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PERSISTENT VISUAL LOSS AS A COMPLICATION OF MENINGOCOCCAL MENINGITIS

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Abstract

Meningococcal disease remains a major cause of morbidity and mortality in childhood. Cerebral infarction complicating meningococcal meningitis is recognized but uncommon. We describe a 3-year-old boy with parieto-occipital infarction secondary to meningococcal meningitis, resulting in permanent visual loss as the sole neurologic sequelae and, consequently, major implications for his subsequent development.

Keywords

meningococcal; meningitis; infarction

A 3-year-old boy presented to the Accident and Emergency Unit at Queen Elizabeth Central Hospital, Blantyre, with a 4-day history of fever, vomiting, decreased consciousness for 24 hours and a generalized convulsion lasting an hour. The convulsion was managed with a single dose of intramuscular paraldehyde (0.2 mL/kg). There was no medical history or family history of note and he was fully immunized. On clinical examination, he was well nourished with a pulse of 108 beats/min, respiratory rate of 44 breaths/min, axillary temperature of 40.3°C, blood pressure of 94/71 mm Hg and oxygen saturation of 94% in air. He was well perfused peripherally. There were no petechiae or bruises. His Blantyre Coma Score1 was 2/5, there was no neck stiffness and tendon reflexes were normal. Kernig's sign was negative. The rest of the physical examination was unremarkable.

Lumbar puncture revealed cloudy cerebrospinal fluid (CSF) and blood film was negative for malaria parasites. Intravenous ceftriaxone (100 mg/kg per day) was started. The blood glucose was 3.1 mmol/L, lactate 5.4 mmol/L, hemoglobin 12.1 g/dL, white cell count 12.3×10^9/mL, neutrophil count 9.1×10^9/mL and platelet 123×10^9/mL. CSF showed a white blood cell count of 3680/mm^3, 96% neutrophils, protein 300 mg/dL and glucose <5 mmol/L (CSF glucose and protein determination performed using Uristix, Bayer, Bridgend, U.K.). Gram-negative diplococci were present on Gram-stained smear. Seven hours after

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admission, the child had become afebrile and the Blantyre Coma Score had improved to 4/5. On day 2, both blood and CSF culture grew fully susceptible *Neisseria meningitidis*, serogroup B. This was subsequently confirmed on both blood and CSF polymerase chain reaction for meningococcal DNA. On day 3, the child was noted to be unable to visually fix and follow objects. Ophthalmology assessment revealed a right gaze palsy, normal pupillary responses and normal fundi. Computed tomography scan demonstrated bilateral parietooccipital infarcts. He received 10 days of intravenous ceftriaxone and was discharged home after 11 days. His visual loss persisted at discharge but hearing was normal and there were no other focal neurologic abnormalities. At 6-month follow up, he was reacting to a bright light but was not fixing or following. The gaze palsy had resolved and fundoscopy was normal. The final diagnosis was cortical blindness with preservation of light response.

DISCUSSION

Cerebral infarction is an uncommon but recognized complication of meningococcal meningitis. There are several reports in the literature of cerebral or cerebellar infarction complicating bacterial meningitis,2-6 Infarction of the basal ganglia and caudate, presenting with hemiparesis and cranial nerve deficits,7 and spinal cord infarction8,9 have been described as complications of meningococcal meningitis in adults but parieto-occipital infarction in childhood meningococcal meningitis has not previously been reported. Persistent visual loss in addition to other neurologic sequelae is also a recognized complication of bacterial meningitis, but this case is unusual in that the persistent visual loss was the sole neurologic complication of meningococcal meningitis.

In the case reported here, the observation of visual loss on day 3 and the bilateral distribution could be consistent with vasculitis as the underlying cause. The child did not have any signs of shock and was not hypotensive at any time, thereby making infarction secondary to cerebral hypoperfusion unlikely.

The pathophysiology of neuronal injury associated with bacterial meningitis involves several mechanisms: 1) raised intracranial pressure secondary to cytotoxic edema and increased vascular permeability with secondary ischemia, 2) leukocyte extravasation into the subarachnoid space, 3) cerebral ischemia secondary to reduced cerebral perfusion pressure or vasculitis and 4) direct toxic effect on neurons from reactive oxygen intermediates, caspases and proteases.7

The use of steroids as adjunctive therapy in bacterial meningitis is controversial. A large multicenter trial in adults in Europe showed an improved outcome with adjunctive therapy, 10 but another large trial involving children in sub-Saharan Africa demonstrated no benefit. 11 Dexamethasone, by virtue of its anti-inflammatory effects, might be expected to reduce neuronal injury as a result of inflammation in the subarachnoid space and also injury secondary to vasculitis. It is impossible to say whether commencing adjunctive steroids in this child on day 3 might have made any difference to the persistent visual loss.

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