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was fully alert. Serum and urinary osmolality became normal, urinary specific gravity was 1005-1025. She recovered from ovarian hyperstimulation syndrome and laparotomy during the next month.

Brain MRI and CT performed during the next five years were normal, as were repeated neurological and psychiatric examinations. The patient's IQ was 126-130.

Serious seborrhöe with ascitis and hydrothorax due to ovarian hyperstimulation syndrome and haemoperitoneum due to tubal pregnancy, with hypovolaemia, anaemia, and hyposomolar serum concentrations masked an SIADH that was heralded by seizures, followed by a prolonged lehargic state. Collateral evidence of SIADH was obtained by normal creatinine clearance1 with urine hyperconcentration. The symptoms of CNS water intoxication, as usual,2 appeared during a sudden decrease in Na+ serum concentration, and were treated slowly to avoid central pontine myelinolysis. During SIADH, CT showed several patchy areas of hypolucency, resembling severe lesions of acute infarction or necrotic encephalopathy with brain oedema.3 4 Hypoxic-anoxic lesions are, however, usually caused by residual neurological or psychiatric deficits, and CT shows evolution of lesions, with ventricular enlargement and leucomalacia.5 In this patient the hypolucencies disappeared, the patient had no neurological or psychiatric alterations, and later CT and MRI did not show residual areas of altered signal corresponding to early hypolucencies. Furthermore, unlike the situation in hypoxic-anoxic lesions,6 the basal ganglia did not seem to be involved, and the ventricular system was not narrowed as in severe brain oedema. We concluded therefore that water intoxication induced CT images of patchy hypolucencies rather than the expected homogenized hypolucency.

Hashimoto's encephalopathy, This was originally postulated to be a distinct disease entity by Brain et al in 19661 and there have subsequently been case reports substantiating the hypothesis that it represents a unique condition.2 3 The characteristic features are a subacute onset of confusion with altered mental status, seizures, and occasionally events that respond to steroids and which occur in the context of high anti-microbial antibody titres.4 To date all the patients reported have been either euthyroid or hypothyroid at the time of presentation. We present a patient with Hashimoto's encephalopathy with pronounced thyrotoxicosis, that was successfully managed with steroids, carbimazole, and propranolol.

A 49 year old woman presented with a six month history of weight loss and a three month history of proximal arm pain and hand tremor. Two weeks before admission she developed a progressive left sided weakness involving the arm and leg in conjunction with a left hemianesthesia. On examination at admission she was flushed, feverish, and had hyperventilation, a hypnagogic state. Her laboratory findings on presentation included a haemoglobin of 12·0 mmol/l, white cell count of 7·4×10⁹, platelets 207×10⁹, liver function test results of aspartate aminotransferase 25 IU/l, alanine aminotransferase 18 IU/l, total bilirubin 1·4 µmol/l, plasma bicarbonate 22 mmol/l, serum creatinine 0·27 mmol/l, and normal sodium, potassium, chloride and urea concentrations. Serum thyrotrophin was 0·006 mU/l and thyroxine 10·6 μg/l. A computed tomographic scan of the brain showed no abnormality.

Thyrotoxic Hashimoto's encephalopathy

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Letters to the Editor

Wasting after aneurysmal subarachnoid hemorrhage recently it has become clear that hyponatremia who showed remarkable changes in urine production during surgery. On day 9 she was found unconscious with respiratory failure and fluid loss were fully compensated by 0.9% NaCl infusion. On day 9 she was developed a progressive polyuria of up to 15000 ml/day and the patient regained consciousness and she gradually recovered from a mild aphasia and right facial weakness. However, from day 12 onwards she again developed a progressive polyuria of up to 21 200 ml per day (on day 22) and a 24 hour renal sodium loss of 2630 mmol. The patient had fully recovered two months after the operation.

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A patient with cerebral autosomal dominant arteriopathy with subcortical infarcts and leuconecephalopathy (CADASIL) confirmed by sural nerve biopsy

“A cerebral autosomal dominant arteriopathy with subcortical infarcts and leuconecephalopathy” (CADASIL)1 is a newly defined syndrome characterised, in the absence of hypertension, by recurrent subcortical ischaemic strokes and by peculiar infarcts and leuco encephalopathy. The diagnosis is based on clinical criteria,2 recent genetic linkage analysis3 has assigned the disease locus to chromosome 19. Around 120 small and large vessels were counted in the endoneurial and epineurial spaces. The largest epineurial arteries (size 14-15 mm) appeared normal. Small epineurial and perineurial spaces were often thickened with the cell membrane of an adjacent pericyte to the cell membrane with the electron microscopy showed changes in the cell membrane. The arteriopathy of CADASIL is apparently not restricted to brain vessels as identified vascular lesions have been found in small myocardioc arterial and myocardioc collateral arteries (fig 2A). Most of these granules were on the abluminal surface of pericytes but without the presence of myelin degradation products along the outer aspects of the vessel walls (fig 2A).

Electron microscopy showed changes in a few epineurial vessels, consisting of electron dense cytoplasmic granules that were connected to the outer aspects of the vessel walls (fig 2A). The density of myelinated fibres was 6600/mm2 (normal range for the sural nerve for this age 6000-8000/mm2). Myelin degradation products were not encountered.

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3 Family history showed that the mother of the patient died at the age of 52 with a history of familial CADASIL. The patient had M1 change to the index patient, and had had recurrent episodes of aphasia, headache, and hemianopia. Six members of this family, three affected and three healthy, have been genotyped with eight chromosome 19 markers spanning the CADASIL interval. No recombinant was found with any of those markers. Maximum lod scores were obtained with markers D19S226, D19S253, and D19S119, strongly suggesting that this family is linked to the CADASIL locus.