was fully alert. Serum and urinary osmolarity 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.

Severe serositis with ascites and hydrothorax due to ovarian hyperstimulation syndrome syndrome and haemoperitoneum due to tubal pregnancy, with hypovolaemia, anaemia, and hyposmolar serum concentrations masked an SIADH that was heralded by seizures, followed by a prolonged lethargic state. Collateral evidence of SIADH was obtained by normal creatinine clearance[2] with urine hyperconcentration. The symptoms of CNS water intoxication, as usual,[2 3] appeared during a sudden decrease in Na^+ serum concentration, and were treated slowly to avoid central pontine myelinolisis. During SIADH, CT showed several patchy areas of hypolucency, resembling severe lesions of acute hypoxic-ischaemic encephalopathy with brain oedema.[4 5] Hypoxic-anoxic lesions are, however, usually caused by residual neurological or psychiatric deficit, and CT shows evolution of lesions, with ventricular enlargement and leucomalacy.[4 5] In this patient instead 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,[4 5] 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 homogeneous hypolucency.

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Thyrotoxic Hashimoto's encephalopathy

Thyroid disease is associated with several neurological disorders,[1] of which one of the rarest and least well understood is Hashimoto's encephalopathy. This was originally postulated to be a distinct disease entity by Brain *et al* in 1966[2] and there have subsequently been case reports substantiating the hypothesis that it represents a unique condition.[3–5] The characteristic features are a subacute onset of confusion with altered consciousness, seizures, and stroke-like events that respond to steroids and which occur in the context of high anti-microsomal antibody titres.[3] 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 propanolol.

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 hemianaesthesia. On examination at admission she was flushed, feverish, and tachycardic with a hyperdynamic circulation. Her thyroid gland was slightly enlarged but there was no associated bruit. Cranial nerve examination disclosed left visual inattention as the only abnormality. Limb examination showed a moderately severe left hemiparesis with left sensory inattention, generalised hypereflexia, and bilaterally extensor plantar responses. She had wasting of the shoulder girdle muscles and adhesive capsulitis of the shoulder joints bilaterally. In the days immediately after admission she became drowsy, confused, and had florid visual hallucinations, while independently having runs of paroxysmal atrial fibrillation. As a result of the original negative findings (see later) dexamethasone (12 mg/day) and acyclovir were started with the presumptive diagnosis of an encephalitis or vasculitis. On this regime she made a dramatic improvement, which was further enhanced by the treatment of her thyrotoxicosis on receipt of her thyroid function tests. The introduction of carbimazole and propanolol was then followed by a reduction in the dexamethasone and a cessation of the acyclovir. Attempted steroid weaning over subsequent days provoked a recrudescence of her focal symptoms on two occasions, with weakness of her right arm. Eventually the patient was stabilised on prednisolone (40 mg/day) and discharged on a slowly reducing course with no relapses three months after discharge.

Investigations performed during her inpatient stay showed that full blood count, erythrocite sedimentation rate, urea, electrolytes, glucose, liver function tests, and serum immunoglobulins were normal. Protein electrophoresis showed an acute phase response with a C reactive protein of 32 mg/l. Her autoantibody screen and VDRL/TPHA serology were negative, but her thyroid function tests showed her to be thyrotoxic with TSH less than 0·03 U/l, free T4 >80 pmol/l, and free T3 41 pmol/l. Her thyroid microsomal antibodies were positive at a titre of 1:6400. Her CSF analysis was normal with negative oligoclonal bands and repeated blood cultures were negative. Her chest radiograph was normal but her ECG showed a sinus tachycardia with episodes of paroxysmal atrial fibrillation. Her EEG showed occasional brief bursts of frontal slow activity which spread posteriorly and brain CT with contrast and MRI with gadolinium were normal. In addition a transthoracic and transoesophageal ECG along with MRI of her heart were all normal.

These results show that she had a pronounced thyrotoxicosis with antimicrosomal antibodies. There was no evidence for any fixed structural lesion within the CNS accounting for her neurological condition as evidenced by her normal brain CT and MRI.

Autoimmune thyroid disease can be considered as a range of clinical disorders reflecting the variety of autoantibodies present. Hashimoto's disease is characterised by the presence of thyroid antimicrosomal antibodies and has rarely been associated with an encephalopathic process of unknown aetiology. All previously described patients have either been euthyroid or hypothyroid and this is the first description of an encephalopathy in combination with thyrotoxicosis. As the mechanism of encephalopathy is uncertain the term thyroid related encephalopathy is preferable. Although atrial fibrillation was present in our patient, the normal heart and head imaging argues against an embolic cause for her condition. Furthermore, her remarkable steroid responsiveness suggests an autoimmune cause for her fluctuating multifocal encephalopathy.

Various mechanisms have been postulated to account for this unusual condition. One possibility is demyelination, which can virtually be discounted on the basis of our results as both MRI and CSF were normal. More likely explanations are either a multifocal abnormality of cerebral perfusion or a patchy defect of metabolism.

This patient completes the repertoire of thyroid states seen in thyroid related encephalopathies and emphasises the need to assess thyroid function and autoantibody status in patients presenting with encephalopathy and stroke-like events in the absence of structural or infective aetiologies.

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Cerebral salt wasting syndrome

Excessive natriuresis, resulting in hyponatraemia and polyuria, is an often recognised complication after subarachnoid haemorrhage. Initially this was attributed to inappropriate antidiuretic hormone (ADH) secretion resulting in water retention, but