

Acute Meningococemia Complicating Epidemic Meningitis In Zaria, Nigeria

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Citation

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Abstract

Neisseria meningitidis is a bacterium known to cause 5-10 years cyclical epidemic meningitis in the meningitic belt of Africa. In these case reports, we highlight the multi-systemic manifestations and management challenges of acute meningococemia, a rare life threatening complication of epidemic meningitis.

INTRODUCTION

Neisseria meningitidis is a bacterium known to cause 5-10 years cyclical epidemic meningitis or cerebrospinal meningitis (CSM) in the meningitic belt of Africa (1). Acute meningococemia is a rare life threatening complication of this infection. We highlight the multi-systemic manifestations and clinical sequelae of acute meningococemia in 3 young patients who were referred to our centre during the 2009 CSM epidemic in Nigeria. All patients were residents of overcrowded rural settlements.

CASE HISTORIES

A 20year old male petty trader (Case 1), a 13year old female pupil (Case 2) and a 16yr old female student (Case 3) each, presented with acute symptoms of headache, fever, vomiting and neck pain associated with microscopic haematuria, haemorrhagic skin lesions, meningeal irritation, oliguria as well as hypotension and altered consciousness. Case 1 had petechiae and purpuric lesions on the right foot while Case 2 had similar lesions on her upper and lower limbs. Both of these patients had mild thrombocytopenia and prolonged international normalised ratio of 2.0. Case 3 had different degrees of purpura, blisters and ulcers all over the body, in addition to recurrent generalized seizures, papilloedema, coma and septic shock. She was bleeding from mucosal orifices with laboratory features of disseminated intravascular coagulation (DIC) (platelet counts of $62 \times 10^9/L$, normal range $100-400 \times 10^9/L$, prolonged prothrombin time by 8s above control and prolonged Kaolin cephalin clotting time by 17s above control).

Gram negative intracellular diplococci were identified from the cerebrospinal fluids (CSFs) of the patients but *Neisseria meningitidis* was not isolated from CSF and blood cultures, probably because all patients were referred from secondary health facilities where they had received antibiotics.

Antigenic detection could not be done due to absence of facilities. Investigations were negative for sickle cell disease and HIV-1/2 infections. Spleen sizes, by abdominal ultrasound, were normal in all patients.

Patients were treated with intravenous fluids, dexamethasone 24mg/day in divided doses, and ceftriaxone 2g daily for 3 weeks. While Cases 1 and 2 recovered in the general ward and were discharged without sequelae up to 3 months of follow-up, Case 3 had to be managed in intensive care unit (ICU) with additional transfusions of two units of fresh whole blood. On the 10th day of treatment, she developed bilateral sterile knee arthritis. However, she recovered after 86 days of hospital care and was discharged with residual bilateral knee arthralgia which persisted 3 months post-discharge.

The features of hemorrhagic skin lesions, septic shock, DIC and acute renal failure seen in these patients with meningitis are consistent with acute meningococemia (1). Endothelial dysfunction, vasculitis, and capillary leakage due to the effects of meningococci endotoxin and endotoxin-induced cytokines such as tumour necrosis factor α and interferon γ , underlie these manifestations (1, 2). The occurrence of sterile arthritis in one of the patients may be due to immune complex deposition as suggested by an earlier study from Zaria, Nigeria (3)

All the patients reported lived in overcrowded poor rural environment which are known risk factors for epidemic meningitis. While some of the risk factors for meningococchemia, such as sickle cell disease, asplenia and HIV infection, were excluded in our patients, investigations for the role of other risk factors, such as complement deficiency, properdin deficiency and polymorphisms of various inflammatory mediators (1, 2), were limited by the absence of facilities.

Acute meningococchemia is a potentially fatal condition with mortality ranging from 10-43% and occurring mainly within the first 12 hours of presentation (4, 5). Less than 1% of patients recovering from acute meningococchemia may progress to the chronic phase characterized by recurrent fever, arthritis or arthralgia and recurrent petechiae rash (1). Long term follow up of patients is therefore advocated.

Figure 1

Figure 1: Various degrees of haemorrhagic skin lesions including purpura and ulcers on Left foot of case 3.



CONCLUSION

In conclusion, to avert the morbidity and mortality associated with epidemic meningitis in Nigeria, strengthening of surveillance measures, improvements in vaccination strategies and tackling the prevailing problems of poverty and overcrowding, are imperatives.

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