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Case Report

A case report on meningococcal septicaemia in Kalabazaar, Thimphu, November 2016

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ABSTRACT

We report here a case study of meningococcal septicaemia caused by *Neisseria meningitidis* in Kalabazaar, Thimphu on November 2016. The one-year old patient had no history of contact with any infected individuals in the past two weeks before his onset, neither did he travel anywhere besides his one-storey traditional residence. The patient was hospitalized and given antibiotic treatment of ceftriaxone at an early course of the disease. The patient did not develop any complications during the time of hospitalization and had recovered well. Close contacts with the case were identified and given post-exposure prophylaxis with 500 mg ciprofloxacin. No new cases were encountered during the course of contact tracing.

Keywords: Meningococcal septicaemia, Close contacts, Active surveillance, Bhutan

INTRODUCTION

Neisseria meningitidis continues to be one of the leading infections to cause long-term morbidity and mortality worldwide, despite improvements in the critical care and availability of effective antibiotics.[1] The infection causes approximately 500,000 cases of septicaemia and meningitis globally every year, although incidence rate vary from < 1 per 100,000 per year in North America and Europe to 10-1000 per 100,000 per year in the meningitis belt of sub-Saharan Africa.[2] The peak incidence of invasive meningococcal disease (IMD) occurs in children between six months and two years of age followed by adolescents and young adults.[3] Lethality of sepsis is over 20% in children and thus prevention is a priority.[4]

In Bhutan, meningitis/encephalitis causes 20-30 deaths every year with case fatality rate of 10% in 2015.[5] On November 7, 2016, Paediatrician of Jigme Dorji Wangchuk National Referral Hospital (JDWNRH) informed Royal Center for Disease Control (RCDC) about the hospitalization of one-year-old boy with clinical suspicion of meningococcal infection with purpura fulminans. The case investigation was started on November 8 with aim to describe the clinical presentation of the patient and to identify close contacts with the patient for providing post-exposure chemoprophylaxis to reduce the incident cases and to prevent further spread of disease.

CASE REPORT

The patient was isolated in cabin on November 4, 2016, before admitting in the pediatric ward to prevent the transmission of infection to others. The vital signs of the patient were recorded as follows: heart rate of 163 beats per minute, SpO_2 of 88%, blood pressure of 100/70 mm of Hg, and temperature of 37.2^{0}C. On physical examination, patient looked pale with fever, cough and purpuric rash covering all over his body. The rash initially appeared in the abdomen and later started to spread to other parts of the body with enlargement in its

size in the early hour of hospitalization. The patient did not complain of photophobia and had no other underlying chronic conditions. The patient was born by normal spontaneous delivery with birth weight of 3.6 kg and as of now, all immunization schedules were completed on time. The admittance weight of the patient was 8.7 kg.

Samples for laboratory investigation including blood cultures were taken upon the admission. On the day of admission, laboratory investigation revealed high leucocyte count of 17400/µl (with predominant neutrophils), raised C-reactive protein of 5 mg/dl, low platelet count of 80000/µl of blood and low prothrombin time of 12.3 seconds as shown in Table 1. Although negative for the specimen taken from rash, gram negative diplococci was seen on the Gram staining made on smear of the incubated blood. Sub-culture from blood culture was made on Mac-Conkey agar, blood agar and chocolate agar and, the isolate was confirmed by analytical profile index (API)-NH.

Table 1: Laboratory results for the meningococcal septicaemic case in Thimphu, November, 2016.

Variables	Reference range	Day 1 (on admission)	Day 2	Day 3	Day 4
White Blood Cell/µl	5 - 15	17.4	21.26	17.67	13.2
Differential count (%)					
Neutrophils	35 - 55	69	59.3	51	43.9
Lymphocytes	35 - 55	28	38.7	46.3	49.3
Monocytes	2 - 10	2	1.9	2.4	5.6
Basophils	0 - 2	1	0	0.2	0.2
Eosinophils	0 - 5	0	0.1	0.1	1
Hemoglobin (g/dl)	11 - 16	10.1	10.3	9.7	9.7
Platelet/µl	150 - 450	80	Nd	70	121
Sedimentation rate	0 -15	40	Nd	84	Nd
CRP (mg/dl)	< 0.6	Nd	Nd	5	Nd
Prothrombin (Seconds)	13.6 - 17.5	Nd	Nd	12.3	Nd
INR	0.8 - 1.2	Nd	Nd	1.04	Nd

Nd: Not done

After administering intravenous (IV) ceftriaxone (100 mg/kg/day), the patient was later moved to the pediatric ward. Lumber puncture was carried out on third day (November 6) to rule out bacterial meningitis and continued with the same antibiotic along with IV fluids (DNS) and antipyretic (paracetamol). Cerebrospinal fluid (CSF) was macroscopically clear with no evidence of white blood cells. Glucose level was within the normal range, while protein was slightly lower than the normal values with 15 mg/dl.

The CSF culture had no growth of bacteria and the antigen test was also found negative. Bacterial growth was observed from blood culture and was identified as *Neisseria meningitidis*. The antimicrobial susceptibility testing was performed using Kirby-Bauer disc diffusion method for the isolated organism.[6] Resistant to ampicillin and ceftriaxone were observed for the organism. However, ceftriaxone was continued as patient's prognosis was improving. Rashes were in regression and bleeding manifestation was not seen. The hematological parameters were also stabilized as shown in table 1 during the course of treatment.

Epidemiological investigation

The patient lived in a one-storey traditional house in a nucleated settlement in Kalabazaar, Thimphu, with his parents along with other family members. None of them had contacts with infected individuals before two weeks of onset of illness, nor had they attended any gathering or travelled outside besides their customary activities.

No active cases were encountered during the contact tracing conducted by the team. Close contacts with the patient were identified for providing post-exposure prophylaxis (PEP). There were twelve contacts among family members (all adults) living in a same house with a patient; five females and six males. All of them were given single dose of ciprofloxacin (500 mg).

Additional ten close contacts were identified, as they had been frequently in contact with a patient. They were relatives and neighbours who lived in vicinity to the house where the patient was living. They were also given 500 mg ciprofloxacin. Attending physicians and nurses also had the same chemoprophylaxis. We could not trace the identification of taxi driver who drove the patient to the hospital. However, the mother of the child explained that driver was not at risk of contact as the patient was properly wrapped with a cloth including his face during the transportation.

DISCUSSION

The investigation describes the case of an infant who mostly stayed at home and did not have active social history or a clear history of exposure to patients with meningococcal disease in the community. Review of medical records in JDWNRH did not show any patients

with similar presentation of clinical manifestation over the past two weeks. The development of disease with no clear history of contact with the infected patients was also reported in other studies.[3,7] This situation is not surprising for meningococcal septicaemia because the transmission of meningococci is conducted mostly by asymptomatic carriers. Moreover, cold and dry weather prevalent in the past few weeks might have predisposed the susceptibility of patient.[8]

The patient visited the hospital within less than twelve hours of onset of illness and got hospitalized on time. Antibiotic treatment can save the lives of 95% of the patients provided that it is started during the first two days of illness.[9] Hospitalization of the case was reported to be a protective factor with > two fold reduction in death compared to those not hospitalized. The hospitalized patients are more likely to have their circulatory status monitored with early and aggressive management of shock.[2]

Meningococcal septicaemia in children develops when the initial host response to infection becomes inappropriately amplified and dysregulated. After the initial presentation of fever and pharyngitis, the disease may progress to fulminant fatal disease with disseminated intravascular coagulation, hypotension, septic shock and ultimately organ failure.[10] Skin hemorrhages (purpura fulminans) are the main manifestation of the disease. During the course of fulmnant meningococcal sepsis, meningococci can cross blood brain barrier and invade the brain uncontrollably, as the main humoral and cellular host response are reduced. The reported mortality in the general population is nearly 10 to 20% despite antibiotic treatment and intensive care support.[11] Complications with hearing loss and seizures were also reported in children below five years of age.[2]

The important risk factor in the disease development is the nasopharyngeal carriage. The human nasopharynx is the only known reservoir for *Neisseria meningitidis* and spread by droplets of respiratory or throat secretions from carriers. The carriage rate in respiratory tract mucosa is approximately 5–10% in the general population. The other risk factors for meningococcemia includes prematurity, children less than two years of age, acute viral infections, complement system deficiency and crowded environment. The pathology is initiated once the bacterium enters the bloodstream where they survive and proliferate.[12]

The transmission of infection is highly likely in the community owing to the nucleated or clustered type of settlement, where the patient was living. The houses were constructed very close to each other and separated by a single narrow footpath. However, the conclusive evidence about the temporal relationship could not be established without any similar cases being seen in the community. It is likely that the patient acquired infection from a healthy carrier living in close contact with him in the community.

CONCLUSION

Meningococcal disease spreads very rapidly and thus high index of suspicion is needed to diagnose the disease in its initial stage. The rapid management of the patient with timely appropriate antibiotics treatment and supportive care is pivotal to prevent complications and death. It is important to conduct active surveillance to identify new cases and close contacts to prevent further spread to the general population.

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