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# ***Staphylococcus aureus***

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## **General information**

- ***Staphylococcus aureus*, Gram-positive**, potentially pathogenic bacteria found in nasal membranes, skin, hair follicles, and perineum of warm-blooded animals. They may cause a wide range of infections and intoxications.

- The word *Staphylococcus* is derived from Greek "grapes". Latin word *aureus* ("gold") refers to color of the *Staphylococcus* colonies growing on agar.
- *Staphylococcus aureus* has remarkable ability to develop resistance to antibiotics: for example, within 5 years after Alexander Fleming's discovery of penicillin and its wide distribution as antimicrobial agent, 50% of all *S. aureus* strains were resistant to it.
- **Methicillin-Resistant *Staphylococcus aureus* (MRSA)** is strain of *Staphylococcus aureus* that is non-susceptible to the action of **methicillin**. The mechanism of resistance usually involves modification of normal or the presence of acquired **penicillin binding proteins**. MRSA strains emerged as a noticeable adversary within a decade of the first introduction of the methicillin as an antibiotic in 1959-1960.
- Approximately 20% of populations have persistent asymptomatic nasal colonization by *S. aureus* and 30% acquire it intermittently.
- Number of MRSA infection-associated deaths in United States is about 19,000 annually which is similar to number of deaths due to AIDS, tuberculosis, and viral hepatitis combined.
- Staphylococcal bacterial chromosome easily incorporates various mobile genetic elements, such as bacteriophages, plasmids, and so-called "pathogenicity islands". The incorporated elements encoding virulence factors can transform commensal microorganism into a pathogenic one. Resistance to methicillin and other beta-lactam antibiotics is caused by the *mecA* gene, which is situated on a mobile genetic element, known as, the **Staphylococcal Cassette Chromosome *mec* (SCC*mec*)**.
- Two basic groups of virulence factors play role in *S. aureus* infections: (1) proteins expressed

on the surface of the bacterial cell (exponential growth phase) and (2) secreted proteins (stationary phase). The following common phases of the infection are usually described: attachment, colonization, invasion or penetration, and evasion of host immunity.

## Types of MRSA infections

- **Hospital-acquired MRSA (HA-MRSA)**. There are five major clones of *S aureus* that are responsible for most cases of HA-MRSA internationally: the Iberian, Brazilian, Hungarian, New York/Japan, and Pediatric clones. Different nomenclatures exist.
- **Community-acquired MRSA (CA-MRSA)**. MRSA infection considered CA-MRSA when it occurred in individual who was not exposed to hospital settings for a while. Outbreaks of the CA-MRSA in communities with limited health care started receiving attention since 1989-1990. Majority of fatal cases involved skin and soft tissue infections and necrotizing pneumonia.  
CA-MRSA strains usually produce **Panton-Valentine leukocidin**, which is usually absent in HA-MRSA strains.  
**USA400** (or MW2) and **USA300** are among most virulent and best studied strains of CA-MRSA.

## Virulence factors

- Presence of numerous **microbial surface components recognizing adhesive matrix molecules (MSCRAMM proteins)** that mediate adherence to host tissues.
- Ability to form **biofilms** (slime) on host tissues and surfaces of implants. Bacteria inside the

biofilms are relatively protected from host defenses and antimicrobial agents by a hydrated matrix of polysaccharides and proteins. It is very difficult, if not impossible, to non-invasively eradicate the biofilm-formed pathogens from infected tissues and prosthetic surfaces.

- Production of **leukocidins**, pore forming proteins, that destroy leukocytes by lysis of the cytoplasmic granules.
- Ability to produce **superantigens** - microbial antigens that induce over-activation of T-cells, their anergy and death. The superantigens cause non-specific systemic immune response that weakens the host facilitating subversion of its defenses whereas normal immune response involves development of specific antibodies which inactivate the pathogen and protect the organism.
- **Agr protein (accessory gene regulator)**, a quorum-sensing system, plays a critical role in the regulation of *S. aureus* virulence. It is responsible for orchestrating the pathogen's invasion strategies (adherence, subversion of host immune responses, formation of biofilm, intracellular invasion, etc) on different stages of infections. The quorum-sensing system is a unique mechanism of modulation of bacterial gene expression in response to increased cell density.
- In conclusion, the *S. aureus* uses so many powerful and complex strategies to invade the host that it is not possible to enumerate them all in short review. Moreover, most probably, some of them are still to be discovered.

## Pathologies

- **Furunculosis**

- **Abscess**
- **Staphylococcal Scalded Skin Syndrome (SSSS)**

## Possible severe complications

- Necrotizing **pneumonia**
- **Empyema** - presence of pus in a hollow organ or body cavity
- **Waterhouse-Friderichsen Syndrome** - overwhelming bacterial infection, leading to hemorrhage and necrosis of the adrenal gland
- **Necrotizing fasciitis** - a serious fulminating infection causing extensive necrosis of superficial fascia (connective tissue)
- **Pyomyositis** - suppurative inflammation of muscle tissue
- **Toxic Shock Syndrome (TSS)**. TSS is usually classified into two categories: (1) menstrual TSS (originally described as associated with tampon use) and (2) non-menstrual TSS (usually, hospital-acquired).
- **Purpura Fulminans** - large, rapidly spreading skin hemorrhages, fever, or shock
- **Bacteremia** - presence of viable bacteria circulating in the blood (mostly HA-MRSA)

## Risk groups and epidemiology of CA-MRSA

- Severe infections occur more frequently in the south (in United States as well in Europe).
- **Prostheses and implants** increase chances of HA-MRSA infections.
- Influenza or pneumonia positively correlate

with cases of CA-MRSA pneumonia.

- Injuries in certain groups such as military personnel, prison inmates, athletes, farmers etc. (where immediate medical help can be delayed) are placing them at higher risk of acquiring the MRSA.
- Other groups with increased risk of infections are veterinarians, pet owners, children younger than 2 years, people older than 65 years, people with concurrent infection and people who are close to an infected individual.

## Treatment

- Antibiotics. There is a number of antibiotics that can be administered to out- and in-patients, such as doxycycline, trimethoprim-sulfamethoxazole, vancomycin, linezolid, and several other. All of them have severe side effects and are not universally effective. Each patient requires unique approach because of differences in age, body mass, pre-existing health conditions, and individual tolerance.
- Incision and drainage of subcutaneous infections, such as abscesses and furuncles, followed by application of antibiotics are very important.

## Prevention

- Diminishing the pathogen's capacity to adhere to the host's tissues. A low-pH cream and a gluco-oligosaccharide were reported to inhibit the attachment of *S. aureus* cells on the epithelial surfaces.
- Vaccination. Despite of many years of research in this direction, and development and testing of a number of patented or trademarked

preparations, there are no commercially available vaccines for human infection and the single available vaccine for the prevention of bovine mastitis has inconsistent results.

- Screening of hospitalized patients for MRSA carriage.
- Limiting exposure to antibiotics to occasions where it is absolutely necessary. Prescribed antibiotics should be used to kill an on-going infection, not merely suppress it. It is important that patients use the prescribed dose fully without stopping or interruptions.
- Frequent hand washing.
- Constant decontamination of inanimate surfaces in department stores, public transportation, public toilets, schools, gyms, and other highly populated areas.

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