Group A streptococci (streptococcus pyogenes):

Important features:

Streptococci are gram +ve cocci arranged in chains or pairs.

All streptococci are catalase- negative (whereas staphylococci are catalase- positive) / streptococci are smaller than staphylococci

to be able to tolerate oxygen reactive species, they contain superoxide dismutase

Classification of streptococci:

1- hemolytic reaction on blood agar

Beta hemolysis (complete hemolysis – clear zone)

alpha hemolysis (partial hemolysis – form a green zone/ Hb metabolism into biliverdin) gamma hemolysis (no hemolysis)

2- carbohydrates (polysaccharide) antigens (peptidoglycans)

Lancefield grouping → groups A, B, C, F and G are human pathogens

3- biochemical reactions

Group A beta hemolytic streptococci

Streptococcus pyogenes (GAS)

- ✓ The most prevalent of human bacterial pathogen
- ✓ GAS are exclusively human pathogens
- ✓ It causes a wide range of suppurative (pus forming infections in:
- 1- the respiratory tract (pharyngitis)
- 2-skin
- 3- soft tissue infections / toxin-associated reactions
- 4- Streptococci have serious hallmark POST infective IMMUNOLOGICAL reactions

Pathogenesis – virulence factors:

Not necessarily all of them present in every strain

Adhesion [+initiation of infection]:

Bronectin is recognized by **surface F protein** on S. pyogenes – facilitates internalization of bacteria into host cells + surface-exposed **lipoteichoic acid and M proteins**

Species	Lancefield Group	Typical Hemolysis	Diagnostic Features ¹
S. pyogenes	A	β	Bacitracin-sensitive
S. agalactiae	В	β	Bacitracin-resistant; hippurate hydrolyzed
E. faecalis	D	α or β or none	Growth in 6.5% NaCl ² Resistant to penicilling
S. bovis ³	D	α or none	No growth in 6.5% NaCl sensitive to penicilling
S. pneumoniae	NA ⁴	α	Bile-soluble; inhibited by optochin
Viridans group ⁵	NA	α	Not bile-soluble; not inhibited by optochin



M proteins

the most important virulence factor – if the strain doesn't have it, it is considered avirulent.

Provide GAS with the ability to resist phagocytosis

Prevent opsonization of C3b

immunogenic [can cause immunological reactions and is important in labs to confirm diagnosis]

Capsule

Not always present

Anti- phagocytic factors

Not immunogenic

C5a peptidase

Present in the surface of all strains Inactivates human C5a (chemoattractant of phagocytic cells)

Streptolysins

Hemolysins – streptolysin O (oxygen labile) (**immunogenic**) and streptolysin S (serum soluble) (**not immunogenic**) We use ASO to detect previous GAS infection [doesn't determine when]

Other virulence factors

SPE-A, SPE-B and SPE-C are pyrogenic (fever inducing) and erythrogenic (rash inducing) exotoxins.

These exotoxins are implicated in scarlet fever and toxic shock.

!nvasion/ escape factor

facilitates tissue invasion (spreading)

GAS secrete hyaluronidase to degrade hyaluronic acid, the ground substance of host connective tissue.

Streptokinase

DNase [neutrophil extracellular traps] immunogenic / skin infections

Transmission:

The most common route of entry of GAS is the upper respiratory tract

- > Skin infections -> direct and indirect contact
- Respiratory infection -> droplets + indirect [common object]

Normal flora in more than 10% of population [healthy carriers]

Clinical features:

Pharyngitis →

- ➤ This is the most common infection caused by streptococcus pyogenes [most common bacterial cause] / sore throat + pain from swallowing [odynophagia] + enlarged tonsils that may show patches of grey- white exudate + tonsillar/ retropharyngeal abscesses may develop → mastoiditis + meningitis.
- ➤ Despite the significant symptoms and clinical signs → differentiating streptococcal pharyngitis ('strep throat') from viral pharyngitis is impossible without microbiological or serological examination.

Scarlet fever →

Erythrogenic exotoxins

The rash develops within 1–2 days after the first symptoms of pharyngitis

strawberry tongue

Sandpaper-like rash [pathognomonic]

Poststreptococcal (nonsuppurative) diseases →

Acute glomerulonephritis

Occurs after 2-3 weeks after skin infection [M protein type 49 causes AGN most frequently]

the antigen- antibody complexes deposit in the glomerular Basement membrane. [type 3 hypersensitivity/ antigen-antibody complexes]

It can be prevented by early eradication of nephritogenic streptococci from skin colonization sites but not by administration of penicillin after the onset of symptoms. hypertension + edema + proteinuria [smoky urine] – most patients recover but some can proceed to renal failure

Diagnosis - anti- DNase B

Acute rheumatic fever

Approximately 2 weeks after a group A streptococcal infection—usually pharyngitis (opposite to AGN) —rheumatic fever can occur.

Fever, migratory polyarthritis, endocarditis, chorea, erythema marginatum

Type 2 hypersensitivity/ antibody + self antigens

Diagnosis – ASO titers

- ⇒ It is also an autoimmune disease, however, unlike post strep AGN, it is GREATLY exacerbated(made worse) by recurrence of streptococcal infections.
- ⇒ After a heart-damaging attack of rheumatic fever, reinfection must be prevented by long-term prophylaxis.

Laboratory diagnosis:

- Gram-stained smears are **useless** in streptococcal pharyngitis because viridans streptococci are members of the normal flora and cannot be visually distinguished from the pathogenic S. pyogenes.
- However, stained smears from skin lesions or wounds that reveal streptococci are diagnostic.
- Cultures 18 to 48 hours (beta hemolytic) (sensitive to bacitracin] gold standard
- Rapid tests antigen-antibody dependent
- Serologic ASO titers, anti-DNase B

Treatment:

Penicillin

erythromycin [in case of penicillin allergy]

✓ S. pyogenes is not resistant to penicillin.

Prevention:

There are no vaccines available against any of the streptococci except S. pneumoniae