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Review Article

# STREPTOCOCCUS PYOGENES: A COMPREHENSIVE REVIEW WITH AYURVEDIC CORRELATION

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## ABSTRACT

Streptococcus pyogenes, also called Group A Streptococcus (GAS), is a well-known human pathogen responsible for a variety of illnesses, ranging from mild conditions like sore throat and impetigo to severe invasive diseases such as necrotizing fasciitis and streptococcal toxic shock syndrome. In addition to these infections, it can trigger immune-related complications including acute rheumatic fever and post-streptococcal glomerulonephritis, which contribute significantly to global health burdens. Although effective antibiotics are available, GAS remains a persistent challenge due to its wide array of virulence factors, complex mechanisms of disease, and the serious complications it can cause. This review offers a detailed examination of S. pyogenes, covering its microbiological traits, key virulence factors, disease mechanisms, and the broad spectrum of clinical presentations it causes. Importantly, this paper integrates traditional Ayurvedic concepts such as *Kantharoga* and *Visarpa* to provide a holistic understanding of the disease, focusing on the role of *Dosha*, *Dhatu*, and *Srotas* in its development. Ayurvedic management strategies, including *Shodhana* (purification), *Shamana* (pacification), and localized therapies, are explored. By combining modern microbiological insights with Ayurvedic perspectives, this synthesis aims to promote a more comprehensive and integrative approach to managing infections caused by S. pyogenes, potentially improving patient outcomes through holistic care.

## INTRODUCTION

*S. pyogenes* is a gram-positive, catalase-negative, oxidase-negative, β-hemolytic streptococci. It is a facultative anaerobe, grows best in 5 to 10% carbon dioxide, and forms pinpoint colonies on blood agar plates. The Lancefield serological grouping system is used to differentiate group A streptococci (GAS) from other streptococci.[1]

*Streptococcus pyogenes* commonly colonizes the pharynx, skin, and, less frequently, the anorectal and genital mucosa. Infections caused by this organism are highly contagious and can spread through multiple routes, including respiratory droplets, direct contact with infected nasal secretions, contaminated surfaces, or skin lesions. Transmission may also occur through contaminated food sources. The organism gains entry through breaches in the skin, such as abrasions or minor injuries, leading to localized infections like erysipelas and cellulitis. In more severe cases, it can invade deeper tissues, cause myositis and necrotizing fasciitis, often following trivial trauma, and may progress to streptococcal toxic shock syndrome. Additionally, *S. pyogenes* can infect the female genital tract, including the vaginal mucosa and uterus, potentially leading to septicemia.[1] Skin lesions are recognized as a major predisposing factor for invasive infections.[2] Furthermore, overcrowded environments such as schools, military camps, and nursing facilities facilitate rapid transmission, often resulting in outbreaks of Group A Streptococcal infections.

Despite the availability of effective antimicrobial therapy, S. pyogenes continues to pose a

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major public health concern worldwide due to its association with complications such as acute rheumatic fever and post-streptococcal glomerulonephritis, which contribute significantly to morbidity, especially in developing countries. The organism exhibits a wide array of virulence factors that facilitate adhesion, immune evasion, and tissue invasion, enabling it to produce diverse clinical manifestations.

In recent years, there has been renewed interest in understanding the epidemiology, pathogenic mechanisms, and evolving clinical patterns of GAS infections, along with efforts toward vaccine development. From an integrative perspective, correlating these conditions with Ayurvedic concepts such as *Kantharoga* and *Visarpa* provides additional insight into their pathophysiology and management.

This review aims to present a comprehensive and updated overview of Streptococcus pyogenes, encompassing its microbiology, pathogenesis, clinical features, diagnostic approaches, and treatment strategies, while also exploring its relevance in Ayurvedic medicine for holistic clinical application.

**Taxonomy and Classification**

*Streptococcus pyogenes* is a Gram-positive bacterium belonging to the genus *Streptococcus* within the family *Streptococcaceae*. Members of this genus are characterized by their spherical shape and typical arrangement in chains. *S. pyogenes* is classified under Lancefield Group A based on the presence of a group-specific carbohydrate antigen (Group A antigen) in its cell wall, which serves as an important basis for serological identification.[3]

Taxonomically, it is categorized as follows:

- **Domain:** Bacteria
- **Phylum:** Firmicutes
- **Class:** Bacilli
- **Order:** Lactobacillales
- **Family:** Streptococcaceae
- **Genus:** *Streptococcus*
- **Species:** *Streptococcus pyogenes*

Based on its hemolytic pattern on blood agar, *S. pyogenes* is classified as a β-hemolytic streptococcus, producing a clear zone of complete hemolysis around its colonies. Further classification is based on antigenic variation of the M protein (emm typing), which plays a crucial role in virulence and is widely used in epidemiological studies to differentiate strains and track outbreaks.[4,5]

**Virulence Factors and Pathogenesis**

*Streptococcus pyogenes* possesses a diverse array of virulence factors that enable it to adhere to host tissues, evade immune responses, invade deeper structures, and produce both localized and systemic disease. These factors include structural components as well as extracellular enzymes and toxins, all of which act in a coordinated manner to enhance pathogenicity.

The M protein is the most important virulence determinant of *S. pyogenes*. It is a surface-expressed protein that inhibits phagocytosis by interfering with complement activation and opsonization. Its marked antigenic variability (emm typing) allows the organism to evade host immune responses and contributes to strain diversity and recurrent infections.[6]

The hyaluronic acid capsule surrounds the bacterial cell and closely resembles host connective tissue, thereby providing a form of molecular mimicry that prevents immune recognition and phagocytosis.[7] Additionally, lipoteichoic acid (LTA) and other surface proteins facilitate adhesion of the organism to epithelial cells, particularly in the pharynx and skin, which represents the initial step in colonization.[7]

In addition to structural components, *S. pyogenes* produces several extracellular enzymes that promote tissue invasion and dissemination. Streptokinase activates plasminogen to plasmin, resulting in fibrin degradation and facilitating the spread of bacteria through tissues. Hyaluronidase, known as the "spreading factor," breaks down connective tissue, allowing deeper penetration of the organism. DNases degrade nucleic acids in pus, reducing viscosity and aiding in bacterial dissemination.[8]

The organism also secretes potent toxins that contribute to tissue damage and systemic manifestations. Streptolysin O (oxygen-labile) and streptolysin S (oxygen-stable) are responsible for hemolysis and cytotoxic effects. Streptolysin O is antigenic and forms the basis of the ASO titre used in the diagnosis of recent streptococcal infection. Furthermore, streptococcal pyrogenic exotoxins (Spe toxins) act as superantigens, leading to excessive T-cell activation and massive cytokine release. This results in clinical features such as fever, rash (as seen in scarlet fever), and severe conditions like streptococcal toxic shock syndrome.

The combined action of these virulence factors enables *S. pyogenes* to effectively colonize host tissues, evade immune defenses, and cause extensive tissue destruction. Moreover, molecular mimicry, particularly involving the M protein, plays a crucial role in the development of autoimmune complications such as acute rheumatic fever and post-streptococcal glomerulonephritis.[9]

**Clinical Manifestations**

*Streptococcus pyogenes* causes a wide range of diseases, which are broadly classified into suppurative and non-suppurative manifestations.

**Suppurative Infections**

These result from direct bacterial invasion and include pharyngitis, the most common presentation, characterized by fever, sore throat, and tonsillar exudates. Other manifestations include tonsillitis, impetigo (with honey-colored crusted lesions), and deeper skin infections such as cellulitis and erysipelas. Severe invasive forms include necrotizing fasciitis and streptococcal toxic shock syndrome (STSS), which are associated with high morbidity and mortality.[10]

**Non-Suppurative Complications**

These are immune-mediated sequelae occurring after infection. Acute rheumatic fever presents with carditis, arthritis, and other systemic features, while rheumatic heart disease results in chronic valvular damage. Post-streptococcal glomerulonephritis manifests with hematuria, edema, and hypertension.

**Invasive Manifestations**

In some cases, *S. pyogenes* may cause invasive infections such as bacteremia, sepsis, pneumonia, and puerperal sepsis, particularly in individuals with predisposing factors.[11]

**Group A Streptococcus is an obligate human pathogen**

Human pathogenicity is unique to group A streptococci as only humans are the natural reservoir for the bacterium, unlike groups B, C, and G streptococci, which also act as veterinary pathogens. Concerns on the life cycle and diseases produced by GAS natural infections in animals are not reported to be existent or other environmental reservoirs and biological means remain. Notably, GAS has been sporadically isolated from environmental samples such as canine faeces and conjunctive discharge [12] , as well as in association with a wild European hedgehog.[13,14] While humans are the only biological host of this pathogen, non-human primates can serve as models for GAS infection.

**Experimental Infection Models**

Although GAS does not naturally infect animals, researchers have developed experimental models to study its pathogenic mechanisms. These models are essential due to the human-specific nature of the pathogen.

Non-human primates, particularly Indian rhesus monkeys, have been used successfully to mimic GAS infections. When exposed to artificially high doses of virulent strains such as M1T1, these animals can develop diseases resembling human conditions like pharyngitis and tonsillitis.[15] This demonstrates that while animals are not natural hosts, they can replicate certain aspects of GAS infection under controlled conditions.

Additionally, laboratory animals such as mice (C57BL/6, BALB/c, FVB/NJ) and rabbits are widely used in research.[16,17] These models help in studying immune responses, virulence factors, and vaccine efficacy. However, they have inherent limitations because they do not fully reproduce the complex host–pathogen interactions observed in humans.

**Transmission and Survival**

GAS spreads primarily through person-to-person transmission via respiratory droplets, making close human contact a major risk factor.[1] This mode of transmission reflects its adaptation to the human respiratory tract.

The pathogen can survive on the skin and within the host for hours to several days, facilitating its spread.[18] Despite this temporary environmental persistence, GAS does not establish long-term survival outside the human host.

This human-dependent transmission cycle explains the high prevalence of GAS infections in crowded environments such as schools and communities.

**Adhesion and Colonization**

A critical step in GAS pathogenesis is its ability to adhere to and colonize human tissues, particularly the nasopharynx and skin.

Several bacterial components contribute to this process, including:

- Hyaluronic acid (HA) capsule
- Pili (fimbrial structures)
- M proteins
- Fibronectin-binding proteins such as SfbI

These structures enable GAS to interact with host tissues and establish infection sites.[19]

Adhesion occurs in a two-step process:

1. **Initial attachment**– Mediated by weak interactions involving lipoteichoic acid binding to epithelial cells via hydrophobic interactions.
2. **Firm attachment**– Involving high-affinity interactions such as protein–protein and lectin–carbohydrate binding mediated by pili and adhesins.[20]

This sequential mechanism ensures stable colonization of host tissues.

Host extracellular matrix (ECM) proteins such as collagen, fibronectin, fibrinogen, laminin, and vitronectin act as binding targets for GAS adhesins.[19]

Among these, fibronectin plays a particularly important role by bridging bacterial adhesins to host cell receptors such as integrins.

Following adhesion, GAS forms microcolonies, which are clusters of bacteria that grow and multiply, eventually leading to infection.

**Colonization Dynamics and Adaptation**

The upper respiratory tract provides a favorable environment for GAS growth, but it also presents competition from normal microbial flora. GAS must therefore compete for space and nutrients while evading host defenses.

Some GAS strains have the ability to invade host epithelial cells and survive intracellularly for 4–7 days.[21] This contributes to persistence and recurrent infections.

Encapsulation is a major determinant of colonization efficiency. Studies have shown that encapsulated GAS strains colonize more effectively than non-encapsulated strains.[22] The hyaluronic acid capsule protects the bacteria from phagocytosis by immune cells.

Interestingly, long-term colonization can lead to genetic changes, such as frameshift mutations in the hasA gene, resulting in reduced or absent capsule production.[23] This indicates that GAS can adapt its virulence mechanisms depending on environmental conditions.

The HA capsule also functions as a non-protein adhesin, binding to epithelial cells via CD44 receptors and enhancing colonization.[24]

**Regulation of Colonization**

The processes of adhesion and colonization are tightly regulated by bacterial genetic systems. Important regulatory components include:

- Control of virulence regulatory systems
- Multiple gene regulators
- RofA-like proteins

These systems control the expression of colonization factors and ensure that virulence determinants are expressed at optimal levels.[25]

Thus, colonization is a dynamic and highly regulated process, allowing GAS to adapt to changing host environments.

**Host Cell Invasion**

Beyond surface colonization, GAS has the ability to invade host cells, including epithelial and immune cells.

This phenomenon was first demonstrated in the 1990s, showing that GAS can behave as an intracellular pathogen.[26] The frequency of invasion is comparable to classical intracellular bacteria such as *Salmonella* and *Listeria*.[27]

**Key proteins involved in invasion include:**

- SfbI (fibronectin-binding protein)
- M proteins

These proteins facilitate entry into host cells by interacting with extracellular matrix components and triggering cytoskeletal rearrangements.[19]

Once inside the host cell, GAS can:

- Survive within intracellular compartments
- Escape from phagolysosomes
- Multiply within the cytoplasm

This intracellular survival allows the bacteria to evade immune detection and persist within the host.

A notable feature of GAS is its ability to "hide" within host cells, delaying immune recognition and allowing asymptomatic colonization.[28]

**Immune Evasion Mechanisms**

GAS employs multiple strategies to evade the host immune system:

- Inhibition of phagocytosis via M proteins and capsule
- Survival within macrophages and neutrophils
- Escape from phagolysosomes
- Modulation of immune responses

For example, GAS can survive within macrophages by escaping into the cytoplasm, a process mediated by streptolysin O.[29,30] Similar mechanisms have been observed in neutrophils.[31]

Clinical studies have shown that live GAS bacteria can be found within macrophages in infected tissues, with higher bacterial loads correlating with increased inflammation.[32]

These mechanisms enable GAS to persist within the host and contribute to disease severity.

**Dissemination Within the Host**

Recent research has revealed that GAS can spread through the lymphatic system, reaching local and distant lymph nodes.[33] During this process, the bacteria can remain extracellular.

This finding provides a new perspective on GAS pathogenesis, showing that dissemination does not always require intracellular survival.

Earlier studies had suggested bacterial movement through lymphatics[34], but recent work has clarified its role in GAS infection.

**Clinical Implications of Human Specificity**

The obligate human nature of GAS has several important implications:

- Epidemiology: Transmission is entirely human-based, making outbreaks dependent on human interaction.
- Pathogenesis: GAS is highly adapted to human tissues, leading to efficient colonization and infection.
- Research limitations: Lack of natural animal hosts complicates the development of accurate disease models.
- Vaccine development: Human-specific immune responses must be targeted, making vaccine design more complex.

**Knowledge Gaps**

Despite extensive research, several aspects remain unclear:

- Potential undiscovered environmental reservoirs
- Complete mechanisms of host specificity
- Long-term colonization dynamics

Further research is needed to address these gaps.

**Ayurvedic Perspective**

In Ayurveda, infections caused by *Streptococcus pyogenes* can be understood under disorders involving *Tridosha* vitiation with *Rakta Dushti*, presenting predominantly in the *Urdhvajatrugata* (upper respiratory tract) and *Twak* (skin) domains as presented in the clinical manifestation of this pathogen. Clinical entities such as pharyngitis and tonsillitis resemble *Kantharoga* (e.g., *Kanthashoola*, *Tundikeri*), while skin manifestations such as impetigo, cellulitis, and erysipelas resemble *Kshudra Kushtha*, *Pidaka*, and *Visarpa*.

***Samprapti* (Pathogenesis)**

The disease process begins with *Agnimandya*, leading to *Ama* formation, followed by the vitiation of the *Vata*, *Kapha,* and *Pitta Doshas*. These vitiated *Doshas* localize in *Rakta Dhatu* and susceptible sites such as the throat and skin, producing *Shotha* (inflammation), *Daha* (burning), *Raga* (erythema), and *Vedana* (pain). Rapidly spreading conditions like *Visarpa* correlate with invasive streptococcal infections due to their rapidly progressive nature.

A classical description highlighting its spread is: **"त्वङ्मांसशोणितगताः कुपितास्तु दोषाः सर्वाङ्गसारिणमिहास्थितमात्मलिङ्गम् | कुर्वन्ति विस्तृतमनुन्नतमाशु शोफं तं सर्वतो विसरणाच्च विसर्पमाहुः||३||"**[35] (*Sushruta Samhita, Nidana Sthana* 10th Chapter) -indicating the rapid spreading nature of *Visarpa*, comparable to invasive skin infections.

***Chikitsa Siddhanta* (Principles of Management)**

Management focuses on: basic principles of *Ayurveda*

- *Shodhana* (bio-purification): *Vamana* and *Virechana* in indicated patients
- *Shamana* (pacification): Use of *Tikta*, *Kashaya rasa* drugs for *Pitta–Rakta Shamana.*
- *Rakta Mokshana*: To correct vitiated blood.[36]
- *Krimighna Chikitsa*: Addressing microbial etiology

Commonly used drugs include *Haridra* (*Curcuma longa*), *Nimba* (*Azadirachta indica*), *Guduchi* (*Tinospora cordifolia*), and *Manjistha* (*Rubia cordifolia*) for their anti-inflammatory and antimicrobial properties.

***Sthanik Chikitsa* (Local Therapies)**

- *Gandusha/Kavala* (gargling): Useful in throat infections.
- *Pratisarana* (local application): In skin lesions
- *Lepa* (topical applications): For inflammatory skin conditions.

Thus, Ayurveda provides a holistic framework integrating *Dosha*, *Dhatu*, and *Srotas* involvement, which can complement modern management, especially in recurrent and inflammatory conditions.

**Table 1: Modern vs Ayurvedic Correlation**

| Modern Condition | Ayurvedic Correlation | *Dosha* Involvement | Key Features |
|---|---|---|---|
| Pharyngitis | *Kanthashoola / Tundikeri* | *Kapha + Pitta* | Pain, inflammation |
| Impetigo | *Pidaka / Kshudra Kushtha* | *Kapha + Rakta* | Skin lesions |
| Cellulitis | *Visarpa* | *Tridosha + Rakta* | Spreading inflammation |
| Sepsis | *Sannipata janya vyadhi* | *Tridosha* | Systemic involvement |

**Table 2: Treatment Overview**

| Aspect | Modern Medicine | Ayurvedic Approach |
|---|---|---|
| Etiological treatment | Penicillin, antibiotics | *Krimighna, Rakta shodhana* |
| Symptomatic relief | Antipyretics | *Shamana* therapy |
| Local treatment | None specific | *Gandusha, Pratisarana* |
| Prevention | Prophylactic antibiotics | *Rasayana*, immunity boosting |

## CONCLUSION

*Streptococcus pyogenes* remains a significant human pathogen due to its wide clinical spectrum, ranging from mild infections such as pharyngitis to severe invasive diseases and immune-mediated complications. Despite the availability of effective antibiotics, the burden of disease continues, particularly in developing countries, due to factors such as overcrowding, delayed diagnosis, and inadequate treatment.

A thorough understanding of its virulence mechanisms, clinical manifestations, and early diagnostic approaches is essential for effective management and prevention of complications like rheumatic fever and post-streptococcal glomerulonephritis.

From an integrative perspective, Ayurvedic concepts provide a holistic understanding of disease pathogenesis through the involvement of *Dosha*, *Dhatu*, and *Srotas*, along with individualized treatment approaches emphasizing *Shodhana*, *Shamana*, and *Rasayana* therapies. Such an approach may be beneficial in improving outcomes, particularly in recurrent and chronic conditions.

Future directions should focus on early detection, rational antibiotic use, improved public health measures, and ongoing vaccine research, along with exploring evidence-based integrative approaches combining modern and Ayurvedic medicine for comprehensive patient care.

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