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Atypical meningococcal meningitis with rashless presentation: A case report

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ABSTRACT

Meningococcal disease is the major health problem in developing world. The clinical presentation is varied, ranging from transient fever and bacteraemia to fulminant disease with death ensuing within hours of the onset of clinical symptoms. The classical clinical manifestations of meningococcal disease have been well described, but atypical presentations if unrecognized, may lead to a delay in treatment and fatal outcome. We here report a case presented with atypical presentation of meningococcal meningitis without classical rash, which was diagnosed and managed successfully.

1. Introduction

Neisseria meningitidis (*N. meningitidisis*) the second most common cause of community-acquired adult bacterial meningitis. [1] It has been recognized as a major health problem specially in developing region of the word. The clinical manifestations of meningococcal disease can be quite varied, ranging from transient fever and bacteraemia to fulminant disease with death ensuing within hours of the onset of clinical symptoms. Though the clinical manifestations of meningococcal disease have been well described, but atypical presentations if unrecognized, may lead to a delay in treatment and fatal outcome. We here report an atypical case of meningococcal meningitis where patient presented without classical rash. The patient was promptly diagnosed and managed successfully.

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2. Case report

A 40 yrs old male presented with one day history of high grade fever and sudden onset of headache. He also had history of altered sensorium and irritability with two episodes of vomiting containing digestive food particles. Physical findings included a pulse rate of 150/min, blood pressure 60 mmHg systolic, respiratory rate of 38/min and oral temperature was 39 ℃. He was unconscious, responding only to painful stimuli. There was severe stiffness in the neck seen. He had a reddish well defined flat lesion on dorsum of right hand which was found to be congenital. Respiratory system examination revealed coarse crepitations on right middle zone of chest on auscultation. No other gross abnormality was seen while examination of other systems. Patient was clinically diagnosed as a case of circulatory shock and aspiration pnuemonitis.

He was immediately admitted in the intensive care unit (ICU). Patient was intubated and kept on mechanical ventilation. Supportive management, including fluid resuscitation and inotropes, were started. Empirical

intravenous antibiotics (vancomycin 1 g IV and ceftriaxone 2 g IV 12 hrly) were started after drawing the blood for cultures. Patients was also kept on low dose corticosteroids (hydrocortisone 50 mg six hourly). Blood examination revealed haemoglobin 10.7 g/dL, total leukocyte count (TLC) was 18×10^3/ L with 86.7% neutrophils and platelet count were 50 000/L. Liver function test, cardiac enzymes, urine routine examination, BUN, serum electrolytes and blood glucose levels were within normal limits. Subsequently blood culture showed growth of staphylococcus aureus sensitive to linezolid and vancomycin antibiotics. Coagulation profile was grossly deranged. Chest X ray showed a heterogeneous patch in right middle zone suggestive of likely aspiration pnuemonitis. Electrocardiogram was found to be normal. His cerebrospinal fluid (CSF) was examined and was found to be turbid with total cell count of 5 500 cells/ L, predominantly polymorphs. CSF glucose was low and albumin was raised. CSF gram staining was negative and culture was sterile. A computed tomography (CT) scan of the head was performed revealing no abnormal pathology. Four units of fresh frozen plasma and platelet concentrates were transfused to correct coagulation profile.

On day 4 in ICU, patient had two episodes of generalized tonic-clonic seizures. Antiepileptic (IV phenytoin sodium) was started immediately. A direct PCR test (DT-PCR) was done and we found *N. meningitidis* DNA in clinical samples of the patient. Hence a final diagnosis of atypical meningococcal meningitis with meningococcemia was made. The case was immediately reported to our nodal officer in charge and the ICU staff dealing with patient received post-exposure prophylaxis (rifampicin 600 mg orally once a day for 2 d) in view of the patient diagnosis.

On day 6 in ICU, patient general condition improved. He regained consciousness. His blood picture improved, total leukocyte count (TLC) was found to be within normal limits and platelet counts rose to 1.2×10^5/ L. On day 8 in ICU, he was extubated and subsequently shifted to medicine ward 24 h after. He recovered without any neurological sequelae and was discharged uneventfully after next 48 h.

3. Discussion

Meningococcal infection has been recognised as a serious global problem since the first outbreak in 1805[1]. It can occur as sporadic cases, focal outbreak, or large epidemic and has an incidence of 0.5-5 per 100 000. Mortality rate of meningococcal disease in developed countries is 10% as compared to whopping 50% in developing countries[2, 3].

The causative organism, *N. meningitidis* is a gram negative aerobic diplococci and an obligate human commensal in 10% of population. There are 13 serotypes of *N. meningitidis* so far recognised with highest incidence seen with serotype A amongst them and is known to cause epidemic in 'Meningitic Belt' region of sub-Saharan Africa[4,5]. It commonly affects young adults and children more than three years. Predisposing factors include overcrowding, smoking, deficiency of immunoproteins (properdin or factor P), asplenia (functional / anatomical), and HIV infection. Meningococcus is transmitted amongst persons through contact with saliva or upper respiratory tract droplets[4]. The persons once infected may have life threatening diseases like pyogenic meningitis or meningococcemia with or without meningitis[1].

Meningococcal meningitis contributes to 50% of the cases of invasive disease[2]. It classically manifests as headache, fever, and stiff neck, however nausea, vomiting, photophobia and altered mental status are not uncommon. Its mortality rate is as high as 5%-10% and amongst the survivors nearly 8%-20% may have long-term sequelae such as sensorineural deafness, cranial nerve palsies, mental retardation, spasticity, seizures, and concentration disturbances[2,6,7].

Fulminant meningococcal septicaemia occurs in 5%-20% of the patients affected with very high mortality rate (20%-80%)[1,2]. It is characterized by abrupt onset of fever and a petechial rash which can progress to hypotension, acute adrenal failure (Waterhouse-Friecderichsen syndrome), coagulopathy and multiorgan failure. The rashes (petechiae or purpura) seen are blanchable and are considered pathognomic of meningococcal infection. They occur from the first to the third day of illness and are prominent in areas subjected to direct pressure like axillary folds, the belt line, or the back. Although some reported that these patients may not present with this classical rash at the time of the clinical presentation[6,8]. Andersen etal in a retrospective study conducted in 255 patients with invasive meningococcal disease reported 40% of patients above 30 yrs of age without rash[8]. Thus meningococcal disease cannot be excluded on the absence of the typical rash presentation. Our patient also had meningococcemia with meningitis with absence of characteristic rash at any time during the clinical course.

Approximately 30% of patients with meningococcal disease have meningococcemia without meningitis. Less common presentations include pneumonia, conjunctivitis, otitis media, epiglottitis, arthritis, urethritis and pericarditis and chronic meningococcemia, a syndrome characterized by prolonged intermittent fever, rash, arthralgias, and headaches[1,2].

Meningococcal disease is a life-threatening infection and requires early recognition and treatment. The diagnosis of *N. meningitidis* involves a combination of clinical suspicion and laboratory confirmation with culture of *N. meningitidis* being

the gold standard diagnostic test. Although the cultures may come negative primarily due to the empirical treatment with broad spectrum antibiotics, faulty sample collection and transport conditions[1,2]. Polymerase chain reaction (PCR) is a potential diagnostic tool in such cases with high sensitivity and specificity[4]. Immunochromatography dipsticks are also available for point of care diagnosis of *N. meningitidis*[2].

Immediate hospitalization and supportive care is required to ensure hemodynamic stability. Antibiotics like penicillin and ceftriaxone is the mainstay of treatment. Chloramphenicol, fluoroquinolones and carbepenams can be used in patients allergic to penicillin[2,4]. Precautions against the droplet spread are necessary for hospitalized patients until 24 h after the initiation of appropriate antibiotics. Role of corticosteroids in treatment of fulminant meningococcemia is still controversial[3]. Many studies demonstrated no benefit of high dose steroids treatments. However it is still advocated as a standard treatment protocol in presence of extensive disseminated intravascular coagulopathy (DIC) and shock or suspicion of Waterhouse-Friderichsen syndrome[4]. Chemoprophylaxis with rifampicin, ciprofloxacin or ceftriaxone is recommended for all close contacts of index case regardless of immunization status. It should be given within 24 h and is of limited or no value if given after 14 d of diagnosis of case[1,2]. Immunoprophylaxis can be used as an adjunct to chemoprophylaxis. Meningococcal quadrivalent polysaccharide vaccine and protein conjugated vaccines are readily available. The duration of vaccine induced immunity is 3-5 yrs[1,2]. It is generally indicated at times of meningococcal epidemic, person with properdin (factor P) deficiency, sickle cell anaemia, asplenia or splenectomy, military recruits, and individuals travelling to sub- Saharan Africa. Recently there is evidence of the development of Guillain Barre Syndrome (GBS) with this vaccine administration and hence should be avoided in patients with suspicion of GBS[2].

Meningococcal disease is a disease process that can become insidious and fatal in a short period of time if not diagnosed promptly. Though the classic signs are well known but few atypical manifestations should also be kept in mind that have been reported in literature, which may pose diagnostic problems and can result in fatal outcome[9-14]. Thus it is vital to have thorough knowledge of all clinical manifestations and facility of high end diagnostic tools to detect and treat this fatal condition early in the course to reduce the associated mortality and morbidity.

Conflict of interest statement

The authors declare that there is no conflict of interest.

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