Hypernatremia-induced Limbic System Damage

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A 58-yr-old woman developed an extreme acute hypernatremia (serum sodium level 200 mmol/l) with generalized seizures and coma after a laparoscopic intervention for a liver hydatid cyst excision complicated by cyst rupture and followed by a peritoneal sterilization of the cyst content with 30% hypertonic saline solution. Hemodialysis was started with correction of hypernatremia at a speed of 1 meq/l/h. Brain magnetic resonance imaging scan, performed within 24 h, is shown in the figure. Fluid attenuated inversion recovery images of the patient (panel A) are compared with those obtained from a normal subject at the same slice level for comparison (panel B). Diffusion weighted images of the patient (panel C) are also compared with those of a normal subject (panel D). Images in panels A and C showed juxtacortical edema in most regions of the limbic system network, namely the amygdala and hippocampus (short black arrows), mamillary body (white arrows), temporal pole, orbitofrontal cortex, insula (short white arrows), anterior cingulum (black arrowhead), and fornix (white arrowhead). The lesions appear hyperintense in diffusion weighted imaging, consistent with prominent cytotoxic edema.

Ten days later, a repeated neuroimaging study showed signs of symmetrical coagulative necrosis in the amygdala, hippocampus, and hypothalamus. The patient’s state of consciousness partially improved, with neurologic sequelae of amnesia and aphasia.

Severe hypernatremia may produce central pontine or extrapontine myelinolysis and neuronal damage, depending on the level and duration of hypernatremia and the rapidity of its onset.1 Neuroimaging findings presented here demonstrate a selective vulnerability of the regions belonging to the limbic system network to the effects of an acute and extreme hypernatremia. This condition might be sustained by a predisposing condition of the limbic system towards glutamate excitotoxicity, as suggested by observational studies.2,3

References