



## Short Note on Neonatal Meningitis

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### DESCRIPTION

Neonatal meningitis is a catastrophic disease. Despite improved antibacterial therapy and increased indicators of suspicion among clinicians caring for affected babies, the prognosis has not improved in decades. One in ten babies die of meningitis, and up to half of the surviving babies develop lifelong complications such as seizures, hearing and visual problems, and basic skills such as speaking and walking.

At this time, it is not possible to predict which infant will suffer from bad results. Diagnosis of meningitis in infants is technically difficult, time consuming and invasive, but early treatment is important to achieve more favorable results. Extensive neuronal damage has long been described in association with neonatal meningitis, as the levels of many pro-inflammatory and anti-inflammatory cytokines are elevated. However, the mechanism of the host immune response that drives the clearance of invading organisms and underlies the brain damage caused by meningitis is not well understood. This describes challenges in the diagnosis, prognosis, and management of neonatal meningitis. With a view to fertile areas for further investigation, this setting highlights transcriptomics, proteomics, and metabolomics data that contribute to the proposed mechanism of inflammation and brain damage. Despite the development of rapid diagnosis of pathogens and new antibiotics, Neonatal Meningitis (NM) contributes to neonatal mortality and morbidity worldwide. Neonatal meningitis is an inflammation of the meninges during the first 28 days of life. It is classified as Early Onset Meningitis (EOM) or Late Onset Meningitis (LOM), depending on when it was diagnosed. In EOM, clinical features appear in the first few weeks of life. LOM occurs between the 8th and 28th days after birth. Incidence of bacterial meningococcal in newborns ranges from 0.25 to 1 per 1000 live births and occurs in 25% of newborns with bacteraemia. In developed countries, Group B *Streptococcus* (GBS) is the most common cause of bacterial meningitis, accounting for 50% of all cases. *Escherichia Coli* (*E. coli*) accounts for an additional 20%. Therefore, detection and treatment of maternal genitourinary infections is an important preventive strategy. In developing countries, Gram-negative bacilli such as *Klebsiella* and *E. coli* may be more common than GBS especially in LOM, stiffness can be more common than GBS. In addition, other organisms involved in causing meningitis include the genera *Enterobacter* and *Citrobacter*.

Gram-negative meningitis is often more severe and is associated with higher mortality and morbidity. Diagnosis of NM is based on both clinical symptoms and examination of Cerebrospinal Fluid (CSF). CSF culture is an excellent test for detecting meningitis. Assessing white blood cell count, glucose, and CSF protein levels can be diagnostic. For neonatal meningitis, we evaluated maternal and neonatal risk factors, clinical symptoms, pathogens, and neurological complications.

Often, only the findings typical of neonatal sepsis (temperature instability, shortness of breath, jaundice, and apnea) appear. CNS symptoms (lethargy, seizures [especially partial], vomiting, hypersensitivity) more specifically suggest neonatal bacterial meningitis. More specifically by diagnosis, there is what is known as paradoxical hypersensitivity. This is because parental hugging and comfort stimulates the newborn rather than comforting it (because the inflamed meningeal movements are painful). Inflated or complete fontanelle occurs in about 25%, and nuchal rigidity occurs in only 15%. The younger the patients are rarer these findings. Cranial nerve abnormalities, especially those that affect the 3<sup>rd</sup>, 6<sup>th</sup>, and 7<sup>th</sup> nerves, may also be present. Group B streptococcal meningitis (GBS meningitis) can occur in the first week of life, with early onset of neonatal sepsis and initially manifesting as a systemic disease with prominent respiratory symptoms. However, GBS meningitis has no previous obstetric or perinatal complications after this period (most commonly 3 months of age) and is a more specific sign of meningitis. Ventriculitis is often associated with neonatal bacterial meningitis, especially when caused by Gram-negative enteric bacilli. Organisms that cause meningitis with severe Ventriculitis, especially *Diversus* and *Cronobacter sakazaki* (formerly *Enterobacter sakazaki*) can cause cysts and abscesses. *Pseudomonas aeruginosa*, *Escherichia coli* K1, and *Serratia* species can also cause brain abscesses. Early clinical signs of a brain abscess are elevated Intracranial Pressure (ICP), often manifested by vomiting, tense fontanelle, and sometimes head enlargement. Deterioration of the otherwise stable neonate with meningitis suggests a progressive increase in ICP caused by an abscess or hydrocephalus, or a ruptured abscess into the ventricular system.

Treatment of Meningitis depends on the cause. Some babies with viral meningitis work well without treatment. However, if you suspect meningitis, take your baby to the doctor as soon as

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possible. Symptoms are similar to other symptoms, so you can't tell what's causing them until your doctor does some tests. If necessary, treatment should be started as soon as possible to get good results.

Despite improved neonatal intensive care and timely administration of appropriate broad-spectrum antibiotics, bacterial meningitis continues to be a major contributor to neonatal morbidity and mortality. Our understanding of the mechanism of neonatal brain damage from meningitis remains limited. The use of cytokines as biomarkers for meningitis has not achieved sufficient accuracy for use in clinical practice, but these data are from currently existing human and model organisms for bacterial meningitis. Efforts to

integrate these datasets and edit pathway maps of genes, proteins, and metabolites altered in bacterial meningitis will be of great help in advancing this area. To understand the many infiltrative and complex interactions between cell types present in the brain affected by bacterial meningitis, analysis of primary human tissues (such as CSF) and targeted studies in animal models. A combined subsequent approach is needed. Such studies can advance the development of new and rapid diagnostic and adjuvant therapies that can prevent the catastrophic lifelong consequences of bacterial meningitis in infants.