Short report

Cerebral ventricular volume during hyponatraemia

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SUMMARY In order to determine if the neurologic manifestations in chronic hyponatraemia result partly from brain oedema, we measured the cerebral ventricular volume before and after correction of hyponatraemia in eight patients with central nervous system manifestations. Only the three patients with seizures showed a clear change in the ventricular size and probably had brain oedema.

Cerebral oedema is thought partly to be responsible for the central nervous manifestations in hyponatraemia.1 In chronic hyponatraemia, neurologic manifestations could also be due to intracellular depletion of potassium and sodium per se.2 In one study, the degree of encephalopathy in acutely hyponatraemic rats was related to an increase in brain water content rather than to a loss of intracellular electrolytes.3 Acutely induced hyponatraemia is an unusual clinical circumstance, frequently iatrogenic, where irreversible brain damage is due to brain oedema and intracranial hypertension, as shown by occasional necropsies.4 In chronic hyponatraemia irreversible brain damage is uncommon.5 In order to determine if the neurologic manifestations in chronic hyponatraemia result partly from brain oedema, we studied the cerebral ventricular volume before and after correction of the hyponatraemia in eight patients with central nervous manifestations related to hyponatraemia. Of our eight patients, three who presented with seizures showed a clear change in the ventricular size. These hyponatraemic patients probably had brain oedema.

Patients and methods

Computed tomography was performed on admission in eight patients (mean age ± SD: 57 ± 12 years) with a serum hypo-osmolality (one diuretic-induced; six associated with a syndrome of inappropriate ADH secretion; one with “beer drinker’s potomania”), who presented central nervous manifestations. The CT scan was performed before the discovery of the hyponatraemia, or to rule out a neurological cause to the syndrome of inappropriate ADH secretion. CT scan was repeated after four days of normal natriaemia (Na ≥ 135 mmol/l) in all the patients. The serum sodium concentration was rapidly corrected in three patients with inappropriate ADH secretion, two of them presenting seizures, by an acute one-day treatment with urea6 followed by a chronic treatment with frusemide.7 Frusemide alone corrected the hyponatraemia in the three other inappropriate ADH secretion patients in three days.8 The last two patients were rapidly treated by salt repletion.

Ventricular volume was measured following the method described by Reid et al.4 The CT scan data were displayed on the television screen. The software “region of interest” facility was used to outline the ventricular region. The upper limit for CSF was a Hounsfield number of 20. The volume of third and lateral ventricles was calculated by determining the “areas” of CSF in each slice and summing the results over the six to eight contiguous slices which comprised third and lateral ventricles. The fourth ventricle was not included, as it was only outlined in one slice. The radiologist (AG) did not know the serum sodium concentration of the patients when he measured the ventricular volume. Two other radiologists were asked to examine the scans for ventricular volume evaluation; their results were in concordance with those rated by their colleague. Repeated measurements in the same patient by the same radiologist did not show a change of more than 18% between the lowest and the highest ventricular volume. The reported ventricular volume was the mean of three determinations for each patient.

Results

The table presents the diagnosis and some biological parameters before and after correction of hypo-osmolality in eight patients with neurological symptoms related to hyponatraemia. Figure 1 shows the
changes in ventricular volume after correction of the hyponatraemia. No significant modifications of the ventricular volume (mean ± SD: 51-6 ± 22 ml before and 51-4 ± 21 ml after serum sodium normalisation) were observed in five of the patients (mean ± SD: 111 ± 3 mmol/l; one patient had a diuretic-induced hyponatraemia, four a syndrome of inappropriate ADH secretion). These five patients presented central nervous manifestations (somnolence and confusion). Serial EEG performed in two of them showed a diffuse theta slowing (4 to 7 Hz) during the hyponatraemic episode which returned to normal after electrolyte correction.

In three patients with similar levels of hyponatraemia (Na = 117, 118 and 118 mmol/l), a clear modification of the ventricular volume appeared (which increased from 14-6 to 33 ml, from 46 to 58 ml and from 71 to 90 ml) after normalisation of the serum sodium concentration. These three patients had been sent to the hospital after seizure. Hyponatraemia was rapidly corrected with urea in the two patients with inappropriate secretion of ADH and with isotonic NaCl in the potomanic subject. This patient with beer-induced hyponatraemia had a low ethanol level on admission (1-2 g/l); his brain oedema was probably not alcohol-induced but related to his low serum sodium concentration, as it is well known that ethanol diffuses rapidly in all the tissues and does not induce an osmotic effect on the cell.

After correction of the serum sodium concentration, the central nervous manifestations regressed totally in all our patients. Although we cannot tell precisely the time each patient took to develop hyponatraemia, it was supposedly over a period of several days.

**Discussion**

The pathogenesis of the encephalopathy associated with chronic hypo-osmolality is probably multiple and is not well understood. Although controversial, the intracellular water increase in chronic hyponatraemia is usually evaluated at least at 7%. The difference in the values reported could be explained by the variety of animal models and methods.

In man, if there is a 7% increase in brain intracellular water content, the CT scan should enable the detection of a decrease of the cerebral ventricular volume during chronic hyponatraemia. An increase in brain volume leads to a compression of the ventricular system and the subarachnoid spaces, among
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other accommodative mechanisms. This compression is of 30% intracranially and of 70% in the spinal canal, if there is no obstruction.10

In five of our patients, no change was seen in the ventricular volume, which apparently suggests the absence of brain oedema. Nevertheless, considering Plum and Posner's suggestion that brain oedema from any aetiology could block the aqueduct and prevent the escape of the cerebrospinal fluid from the ventricular system, it is quite possible that the absence of decrease in the ventricular volume could be explained by a concomitant obstructive hydrocephalus.

The reason why some patients with similar hyponatraemic states develop a decrease in ventricular volume and some do not is not clear. It could be due to differences in the efficacy of cellular accommodative mechanisms, or more likely to a faster decrease in serum sodium concentration in the patients with seizures. But the question of whether it was the seizures that generated the cerebral oedema, or the oedema that generated the seizures is left unanswered.

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References