cause SSSS and cytotoxins such as hemolysins, leukotoxins, and PSMs cause cell lysis. The Agr-QS system controls the expressions of virulence factors and biofilm formation, and in order to eliminate antibiotic resistance caused by biofilm formation, alternative therapeutic drugs are urgently needed. Anti-virulence agents can inhibit the activities of virulence factors and pathways that mediate virulence. Recently, anti-virulence therapy has attracted considerable interest, and natural products with anti-toxin properties or that target the agr-QS system have been shown to inhibit S. aureus virulence and control staphylococcal diseases without affecting bacterial growth or viability. For this reason, natural product-based anti- virulence therapies offer the possibility of blocking the progression of staphylococcal diseases without developing antibiotic resistance. Although the molecular mechanisms responsible for the activities of these natural compounds have yet to be determined, these compounds provide starting points for the development of novel anti-virulence agents that target S. aureus.

Recommendations:

- Attempt to isolate your Staphylococcus aureus teriasis from different sources (environmental, food) especially cases of food poisoning with Staphylococcus aureus and a comparison of factors the virulence possessed by virulence factors of bacteria isolated from healthy carriers.
- should perform studies about exogenous and endogenous staph aureus in patients with burn, tonsillitis and other clinical source.
- studies must be done to make continuous surveillance of β-Lactamases-producing strains.
- Study other plasmidic genes in burn, tonsillitis and others patients.
- Studying the resistance of bacteria isolated from the nose, skin and ear to antibiotics, especially Vancomycin and methicillin.

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