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Please read this bit first

The HPCSA and the Med Tech Society have confirmed that this clinical case study, plus your routine review of your EQA reports from Thistle QA, should be documented as a "Journal Club" activity. This means that you must record those attending for CEU purposes. Thistle will **not** issue a certificate to cover these activities, nor send out "correct" answers to the CEU questions at the end of this case study.

The Thistle QA CEU No is: **MT00025.**

Each attendee should claim **THREE** CEU points for completing this Quality Control Journal Club exercise, and retain a copy of the relevant Thistle QA Participation Certificate as proof of registration on a Thistle QA EQA.

Cycle 22 Organism 2:

Streptococcus agalactiae

Fry first reported group B streptococci as human pathogens in 1938, and described three cases of fatal puerperal sepsis[1]. By the 1970s, group B streptococcus had become a predominant pathogen causing septicaemia and meningitis in neonates and infants younger than 3 months. Implementation of chemoprophylaxis in the mid-1990s and has been associated with a dramatic decrease in the incidence of early-onset disease in neonates and with significant decline in the incidence of invasive disease during pregnancy[2].

S. agalactiae is the species designation for streptococci belonging to Lancefield group B. Group B streptococci are facultative, Gram-positive cocci that grow on a variety of bacteriological media. Isolated colonies on sheep blood agar are 3 to 4 mm in diameter and grayish white. A narrow zone of beta-haemolysis surrounds the flat, somewhat mucoid colonies. One to 2 percent of strains are non-haemolytic. Definitive identification of group B streptococci is based on detection of group B-specific cell wall antigen common to all strains. A number of serologic methods have been developed for the detection of group B antigen. Latex agglutination is the most widely employed. Biochemical methods permit presumptive identification. They are resistant to bacitracin and trimthoprim-sulfamethoxazole. The production of the Camp factor, which is a thermostable extracellular protein that results in synergistic haemolysis on sheep blood agar with the beta-lysin of *Staphylococcus aureus*[3].

Group B streptococci have been isolated from genital or lower gastrointestinal tract cultures of pregnant and non-pregnant woman at rates ranging from 10% to 40%. The principal reservoir for group B streptococci is the lower gastrointestinal tract and supports the premise that genital colonization is probably acquired from the rectal site[4].

Mucus membrane colonization of newborns results from vertical transmission of the organism from the mother, either in utero by the ascending route or at the time of delivery. To cause disease, group B streptococci must colonize mucosal surfaces and then breach these surfaces to enter normally sterile sites such as the blood stream. The most common diseases caused by these organisms are neonatal meningitis and septicaemia[2].

The antimicrobial regimens recommended for treatment of group B streptococcal infections in infants and adults are ampicillin plus an aminoglycoside (gentamicin). Penicillin G is the drug of choice once the diagnosis is established[5].

References:

1. Fry RM. Fatal infections by haemolytic streptococcus group B. Lancet. 1938;**1**:199-201.
2. Schrag SJ, *et al*. Group B streptococcal disease in the era of intrapartum antibiotic prophylaxis. N Eng J Med. 2000;**342**:15-20.
3. Facklam RR, *et al.* Presumptive identification of group A, B and streptococci on agar plate medium. J Clin Microbiol. 1979;**9**:665-672.
4. Dillon HC, *et al.* Anorectal and vaginal carriage of group B streptococci during pregnancy. J. Infect Dis. 1982;**145**:794-799.
5. Fernandez M, *et al.* Antimicrobial susceptibilities of group B streptococci isolated between 1992 and 1996 from patients with bacteraemia and or meningitis. Antimicrob Agents Chemother. 1998;**42**:1517-1519.

Questions:

1. How will you isolate and identify a *Streptococcus agalactiae* (Group B *Streptococcus*)?
2. What is the main source/site of *S. agalactiae* in humans?
3. Why is it important to identify colonization by *S. agalactiae* in pregnant women?
4. What antibiotics should be used to treat infections caused by *S. agalactiae*?