A CASE OF MENINGOCOCCAEMIA WITH DEAFNESS

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INTRODUCTION

Nisseria meningitidis is the second most common cause of community-acquired adult bacterial meningitis [1]. The clinical manifestations of meningococcal disease can be quite varied, ranging from transient fever and bacteremia to fulminant disease with death ensuing within hours of the onset of clinical symptoms. It is the most common cause of meningitis associated with septicemia [2]. Acute systemic meningococcal disease is most frequently manifest by three syndromes, Meningitis, Meningitis with accompanying meningococcemia and Meningococcemia without clinical evidence of meningitis.

Because of the trend for endemic meningococcal infection to occur during the late winter when concurrent influenza virus is in the community, many cases of meningococcal disease are mistaken initially for severe "flu." It is also not uncommon for other cases to have been reported in the region or for the patient to have been a contact of a previously diagnosed case.

CASE REPORT

A 19 year old young soldier was brought with one day history of high grade fever with rigors and chills and five hour history of confusion and irritability. There was also one episode of vomiting containing digested food particles.

Physical findings included a pulse of 124 per minute, blood pressure 80/60 mmHg and a respiratory rate of 34/min. He was unconscious responding only to painful stimuli having generalized purpuric rash all over his body (Figure). He was pale. Neck was supple. There was right sided ptosis, pupils were reacting and planters were equivocal.

Provisional diagnosis of "meningococcal meningitis with meningococcaemia" was made. He was admitted to intensive care unit, where he was given intravenous fluids along with Injection Ceftriaxone 2g intravenous BD plus Injection Benzylpencillin 2 million units intravenous 2 hourly. Intravenous Injection Dexamethasone 8 mg stat and 4 mg intravenous eight hourly was also given for the first 48 hours.

Blood examination revealed haemoglobin 10.8 g/dL. total leucocyte count was $7.0x10^3$ L with 86.7% neutrophils & platelets were 20,000/L. Liver function test, cardiac enzymes. urine routine examination. urea. electrolytes and blood glucose levels were within normal limits. X-ray chest was clear. Coagulation profile was deranged & serum D-dimers were raised. His cerebrospinal fluid was turbid & revealed cell count of 4250 cells L with predominantly polymorphs. Cerebrospinal fluid gram staining showed Gram negative diplococci morphologically resembling Nisseria meningititides. Cerebrospinal fluid glucose was low & albumin was raised. Four units of fresh frozen plasma and platelet concentrates were transfused to correct coagulation profile.

Two days later he had a generalized tonic clonic seizure, for which he was started on Sodium valporate.

On 6th day of his treatment he regained consciousness. After 01 week his blood picture revealed total leucocyte count within normal limits & platelets raised to 83,000/L. His ECG showed ventricular ectopics. Tablet Metoprolol twenty five mg BD was added. Echocardiogram was done & found to be normal. He was shifted to the medical ward on the 10th day of admission.

After regaining consciousness he was found to have bilateral deafness. On examination he was found to be suffering from total sensorineural deafness. Audiogram showed bilateral symmetrical hearing loss with hearing threshold of 75 decibels. CT scan brain was advised & found to be normal. He improved further with treatment and his hearing also improved. His subsequent audiogram showed that hearing threshold improved to 45 decibels. He was subsequently discharged with advice to follow up in out door.

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DISCUSSION

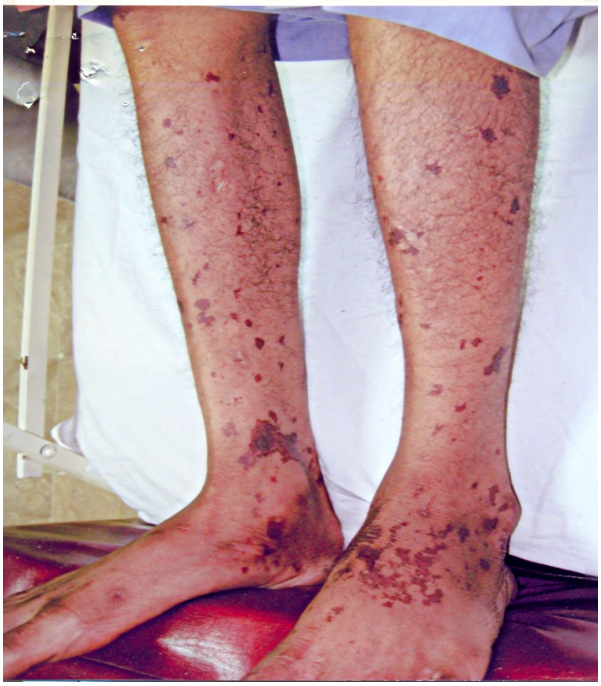

Figure: Purapuric Rash Legs seen in patient of Mengingococaemia.

A number of complications have been documented in patients with meningococcal meningitis more commonly, later in the recovery phase of illness. These include immune complex associated complications including arthritis without the recovery of organisms, pleurisy, vasculitis and pericarditis. Epiglottitis, conus medullaris syndrome, and cranial nerve dysfunctions especially sixth, seventh, and eighth can also occur.

Sensorineural hearing loss after bacterial meningitis may be transient or permanent. Transient hearing loss in many patients is due to conductive disturbance. However, permanent hearing loss may be due to damage to the cochlea, labyrinth, or eighth cranial nerve from direct bacterial invasion or the inflammatory response elicited by the infection. Sensorineural hearing loss has been reported in 6 percent of patients suffering from meningococcal meningitis [3]. In another study total incidence of impaired hearing was 5.8%. This incidence was higher in the younger age groups, females, severe cases of meningitis and in patients who received specific therapy late after the onset of signs and symptoms of the disease. [4] It has been found that hearing loss is two to three times more common in pneumococcal meningitis than meningococcal meningitis [5].

However incidence is less in Haemophilus influenzae meningitis than meningococcal meningitis [6]. A study showed that length of hospitalization, development of seizures, elevated cerebrospinal fluid protein, and decreased cerebrospinal fluid glucose were significant predictors for hearing loss with bacterial meningitis[7]. In meningitis permanent deafness is rare but 10% of the patients may have a rapidly reversible cochlear dysfunction [5]. Our patient also showed recovery of hearing function progressively with treatment.

Acute meningococcaemia is a serious life threatening infection associated with a variety of complications including sensorineural deafness. In meningitis permanent deafness is rare and majority of the patients may have a rapidly reversible cochlear dysfunction.

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