Streptococci are Gram-positive cocci that grow in chains. Group A and B streptococci are two groups of beta-haemolytic streptococci. Group A streptococci are *Streptococcus pyogenes*, and Group B are *Streptococcus agalactiae*. Both may cause invasive disease. 

**Group A**

Group A streptococci (GAS) are a cause of many infectious diseases both invasive and non-invasive, and may also asymptptomatically colonise the nose, throat, vagina and rectum. Carriage in the throat is common and may follow from non-eradication of bacteria after infection.

The two most prominent infections with GAS, strep throat and scarlet fever, are non-invasive. If infection is not adequately treated these disorders can seed complications including rheumatic fever and endocarditis.

Over one third of invasive GAS diseases are skin and soft tissue infections, the most severe being necrotizing fasciitis. Bacteraemia without identified focus is the second most common manifestation of severe GAS disease. GAS can be isolated from the blood in over 70% of invasive GAS infections.

67% of all patients with invasive GAS disease have an underlying condition.

GAS meningitis is uncommon, with 50% of cases occurring in neonates. It may spread from a non-invasive infection such as otitis media but often has no obvious point of entry.

Streptococcal toxic shock syndrome (STSS) typically presents in people with pre-existing skin infections with *S. pyogenes*. These individuals often experience severe pain at the site of the skin infection, followed by rapid progression of classical sepsis symptoms including fever, hypotension, malaise and confusion. In contrast to TSS caused by *Staphylococcus*, streptococcal TSS less often involves a sunburn-like rash.
Group B

Group B streptococci (GBS) are common commensal bacteria in the vagina, cervix, rectum, perianal area, urethra and may also colonise the skin and pharynx\textsuperscript{10}.

GBS remains the most common cause of both meningitis and neonatal sepsis, causing greater than 40% of all early-onset infections\textsuperscript{11}. Neonatal GBS infection may present as meningitis, sepsis, pneumonia or focal infection. Early-onset infections are more likely to present as sepsis or pneumonia\textsuperscript{10}. Early-onset infection is most often caused by vertical transmission of commensal GBS from the mother’s genital tract (less commonly from the placenta).

Most cases of neonatal sepsis and meningitis, however, are late onset\textsuperscript{12}, where acquisition occurs after the first 72 hours of life. In these instances, infection is acquired from the environment (eg IV lines, hands of care workers in the hospital or at home). Risk factors for neonatal sepsis include low birth weight and prematurity.

GBS may also cause amniotic and endometrial infection in pregnant and postpartum women, which may then cause sepsis. Rarely, this may lead to meningitis\textsuperscript{13}. GBS amniotic infection may lead to intrauterine foetal death\textsuperscript{8}.

GBS infections in non-pregnant adults are increasing in the UK, but mostly occur in patients with underlying disease. The most common manifestations are bacteraemia and soft tissue infection such as cellulitis\textsuperscript{10}.

Detection

Detecting GAS and GBS by culture can be difficult, as other organisms may be present, depending on the sample type. Also, some GBS strains are non-haemolytic or hyperhaemolytic, causing confusion when trying to identify the pathogen\textsuperscript{14}. For both GAS and GBS, culture may take 48hrs, and is unreliable after antibiotic therapy has started, which may cause problems in the case of sepsis or meningitis. Latex agglutination and immunoassays are rapid but have lower sensitivity than culture.

DNA detection by polymerase chain reaction (PCR) has a higher sensitivity than culture and is more rapid. PCR also has the advantages of not being affected by the presence of other organisms, and it can still be used after antibiotic therapy has started\textsuperscript{15}.

At Micropathology Ltd we use single-round hot-start molecular amplification assays for GAS and GBS, targeting the $MF$ gene in GAS and the $cfb$ gene in GBS.
References


