CASE REPORT

Subdural effusion in a case of meningococcemia

Heera Hassan, Reena John, K Prithi Nair

Department of Microbiology, Government Medical College, Thrissur, Kerala, India

ABSTRACT

Neisseria meningitidis is one of the primary pathogens of pyogenic meningitis and has the potential to cause large epidemics. There are 13 serogroups of *N. meningitidis* that have been identified, six of which (A, B, C, W, X and Y) can cause epidemics. We report a case of acute pyogenic meningitis caused by *N. meningitidis* in a six-month-old male child who presented with fever of four-day duration and irritable cry for one day. *N. meningitidis* was isolated from his blood using automated blood culture system. Latex agglutination test for *N. meningitidis* antigen was positive from cerebrospinal fluid. The baby responded well to Ceftriaxone but developed subdural effusion.

Key words: Acute pyogenic meningitis, latex agglutination test, meningococcemia, *Neisseria meningitidis*

INTRODUCTION

Meningococcal meningitis is a life-threatening condition caused by intracellular Gram-negative diplococci, *Neisseria meningitidis*. Meningococcal meningitis cases occur throughout the world. However, large recurring epidemics affect an extensive region of Sub-Saharan Africa known as the 'meningitis belt'.[1] It is spread by person-to-person contact through respiratory droplets of infected people. *N. meningitidis* inhabits the mucosal membrane of the nose and throat. Up to 5%–10% of a population may be asymptomatic carriers. These carriers are crucial to the spread of the disease as most cases are acquired through exposure to asymptomatic carriers. The highest incidence of cases occurs in children less than four years old. There is also a 'meningococcal season' with 60%–65% of cases occurring in the first four months of the year.[2] The onset of symptoms is sudden and death can follow within hours. The incubation period averages three to four days, during which the person is infective. Meningococci colonise the upper respiratory tract and the non-ciliated mucosal cells internalise them. They traverse the mucosal cells, enter the submucosa and in approximately 10%–20% cases make their way to the bloodstream. In the vascular compartment, they may be killed by the bactericidal antibodies, complement and phagocytic cells or may multiply and initiate bacteraemic phase. The bacteria may seed local sites such as meninges, joints or pericardium and/or multiply in the bloodstream, causing meningococcemia with or without petechiae. Neurological complications include seizures, raised intracranial tension and hydrocephalus, and late complications include communicating hydrocephalus, subdural effusions in children and deafness. The fatality with treatment is 13% and without treatment is 50%–90%. Sequelae are seen in 3%–4% of survivors mostly in children.[3]

Access this article online

Quick Response Code:

Website: www.jacmjournal.org

DOI: 10.4103/0972-1282.194947

CASE REPORT

A previously healthy six-month-old male infant from Palakkad was brought to Government Medical College, Thrissur, Kerala, with a history of fever and nasal discharge of four-day duration and irritable cry of one-day duration. He had occasional cough. The child developed an episode of seizure when being admitted to the casualty. He had an uneventful history. He was fully immunised according to the Universal Immunization Schedule.

Address for correspondence: Dr. Heera Hassan, E-mail: drheerahassan@gmail.com

How to cite this article: Hassan H, John R, Nair KP. Subdural effusion in a case of meningococcemia. J Acad Clin Microbiol 2016;18:121-3.

The perinatal history of mother was uneventful. There was no developmental delay for the child. There was a contact history of fever in a four-year-old sibling attending Anganwadi four days before the onset of fever in the infant.

On examination, pulse rate was 148/min and respiratory rate was 38/min. The body temperature was 100°F. The anterior fontanelle was tense and pulsating, and there were no rashes. Examinations of the central nervous system were within normal limits. The blood investigations and cerebrospinal fluid (CSF) for culture and sensitivity were sent.

Blood haemogram revealed haemoglobin of 10 g%; total white blood cells count of 14,200 cells/mm^3 with polymorphs of 55%, lymphocytes 39% and eosinophils of 6% and erythrocyte sedimentation rate 32 mm/first hour. The platelet count was 2.8 lakhs/cumm. The peripheral smear showed normocytic normochromic anaemia and neutrophilia with neutrophils showing toxic granules and shift to left. The random blood sugar was 82 mg/dl. No obvious pathology was seen in the neuro-sonogram. CSF protein was 155 mg/dl and sugar was 16 mg/dl. CSF cell count revealed 800 cells with 70% polymorphs and 30% lymphocytes and four to six red blood cells. CSF Gram staining revealed five to six pus cells/oil immersion field with no organism. Rapid diagnostic test for bacterial antigen detection was done using Wellcogen™ Bacterial Antigen Kit[4] and was positive for *N. meningitidis* group ACYW135. The culture was sterile after 48 h of incubation in blood agar and chocolate agar. Blood culture from BacT/Alert beeped positive on day two and culture and direct sensitivity with Ceftriaxone 30 mcg, Penicillin 10 U and Ciprofloxacin 5 mcg discs (Kirby–Bauer disc diffusion method) was done on the same day. The culture yielded 0.5–1 mm grey translucent convex glistening elevated non-lytic colonies in blood agar and chocolate agar which were incubated at 37°C in 5%–10% CO_2. Gram staining of culture smears revealed Gram-negative diplococci. The isolate was oxidase positive and gave positive fermentation for glucose and maltose. The isolate was sensitive to Penicillin, Ceftriaxone and Ciprofloxacin.

The patient responded well to Ceftriaxone 330 mg twice daily and the patient was afebrile and better for three days. However, again, he developed fever spikes to the peak of 102° F and a possible episode of seizures. The patient was given symptomatic treatment and again a repeat lumbar puncture was done. The CSF again turned out to be sterile, and blood culture reports were also negative. The patient was subjected to a repeat neuro-sonogram which revealed bilateral minimal frontal subdural effusion. Magnetic resonance imaging report of the patient (as shown in Figure 1) suggested the possibility of post meningitis subdural hygroma . The baby was playful and active and sucking well at breast, except for the occasional fever spikes. Neurosurgery consultation was done for the subdural hygroma and conservative management was advised. Hence, without a subdural diagnostic tap, Meropenem 40 mg/kg intravenous every eighth hourly was added. The patient became afebrile after two days. The baby was discharged after ten days of antibiotic. During follow-up after one month, there was decrease in size of the subdural effusion.

DISCUSSION

In the absence of immunodeficiency disorders, the most common cause of bacterial meningitis in age group between one and 23 months of age are *Streptococcus agalactiae*, *Escherichia coli, Haemophilus influenzae*, *Streptococcus pneumoniae* and *N. meningitidis*.[5] To the best of our knowledge, this is the first report on meningococcal meningitis complicated with subdural effusion in an infant in Kerala. In infants and children younger than five years, in countries where universal vaccination against *H. influenzae* Type B and pneumococcus is not done, these microorganisms are usually the most frequent cause of meningitis. There are 13 serogroups of N. *meningitidis* that have been identified, six of which (A, B, C, W, X and Y) can cause epidemics.[6]

N. meningitidis is endemic in the USA, England, Spain and Africa, where several serogroups are predominant. India, though not in the endemic list, reports of sporadic cases of meningococcal meningitis[7,8] and its complications exist.[7,9]

The child would have probably acquired infection from his elder school-going sibling. The patient is said to have developed fever four days after onset of fever in sibling

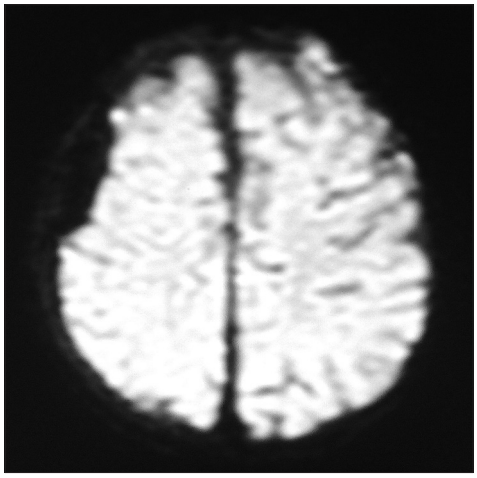

Figure 1: Magnetic resonance imaging scan showing left frontal subdural effusion

with coincides with the average incubation period for meningococcus transmission.[6]

Single dose of oral Ciprofloxacin (500 mg) is effective in elimination of nasopharyngeal carriage of meningococci and was administered to close contacts. Other effective chemoprophylactic agents are Rifampicin, Ceftriaxone intramuscular, oral single dose of Azithromycin.

Post-meningitis subdural fluid collection is a classical[10] but rare complication of bacterial meningitis in infants.[11] Its incidence has been estimated to be as high as one-half of the cases of meningitis, with *H. influenzae* being the most common bacterial cause. *N. meningitidis* has become more prevalent since the introduction of vaccination against *H. influenzae* and *S. pneumoniae*, and many authors now estimate that 5% of *N. meningitidis* infection in infants is complicated by a significant subdural effusion.[11] Subdural hygroma is a subdural body of CSF collection, without blood. They can be caused by leakage of CSF following minor trauma in the setting of cerebral atrophy, following meningitis in children or more commonly after ventricular shunting.[12] Most of the subdural hygroma is small and clinically not significant. They resolve spontaneously and do not require surgical intervention. However, some of them can be large and cause compression and secondary neurological symptoms. Such patients will require surgical intervention to avoid permanent neurological sequelae.[13]

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. WHO Weekly Epidemiological Record 2015; 27 March, 2015, 90th Year; No. 13. Vol. 90. 2015. p. 121-32. Available from: http://www.who.int/wer. [Last cited on 2015 Apr 22].
2. Meningococcus. Medical Staff Conference, University of California, San Francisco; October, 1977. p. 314-24.
3. Johri S, Gorthi SP, Anand AC. Meningococcal meningitis. Med J Armed Forces India 2005;61:369-74.
4. Wellcogen™ Bacterial Antigen Kit Manual REF ZL26/R30859602; IFU X77138 Manufactured by Remel Europe Ltd., UK.
5. Mandell GL, Bennett JE, Dolin R, editors. Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. 7th ed., Vol. 1. Philadelphia: Elsevier Churchill Livingstone; 2010. p. 1189-229.
6. Procop GW, Church DL, Hall GS, *et al*. Koneman's Colour Atlas And Textbook of Clinical Microbiology. 7th edition. Philadelphia: Wolter Kluwer Health;2017: p620
7. John TJ. Letters to the editor – Subdural effusion or empyema in infants. Indian Paediatr 2004;41:968-70. Available from: http://www.indianpaediatrics.net/sep2004/sep-968-970.htm. [Last cited on 2016 Apr 07].
8. Rafeeda KM, Manjusree S, Raveendran G, Ramani Bai JT. Meningococcal meningitis without rash. J Acad Clin Microbiol 2015;17:130-1. Available from: http://www.jacmjournal.org/text.asp?2015/17/2/130/171905. [Last cited on 2016 Jun 28].
9. Steinberg SH, Murphy JP. Subdural hygroma complicating meningococcic meningitis. J Neurosurg 1951;8:671-4.
10. Perez-Pico V, Llausas-Magana E, Leon-Ramirez A, Perez-Gaxiola G, Leon-Sicairos N. Meningococcal meningitis complicated with subdural effusion: A report of two infant cases. J Infect Dev Ctries 2007;1:74-7.
11. Updated by: Campellone JV. A Subdural Effusion: Medline Plus Medical Encyclopedia; 2016. Available from: http://www.nlm.nih.gov/medlineplus/ency/article/001422.htm. [Last cited on 2016 Apr 11].
12. De Bonis P, Sturiale CL, Anile C, Gaudino S, Mangiola A, Martucci M, *et al.* Decompressive craniectomy, interhemispheric hygroma and hydrocephalus: A timeline of events? Clin Neurol Neurosurg 2013;115:1308-12.
13. Sharma P, Mishra A, Arora G, Tripathi M, Bal C, Kumar R. Post meningitis subdural hygroma: Anatomical and functional evaluation with ^{99m}Tc-ehylene cysteine dimer single photon emission tomography/computed tomography. Indian J Nucl Med 2013;28:23-5.