Cortical sclerosis presenting with dementia as a sequel of rapid correction of hyponatraemia

(A) (right) axial; (left) coronal enhanced T1 weighted MRI. Gyriform cortical enhancement: cortical laminar sclerosis. On the coronal scan, postoperative changes and enhancing tumour remnants of a pituitary macroadenoma can be seen. (B) Global cortical hypofixation on brain HMPAO scintigraphy.
We report the case of a previously healthy 53 year old woman who became chronically vegetative after transphenoidal pituitary surgery complicated by hyponatraemia. She was admitted to our hospital because of a speech disturbance and confusion. Ten days previously, she had developed a headache and isolated bitemporal hemianopsia. Brain MRI showed a heterogenous pituitary mass raising the optic chiasma. Transphenoidal partial hypophysectomy was performed. Two days later, MRI showed a hypersignal on T1 weighted images, exclusively inside the remaining pituitary tumour. Her level of consciousness after surgery was good. Histopathology of the removed pituitary tissue was consistent with a chromophobic adenoma. Three days later, the patient began to be confused. Intratable and inappropriate secretion of antidiuretic hormone developed, associated with hyponatraemia (serum sodium: 110mEq/l). She was treated with fluid restriction and infusion of physiological serum saline. Over 48 hours, serum sodium was normalised to 142 mEq/l; she experienced dysarthria and bradykinesia. She presented a global aphasia and severe frontal syndrome. Her speech output was reduced and she did not respond congruously to simple commands. Their was a lability of affect with stereotyped and perseverative behaviour. She put all objects she could reach in her mouth. The severity of her neuropsychological deficit did not permit us to perform any memory tests. No posturing was noted, but bilateral Babinski’s signs were present. Brain MRI disclosed no abnormal finding in the pons and basal ganglia but showed a gyriform cortical enhancement on the coronal enhanced T1 weighted images (figure (A)). Cerebrospinal fluid analysis was normal. HMPAO-tomography disclosed a complete cortical hypofixation in both frontal, parietal, and temporal lobes (figure (B)). Surprisingly, the passive P300 was recorded over midline derivations and the latency was significantly increased. Six months later, there was no improvement at neurological examination, or on brain MRI and SPECT. The clinical presentation and the functional imaging strongly suggest a new case of laminar sclerosis of Morel, without centropontine myelinolysis, due to rapid correction of hyponatraemia. To our knowledge, cortical sclerosis is a complication of chronic ethanol intoxication and has never been described after pituitary surgery with MRI and SPECT studies.

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