Hashimoto's thyroiditis associated with acquired idiopathic demyelinating polyradiculoneuropathy

Sir,

Simultaneous presentation of acquired idiopathic demyelinating polyradiculoneuropathy (IDP) and Hashimoto's thyroiditis suggests that IDP has an autoimmune pathogenesis.

A 51 year old man presented with a 5-week history of weakness, initially in the legs and progressing to involve the arms. No recent viral infection or immunization had occurred. Two weeks before admission he had seen his general practitioner for the leg weakness and tiredness but had no other symptoms of hypothyroidism. At that time, thyroid stimulating hormone was raised (34.8 mU/l, normal range 0.4–6.0) and free thyroxine was within normal limits (10.4 pmol/l, normal range 8.8–23). There was no family history of thyroid or neurological disease and previously he had been healthy except for mild hypertension treated with atenolol. Thyroxine 50 μg/day was commenced one week prior to hospital admission but his weakness progressed. On examination, the cranial nerves were normal. The limbs displayed symmetrical, distally predominant weakness affecting the legs more than the arms, with areflexia and flexor plantar responses. He was unable to stand unaided. Light touch and pain sensation were decreased on the anterior aspects of both thighs. Forced vital capacity was 3.3 litres. The remainder of his examination, including the thyroid gland, was unremarkable. The cerebrospinal fluid (CSF) contained 2.24 g/l protein and 4×10^6 lymphocytes/l. Electrophysiology showed absent F waves in the right and left tibial nerves, with slight delay in the right median nerve (33.2 ms), and mild slowing of motor conduction velocity of 47 m/s in the right median nerve and 35 m/s in the right medial popliteal nerve. Sensory action potentials were normal in the right sural (12 μV) and radial (30 μV) nerves. Routine haematological and biochemical parameters were normal. Autoantibodies were found against thyroglobulin (titre $>$ 1:5120) and thyroid microsomes (strongly positive). Antibodies against parietal cells, mitochondria, smooth muscle cells, reticulin and nuclei were absent. He was treated with prednisolone 60 mg/day and after 6 days the strength of his hands improved. Three months later he became able to walk 5 m without aid and has continued to improve whilst slowly decreasing the prednisolone. Four months after the onset he had not relapsed.

The clinical and neurophysiological picture and CSF results supported the diagnosis of an acquired IDP. The initial 5-week history of progressive weakness suggested a subacute form of acquired IDP rather than Guillain-Barré syndrome.[1] The occurrence of hypothyroidism with a high titre of antibodies to thyroid tissue is pathognomonic of Hashimoto's thyroiditis.[2] There has been one previous report of an association between 'autoimmune' hypothyroidism and chronic IDP,[3] and three reports of hypothyroidism and Guillain-Barré syndrome.[4–6]

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Fatal rupture of a subcapsular liver haematoma in a patient treated with anisolylated plasminogen streptokinase activated complex

Sir,

Haemorrhage is the major specific adverse effect of thrombolytic therapy.[1] In a recent AIMS Study[2] on the long term effects of intravenous (i.v.) anistreplase in acute myocardial infarction (AMI) bleeding was usually minor and related to venepuncture sites. This was easily controlled by minor pressure and in only 10 out of 1258 randomized patients was transfusion for such episodes necessary with no observed difference between treated and placebo groups. Major haemorrhagic complications associated with significant mortality are usually intracranial or intraperitoneal.[1,3] We report a case of massive intraperitoneal bleeding from a spontaneous rupture of a subcapsular liver haematoma as a direct cause of death.

A 77 year old man was admitted (0 hours) with an acute inferior myocardial infarction and entered into ISIS 3.[4] He was randomized to treatment with aspirin, heparin and anisolylated plasminogen streptokinase activator complex (APSAC). His clinical course was complicated in the initial 6 h by refractory chest pain which was successfully treated with 24 h of i.v. heparin and ventricular tachycardia which was controlled with lignocaine. Clotting screen during this time was normal. After 36 h he was well and ISIS heparin was reintroduced. Aspirin was stopped after 72 h for an episode of nausea, vomiting and abdominal pain. His subsequent clinical course was uneventful, he was mobilized and preparation for discharge was made. However, 120 h after admission he suffered a cardiac arrest from which resuscitation was unsuccessful.

At autopsy there was a haemoperitoneum which was due to a rupture of one of several subcapsular liver haematomas. Death was thought to be due to primary rupture rather than resuscitation since the quantity of blood in the abdominal cavity was too large to be accounted for by the size of the haematoma. The remaining abdominal viscera were normal with no other evidence of haemorrhage.