Letters

CT brain scan in acute water intoxication

Sir: Cerebral computed tomography (CT) in acute water intoxication has not yet been described, although clinical and EEG features of this condition are well known.¹⁻⁴ We present a typical case of this entity, in which CT scans were performed.

A 52-year-old female was admitted to a surgery ward, because of abdominal pain, headache and repeated vomiting. Her past medical history was negative, except for moderate arterial hypertension which had long been treated by diuretics (frusemide 40 mg/d). On admission, blood pressure was 160/100 mm Hg and the physical examination was normal except for mild tenderness in the right upper abdomen. Laboratory tests, including CBC, BRS, BUN, electrolytes, creatinine, glucose, diastase, proteins, triglycerides and enzymes were normal. Because of repeated vomiting she was intravenously infused with 4,400 ml of 5% glucose solution, within the first 24 hours.

The next morning she became suddenly restless and drowsy; her headache became severe and grand mal convulsive seizures appeared. On neurological examination, the patient was confused, changing from somnolence to psychomotor agitation, with mild neck rigidity and Kernig and Brudzinsky signs bilaterally. Her pupils were equal; there was no papilloedema. She showed conjugate eye deviation to the right. There was no paresis, deep tendon reflexes were brisk and there were bilateral extensor plantar reflexes. Lumbar puncture revealed clear, normal CSF. Laboratory tests showed hyponatraemia 114 mg/dl and hypokalaemia 2.6 mg/dl. The rest of the blood tests were normal. EEG showed diffuse irregular slow activity. CT showed narrowing of the third ventricle and small lateral ventricles representing diffuse brain oedema (fig a).

Following the clinical and CT diagnosis of water overload and brain oedema, the fluid intake was limited to 600 ml per 24 hours of hypertonic saline. Serum sodium increased gradually to 133 mg/dl within 48 hours, and the potassium level became normal. Her clinical and neurologic condition improved dramatically. Seizures dis-

appeared and her mental condition returned to normal with no meningeal or focal neurological signs. Follow-up EEG and CT examinations (fig b) ten days later were normal. Both EEG and CT examinations were performed in identical technical conditions.

This was a classical case of iatrogenic water intoxication. For several years the patient had been on diuretics. It is well known^{1,2} that the long standing use of diuretics leads to relative inappropriate ADH secretion with an increase of the extracellular volume and tendency to chronic hyponatraemia. This patient's water electrolyte balance was partially maintained until she vomited profusely, thus losing sodium and aggravating her latent hyponatraemia. Furthermore, she was given 4,400 ml of glucose solution without sodium or potassium and hence developed severe hyponatraemia and hypokalaemia with rapid expansion of the brain. EEG changes were those described in water intoxication.^{3,4} We present this case to demonstrate the CT confirmation of the diagnosis and prognosis. The first CT scan during the onset of the neurological picture, revealing diffuse brain oedema

Fig 1 (a) Cerebral CT, 30 June, 1983 (Plasma Na level 114 mg/dl). CT scan at the mid-convexity level shows small symmetrical lateral ventricles due to diffuse brain edema. (b) Cerebral CT, 11 July, 1983 (Plasma Na level 133 mg/dl). CT scan at the same level. The lateral ventricles have expanded to normal size.

with compressed ventricles, confirmed the diagnosis. In this CT scan the basal cisterns were not compressed. Recently this sign was described as a predictor of outcome in severe head injury.⁵ The second CT scan when the patient recovered was normal. From the radiological point of view, the entity of acute water overload should be included among the differential diagnosis of small lateral brain ventricles.

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Accepted 24 December 1984.

Cortical vasoactive intestinal peptide in relation to dementia in Parkinson's disease

Sir: Accumulating evidence has suggested analogies in the dementia of Parkinson's and Alzheimer's diseases. At least one third of the Parkinsonian population presents signs of intellectual deterioration.¹ Histopathological stigmata associated with Alzheimer's disease have been also observed at necropsy in brains from demented Parkinsonian patients.² Biochemical investigations on post-mortem material have revealed a decrease in choline acetyl-transferase (CAT) activity as well as in somatostatin concentrations in the cerebral cortex of both

Alzheimer³⁻⁵ and demented Parkinsonian patients.⁶⁻⁸ The biochemical comparison between the two diseases was further extended to vasoactive intestinal peptide (VIP), a neuropeptide highly concentrated in the human cerebral cortex.⁹ Since cortical content of VIP was reported to be unaffected in brains of patients with Alzheimer's disease,¹⁰ it appeared of interest to determine VIP levels in the frontal cortex of Parkinsonian subjects with and without dementia.

Brains from 24 control subjects with no evidence of neurological or psychiatric disease (mean age 73.2 ± 2.5 years, necropsy delay 12.3 ± 1.2 hours) and 23 Parkinsonian patients (mean age 72.6 ± 1.6 years, necropsy delay 14.8 ± 1.6 hours) were examined. Fourteen Parkinsonian patients were affected with intellectual deterioration diagnosed as previously reported.⁷ Tissue collection and dissection have been described elsewhere.⁷ The concentrations of VIP-like immunoreactivity (VIP-LI) were measured by means of a sensitive double-antibody radioimmunoassay method.¹¹ The VIP antiserum used for this study recognized the region 15-28 of the VIP molecule. CAT activity was estimated by radioenzymatic assay as previously described.⁶

As expected^{6,7} CAT activity was lower in the frontal cortex of Parkinsonian subjects when compared with controls (table). This increase probably reflected degeneration of the cholinergic pathway originating in the substantia innominata and projecting into frontal cortex.⁷ The deficit was greater in Parkinsonians with dementia suggesting that the lesion of the subcortico-cortical cholinergic system was implicated in the process of intellectual deterioration.⁷ Cortical VIP-LI concentrations were not significantly different from controls in Parkinsonian patients (table). No further significant difference was found when Parkinsonians were subdivided in non-demented or demented patients. These

data suggest that neurons containing VIP in the frontal cortex are neither affected in Parkinsonian patients nor involved in dementing process in Parkinson's disease, as previously reported for Alzheimer's type dementia.¹⁰

These observations emphasize the neurochemical similarities between Alzheimer's and Parkinson's types of dementia as far as cortical neurotransmitter systems are considered. Besides VIP other neuropeptides (CCK-8, Substance P, enkephalins) have been found in normal concentrations in brains from patients with both types of dementia.^{4,5,12} These results contrast with the damage of intra-cortical neurons, such as somatostatin containing neurons⁸ and subcortico-cortical (cholinergic, adrenergic) neuronal systems^{4-7,13} which are probably implicated in the dementing process for the two diseases.

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Table VIP-LI and CAT activity in control and Parkinsonian frontal cortex (Brodman area 9)

	Control	Parkinson's disease		
		all cases	non-demented	demented
CAT activity nmol/h/mg protein	5.98 $\pm$ 0.28 (12)	3.70 $\pm$ 0.28*	4.81 $\pm$ 0.39*	3.10 $\pm$ 0.26*
VIP-LI pg/mg protein	182.9 $\pm$ 9.94 (24)	176.5 $\pm$ 14.80 (23)	189.5 $\pm$ 29.99 (9)	168.1 $\pm$ 15.62 (14)

Values are the means $\pm$ SEM; the number of brains is indicated in parentheses. *Significantly different from control (student's *t* test; $p \leq 0.05$).