Successful treatment of a case of primary amoebic meningoencephalitis: How important is history taking

Sir,

*Naegleria fowleri* is a rare cause of meningoencephalitis leading to a fatal fulminant primary amoebic meningoencephalitis (PAM) acquired after diving, swimming in warm fresh water. Very few patients are reported to survive after acquiring this infection.[1,2] Timely diagnosis requires a high index of suspicion in any individual presenting with a sub‑acute onset meningitis after exposure to standing water in lakes and pools.

A 6-year-old male child presented with a history of high grade fever, headache, vomiting for 10 days and altered sensorium for 1-day. There was no history of seizures, any other complaints or contact with tuberculosis. There was a history of playing and swimming in water stored in a cement tank, collected from a stream of water (Koohl).

On examination, the child was in delirium, febrile with stable vitals. Central nervous system (CNS) examination revealed signs of meningeal irritation with the early papilledema. Rest of the systemic examination was normal. On admission possibilities of acute pyogenic meningitis, rickettsial meningoencephalitis, as this region is endemic for scrub typhus and tubercular meningitis, was kept. Intravenous (IV) ceftriaxone, azithromycin, mannitol, maintenance fluid and supportive care were started.

Cerebrospinal fluid (CSF) analysis after lumbar puncture that was done next day, showed 415 cells/mm³ with 30% neutrophils and 70% lymphocytes. Gram-staining did not reveal any bacteria, but the wet mount smear showed plenty of motile, amoeboid flagellate organisms, 15–20 µm in size, *Naegleria* species. The water that was later on collected from water tank and koohl also showed similar *Naegleria*. CSF culture was negative for bacteria and computed tomography head done on 2nd day showed mild cerebral edema.

The child was started on IV amphotericin B (1 mg/kg/d IV for 21 days), fluconazole (12 mg/kg/d PO on day 1 followed by 6 mg/kg/day for total of 21 days), oral rifampicin (15 mg/kg/d PO for 21 days) and supportive care for 3 weeks. The child showed gradual improvement with normal sensorium in 3 days, afebrile and disappearance of neck rigidity after 6 and 8 days of treatment respectively. He was discharged and followed-up for 3 months with no residual CNS abnormality.

Primary amoebic meningoencephalitis is a fulminant and rapidly fatal disease that principally affects children and young adults caused by *N. fowleri*, an amoeba-flagellate found in most soil and freshwater habitats. Most of the cases reported in children as well as in adults has been fatal.[1,3] One of the key and most important factors for a successful outcome is the promptness in diagnosis and treatment of the patient. Delay in diagnosis and the fulminant nature of the disease results in few survivors.[4] In our case, the child presented with features of meningitis and altered sensorium with sub-acute course with history of swimming in stored fresh water. In a case of meningitis, we routinely do not perform wet mount microscopy but in our case history of swimming in stored fresh water with a sub-acute course of meningoencephalitis led to the investigation and early diagnosis, which is the most important factor for successful treatment of PAM. Early treatment was started, and the child recovered completely. Vargas-Zepeda et al.[2] also successfully treated a 10-year-old boy of PAM with IV amphotericin B, fluconazole, rifampicin, and dexamethasone. So, history taking that is perhaps a dwindling art can create a difference perhaps between death and survival and wet mount microscopy should be routinely done in a case of meningitis or meningoencephalitis with sub-acute course.
Abnormal U-shape course of central venous catheter

Sir,

A 50-year-old male patient with shortness of breath was referred from other hospital to our center. After doing all routine blood investigations and bedside chest X-ray in the casualty ward, patient was transferred to intensive care unit. Patient had triple lumen central venous catheter (CVC) in right internal jugular vein (IJV) which was inserted in outside hospital. However, there was no back flow in all 3 lumens, so it was removed. Chest X-ray revealed abnormal U-shape course of CVC [Figure 1]. Before inserting new central venous line, ultrasound of right IJV was done which showed large valve in right IJV [Figure 2]. Central line was inserted in left IJV under ultrasound guidance. Back flow in all 3 lumens of CVC was present.

Valve in IJV is present in 88–100% of cases. It inhibits retrograde flow from the right atrium to the brain. This valve prevents a sudden increase in the jugular venous pressure during positive pressure ventilation or conditions with raised abdominal pressure (e.g., ascites). Thereby it prevents cerebral congestion by avoiding excessive backward flow to the brain.

The types of valve leaflet can be unicuspid (1.4–16%), bicuspid (77–98%) or tricuspid (0–7%). There is a risk of persistent incompetence of the IJV valve by CVC. Valve damaged during IJV catheterization can be a site for thrombus formation. Large venous valve causing difficult central catheter placement has been reported. In the present case, large valve in IJV might have changed the course of guidewire in the reverse direction. CVC rolled over the guidewire acquired the same U-shape course of

References