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Neonatal Sepsis Remains an Enigmatic Area for Neonatologists Due to Epidemiology Changes

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Introduction

The incidence of neonatal sepsis or bacteria in asymptomatic infants is low, but not negligible. Voora reported a prevalence of fever in term new-borns with the febrile infants having culture-proven sepsis [1]. While term new-borns were described as being more likely to react to a bacterial infection with fever, preterm new-borns were more likely to react with hypothermia, because of transitional difficulty with temperature control especially in the first few days [2]. In contrast, the lack of clinical relevance of body temperature in diagnosing sepsis later in preterm infants might be attributable to the use of incubators. However, neonates with core body temperature elevation sustained for more, not due to environmental causes and greater than more likely to have bacteria, meningitis, pneumonia, and also associated with viral disease, particularly herpes simplex encephalitis and therefore evaluation should include lumber puncture. Respiratory distress including tachypnea, grunting, nasal flaring, and retraction of respiratory muscles can be the sole manifestation of sepsis with or without pneumonia and can be confused with transient tachypnea of new-born initially [3]. Rapid clinical deterioration ensues unless prompt antibiotic management is started in neonates with sepsis. Neonatal sepsis can be complicated by metastatic foci of infection, disseminated intravascular coagulation, congestive heart failure and shock. Necrotizing enterocolitis is an acute inflammatory necrosis of the bowel and may be the underlying cause of neonatal sepsis [4]. The probability of the latter is high when a neonate presents with gram-negative sepsis and has nonspecific intestinal and radiological signs. Chaaban reported neonates with nonspecific abdominal findings had positive blood cultures. With improved survival of preterm infants, late-onset sepsis has become an important cause of morbidity and mortality among low birth weight infants [5]. Late-onset sepsis is mainly associated with the organisms acquired from the environment after birth. In a study on infants admitted to National Institute of Child Health and Human Development Neonatal Research Network centres, first episode late-onset infections were caused by gram-positive organisms, with coagulase-negative staphylococci accounting for the infections. Death rates were highest for infants infected with Pseudomonas aeruginosa, Candida albicans, Serratia marcescens, and *E. coli* [6]. The incidence of late-onset Group B Streptococcal disease has remained unchanged despite intra-partum antibiotic prophylaxis. Meningitis remains a common presentation of late-onset Group B Streptococcal disease, with serious neurologic sequel and permanent impairment among many survivors [7]. Early onset Group B Streptococcal infection has a case fatality: a multistate active surveillance system demonstrated that early onset Group B Streptococcal infections resulted in death. Neonatal infection can occur when Group B Streptococcal ascends from the vagina to the amniotic fluid after the onset of labour or rupture of membranes [8]. It is more commonly the result of vertical transmission from mother to infant in women with recto-vaginal colonization. Colonization with Group B Streptococcal occurs in roughly pregnant woman in their vagina or rectum. The only intervention proven to decrease the incidence of early-onset neonatal Group B Streptococcal sepsis is maternal treatment with intra-partum intravenous antibiotics [9]. Erythromycin is no longer recommended for prophylaxis because of high resistance rates. In those women with a non-serious penicillin allergy, cefazolin is the drug of choice. For a mother with a history of life-threatening penicillin allergy, clindamycin is the substitute for penicillin, but should only be used if the recto-vaginal Group B Streptococcal isolate is tested and found to be susceptible [10]. If the clindamycin sensitivity is unknown or the Group B Streptococcal isolate is resistant to clindamycin, vancomycin is the substitute for prophylaxis.

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Conflict of Interest

None

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