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Staphylococcus epidermis and acne scar inflammations in young people

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ABSTRACT: Staphylococcus epidermidis is a resilient microbe distinguished by its immobile, Gram-positive spherical cells that aggregate in clusters resembling grape clusters. Biochemical examinations reveal a faintly positive outcome in the nitrate reductase test for this microbe. It generates urease while lacking oxidase production. S. epidermidis possesses a transferrin-binding protein that facilitates the acquisition of iron from transferrin. The formation of biofilms on plastic devices plays a pivotal role in the pathogenicity of S. epidermidis and its connection to acne. This adaptable bacterium is frequently encountered as a nosocomial and commensal pathogen, recognized for its opportunistic behavior and its global impact on infections, primarily contracted within healthcare facilities. It displays a remarkable ability to establish strong biofilms on various surfaces, contributing to infections linked to catheters and heart valve implants. The transmission of the bacterium is easily facilitated through the contamination of the skin of hospital visitors and healthcare personnel.

Keywords: Acne scar, skin inflammation, Staphylococcus epidermidis, biofilm

1. INTRODUCTION

Us Staphylococcus epidermidis has long been recognized as the predominant bacterial colonizer of human skin [1]. A study of the healthy human microbiome has reinforced this, with S. epidermidis found to be universally present on the skin of study participants [2]. Due to this ubiquitous presence and fewer virulence factors compared to its more pathogenic relative, Staphylococcus aureus, historically S. epidermidis was perceived as non-pathogenic. The growing trend in modern healthcare towards interventional and invasive techniques has contributed to the rise of S. epidermidis as a notable pathogen primarily acquired within healthcare settings, particularly concerning prosthetic devices [3]. The capacity of this organism to develop biofilms on foreign objects plays a crucial role in the progression of disease. Within these biofilms, bacteria are encased in a protective extracellular polymeric matrix, providing resistance against both antibiotics and the host immune system [4]. Although recognized as a significant nosocomial pathogen for over 30 years, knowledge bout the epidemiology, transmission and molecular biology of S. epidermidis is limited compared to S. aureus [5]. Scientific review of S. epidermidis inevitably involves discussion of S. aureus. Historically, descriptions of S. epidermidis have been shaped by comparisons to S. aureus [6]. Furthermore, the genetically intractable nature of S. epidermidis and the absence of molecular methods specific for the manipulation of the species have hindered its study. In the absence of specific knowledge, there has been a tendency to assume that findings about S. aureus can be extrapolated. However, as multidrug resistance proves increasingly common and the clinical impact and associated economic burden posed by S. epidermidis continue to increase [7], the time for the dedicated study of this organism is long overdue [8].

2. Basic Microbiology

S. epidermidis is a non-motile, non-spore-forming, gram-positive coccus, 0.8 – 1.0 mm in diameter, arranged in pairs and tetrads with occasional single cells (figure 1) [9]. Colonies are circular, 2.5 – 4.0 mm in diameter, smooth, raised, glistening and translucent to slightly opaque; typically, grey or greyish-white in color, rare colonies possess a slightly yellow or brownish pigment [10]; sticky consistency increases with age [11]. The cell wall is composed of peptidoglycan, teichoic acid and lipoteichoic acid. Certain strains can also produce polysaccharide intercellular adhesin (PIA) which can coat the cell and stimulate biofilm formation [12]. S. epidermidis can grow from 15 – 45oC, with optimal growth at 30 – 37oC [11]. Although a facultative anaerobe, it grows best under aerobic conditions. The G+C content of its DNA is 33.5 + 0.2% [13].

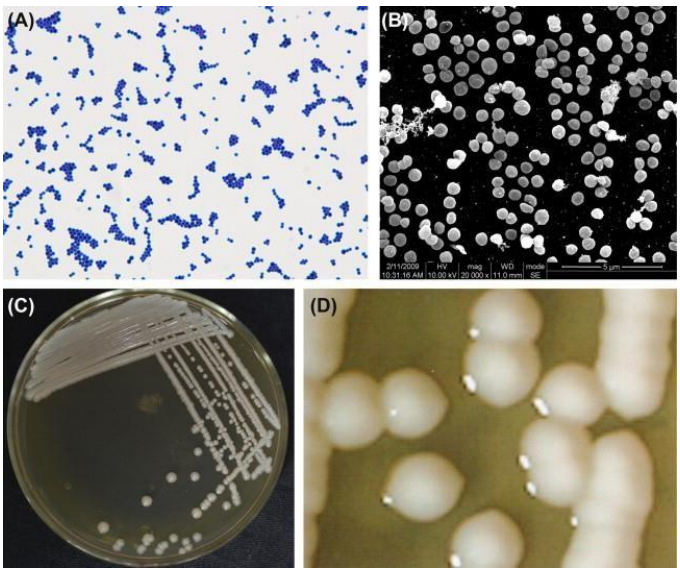

FIGURE 1. - (A) *Staphylococcus epidermidis* cells (Gram stain). (B) *S. epidermidis* cells (SEM). *S. epidermidis* cells are spherical (0.5–1.5 µm in diameter) and gram-positive

The division of staphylococcal species on the basis of coagulase testing was proposed by Fairbrother in 1940 [14]. For a number of decades, medical microbiologists only focused on identifying the coagulase-positive species, S. aureus, based on the assumption that all coagulase negative staphylococci (CNS) were non-pathogenic commensals [11]. In 1974,Kloos and Schleifer introduced the biochemical classification scheme for the differentiation of Staphylococcus species, on which current bio typing schemes are based[15]. Consequently, S. epidermidis became recognised as the major cause of all CNS infections except those of the urinary tract [15,16-17], with Staphylococcus saprophyticus was found to account for 80% (12/15) of female urinary tract infections [18] . The original Kloos and Schleifer scheme described 11 different staphylococcal species [19]. Since then, 54 staphylococcal species have been catalogued by the List of Prokaryotic Names with Standing in Nomenclature (LPSN) database [20].

3. Clinical impact of S. epidermidi

Consistently recognised as a leading cause of nosocomial infections, S. epidermidis posessignificant morbidity and mortality to individual patients and economic burden on healthcare systems. This impact is reflected in the summary data on Antimicrobial-resistant pathogens associated with healthcare-associated infections reported by the Centers for Disease Control and National Healthcare Safety Network, in which CoNS predominantly represented by S. epidermidis, were the identified as the: most common cause of central line associated bloodstream infections (CLABSI); second-ranked cause of surgical site infections; and third most reported pathogen for hospital-acquired infections [21].

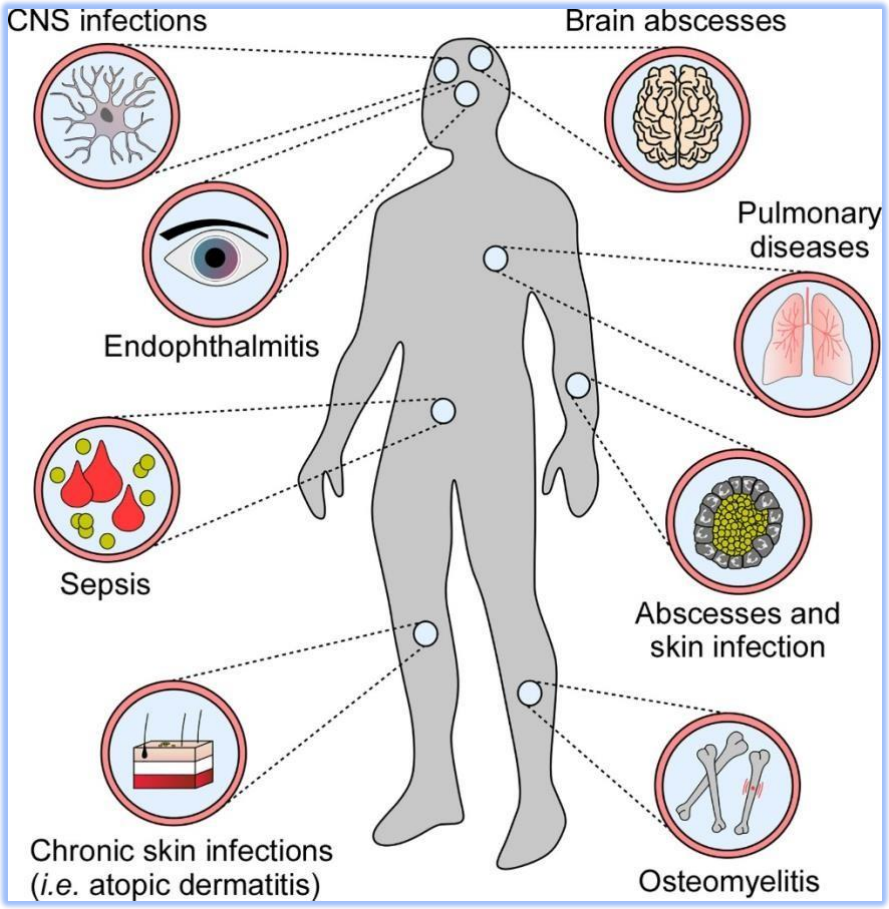

FIGURE 2. - Clinical impact of *S. epidermidis* (31)

4. Epidemiology of S. epidermidis

S. epidermidis is characterized by marked genetic diversity, greater than that observed in other staphylococcal species [3]. To date, the majority of epidemiological studies of S. epidermidis have utilized pulsed-field gel electrophoresis (PFGE) as the typing method [22] Despite pronounced genetic diversity within the species, as demonstrated in community-based colonization studies hospital- based investigations suggest the predominance of a few hospital- associated, epidemic clonal lineages [23].

Individually, most nosocomial transmission studies have mostly focused on the presence of clones within specific high-risk sites, such as the ICU [24], neonatal ICU (NICU) cardiac care centers and haemodia lysis unit [25]. Typically, these studies have demonstrated the local prevalence of a few dominant clones. One NICU-based study documented the emergence of a particular S. epidermidis clone responsible for 31% (20/65) of bloodstream infections occurring within the unit over a year, as wellas the longitudinal persistence of the second clone over the 11-year period of the study [26]. While another prospective Italian study reported S. epidermidis to account for 30.4% (56/184) of all NICU-acquired infections over three years, with four main clones noted to cause most infections [26]. Compared to sporadic strains these clones were more antibiotic resistant, particularly to the agents treatment within the unit. This observation of disseminated clones possessing more resistant antibiotic profiles is common to several studies, with methicillin-resistant S. epidermidis reported to comprise up to 79% [27] to 92% of hospital isolates. The classification of S. epidermidis was revised in 2007, with introduction of an improved multi locus sequence type (MLST) scheme [5]. This saw the reclassification of ST27, previously identified as the major disease- associated strain type, present in hospitals in both the US and Europe [28].

Mode of transmission A limited number of studies have attempted to address the mode of transmission of S. epidermidis clones within the hospital environment. These investigations have yielded similar results, indicating the replacement of less drug-resistant colonizing strains with locally prevalent, drug-resistant S. epidermidis strains during the course of admission [29]. Both healthcare workers [5,30,31] and the hospital environment [31] have been implicated as potential reservoirs for hospital- adapted S. epidermidis clones (figure 3). A study conducted in 1982 by Archer et al. on patients undergoing cardiac surgery at the Medical College of Virginia found that only one in 26

patients who were not previously hospitalized were pre-operatively colonized with multidrug- resistant S. epidermidis (MDRSE; defined as resistance to methicillin and gentamicin), however, by day ten post-operatively 80% of patients were colonized [5].

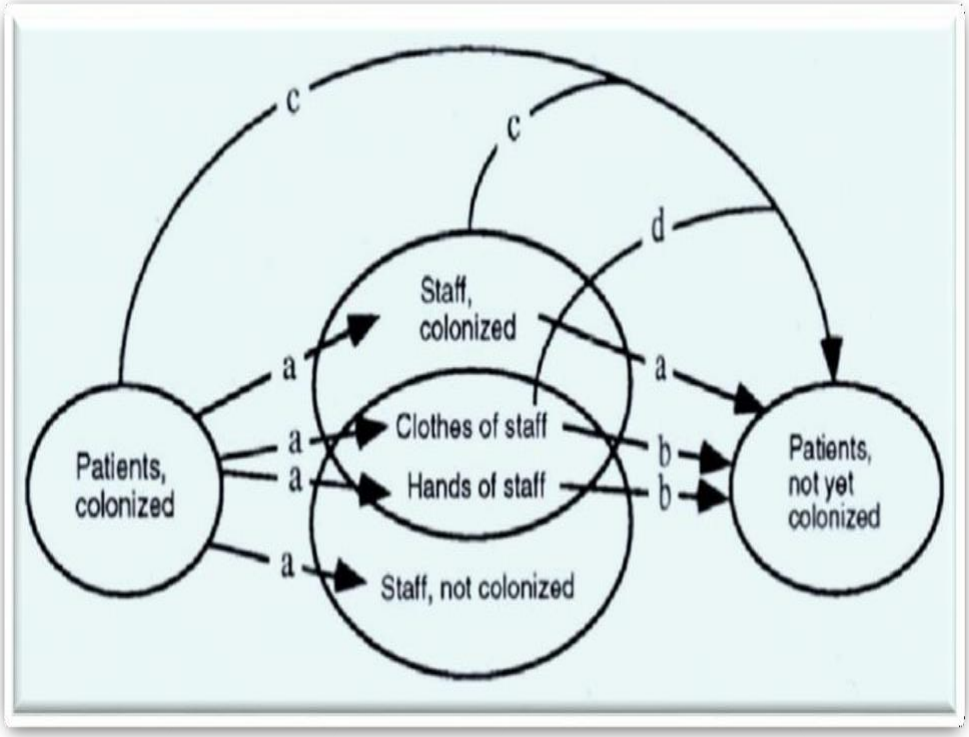

FIGURE 3. - Mode of transmission of *S. epidermidis*

The understanding of virulence factors in S. epidermidis remains limited due to its high genetic variability, which may serve as a potential marker and therapeutic target against its invasiveness. Among the various virulence factors identified, our focus in this review primarily revolves around the significant role played by biofilm formation. Furthermore, the ability of S. epidermidis to invade human immune defenses and exist as a commensal on the human skin, while still not fully comprehended, renders it as an occasional "accidental pathogen" [2,5].

In contrast to many toxin-producing bacteria, S. epidermidis exhibits unique amphipathic peptides called phenol-soluble modulinos (PSMs) that serve multiple functions. PSMs interact with biofilm formation, are regulated through quorum sensing, and influence the human innate immune system response, thereby contributing to the development of sepsis. Another noteworthy virulence factor is the glycoprotein hemagglutinin, which induces coagulation of red blood cells (RBCs). S. epidermidis hemagglutinin displays resistance to pH variations, temperature changes, protease concentrations, serum proteins, cleanser detergents, and sub-inhibitory antibiotics. These factors play a pivotal role in the pathogenesis of S. epidermidis by directly aiding adherence to polymers, which is the initial step in the formation of biofilms and the occurrence of biomaterial-associated infections [32].

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CONFLICTS OF INTEREST

The authors declare no conflict of interest

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