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Staphylococcus aureus: Review of literature in brief

Dr.Parul Parvesh Verma[1], Dr. Anisha Yadav[2], Dr. Ashu Gautam[3], Samim Ali[4]
Senior Resident, MD Microbiology, Department of Microbiology, Kalpana Chawla Government Medical College, Karnal
PG resident, Department of Microbiology, Kalpana Chawla Government Medical College, Karnal
PG resident, Department of Microbiology, Kalpana Chawla Government Medical College, Karnal
Research Assistant, Department of Microbiology, Kalpana Chawla Government Medical College, Karnal

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ABSTRACT
Staphylococcus aureus (S. aureus) is both a commensal bacterium and a major human pathogen causing a wide range of clinical infections. S. aureus has been identified as a risk factor for the development of various infections. It causes blood stream infections (BSIs), skin and soft tissue infections (SSTIs), osteomyelitis, endocarditis and nosocomial infections and is the major cause of community-acquired infections. This review comprehensively covers the discovery, taxonomy, virulence factors and pathogenesis. The mortality of S. aureus bacteremia remains approximately 20 to 40% despite the availability of effective antimicrobials. Introduction of penicillin in the early 1940s dramatically improved the prognosis of patients with Staphylococcal infection. However, as early as 1942, penicillin resistant Staphylococci were recognized, first in the hospitals and subsequently in the community. This pattern of resistance first emerging in hospitals and then spreading to the community, is now a well-established pattern that recurs with each new wave of antimicrobial resistance. Therefore, accurate and early detection of S.aureus is mandatory for effective management of infections caused by it.

KEYWORDS: S.aureus **,** bloodstream infections, antibiotic resistance, skin and soft tissue infections

STAPHYLOCOCCUS AUREUS: REVIEW OF LITERATURE IN BRIEF

Infections have been one of the major causes of morbidity and mortality worldwide among the human population. All the microorganisms including bacteria, viruses, parasites and fungi cause a variety of infections affecting every organ of the body. The immune system is an effective barrier against these infectious agents.

Staphylococcus aureus (S. aureus) is both a commensal bacterium and a major human pathogen causing a wide range of clinical infections. S. aureus has been identified as a risk factor for the development of various infections. This organism is extensively studied in patients with surgical site infections (SSIs), patients undergoing hemodialysis and in patients on continuous ambulatory peritoneal dialysis (CAPD). It causes blood stream infections (BSIs), skin and soft tissue infections (SSTIs), osteomyelitis, endocarditis and nosocomial infections and is the major cause of community-acquired infections. Staphylococci are generally found on the skin and mucous membrane of the humans. It predominantly colonizes the anterior nares (vestibulum nasi). Persistent carriage is more common in children than in adults. The prevalence and incidence of S. aureus varies according to the population studied. Treating the anterior nares with topical antibiotics, may cause the organism to disappear. Penicillin was the original drug for the treatment of infections caused by S. aureus and the emergence of resistance was due to the acquisition of plasmid-borne genetic elements encoding β-lactamases. Penicillinase-resistant penicillins were developed for the treatment. S. aureus has been recognized as an important human pathogen. Staphylococcal infections occur regularly in hospitalized patients and have severe consequences, despite antibiotic therapy.[1,2]

Discovery

S. aureus was discovered in 1880 by a surgeon, Alexander Ogston, who described Staphylococcal disease and its role in sepsis and abscesses. S. aureus remains a versatile and dangerous pathogen to human health over the last 100 years and has become one of the leading causes of hospital-acquired infection worldwide.[3,4,5]

Taxonomy

The genus Staphylococcus belongs to the family Staphylococcaceae, class Bacilli and order Bacillales. The term Staphylococcus is derived from the greek term staphyle, meaning “a bunch of grapes.” Under the microscope, Staphylococcus appears as gram-positive cocci (0.5-1.5µm) arranged in single cells, tetrads and short chains,

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but predominantly as "grape like" clusters. They are facultative anaerobes (except S. aureus subsp. anaerobius and S. saccharolyticus), non-motile, non-spore forming and catalase positive. They don't produce gas from carbohydrates. The organisms are resistant to high temperatures (50°C), to high salt concentrations, and to drying.[6]
A major genotypic criterion of this genus is G+C content of 30 to 39 mol%. Whole genome sequencing has been performed for many Staphylococcal strains and complete genome sequences are available for S. aureus. The S. aureus genome is composed of a single chromosome ranging in size from 2.8 to 2.9 Mbp.

Carriage of S. aureus

Staphylococci are ubiquitous colonizers of skin and mucous membrane. S. aureus can exist as normal flora. Primary reservoir of Staphylococci is anterior nares. Three patterns of carriage can be distinguished: [1]

1. **Persistent carriers:** About 20% of individuals almost always carry one type of strain (two S. aureus positive culture)
2. **Intermittent carriers:** About 60% of individuals harbors S. aureus intermittently and the strains change with varying frequency (one S. aureus positive culture)
3. **Non-carriers:** A minority (20%) of people never carry S. aureus (no S. aureus positive culture).

The reasons for these differences in colonization patterns are unknown.

Factors influencing the rate of S. aureus nasal carriage [1]

1. Adherence to epithelia which is mediated by: lipoteichoic acid, surface associated proteins, carrier versus non-carrier state and viral infections of the upper respiratory tract.
2. Nasal abnormalities
3. HLA type
4. Ecology of nasal flora
5. Race
6. Age
7. Genetic makeup
8. Immunological status
9. Hospitalization
10. Repeated needle injections
11. Hormonal status in women

Incidence

In the past 30 years, both the community-acquired and hospital-acquired Staphylococcal infections have increased. According to the data from national nosocomial infections surveillance system, centers for disease control and prevention (CDC), during the period from 1990 to 1992, S. aureus was the most common cause of pneumonia and surgical wound infections and the second most common cause of nosocomial BSIs. Another data from national nosocomial infections surveillance system during the period from 1989 to 1997 showed that the number of infections in intensive care units has continued to increase.[3]

Virulence factors

A large number of virulence factors have been identified for S. aureus and their possible role in pathogenesis. These include the slime layer, capsular polysaccharides, cell wall constituents (peptidogylycan, teichoic acid, protein A and adhesions), exoenzymes and exotoxins.[3,7]

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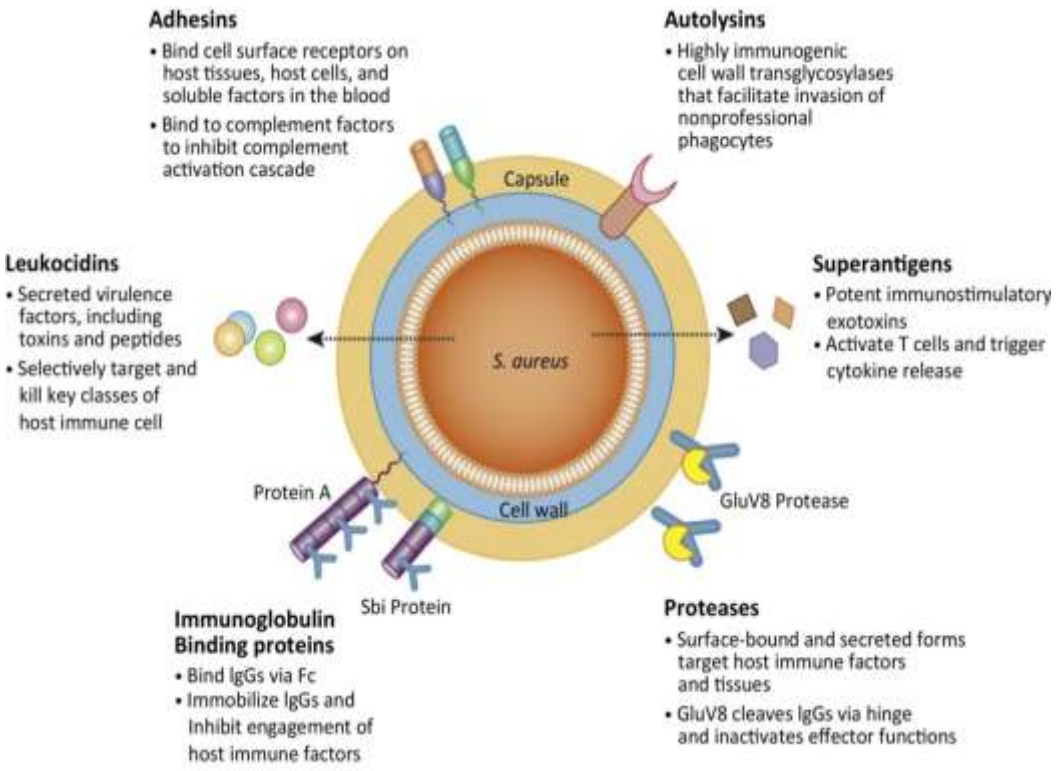

Figure 1: Virulence factors in Staphylococcus aureus[8]

"Although this organism is frequently a part of the normal flora, it can cause significant infections under appropriate conditions."

Predisposing factors to S. aureus infection

- Defects in leucocyte chemotaxis;
- Defects in opsonization by antibodies secondary to congential or acquired hypogammaglobinemias or complement component deficiencies;
- Defects in intracellular killing of bacteria;
- Skin injuries;
- Presence of foreign bodies;
- Viral infections;
- Chronic underlying diseases like malignancy;
- Therapeutic or prophylactic antimicrobial administration.

Pathogenesis

S. aureus has a diverse arsenal of components and products that contribute to pathogenesis of infection. These components and products have overlapping roles and can either in concert or alone. The organism may cause disease through tissue invasion and toxin production.[3]

1. **Tissue invasion:** The postulated sequence of events that leads to S. aureus infection is initiated with endothelial cell injury which is the potential target of S. aureus. Staphylococcal avidly adhere to endothelial cells and bind through adhesion receptor interactions. In vitro studies demonstrate that after adherence, Staphylococci are phagocytosed by endothelial cells.[6]The hallmark of Staphylococcal infection is the abscess, which consists of a fibrin wall surrounded by inflamed tissues enclosing a central core of pus containing the organisms and leukocytes. The organism from the focus may disseminate hematogenously. This may result in pneumonia, bones and joints infection, and infection of heart valves.

2. **Toxin mediated disease:** the organism elaborates toxins that cause specific diseases. Pyrogenic toxin being a superantigen can cause life threatening disease that is characterized by rapid onset of high grade fever, shock, capillary leak, and multiorgan dysfunction. Superantigens are T-cell mitogens that bind directly to invariant regions of major histocompatibility complex (MHC) class II molecules, bypassing intracellular protein ingestion and digestion and subsequent peptide presentation by the antigen presenting cells. The MHC bound superantigens attach to T cells according to the composition of the variable region of the T cell receptor β-chain.

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Toxic shock syndrome toxin 1 (TSST-1) binds all the variable region of β2-positive T cells, causing an expansion of clonal T cells, resulting in the massive cytokines release. These cytokines mediate the toxic shock syndrome (TSS).[3,9]

Infections associated with S. aureus:

1. **Folliculitis** is a benign infection of superficial dermis (Ostia of the hair follicles) characterized by presence of small, reddish, painful lesions.
2. **Impetigo** is a superficial infection of the dermis most commonly seen in children. Two forms: nonbullous and bullous. S. aureus accounts for 80% to 90% cases of impetigo.[10]
3. **Cellulitis** refers to rapidly spreading inflammation and infection of the soft subcutaneous tissues. Erysipelas is a type of cellulitis occasionally caused by S. aureus. Necrotizing fasciitis is another cutaneous infection caused by S. aureus.
4. **Endocarditis:** S. aureus is a cause of native valve endocarditis and is also a leading cause of prosthetic valve endocarditis.[11]
5. **Skin and soft-tissue infections (SSTIs)** occur after 2 to 5% of all surgeries. According to 2009-2010 U.S. National Healthcare Safety Network data, S. aureus was the most common cause of SSIs accounting for 30% of infections.[12]
6. **Staphylococcal food poisoning** follows the ingestion of preformed enterotoxins produced in the food contaminated with enterotoxigenic Staphylococci and then left at 28°C for 2-4 hours. Commonly incriminated foods include cooked or processed meat or dairy products. Ham is most frequently incriminated, accounting for 24% of outbreaks reported to the CDC from 1921 to 1981.[13]
7. **TSS:** With the introduction of superabsorbent tampons used during menstruation Staphylococcal toxic shock syndrome came into prominence. The disease is characterized by fulminant onset. Clinical findings include high fever, erythematous rash with subsequent desquamation, hypotension and multiorgan damage. It often develops from the site of colonization rather than infection.[14]
8. Staphylococcal bacteremia seed to distant sites, leading to endocarditis, osteomyelitis, polyarthritis, and metastatic abscess formation.[15,16]

In the early 1970s, physicians were finally forced to abandon their belief that, given the vast array of effective antimicrobial agents, virtually all bacterial infections were treatable. Their optimism was shaken by the emergence of resistance to multiple antibiotics among such pathogens as S. aureus, Streptococcus pneumoniae, Pseudomonas aeruginosa, and Mycobacterium tuberculosis. The evolution of increasingly antimicrobial resistance bacterial species stems from a multitude of factors that includes the widespread and sometimes inappropriate use of antimicrobials, the extensive use of these agents as growth enhancer in animal feed, and with the increase in regional and international travel, the relative.[2]

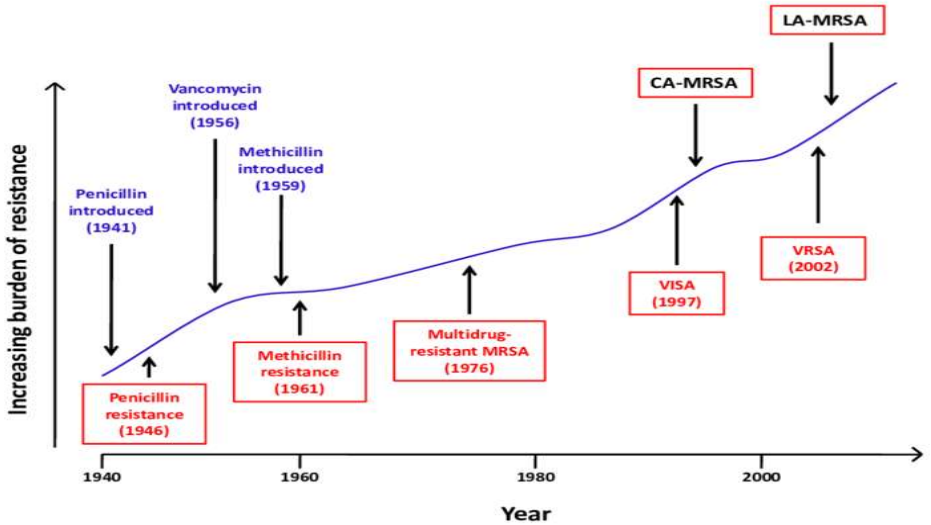

Figure 2: Emergence of antibiotic resistance against S. aureus[17]

The mortality of S. aureus bacteremia remains approximately 20 to 40% despite the availability of effective antimicrobials. S. aureus is now the leading overall cause of the nosocomial

infections. The mortality of patients with S. aureus bacteremia in the pre antibiotic era exceeded 80% and over 70% developed metastatic infections. Introduction of penicillin in the early 1940s dramatically improved the prognosis of patients with Staphylococcal infection. However, as early as 1942, penicillin resistant Staphylococci were recognized, first in the hospitals and subsequently in the community. By the late 1960s, more than 80% of both community and hospital acquired Staphylococcal isolates were resistant to penicillin. This pattern of resistance first emerging in hospitals and then spreading to the community, is now a well-established pattern that recurs with each new wave of antimicrobial resistance.[2]

Accurate and early detection of S.aureus is mandatory for effective management of infections caused by it.

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