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Review on *Streptococcus pyogenes* Biology, Epidemiology, Pathogenesis and Diagnosis

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Abstract

The Streptococcus genus contains greater than fifty named species that inhabit a broad range of hosts, including humans and domesticated animals. The numerous species of streptococci may be described on the basis of the nature and extent of their hemolytic activity as alpha-hemolytic, beta-hemolytic, and non-hemolytic species. *S.pyogenes* (group A streptococcus, GAS) is beta hemolytic an important human pathogen ranked number nine on the list of global killer pathogens. Before World War II, this species was mostly known for causing severe puerperal infections, scarlet fever, and for being a major cause of death in burn patients, but the introduction of penicillin was responsible for a marked decline in the incidence of these infections in developed countries. However, since the 1980s, a resurgence of severe invasive was reported in several industrialized regions. GAS is responsible for a broad spectrum of diseases ranging from benign, self-limiting infections (pharyngitis or strep throat, impetigo) to life-threatening invasive disorders (necrotizing fasciitis, streptococcal toxic shock syndrome) to immune sequelae (acute rheumatic fever). GAS is distributed directly to other persons via droplets from the respiratory tract or skin. The nasal discharges of a person harboring GAS are the most dangerous source for spread of these organisms. *S.pyogenes* has several virulence factors that enable it to attach to host tissues, invade immune response and spread by penetrating host tissue layers. The capsule protects the bacterium from phagocytosis by neutrophils. In addition, the capsule and several factors embedded in the cell wall, including M protein, lipoteichic acid and protein F facilitate attachment to various host cells. Streptococci are generally grown on agar media supplemented with blood. Penicillin is first choice for infections caused by hemolytic streptococci. Cephalosporin, or erythromycin or clindamycin in the case of allergy to penicillin, should be prescribed in age-related doses.

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Introduction

The Streptococcus genus contains greater than fifty named species that inhabit a broad range of hosts, including humans and domesticated animals, where they often colonize as part of the normal flora and/or cause infection (1). The streptococci are gram-positive, catalase-negative, facultative anaerobic, coccoid, or ovoid bacteria. Streptococci occur in mouth and intestinal tract and also on the skin and in the upper respiratory and genital tracts. The numerous species of streptococci may be described on the basis of the nature and extent of their hemolytic activity as alpha-hemolytic (α-hemolytic), beta-hemolytic (β-hemolytic), and non-hemolytic species(γ-hemolysis) (2). In α-hemolysis the red blood cells are partly lysed and that can be seen as green circle around bacterial colonies. In β-hemolysis, the red blood cells are completely lysed, and a clear zone can be seen around colonies. When there is no red blood cell lysis seen, it is called the γ-hemolysis (3).

Streptococcal diseases have been recognized in recorded history for over two thousand years and remain today as a serious cause of worldwide health problems (4) and infections have been documented in all races, sexes and age groups (5). The most clinically significant of the human pathogens are *Streptococcus pyogenes*, the causativeagent of ‘‘strep’’ throat, and *S. pneumoniae*, a leading cause of bacterial pneumonia (1).

Streptococcus pyogenes (group A streptococcus, GAS) is an important human pathogen ranked number 9 on the list of global killer pathogens (7). The disease manifestations can range in severity from mild upper respiratory tract infections and skin and soft tissue infections to the severe invasive infections, such as bacteremia, pneumonia, necrotizing fasciitis, and streptococcal toxic shock syndrome (8). *S. pyogenes* is the main human pathogen associated with local or systemic invasion and poststreptococcal immunologic disorders.

S pyogenes typically produces large (1 cm in diameter) zones of β hemolysis around colonies greater than 0.5 mm in diameter (6).

Historically, the first record of an *S. pyogenes* infection is attributed to Hippocrates (5th-4th century B.C.) who described a scarlet fever epidemic (10). Later studies by Billroth in 1874 and Pasteur in 1879 described *S. pyogenes* cocci from wound infections and from the blood of a patient with puerperal sepsis, respectively. The name streptococcus came from Friedrich Julius Rosenbach in 1884, who examined bacteria isolated from suppurative lesions, and the species was named *Streptococcus pyogenes* (Gr., pyo, pus, and genes, forming) (11).

Before World War II, this species was mostly known for causing severe puerperal infections, scarlet fever, and for being a major cause of death in burn patients, but the introduction of penicillin was responsible for a marked decline in the incidence of these infections in developed countries. However, since the 1980s, a resurgence of severe invasive GAS disease was reported in several industrialized regions. Currently, in developed countries *S. pyogenes* is well known for causing mild superficial infections of the upper respiratory tract and skin, like sore throat and impetigo, but also severe and rapidly progressing invasive infections, like necrotizing fasciitis, which made this organism popularly known as the “flesh-eating” bacterium due to the extensive necrosis of deep soft tissue (12).

The importance of *Streptococcus pyogenes* as a human pathogen led to development of well-used clinical microbiology tools for its identification. Most notably, *S. pyogenes* forms large colonies and produces β-hemolysis following growth on blood agar, and is serologically distinguished from many other streptococcal species by its group carbohydrate that is covalently linked to the peptidoglycan cell wall (14).

GAS diseases remain a major public health problem in developing countries, reaching 600 million cases per year and thus constituting an important cause of morbidity and mortality (15). At least 517, 000 deaths each year are estimated to result from severe GAS infections, and its prevalence is at least 18.1 million cases, with 1.78 million new cases arising each year. Since the advent of antibiotic therapy in the 1940’s, the incidence of invasive GAS infections decreased significantly worldwide, only to re-emerge during the 1980’s (16). *S. pyogenes* causes the infection commonly known as “strep throat” and is the cause of 90% of bacterial pharyngitis cases. Untreated infections may lead to acute glomerulonephritis, scarlet fever, or rheumatic fever. It has many virulence factors that contribute to its pathogenicity, such as lipoteichoic acid, M protein, hyaluronidase, protease, streptokinase, DNase/RNase, C5a peptidase, and Streptolysins O and S (17).

A serotyping system based on antigenic variation of a surface exposed M protein and developed by Rebecca Lancefield has been in use since 1928 (18). The genetic diversity of group A streptococcal GAS isolates obtained in 1990 from Ethiopian children with various streptococcal diseases was studied by using *emm* gene sequence analysis.

Biology

S. pyogenes is Gram-positive, non-motile, and non-spore forming spherical cocci with 0.5 to 1.0 μm, which can form long chains in liquid medium, although they occur in short chains or pairs in clinical specimen (18*).GAS colonies,* exhibits a spectrum of encapsulation morphologies, ranging from highly mucoid to non-mucoid, depending on the level of hyaluronic acid capsule production (10).

Taxonomy of *S.pyogenes*;
Domain: Bacteria
Phylum: Firmicutes
Class: Bacilli

Order: Lactobacillales
Family: Streptococcaceae
Genus: Streptococcus
Species: pyogenes (19)

S. pyogenes is a facultative anaerobe and is grown at 37°C in either ambient air or in5% to 10% CO_2. Like all streptococci, GAS is both catalase and oxidase negative. GAS lacks the necessary enzymes for a functional TCA cycle and oxidative-cytochromes forelectron transport; therefore, it relies completely on fermentation of sugars for growth and energy production. It is a member of the lactic acid bacteria and is homofermentative for lactic acid production from glucose fermentation (20).The human oropharynx is the primary anatomic site for GAS colonization and infection, and saliva is the first material encountered (21).GAS is distinguished from other groups of β-hemolytic streptococci by a group-specific polysaccharide (Lancefield carbohydrate C) located in the cell wall. Serologic grouping by the Lancefield method is precise, but group A organisms can be identified more readily by any one of a number of latex agglutination, co-agglutination, or enzyme immunoassay procedures.

GAS can be subdivided into >100 serotypes by the M-protein antigen that is located on the cell surface and by fimbriae (hairlike fuzz) that project from the outer edge of the cell. Classically, typing of the surface M protein relied upon available polyclonal antisera.The GAS cell is a complex structure. In rapidly dividing strains (e.g., young cultures, epidemic strains), the cell is covered with a hyaluronic acid capsule that gives the colonies a mucoid or water drop appearance. Protruding from the cell surface and into the hyaluronic capsular layer are microscopic hairlike fimbriae, which promote adherence of GAS to epithelial cells and extracellular matrix proteins (22).

Epidemiology

The ultimate source of group A streptococci is a person harboringthese organisms. The individual may have a clinical or subclinical infection or may be a carrier distributing streptococci directly to other persons via droplets from the respiratory tract or skin. The nasal discharges of a person harboring *S.pyogenes* are the most dangerous source for spread of these organisms (6).Different clinical manifestations of this bacterium are more common in different parts of the world. *S.pyogenes* is responsible for a wide range of both invasive and non-invasive infections (23).

Worldwide, *S.pyogenes* causes over 18 million cases of severe diseases resulting a half million deaths. There are 616 million cases of pharyngitis caused by *S, pyogenes* worldwide each year ((24).Streptococcal pharyngitis is predominant in temperate areas and peaks in late winter and early spring. About 15-20% of school aged children has *S.pyogenes* in its carrier form in their throats and is more at risk of having the disease. Impetigo is common with children warm humid climates. Crowding and poor hygiene increase the chance of an outbreak of GAS infection (1).

Epidemiologic studies showed that resurgence of severe invasive GAS infection was not limited to sporadic cases; rather, it represented a global spread, ushering in a new pandemic similar to that of 20th century. An important feature of this latest pandemic is its association with distinct epidemiological shift in GAS serotypes (16).

There are 115.6 million cases of RHD yearly and at least 18.1 million cases of invasive infections, predominantly in older populations. Annually there are 400, 000 deaths and hundreds of thousands of children died due to ARF and RHD (25).

Pathogenesis and Virulence factors

S. pyogenes is a strict human pathogen that cans colonize asymptomatically, but is also a common cause of clinical diseases (19). *S.pyogenes* has several virulence factors that enable it to attach to host tissues, invade immune response and spread by penetrating host tissue layers. The capsule protects the bacterium from phagocytosis by neutrophils. In addition, the capsule and several factors embedded in the cell wall, including M protein, lipoteichic acid and protein F facilitate attachment to various host cells. M proteins also inhibitsopsonization by alternative complement pathway to host complement regulators. The M protein found on some serotype is able to prevent opsonization by binding to fibrinogen (26).

The first step in GAS disease pathogenesis involves successful colonization of the upper respiratory mucosa or skin of human host.As needs to overcome many obstacles to establish infection, including circumventing the innate immune defences, colonizing epithelial surfaces and penetrating the mucosal layer. It has developed an impressive array of surface and secreted virulence factors to exploit host immune defences to

adapt, thrive and/or persist (27). Some of these virulence factors are strain specific and confer tissue tropism are;

M protein: encoded by the *emm*gene, is the major virulence factor and an important epidemiological typing tool (28). In electron microscopy, they appear as fine hair-like projections radiating from the streptococcal surface. The M-protein inhibits phagocytosis, which is a primary virulence mechanism for survival in tissue. Absence of the emm gene allowed rapid phagocytosis of the streptococcus.

Lipoteichoic acid: In *Streptococcus pyogenes*, lipoteichoic acid is associated with M protein that protrudes from cell membrane through peptidoglycan layer.The long molecular M protein together with the liopteichoic acid from microfibrils that facilitate the attachment of S pyogenes to animal cells (29).

Hyaluronic Acid Capsule: Capsules are major virulence factors of many pathogenic bacteria as they are anti-phagocytic in nature. The hyaluronic acid capsule of S. pyogenes is non antigenic because of its chemical similarity to host connective tissue. Capsule of S. pyogenes thus assists the bacterium to hide its own antigens and to go unrecognized as antigenic by its host. The hyaluronic acid capsule also prevents opsonized phagocytosis (30).

GAS produces two hemolysins, namely streptolysin O (SLO) and streptolysin S (SLS). SLO is a well-characterized oxygen labile bacterial exotoxin. Early studies of purified SLO and SLS demonstrated that these hemolysins were toxic to the variety of human cells in vivo and in vitro. Recently, cytotoxic effects of SLO and SLS have been described to protect GAS from phagocytic killings and enhance bacterial virulence (31)

Streptokinase: Streptokinase acts on plasminogen, which is converted to plasmin, active proteolytic enzymes that lyse fibrin. Thus streptokinase appears to play a biological role in streptococcal infections by breaking down the fibrin barrier around the lesions and facilitates the spread of infection.

Diseases caused by S. pyogenes

A wide range of clinical infections are recognized as being caused by Streptococcus pyogenes, including respiratory, cutaneous, soft tissue and systemic infections.

Pharyngitis

Pharyngitis is defined as an acute inflammation of the pharynx, tonsils or both. A sore throat is the most common symptom of pharyngitis. The terms 'pharyngitis', 'tonsillitis' and 'sore throat' are often used interchangeably (32). Pharyngitis is the most common manifestation of infection with GAS (33). Infection with this bacterium is diagnosed in 20 to 40% of pharyngitis cases in children and in 5 to 15% in adults (34).

The peak incidence of GAS pharyngitis occurs in children aged 5 to 15 years old (35). Pharyngitis is more common to occur during winter and spring and can be transmitted by direct or indirect contact with an infected person via large respiratory secretions droplets, or through contaminated objects and food (33).

Non-infectious auto-inflammatory complications of pharyngitis can include acute rheumatic fever and post-streptococcal glomerulonephritis, both of which are thought to result from immune responses to streptococcal infection.

Acute rheumatic fever (ARF)

ARF is an autoimmune disorder resulting from infection with GAS; in which heart valves may be severely damaged (rheumatic heart disease) (32).ARF and its sequelae rheumatic heart disease (RHD) are the major health issues, which affect children between 5-15 years of age mainly in developing countries as well as in aboriginal population of developed countries (36). ARF and RHD are estimated to affect over 20 million people globally; they lead to cause cardiovascular death during the first 5 decades of life in developing countries (37). ARF, which is the precursor to RHD, results from an abnormal autoimmune response to GAS infection in a genetically susceptible host; it affects the heart, joints, brain, and subcutaneous tissue (38).

Glomerulonephritis

This sometimes develops 1–4 weeks after *S.pyogenes* skin infection (pyoderma, impetigo) or pharyngitis. Some strains are particularly nephritogenic, principally with M types 2, 42, 49, 56, 57, and 60 (skin) (6). It is manifested in the form of post streptococcal disease. Acute post infections glomerulonephritisis characterized by inflammation, hematuria, fever, edema, hypertension,

urinary sediment abnormalities and severe kidney pain (39).

Impetigo

Impetigo is group A streptococcal superficial infection of the outer layers of skin. It commonly affects young children (40). Impetigo is a highly communicable superficial skin infection commonly caused by gram-positive bacteria that includes either Staphylococcus aureusor Streptococcus pyogenes (41). The estimations show that more than 162 million children can be affected by impetigo at any time (42). The burden of disease is highest in the low-income and developing countries. This infection is caused by invasion of the epidermis by GAS colonizing the skin following minor trauma. Autoinoculation is common and the infection is highly transmissible (42).

Factors that play a role in frequent transmission of the diseases in epidemic areas include hot and humid climatic conditions, poor access to water and crowded areas (43).

Necrotizing fasciitis

Necrotizing fasciitis (Streptococcal Gangrene) is an infection of the deeper subcutaneous tissues and fascia; it is characterized by extensive and rapidly spreading necrosis (gangrene) of the skin and underlying structures (44).

GAS necrotizing fasciitis is often a fulminate and devastating infection. Such infections can progress within hours to necrosis of an entire limb, often culminating in amputation or death. GAS necrotizing fasciitis is relatively uncommon, but when it occurs, it requires fast action to provide the best outcome for the patient (45).

Staphylococcal toxic shock syndrome (STSS)

STSS may begin at the site of any GAS infection even at site of seemingly minor trauma. The systemic illness starts with vague myalgia, chills and severe pain at the infected site, most commonly in the skin and soft tissues and leads to necrotizing myonecrosis. It continues with nausea, vomiting and diarrhea followed by hypotension, shock and organ failure. The laboratory findings are lymophocytosis. Impaired renal function (azotemia) and in over half the cases bacteremia. Some patients are in irreversible shock by the time they reach a medical facility. Many survivors have been left as multiple amputees as result of metastic spread of streptococci (46).

Bacteremia/Sepsis

Infection of traumatic or surgical wounds with streptococci results in bacteremia which rapidly can be fatal. *S.pyogenes* bacteremia can follow skin infections such as cellulitis and rarely pharyngitis (47).

Laboratory diagnosis

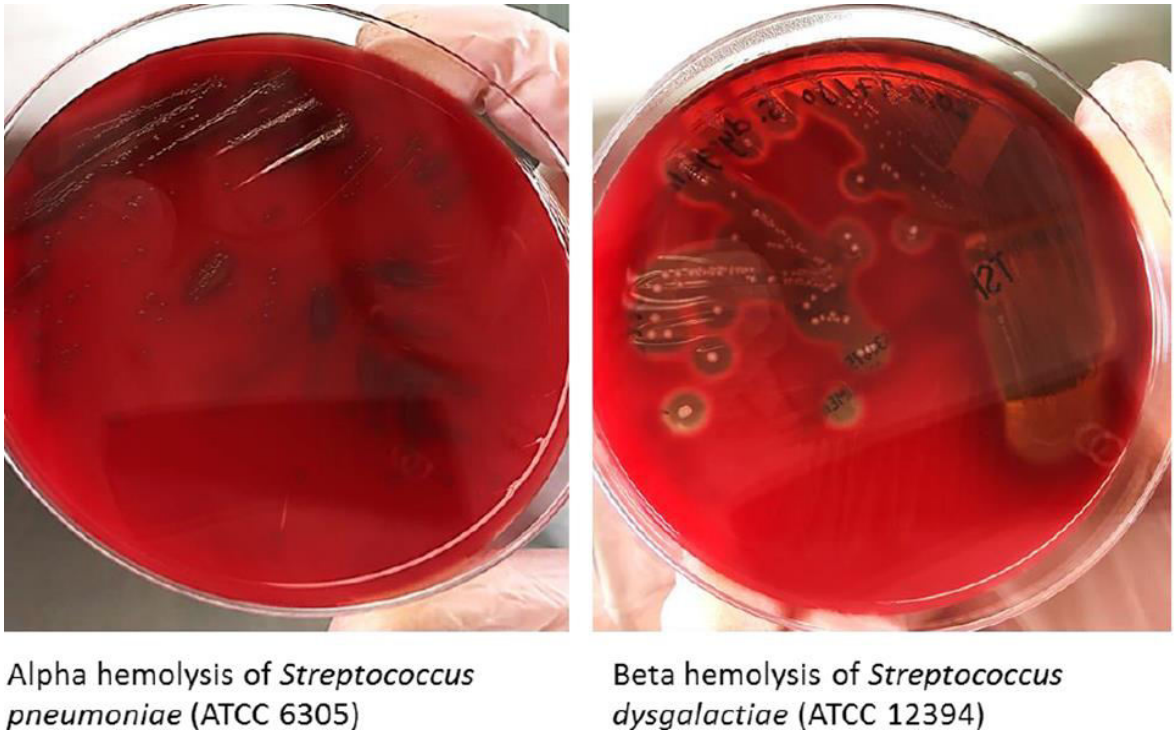
Streptococci are generally grown on agar media supplemented with blood. This technique allows the detection of β-hemolysis, which is important for subsequent identification steps, and enhances the growth of streptococci by the addition of an external source of catalase. Selective media for culturing Gram-positive bacteria (such as agar media that contains phenylethyl alcohol or Columbia agar with colistin and nalidixic acid) also provide adequate culturing conditions for S. pyogenes. To identify S. pyogenes in clinical samples, blood agar plates are screened for the presence of β-hemolytic colonies. The typical appearance of S. pyogenes colonies after 24 hours of incubation at 35-37°C is dome-shaped with a smooth or moist surface and clear margins. They display a white-greyish color and have a diameter of > 0.5 mm, and are surrounded by a zone of β-hemolysis that is often two to four times as large as the colony diameter. Microscopically, S. pyogenes appears as Gram-positive cocci, arranged in chains (48).

Specimens: Specimens to be obtained depend on the nature of the streptococcalinfection. A throat swab, pus, or blood is obtained for culture. Serum is obtained for antibody determinations (6).

Smears from pus often show single cocci or pairs rather thandefinite chains. Cocci are sometimes gram negative because the organisms are no longer viable and have lost their ability to retain the blue dye (crystal violet) and be gram positive. If smears of pus show streptococci but cultures fail to grow, anaerobic organisms must be suspected. Smears of throat swabs are rarely contributory because viridans streptococci are always present and have the same appearance as group Astreptococci on stained smears. Culture media: *S.pyogenes*grow readily on 5% sheep blood agar, chocolate agar, Columbia agar with colistin and nalidixic acid, phenylethyl alcohol agar, crystalviolet blood agar but they can be isolated more

easily using selective media that inhibit the growth of normal pharyngeal flora e.g 5% sheep blood agar containing trimethoprim-sulfamathoxazole and enrichment broth e.g. serum blood or blood broth (49).

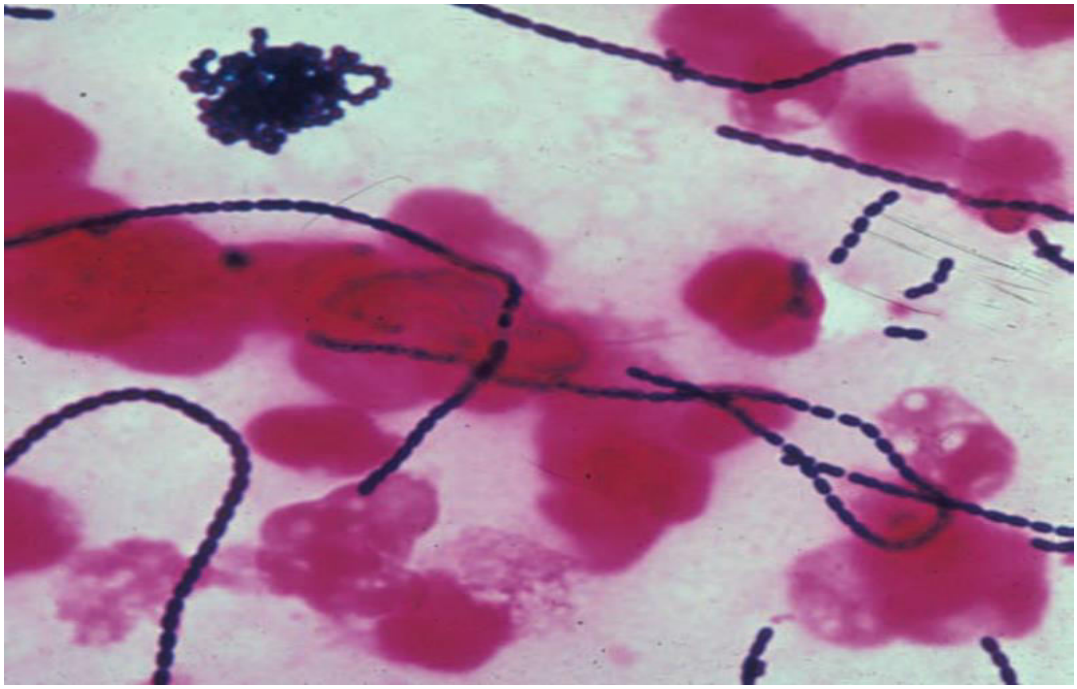
Figure.1 Alpha and beta hemolysis seen on blood agar



Alpha hemolysis of *Streptococcus pneumoniae* (ATCC 6305)

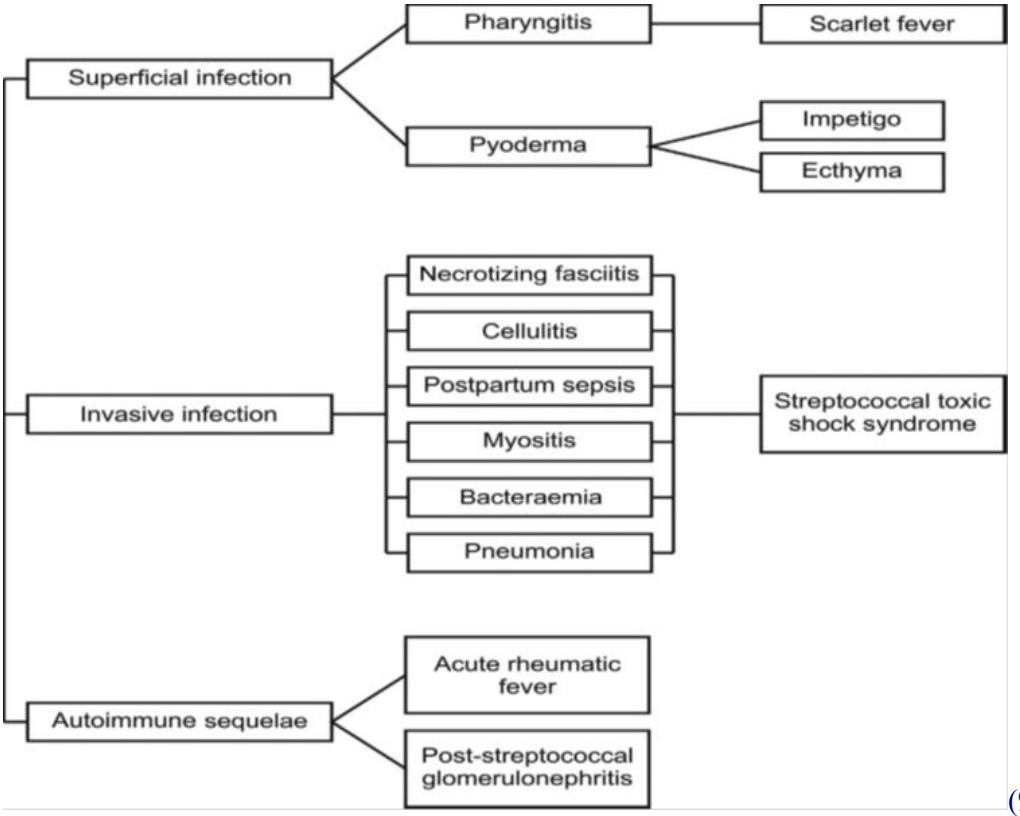
Beta hemolysis of *Streptococcus dysgalactiae* (ATCC 12394)

Fig.2 Streptococci grown in blood culture showing gram-positive cocci in chains



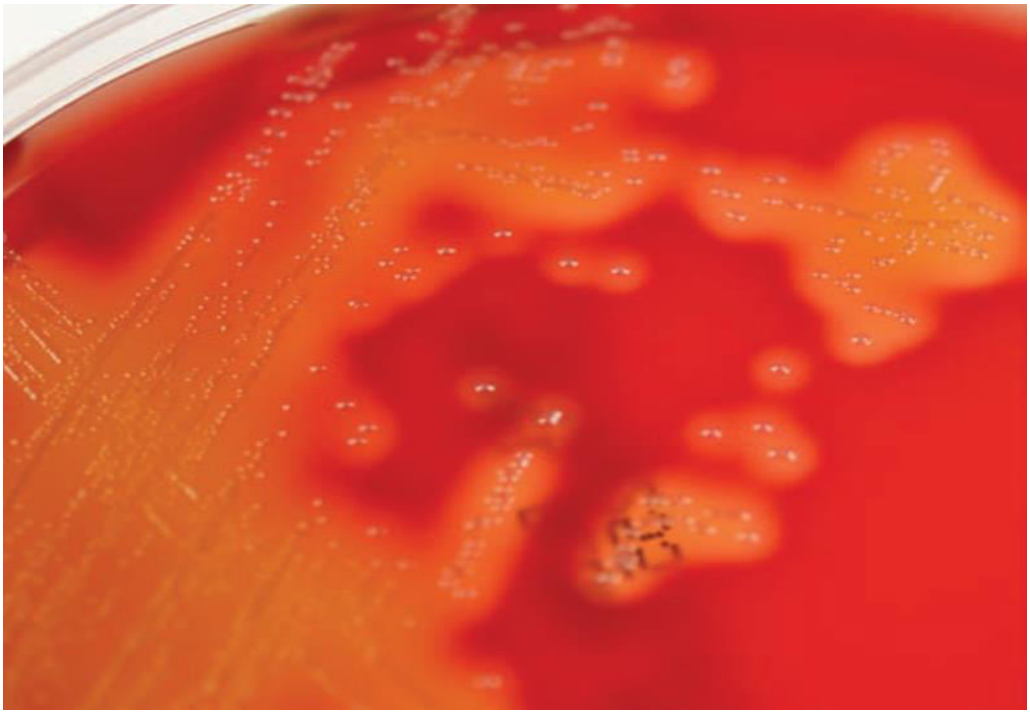
(6)

Figure.3 The major pathologies associated with group A streptococcal infection



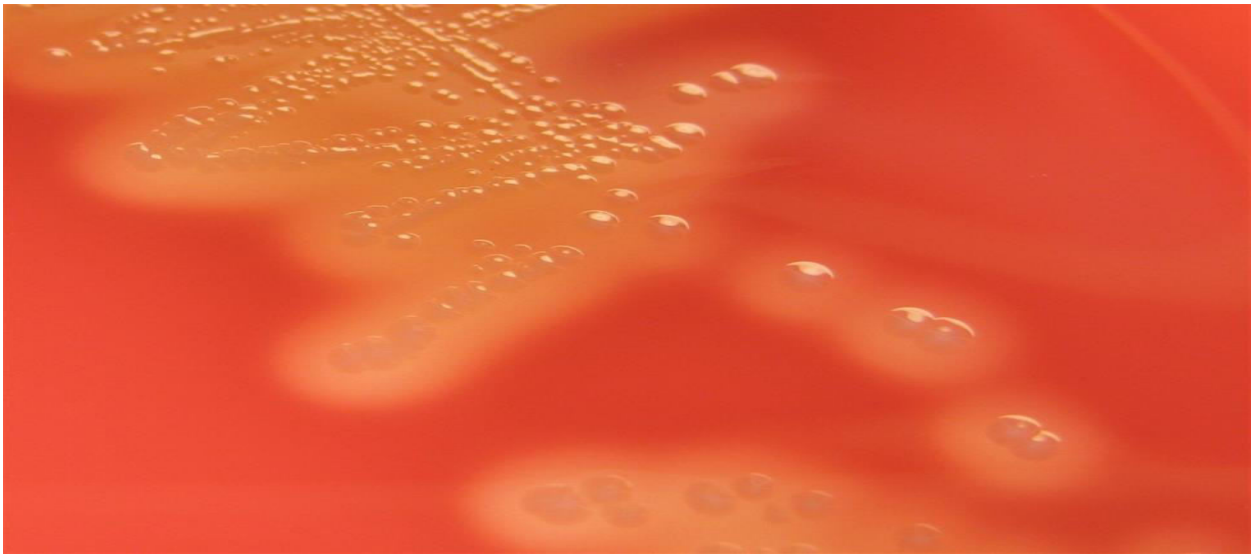
(9)

Figure.4 Group A β-hemolytic streptococci (*S. pyogenes*) after growth overnight on a 10cm plate with 5% sheep blood agar.



(6)

Figure.5 Typical appearance of S. pyogenes on sheep-blood agar plates, following 24 hour incubation under aerobic conditions. (48).



To accurate hemolysiscolonizes should be inoculated on 5% agar sheep blood agar by stabbing the inoculatory loop into agar several times.As visualization of beta-hemolysis is enhanced by anaerobic conditions, plates should be colorless shiny with mucoid appearance and produce complete haemolysis (50).

Culture of throat Specimen

Culture of a throat swab on a sheep-blood agar plate remains the gold standard for the confirmation of the clinical diagnosis of acute streptococcal pharyngitis and carriers. If done correctly, culture of a single swab on a blood agar plate has a sensitivity of 90-95% for the detection of the presence of GAS in the pharynx (51). Several variables affect the accuracy of throat culture results. For example, the manner in which the swab is obtained has an important impact on the yield of streptococci from the culture. Throat swab specimens should be obtained from the surface of both tonsils (on tonsillar fossae) and the posterior pharyngeal. A test for bacitracin susceptibility and/or PYR (pyrrolidonylaminopeptidase) are helpful for presumptive identification of GAS among b-hemolytic streptococci, which then can be confirmed by group specific antibodies (52).

Rapid Antigen Tests: The major disadvantage of culturing a throat specimen on blood agar plates is the delay (overnight or longer) in obtaining the result. Rapid antigen detection tests have been developed for the identification of GAS directly from throat swabs. Although these rapid tests are more expensive than blood agar culture, they provide results faster. Almost all such rapid tests involve an acid extraction step to solubilize GAS cell wall carbohydrate and to identify the presence by an immunologic reaction. The great majority of the rapid antigen detection tests currently available have an excellent specificity of 95%, compared with blood agar plate culture (53).

Serological Diagnosis

The host responds immunologically to streptococcal infections with a plethora of antibodies against many cellular and extracellular components. Serological diagnosis of GAS infection is based on immune responses against the extracellular products streptolysinO, DNase B, hyaluronidase, NADase, and streptokinse, which induce strong immune responses in the infected hosts. Anti-streptolysin O (ASO) is the antibody response most often examined in serological tests to confirm antecedent streptococcal infection, and helps in the diagnosis of rheumatic fever (30).

Treatment

Penicillin agents are representative drugs used as the agents of first choice for infections caused by hemolytic streptococci. However, many reports on the sensitivity of S. pyogenes to antimicrobial agents pertain to strains isolated from patients with pharyngitis or tonsillitis, and there are very few reports focusing on strains isolated from patients with invasive infections. (54).

Cephalosporin, or erythromycin or clindamycin in the case of allergy to penicillin, should be prescribed in age-related doses. Close follow-up is necessary, especially in young children (55).

Streptococcus pyogenes Vaccines

To avoid pandemic situations and to reduce the fatality rate of both GAS infections and post-streptococcal diseases, initial preventative measures to impede primary infections caused by GAS bacteria need to be considered. Vaccination, remains one of the most effective and cost-efficient methods in infectious disease prevention and control. Some models are based on developing vaccines targeting the M protein (N- and C-terminal portions), the major antigen of S. pyogenes (6).

In conclusion, Streptococcal diseases have been recognized in recorded history for over two thousand years and remain today as a serious cause of worldwide health problems. The Streptococcus genus contains many named species that inhabit a broad range of hosts, including humans and domesticated animals. GAS is an organism that is able to cause a wide array of infections in humans. GAS is an important human pathogen ranked number 9 on the list of global killer pathogens. The disease manifestations can range in severity from mild upper respiratory tract infections and skin and soft tissue infections to the severe invasive infections, such as bacteremia, necrotizing fasciitis and streptococcal toxic shock syndrome.

Given its considerable importance to human health and substantial financial burden, GAS pathogenesis has long been an area of active investigation. The overall control and prevention of streptococcal infections needs to be an area of emphasis for future research in order to reduce morbidity and mortality. Vaccines would be one answer, and understanding the role of the extracellular products in the disease process may provide new ways for looking at the development of such vaccines.

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