MATERNAL AND NEONATAL GROUP B STREPTOCOCCAL BACTERIURIA

Pages with reference to book, From 201 To 202

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Group B Streptococci (Strept agalactiae) are the etiologic agents of bovine mastitis, and until recently were not believed to cause serious human diseases. However it has been established that Group B Streptococci (GBS) are present in the vaginal flora of approximately 25% women and have also been frequently isolated from rectal swabs and less frequently from throat and male urethra[1]. Colonies on sheep blood agar typically produce small zones of beta hemolysis. They are usually bacitrecin sensitive and give a positive response to CAMP test[2].

Lancofield Group B Streptococci cause skin infections, endocarditis, puerperal infections, neonatal septicemia and meningitis. Wound infections are most common in patients with diabetes mellitus and peripheral vascular disease. GBS are currently the commonest pathogen causing serious neonatal infections.[3] These organisms have special importance in female genital tract. They were present in 5 — 35% of cultures obtained from asymptomatic pregnant women[4] and 2 — 14% of women with endometritis[5].

Pasteur first observed those organisms in patients with puerperal fever and other investigators later reognized them as the commonest genital isolate in women with endocarditis[6]. The rate of harbouring this organism ranges from 4.6 — 9%[7,8]. In addition GBS commonly cause intrapartum infection (chorio-amnionitis). Infections with GBS also occur frequently in patients delivering preterm infants[6].

Certain reports have emphasized the opportunistic nature of Group B Streptococcal infections with diseases like diabetes mellitus, immunosuppression and urinary tract infections[7,9,10]. The association between diabetes mellitus and Group B Streptococcal infection is well known[7,10] Patients with diabetes are at greater risk for colonization or infection by GBS than others without this disease 11. GBS. is also a potential pathogen of urinary tract during pregnancy. Group B Streptococcal isolates were identified from urine specimens of patients with asymptomatic bacteriuria, cyst itis, and pyelone phritis[12].

Lancofield Group B Streptococci is the most common gram's positive bacterium causing neonatal meningitis and sepsis[13]. An increase in the incidence of neonatal Group B Streptococcal disease appeared in later 1950s and early 1960s[14].

There are two ways in which neonatal Group B Streptococcal disease may present clinically 1 The early onset form of disease may either be bacterimic or meningitic and tends to affect premature and low birth weight infants, and is often associated with prolonged labour after rupture of membranes. Late onset form of disease affects otherwise healthy infants between 2 — 4 weeks of age and is largely meningitic[14] .

Early onset Group B Streptococcal disease in the infants is acquired by vertical transmission from mothers colonized with GBS[15,16]. The presentation of early onset disease is mainly that of respiratory distress and shock. Mortality rate varies from 50 — 90%. Approximately half of the survivors with meningitis will have neurologic sequelae. Attack rate of early onset disease vary from 1- per 1000 live births[3]. Serotype of GBS, isolated from infants with early onset disease are identical to those isolated from genital tract of their mothers[16]. Adults rarely get severe infections and meningitis' due to GBS. Majority of infants with late onset disease have meningitis and mortality rate is approximately 20%. The route of infection of these infants is not clear; nosocomial acquisition of organisms is suggested[18].

Antibiotic sensitivity pattern of GBS are generally similar to Group A Streptococci. Penecillin G is the

drug of choice[19]. Majority of GBS strains are also sensitive to Erythromycin, Chloramphenicol, Cephalosporin,Lincomycin and Clindamycin[20].

Ptevention of neonatal disease by prophylactic administration of penicillin or by immunization is controversial[20]. Seigel et al. showed that prophylaxis with benzl penicillin (which is highly sensitive against GBS, crosses the placenta and thus protects the fetus directly) given at birth reduced the incidence of both colonization and infection with GBS. It was also suggested that mass prophylaxis might lead to the emergence of neonatal disease resistant to penicillin[2] 1 Administration of penicillin during labour appears to be the most efficacious method of preventing neonatal Group B Streptococcal Sepsis[21,22].

Yow et at achieved good results by using Ampidillin Sodium intravenously during labour arid delivery to women colonized with GBS. Intravenous administration is an effective method of preventing intrapartum transmission of GBS from mother to infant. Intravenous route is preferable to the oral route because of the rapid and reliable concentration of the drug in the serum, tissues and amniotic fluid. Treatment of colonized women by intravenous route at term is a suitable method of preventing GBS infections in neonates[16].

Antibiotic therapy alone seems to be of only limited value in early onset disease. The infected baby may be deficient in compliment and phagocytic function, in other words it is an immunocompromised host. Overwhelming GBS bacterimia is often associated with neutropenia. The combination of chemotherapy and immunotherapy (plasma and granulocyte transfusions) has been used successfully in the management of some immunocompromised patients[14].

The successful treatment of GBS diseases needs early diagnosis and effective therapy, and it is especially important to screen all the pregnant, especially diabetic pregnant, women for GBS bacterimia to minimize adverse maternal and neonatal consequences of infection.

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