International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Index Copernicus Value (2013): 6.14 | Impact Factor (2014): 5.611

A Case Statement of Meningococcal Infection in a Neonate

Gjovalin Valca[1], Enkelejda Shkurti[2]

Abstract: ***Early beginning and tardy onset types of meningococcal sepsis in neonates have been described while neonatal meningococcal meningitis (NMM) is atypical. The result of meningococcal illness can be critical and depends on the native immune system, age, serogroups, pre-existing antibodies, and other unidentified host issues. The presentation of NMM varies from that in kids and youngsters and may be characterized with fever, poor nourishment, diminished activity, spasms, distorted perception and respiratory suffering. Punctual detection and commencement of antibiotics is decisive to survival. In the literature, very a small number of cases of neonatal meningococcal disease have been described in the United States. The regular annual occurrence of meningococcal meningitis in neonates is very small in contrast with the incidence of group B streptococcal meningitis. We represent the little acknowledged case of neonatal meningococcal meningitis in Albania. In addition, we present a review of the accessible literature.***

Keywords: meningoccocal infection, neonate, Albania

1. Introduction

Meningococcal meningitis is a uncommon disease in the primary 4 weeks of life. The contributing cause is *Neisseria meningitidis*, an encapsulated gram-negative, aerobic, intracellular diplococcus. The spectrum of illness varies from mild fever to septic shock, purpura fulminans, coma, and death.

In 1916 it was observed the initial case of neonatal meningo-coccal meningitis (NMM) in a 3-day-old baby born subsequent to a prolonged effort. The baby lived, but afterward expanded hydrocephalus. Since 1916, there have been 48 cases of NMM made obvious (Koplik H et al 1916). The occurrence of NMM varies from 0.8 to 1.3 per 100,000 in the US inhabitants (Granoff DM 2004). We represent what is, to the top of our comprehension, the newest acknowledged case of NMM in the Albania. This subject was presented with fever and rash, developed quickly to purpura, unbalanced septic shock, various organ dysfunction syndrome and died within 12 hours of appearance.

2. Case Presentation

A 40-week male, with a labor weight of 3380 grams, was born in a the Gynecology Obstethric hospital by standard natural vaginal vertex delivery to a 27-year-old female with 1 earlier pregnancy ensuing in live labor. Her prenatal screening analysis (involving gonorrhea, syphilis, rubella, group B streptococcus, and complete blood count) were ordinary. Her delivery was regular and simple, enduring 12 hours. She got a dose of intravenous ceftriaxone after her postpartum settle for fever. The baby's Apgar results were 7 and 8 at labor, and he was released to his house in 24 hours of birth.

The baby's hereditary screen was regular. He had a standard assessment 5 days after release. His one year old brother had right foot cellulites and cold signs that were cured with cephalexin at the period of this baby's disease.

The child's mother called the pediatric unit, reporting a fever of 39ºC, petulance, rash, reduced nourishing, complaining and weeping for the precedent 3 hours. She stated that the baby had been healthy until that morning, when he would cry at nourishing efforts. She explained the rash as restricted to the abdomen, with erratically sized pink to dark pink marks. No respiratory difficulties were observed. She was counseled by the pediatric care unit to get the baby to the close emergency department (ED).

The mother had gone to the ED with the 10-day-old kid an hour of her call to the nurse line. The kid had a temperature of 39 ºC, and the rash had extended to his extremities, with petechial injuries in the groin that were purple to black in color. He seemed visibly sick, looking pale and tired with some moaning. On physical assessment, his frontal fontanel was soft and open, but hollow; his eyes looked drawn. His lungs were lucid without whispers or rhonchus. His reaction to aching stimulus was reduced. They gave him acetaminophen that decreased his fever to 37.30ºC. An entire blood count confirmed a white blood cell (WBC) of 3800/uL, with 52% lymphocytes, 12% bands, 23% segmented neutrophils, and bunch platelets. Erythrocyte sedimentation rate, C-reactive protein, and electrolytes were normal. A chest radiograph divulged no acute pulmonary development and **a** standard cardio thymic outline. Blood and urine cultures were acquired, but they finally returned with no enlargement.

3. Results

On entrance to the pediatric floor, the baby seemed obviously sick, was stated as flaccid and hypotonic with complete capillary refill time and purpuric damage covering his body. Subsequent to lumbar puncture, in one hour of entrance it was offered Ampicillin (200 mg/kg/day), gentamicin (5 mg/kg/day), and acyclovir (60 mg/kg/day). He got a 30 cc standard saline bolus to cure hypotension (blood pressure 52/31 mmHg) and was removed to the pediatric intensive care unit (PICU) because of his serious situation. A communicable disease expert was consulted, and the antibiotic treatment was modified to ceftriaxone and linezolid. Due to respiratory malfunction, he was intubated 3 hours upon entrance in the PICU. He began to worsen, and the initial cardiac arrest arised 3 hours upon PICU access.

Laboratory outcomes disclosed distributed intravascular coagulation (DIC) with D-dimer 4.00 µg/dL, fibrinogen 40 mg/dL, platelets 43500/ml and harsh metabolic acidosis with lactate >15 mmoL/L and potassium 7.4 mmoL/L. Cerebral spinal fluid (CSF) assessment demonstrated 3 WBCs/uL, 13 red blood cells/uL, total protein 82 mg/dL, and glucose 39 mg/dL. Primary gram stain of the CSF confirmed a small number of neutrophils and no microorganisms, but it consequently cultivated *N meningitides*. Successive blood and viral cultures were negative. Regardless of numerous boluses of saline, new frozen plasma, and combinations of dopamine and epinephrine, the baby depart this life 11 hours following his mother's ring to pediatric care unit and 8 hours after hospital entrance.

4. Discussion

While the occurrence of meningococcal disease is moderately elevated in the primary 2 years of life in relation to further age ranges, but the frequency in the month of life is very short. From 1990 to 2005, there were 3400 deaths because of meningococcal illness reported in the United States, with the utmost disease mortality rate stated in patients under 2 years of age (Sharip A & Sorvillo F 2006). With the application of antibiotics, the rate of problems has reduced to 14% (Chiu CH, Lin TY 1994). In 2003, a statement (Shepard C 2003) of population-based supervision records in the United States from 1990 to 1999 established an elevated occurrence rate of neonatal meningococcal illness than earlier expected, but a rate close to that set up in patients aged 6 to 24 months.

Regarding the literature reports, the case fatality rate is no less than 52 % in those who came out with harsh purpura, MODS, and/or DIC (Bhutta ZA 1991). Whereas the hurdle rate may be diminished with the application of antibiotics, the frequency of meningococcal disease has not altered.

The medical spectrum and creatures that cause neonatal meningitis vary in babies from elder kids and adults. *Neisseria meningitidis*, a gram negative, put in a nutshell, is the contributory creature for meningococcal meningitis. The serotypes in charge for neonatal meningitis may be B, C, Y serotypes. There are so few cases that virulence models are unattainable to conclude; though, serotype B was set up to be the main frequent reason of meningitis in all age groups (Nizet V, Klein JO 2011). Although *N meningitidis* usually causes sepsis and meningitis in kids and teenagers, it hardly ever is correlated with insidious disease in neonates. Principal reasons of neonatal septicemia and meningitis are group B streptococci, *Escherichia coli*. These pathogens usually settle the maternal rectovaginal region and are therefore mainly connected with neonatal contagion. Even if *N meningitidis* may as well inhabit the woman genital area, it does so with much less incidence, and is consequently less frequently a source for neonatal disease (Granoff DM 2004; Nizet V, Klein JO 2011)

The main common outlines of meningococcal illness are meningitis and meningococcemia. The moment from the beginning of fever until decease in harsh meningococcemia is regularly as tiny as 12 hours (Huang HR et al 2006). Meningitis may firstly present with fever, bad temper, reduced nourishing, or deprived activity with or exclusive of meningeal symptoms. Even if the maculopapular inflammation is the characteristic indication of meningococcal disease, it is noted in simply 9 % of cases.4 The rash may quickly develop into important petechiae and purpura and may advance to purpura fulminate, a necrosis of the skin because of thrombosis. Meningococcemia is a fulminate outline of sepsis characterized by harsh septic shock and acidosis. Regardless of fast analytic testing, antibiotic cure and common sustain care in the PICU, mortality rates stay elevated. Subjects with purpura fulminans, shock, acidosis, hyperpyrexia, DIC, and positive blood culture have a very deprived prediction, and the majority of deaths arise in 24 to 48 hours of hospitalization(Chiu CH, Lin TY et al 1994). Meningitis can be complicated by cerebral abscess, disruptive hydrocephalus and ventriculitis. The main common neurological outcome is deafness.

Diagnosis is made by isolation of *N meningitidis* from normally sterilized body liquids.4 Spinal liquid cultures may be positive exclusive of pleocytosis, as illustrated in our patient(Malley R, Inkelis SH 1998). Subjects with CNS disease without CSF pleocytosis are at considerably elevated danger of unfavorable results like death and limb failure than meningococcal bacteremia alone. Malley, R, Inkelis SH 1998). Lack of CSF pleocytosis can be judged as a prognostic issue (Malley, R, Inkelis SH Penicillin G or cefotaxime stay the primary treatments of choice (Pickering LK 2012). The period of treatment depends on the patient's medical reaction, but a minimum of 10 days is designated for a neonate. Before release, a brain sonogram should be done to evaluate for encephalomalacia, while neonatal meningitis can have intense inferences for a kid's neurodevelopment. Recurrent outcome comprise deafness, hydrocephalus, language confusion, and mental and motor disabilities. Due to the small occurrence of this fastidious organism, it is hard to explicitly establish if there is relatively inclination for creating each of the ordinary sequel (Nizet V, Klein JO 2011). Important outcomes expand in up to 60% of babies in existence. To facilitate the prognosis, it is essential that a repeat LP be done at the conclusion of healing to reveal that the CSF is definitely sterilized, and that imaging be made for the presence or lack of abscesses and/or thrombosis. The subject should be pursued for lasting problems with developmental, neurologic, and hearing assessments.

5. Conclusion

Meningococcal illness should be taken into account in the differential analysis of rash in the neonate, particularly when rash is accompanied by other symptoms of disease such as fever, deprived hunger, and atypical look. Development of the syndrome is more quick than in other sorts of meningitis, and the primary 24 to 48 hours are serious.

Fast detection of meningococcal illness, along with antibiotic cure and helpful care are the keys to successful management of persistent meningococcal infection. In the majority of patients, the initial symptom of disease is fever that can be pursued by reduced appetite, nausea, and queasiness. Nevertheless, some researchers have discovered

that the primary signs of sepsis are fever, atypical skin color, cold hands and feet, leg ache, and dehydration.

References

[1] Koplik H. 1916. Meningitis in newborn and in infants under three months of age. *Arch Pediatr*;33(7):481–500.
[2] Granoff DM, Gilsdorf JR 2004. Neisseria meningitidis (Meningococcus). In: Kliegman RM, Behrman RE, Jenson HB, Stanton BM. *Nelson Textbook of Pediatrics.* 19th ed. Philadelphia, PA: Saunders; 929–935.
[3] Sharip A, Sorvillo F, Redelings MD, Mascola L, Wise M, Nguyen DM 2006. Population-based analysis of meningococcal disease mortality in the United States: 1990–2002. *Pediatr Infect Dis J* ; 25(3):191–194.
[4] Chiu CH, Lin TY, Yang PH, Hwang MS 1994. Neonatal meningococcal meningitis: report of two cases. *Zhonghua Min Guo Xiao Er Ke Yi Xue Za Zhi.*;35(6):542–545.
[5] Shepard C, Rosenstein N, Fischer M; 2003.Active Bacterial Core Surveillance Team. Neonatal meningococcal disease in the United States, 1990 to 1999. *Pediatr Infect Dis J*;22(5):418–422.
[6] Falcão MC, Andrade SB, Ceccon ME, Costa Vaz FA 2007. Neonatal sepsis and meningitis caused by Neisseria meningitidis: a case report. *Rev Inst Med Trop Sao Paulo*;49(3):191–194.
[7] Chiu CH, Lin TY, Huang YC 1995. Cranial nerve palsy and cerebral infarction in a young infant with meningococcal meningitis. *Scand J Infect Dis* ;27(1):75–76.
[8] Huang HR, Chen HL, Chu SM 2006. Clinical spectrum of meningococcal infection in infants younger than six months of age. *Chang Gung Med J*;29(1):107–113.
[9] Tinsa F, Jallouli M, Ben Lassouad M, Smaoui H, Brini I, Bousseta K, Bousnina S 2008. Neonatal meningitis by Neisseria meningitidis B. *Tunis Med.*;86(11):1014–1015.
[10] Bhutta ZA, Khan IA, Agha Z 1991.Fatal intrauterine meningococcal infection. *Pediatr* Infect Dis J;10(11):868–869.
[11] Nizet V, Klein JO 2011. Bacterial sepsis and meningitis. In: Remington JS, Klein JO, Wilson CB, Nizet V, Maldonado Y, eds. *Infectious Diseases of the Fetus and Newborn Infant.* 7th ed. Philadelphia, PA: Elsevier; 222–275.
[12] Malley R, Inkelis SH, Coelho P, Huskins WC, Kuppermann N 1998. Cerebrospinal fluid pleocytosis and prognosis in invasive meningococcal disease in children. *Pediatr Infect Dis J*;17(10):855–859.
[13] Pickering LK, Baker CJ, Kimberlin DW, Long SS 2012. *Red Book: Report of the Committee on Infectious Disease.* 29th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2012.
[14] Malley, R, Inkelis SH, Coelho P, Huskins WC, Kuppermann N 1998. Cerebrospinal fluid pleocytosis and prognosis in invasive meningococcal disease in children. *Pediatr Infect Dis J*;17(10):855-859.