Inappropriate antidiuretic hormone secretion in Wernicke's encephalopathy

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Summary
A case of the syndrome of inappropriate secretion of antidiuretic hormone in association with Wernicke's encephalopathy is described, where both responded to intravenous vitamin B complex. Thiamine may be life-saving in hyponatraemia with neurological signs due to Wernicke's encephalopathy.

Introduction
It has been suggested that the syndrome of inappropriate antidiuretic hormone secretion might occur in Wernicke's encephalopathy (Shalhoub and Antoniou, 1969; Ebels, 1978; Leading Article, 1979). However, the authors are unaware of published evidence for this although Ebels mentions he has such evidence in a patient. A case is reported in which reversal of the syndrome of inappropriate antidiuretic hormone secretion occurred with intravenous vitamin B complex.

Case report
A 58-year-old white man, a known alcoholic, was admitted with a story of progressive unsteadiness and confusion over 3 weeks. He had been living rough and was drowsy, disoriented, malnourished and unkempt. He was on no therapy and unable to give a history.

The dramatic physical signs were severe truncal ataxia such that the patient could not sit up in bed, a bilateral abducens nerve palsy and absent pupillary reflexes to light and accommodation. There were no stigmata of chronic liver disease or evidence of acute liver failure. He was apyrexial without meningeal irritation. There was no evidence of fluid depletion or cardiac failure but there was slight pitting oedema at the ankles. His pulse was 95/min, regular of good volume and his blood pressure 160/85 mmHg.

Investigations revealed severe hyponatraemia (115 mmol/l), a low urea (3-4 mmol/l), a low serum osmolality (235 mmol/kg) and a high urinary osmolality (424 mmol/kg). The serum urate was low at 0-18 mmol/l. The serum aspartate aminotransferase, alkaline phosphatase, bilirubin, B12, folate, cholesterol, thyroxine, and TSH were normal. The albumin was low at 32 g/l and the y-glutamyl transferase slightly raised at 50 u/l.

The creatinine clearance was 82 ml/min. The 24-hr urinary sodium the day after admission was 85 mmol. A chest X-ray showed emphysema with a normal sized heart. A tetracosactrin (Synacthen) test was normal. An emergency pertechnetate brain scan showed possible increased uptake in the right lobe. A CT brain scan revealed considerable cortical atrophy and excluded a subdural haematoma.

A putative diagnosis of Wernicke's encephalopathy was made and intravenous therapy with vitamin B complex begun. Fluid restriction to 500 ml/day was instituted. The patient's conscious level and ataxia and eye signs rapidly improved through a phase of nystagmus. He also developed a diuresis as his plasma: urine osmolality ratio reversed. The serum sodium steadily rose to normal over 3 weeks while daily vitamin B therapy continued. Water restriction was abandoned after 48 hr.

At discharge 3 weeks after admission the patient had a slightly wide-based gait with some residual impairment of short-term memory and a tendency to confabulate.

Discussion
The characteristic clinical picture in a vagrant with alcoholism, cortical atrophy on CT scan and a rapid improvement following B vitamin complex, was felt to be diagnostic of Wernicke's encephalopathy, although studies for pyruvate concentrations and transketolase were not performed.

The inappopriate urinary osmolality in the face of serum hypo-osmolality, the low urate (Beck, 1979), absence of fluid depletion with normal renal, thyroid, hepatic and adrenal function is considered good evidence for the syndrome of inappropriate antidiuretic hormone secretion.

It has been suggested (Shalhoub and Antoniou, 1969; Ebels, 1978; Leading Article, 1979) that the syndrome of inappropriate antidiuretic hormone secretion might be expected to occur occasionally in Wernicke's encephalopathy as does hypothermia
(Philip and Smith, 1973). Victor, Adams and Collins (1971) found the characteristic lesions in the supra-optic nuclei of the hypothalamus in 16-7% and in the paraventricular nucleus in 35% of their patients with Wernicke’s encephalopathy at post-mortem.

The syndrome of inappropriate antidiuretic hormone secretion has been observed in various cerebral conditions and may present with confusion, fits and upgoing plantar responses (Bartter and Schwartz, 1967). It has been observed in hypothalamic lesions in particular, such as in acute intermittent porphyria with neuronal loss in the supra-optic and paraventricular nuclei (Perlroth et al., 1966), in neurohypophyseal choristoma (Haslett and Douglas, 1978) and in hypothalamic glioma (Brisman and Chuturian, 1970). Similarly the syndrome of inappropriate antidiuretic hormone secretion has been produced in goats by inducing lesions in the region of the supra-optic nuclei (Rundgren and FYhrquist, 1978).

Although hyponatraemia may give rise to neurological signs, an underlying neurological lesion causing the hyponatraemia should not be forgotten, least of all Wernicke’s encephalopathy where intravenous vitamin B complex may be life-saving. There are several cases in the literature of the syndrome of inappropriate antidiuretic secretion where Wernicke’s encephalopathy was not apparently excluded (Hamilton, 1978; Decaux et al., 1981).

**Acknowledgment**

We thank Dr Michael J. Williams for permission to report this patient who was under his care.

**Clinical reports**

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<th>Day</th>
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*On 24-hr urinary collection.

**References**


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