ISSN: 2581 – 3633
PubMed - National Library of Medicine - ID: 101745081

International Journal of Medical Science and Advanced Clinical Research (IJMACR)

Available Online at:www.ijmacr.com

Volume – 8, Issue – 5, September - 2025, Page No.: 75 – 80

Neonatal Meningitis Presenting As Abdominal Distension: A Case Report

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How to citation this article: Dr. Kirankumar Haridas, Dr. Kala Shanker Dussa, Dr. Manga Balakrishna, Dr. Polasa Ramesh, "Neonatal Meningitis Presenting As Abdominal Distension: A Case Report", IJMACR- September - 2025, Volume – 8, Issue - 5, P. No. 75 – 80.

Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract

Neonatal meningitis is the common cause of morbidity, mortality and long term sequel in newborns. The incidence ranges from 0.25-0.32 per 1000 live births. The diagnosis remains challenging as the clinical feature are varied and nonspecific in most of the cases. They present as lethargy, decreased feeding ability and nonspecific lab abnormalities. The clinical indicator which points towards meningeal involvement is presence of seizures. Any newborn with seizures should undergo lumbar puncture to rule out presence of meningitis. Lumbar puncture remains gold standard in resource poor settings for identifying the presence of meningitis in suspected cases. The blood and CSF culture and sensitivity should be done to identify the causative organism and to institute specific antimicrobial therapy. Early identification of neonatal meningitis and its treatment forms the mainstay in preventing the morbidity and mortality of the disease in neonates.

Keywords: Antimicrobial Therapy, Caesarian**,** Global Mortality Neonatal Meningitis.

Introduction

Neonatal sepsis and meningitis are major contributors to morbidity and mortality during the neonatal period, accounting for substantial health burdens globally, particularly in developing countries.[1] EOS can present as sepsis, pneumonia and meningitis in newborns.[2] Despite advances in neonatal intensive care, bacterial meningitis

continues to have devastating outcomes in 20 to 60% among its survivors. [3]. Global mortality estimates are approximately 190,000 cases per year.[4]

The incidence and mortality have declined over the past few decades, it remains challenging to diagnose due to pathogens varying with gestational age at birth, age at presentation, and geographic location, the often subtleness of clinical presentation, and inconsistent findings on clinical examination.[5] We are reporting a case of premature newborn with early onset meningitis presenting with abdominal distension which was admitted in our unit.

Case report

A 36^{+5} week baby is delivered to a primigravida mother via emergency caesarian section on 18/05/23 at 7:51am, indication being decreased fetal movements. Baby cried immediately after birth with an APGAR score of 8 and 9 at 1 and 5 min of life, weighing 2490gms. Soon after birth baby developed respiratory distress for which baby was shifted to NICU and kept on CPAP. Baby showed improvement in respiratory distress with decreasing Downes score and baby was weaned off from CPAP after 12 hours. Baby was not started on feeds initially as the abdomen was distended and tense. Even after weaning off from CPAP the distension persisted which ruled out the suspicion of CPAP belly. The baby was also lethargic and had decreased bowel sounds on auscultation. The first line antibiotics were started on suspicion of early onset sepsis (Gastrointestinal sepsis) after collecting the required samples and baby was continued nil per orally. Initial investigations did not reveal the presence of sepsis. The baby continued to have abdominal distension, hence feeds were not started. Repeat sepsis screening was sent after 24hours which showed elevated WBC and CRP suggesting presence of infection.

The antibiotics were continued, but baby did not show clinical improvement and continued to have persistent dull activity in the range of severe stupor to coma. Hence antibiotics were upgraded according to unit policy. On day of life 5 baby had multiple episodes of apnea which was associated with tachycardia and desaturation. The episodes were suggestive of convulsions for which baby was loaded with antiepileptic medication. The presence of convulsions made us to suspect meningitis, for which the antibiotics were immediately upgraded to meropenem and vancomycin, pending CSF examination, based on the unit policy. Later on the same day lumbar puncture (LP) examination performed, revealed high protein of 170mg/dl and lower sugars of 12mg/dl with elevated white blood cell (WBC) counts of 62 with neutrophil predominance, confirming the diagnosis of meningitis. The blood culture showed growth of Acinetobacter baumannii which was sensitive to cotrimoxazole. CSF culture was negative for growth of microorganism. Cotrimoxazole was added to the antibiotic regimen and meropenem was continued along, as the baby showed improvement. During the same period baby also had deranged prothrombin (PT) and activated partial thomboplastin (APTT) time requiring one unit of FFP transfusion.

Baby showed improvement in the level of activity on day 10 of life and abdominal distension was decreased on day 11 of life. Feeds were started on day 11 with trophic feeds, which baby tolerated well. The feeds were then increased and reached to full feeds by day 16 of life (150ml/kg/day). Later the baby was started on palladai feeds on day 14 of life. Direct breastfeeds were started

on day 20 of life. The baby was also started on early stimulation therapy and was taught to mother as well.

The head circumference was monitored weekly during the hospital stay which showed adequate growth. The MRI at discharge showed T1 hyperintensities in frontal, parietal and occipital cortex. Baby was examined neurologically on daily basis which showed no abnormality. OAE was normal at discharge. ROP screening showed zone 3 immature retina in both eyes. Baby was discharged after 28 days of hospital stay with no neurological deficits on examination at discharged.

Discussion

Globally, the reported incidence of newborn meningitis is significantly greater in underdeveloped regions, ranging from 0.8 to 6.1 per 1000 live births, while the estimated incidence in industrialized countries varies between 0.25 and 0.32 per 1000 live births. Approximately one-third of neonates with clinical sepsis are likely to develop meningitis in these regions, underscoring the critical necessity for prompt diagnosis and appropriate management strategies.[6] In a multicenter study conducted in India found that the meningitis was present in 200 (1·5%) of neonates included in the study. The most frequent organisms causing neonatal meningitis was found to be Gram-negative pathogens, the most common being Acinetobacter spp, Klebsiella spp, Escherichia coli, Pseudomonas spp, and Enterobacter spp. The predominant Gram-positive pathogens were coagulase-negative staphylococcus (CONS), Staphylococcus aureus, and Enterococcus spp. Group B streptococci were isolated in only a few neonates.[7]

The clinical presentation may vary based on the birth weight and gestational age at birth. Nonspecific findings of temperature instability, lethargy, feeding intolerance, and poor perfusion have been reported as the most common presenting signs.[8,9] The most common signs in neonates weighing over 2500 grams include fever, irritability, seizures, and bulging fontanels. In contrast, apnea, jaundice, and abdominal distention are most common in those weighing less than 2500 grams.[9] The presenting clinical features may differ in term and preterm infants. The common features of meningitis in term born newborn include convulsions, irritability and feeding intolerance. Preterm infants will often have apnea, bradycardia, and cyanosis as the first sign of infection.[10]

Classical findings such as convulsions, bulging fontanels, coma, and neck stiffness were found in 28%, 22%, 6%, and 3% of cases in a descriptive study conducted by okike et al.[8] A study by Lim et al. reported a high incidence of poor activity and respiratory distress in preterm infants.[10] The case fatality rate in neonates with sepsis is also high. The mortality due to gram-negative pathogens is 59% and that of neonates infected with Gram-positive pathogens is 33%. Neonates infected with Pseudomonas spp had the highest case fatality rate.[7]

All newborns with sepsis or bacteremia should have a LP, according to the 2012 AAP Committee on Fetus and Newborn.[11] The gold standard for diagnosing newborn meningitis is LP with cultures, whether or not the CSF has molecular diagnostics. As of now, all neonates with confirmed or suspected sepsis should have an LP performed.[5]

Yikilmaz et al[12] recommended a neurosonographic evaluation of every infant with evidence of meningitis. Magnetic resonance imaging (MRI) of the brain is the recommended follow-up study in stable patients. This is to identify organic complications of the infection.[12]

PCR panels are a dependable method for identifying CSF infections that are culture-negative, particularly in infants who were given antibiotics prior to LP. Even after starting antibiotics, the RT PCR had a greater detection rate than standard cultures (72% vs. 48%).[5]

Treatment

The presence of MDR organisms have made the empiric antibiotic of choice difficult. In multicenter DeNIS trial which was conducted in India**,** the most common organism Acinetobacter baumanii was found to be resistant to cephalosporins and carbapenems. Other gram negative organism was also found to be MDR to more than two class of drugs. These findings alarm the treating clinician in finding the appropriate empiric antibiotics. The empiric antibiotic should be selected based on the unit's culture and sensitivity reports which results in maximum chance of better response.[7]

Recommendations for empiric therapy for neonatal meningitis vary by geographic region, local resistance patterns, and expert opinion. For most patients with suspected early-onset neonatal meningitis, ampicillin plus an aminoglycoside or an expanded spectrum cephalosporin should be started empirically. In late-onset meningitis, vancomycin should be added to the above empiric regimen when the suspicion of nosocomial pathogens is high.[5] Definitive therapy should be instituted when the causative organism and its susceptibilities have been determined. The duration of antibiotic treatment for newborn meningitis cannot be determined from the available data. According to European criteria, infections caused by L. monocytogenes and GBS must last at least 14 days, whereas infections caused by gram-negative organisms must last at least 21 days.[14] Some specialists advise that if there is clinical improvement, there is no need to repeat the LP to demonstrate CSF clearance.[5] Others recommend ensuring CSF pathogen clearance after 2 to 3 weeks of continued antibiotic therapy.[14] In standard practice, it is not advised to use intraventricular antibiotics, dexamethasone, intravenous immunoglobulins, granulocyte or granulocyte-macrophage colony-stimulating factor, or oral glycerol.[5]

A study from Tunisia revealed a neurologic complication rate of 21.6% in neonatal meningitis cases.[15] Neonatal meningitis can result in a number of problems, such as multiple small artery thrombi, subdural empyema, intracranial abscesses, parameningeal abscesses, and obstructive ventriculitis that causes hydrocephalus. Concerns about meningitis consequences should be raised by persistent bacteremia or CSF infection.[12,14] Hearing loss is a long-term consequence of neonatal meningitis.[4]

The mortality associated with meningitis in neonates has decreased drastically with appropriate antibiotic treatment but it continues to have high morbidity.[4] In low-income countries, the mortality rate is as high as 58%, with moderate to severe neurodevelopmental impairment in approximately 23% of survivors.[16] In high-income countries, the rate of neurological squeal is 20 to 50%.[4,17]

Conclusion

Our case was unique in that the initial presentation of abdominal distension was suspected of having a simple feed intolerance is turned out to be a case of meningitis. The presence of abdominal distension in most cases is considered as the feed intolerance or gastrointestinal sepsis as the first possibility by the treating clinician. This correlation was not present in our case, though our baby had abdominal distension initially but later turned out to be a case of meningitis which responded well to

appropriate antibiotics. Hence we suggest to keep a high index of suspicion for meningitis when the premature infants' presents with abdominal distension and dull activity during early postnatal period.

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