Meningococcal Septicemia

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Meningococcal disease is an illness caused by an organism called Neisseria meningitidis. The disease is highly contagious as it spreads through close contact. Immediate medical recognition of the disease is vital once it is suspected. Any person could be infected with meningococcal disease; however, certain groups are at practical risk.

A healthy middle aged man presented with symptoms of fever, general fatigue, and widespread rash. The patient deteriorated rapidly and passed away. This case report is a typical presentation of meningococcal disease. The presentation could be vague and if not recognized early, might lead to rapid decline in health and eventual death.

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Meningococcal disease is caused by Neisseria meningitidis, a gram-positive intracellular diplococci organism. It is commonly seen in Asia and Africa. Its presentation could range from a transient fever and bacteremia to fulminate disease, which eventually leads to death within hours of the disease process. The three clinical features of the disease include meningitis, meningitis with meningococcemia and meningococcemia without clinical evidence of meningitis.

The initial manifestations of the disease are nonspecific and can be confused with other transient viral illnesses. It could present with fever, loss of concentration, nausea and vomiting, and myalgia. The first classic symptom of the disease is rash, which may progress from a nonspecific rash to petechia to hemorrhage within several hours[1]. The condition is characterized by its rapid evolvement from minor symptoms to hypotension, septic shock, and disseminated intravascular coagulation. Therefore, a high index of clinical suspicion of the disease at its early stages is crucial.

The aim of this report is to present a case of a healthy middle aged man who complained of fever and rash, which was later confirmed to be meningococcemia. The importance of early diagnosis and treatment is the cornerstone of such condition.

THE CASE

A forty-nine-year-old Indian male presented with fever of three days' duration, chest pain radiating to the left arm, shortness of breath and orthopnea of 48 hours' duration. He had a generalized rash spreading to the mucous membranes and vomiting for two days. He visited a private medical center upon the onset of symptoms and he was assured and discharged on analgesia.

On examination, the patient was conscious and oriented to time, place and person. He looked generally sick with a respiratory rate of 22/minute. Vitally, he was febrile with a temperature of 39 degrees, blood pressure of 105/60 mmHg, heart rate of 100/min and oxygen saturation of 92% on 6L FM. He had a widespread non-blanching petechial purpuric rash mainly concentrated on the upper and lower limbs. He had petechial lesions on the mouth mucus membranes with surrounding erythema.

The patient had equal reactive pupils bilaterally, was able to move all limbs on command, and was communicating normally. All limb reflexes were 2+, Bibinski sign negative, no neck stiffness and both Kerning's and Brudizinski's signs were negative. He had bilateral basal inspiratory coarse crepitation.

Initial laboratory investigations were normal except for bandemia of 12% found on complete blood count. Arterial blood gas revealed as follows: PO_2 63.7 mmHg, PH 7.47 and CO_2 29.3, HCO_3 21.6. Chest X-ray revealed bilateral reticulonodular infiltrates.

During assessment, the patient's consciousness level was decreasing and deteriorating rapidly. He was shifted to the high dependency unit for further management. Echocardiogram revealed ejection fraction of 45%, no regional wall motion abnormality, no evidence of vegetation and no pericardial effusion.

Blood culture was requested. He was started on a high dose of ceftriaxone 2g twice daily and vancomycin 1g twice daily.

Other investigations including malaria smear, Widal test, HIV, inflammatory markers and deep tracheal aspiration for gram stain, culture and Acid Fast Bacilli were requested. Ultrasound

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abdomen revealed some perinephric fluid collection and renal parenchymal changes and no other abnormalities. CT abdomen and brain showed no remarkable changes. Lumbar puncture was performed, see table 1.

Table 1: CSF Analysis

RBC	670000
WBC	520
Neutrophil	77
Lymphocyte	16
Eosinophil	0
Monocyte	7
Blast	0
Other microscopic observation	Bloodstained
Initial gram stain	negative

The patient was intubated because of gasping and drowsiness. The patient started to bleed inside the airway during intubation. He had bled from the oral cavity and nasogastric tube. Urgent fresh frozen plasma and packed RBC were transfused and the repeated coagulation profile showed prolonged value suggesting disseminated intravascular coagulation and a drop in platelet count to 33.

Blood culture revealed gram-negative diplococci highly suggestive of meningococcus, which proved to be *Neisseria meningitidis* serogroup B. The patient's blood pressure dropped despite boluses of fluids and inotropic support.

Ten hours after presentation, the patient became asystole and CPR was initiated according to BLS protocol; however, it was unsuccessful and the patient expired.

DISCUSSION

Meningococcal disease is an infection with high mortality, particularly among children and young adolescents. The disease is transmitted through close contact with infected nasopharyngeal droplets. Close contacts with infected patients are 300-1000 times prone to be infected compared to the rest of the population[2]. Patients with immunodeficiencies such as HIV, the elderly, diabetics, asplenic or complement system defect patients are more prone to the rapid deterioration in the clinical course.

The organism can be detected by gram stain in a blood sample or skin biopsy. Since antibiotics use is widespread, it could, on occasion, impede the growth of the organism on blood culture and result in a false negative result. Skin biopsy, polymerase chain reaction (PCR) technique or antigen detection are not affected by antibiotic use. However, hospitals depend on blood culture because it is easier and cheaper[3].

The poor prognostic indicators in infected patients are the extreme of age, widespread skin lesions, acute onset of the infection and absence of signs of meningitis, elevated inflammatory markers and the presence of the signs of shock[4]. Disseminated intravascular coagulopathy is a common complication of the infection.

Third generation cephalosporin is the drug of choice (particularly ceftriaxone which has a good CNS penetration). Chloramphenicol is an alternative choice in cases of hypersensitivity to penicillin or cephalosporin[5].

Prevention is most effective with vaccination. The most common serotypes causing the infection are B, C and Y. The polysaccharide vaccination available covers infections with serotypes A, C, W135 and Y[6]. Healthcare workers who are in close contact with patients confirmed to be infected with meningococcal disease should receive a prophylaxis with ciprofloxacin, rifampicin or ceftriaxone if pregnant. However, prophylaxis is meant only to eliminate the nasopharyngeal infection and does not prevent it if the person was already infected[7].

CONCLUSION

Meningococcal disease is a serious condition that progresses rapidly. Therefore, a high index of clinical suspicion from the initial presentation is vital. The disease can present as sepsis, meningitis or both. Rapid and aggressive treatment with fluids and antibiotics is the cornerstone of treatment.

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