Hydrocephalus Caused by H3N2 Type A Influenza Virus or Cerebellopontine Angle Schwannoma?

Sir,

We report the case of a 51-year-old woman who was admitted to the emergency department at the University Hospital Center Zagreb, Croatia, presenting with progressively worsening headache, nausea, dizziness, and ataxia. Ten days before the onset of symptoms she was discharged from another hospital, where she was treated with oseltamivire after type A influenza virus had been isolated from her saliva. An urgent brain magnetic resonance imaging (MRI) showed enlarged ventricles with radiographic features of hypertensive hydrocephalus. In addition, a medium-sized (2.5 cm × 3 cm) tumor, characterized radiologically as schwannoma, was noted in the left cerebellopontine angle, without signs of fourth ventricle compression or aqueductal stenosis [Figure 1a-d]. Lumbar puncture results were not suggestive of meningitis/encephalitis-cerebrospinal fluid (CSF) biochemistry values were within reference ranges. Immediately before the planned surgery, the patient became somnolent, with inadequate verbal responses, and developed a series of generalized tonic-clonic seizures, after which she was transferred to intensive care unit. External ventricular drainage (EVD) was inserted preoperatively. Gross total tumor resection with facial nerve preservation was performed through a retrosigmoid craniotomy, using intraoperative evoked potential monitoring. Histopathology confirmed that the tumor was a schwannoma (predominantly Antoni A tissue). Influenza virus type A, H3N2 subtype, was identified in the CSF obtained from the EVD using the polymerase chain reaction method (considered effective and sensitive).[1] Postoperative brain MRI showed a small tumor remnant and persisting ventricular enlargement. There were no radiological signs of encephalitis [Figure 2a-c]. The patient did not tolerate EVD weaning, requiring the placement of a ventriculoperitoneal CSF shunt with a programmable valve system (at 110 cm H₂O). Follow-up computed tomography showed a reduction in the size of the ventricular system [Figure 2d]. As her condition improved, she was transferred to a neurosurgical unit and subsequently dismissed from hospital neurologically improved: Glasgow Coma Scale 15, House-Brackmann grade II facioparesis, without headaches or other neurological symptoms.

To the best of our knowledge (PubMed search, August 2017, no language restrictions: “hydrocephalus” AND “influenza”), only one similar case of H3N2 type A influenza virus-related hydrocephalus has been described in the literature. De Santis et al.[2] reported a case of influenza-related cerebellitis with secondary obliteration of the sylvian aqueduct and triventricular hydrocephalus in a patient with serious systemic comorbidities who was discharged in a persisting coma state. Possible pathogenetic mechanisms underlying the
association between viral infections and hydrocephalus include ependymal destruction and aqueductal stenosis through selective ependymal cell infection, and periventricular and periaqueductal gliosis. Different influenza virus-related central nervous system infections have been described in the literature, including fatal encephalopathy, acute cerebellitis and severe neurological sequelae. Our patient had coincidental vestibular schwannoma, a condition known to be associated with changes in CSF dynamics and hydrocephalus – it is possible that the schwannoma acted as a predisposing condition and the viral infection triggered the acute hydrocephalus.

Influenza-associated hydrocephalus is an extremely rare condition that can trigger serious neurological disorders. Patients with comorbid conditions are at a greater risk of developing such complications.

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Conflicts of interest
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