hepatitis were all negative and serum copper was within the normal range.

All the initial tests for common causes of metabolic encephalopathy were thus negative. A diagnosis of Hashimoto’s encephalitis was based on clinical picture, high antithyroid antibody titres, altered thyroid functions and raised CSF proteins. After 4 days’ treatment with oral prednisolone 60 mg per day he was oriented and behaving much more normally. On repeat testing TSH had risen to 85 mU/L, FT4 had fallen to 3.2 nmol/L. He was started on thyroxine. Before discharge his Mini Mental State Examination score was 29/30 and the EEG was within the normal range.

COMMENT

The first case of ‘Hashimoto’s encephalopathy’ was reported in 1966.1 In the few cases on record since then, the mean age at presentation was 41 years. This condition has been reported in children and has a female preponderance (ratio 3.6:1).2

The diagnosis is challenging to make since the underlying thyroid disease is often subclinical and the symptoms mimic other neurological conditions. The encephalopathy tends to present acutely with confusional state, focal or generalized seizures and stroke-like episodes.3 Other presentations are dysarthria, hallucinations, stupor, headaches and myoclonus.3 The diagnosis is supported by the finding of raised antithyroid antibodies (antithyroglobulin, antithyroid peroxidase, anti-TSH-receptor, anticyttoplasmic).3 The main EEG abnormalities are generalized slowing, frontal rhythmic slowing and triphasic waves. The cerebrospinal fluid protein is above normal in up to 75% of patients and oligoclonal bands may be seen.2 MRI scans are normal in most of the patients and the reported abnormalities include generalized cerebral atrophy and reversible subcortical signal abnormalities. Single photon emission computed tomography has shown multiple areas of hypoperfusion in a few cases.

Hashimoto’s encephalopathy is thought to be due to an autoimmune vasculitis, and this notion is backed by the identification of z-enolase autoantigen. Anti-z-enolase antibodies are associated with other forms of autoimmune vasculitic diseases including systemic lupus erythematosus.4 Some workers object to the term Hashimoto’s encephalopathy on the argument that thyroid autoantibodies may simply be a marker for other autoantibodies, as yet unidentified, that cause cerebral vasculitis. In the absence of proof of a causal connection between thyroid autoantibodies and encephalopathy, a more appropriate name might be encephalopathy associated with autoimmune thyroiditis.5 Steroids seem an effective treatment in these conditions, though no formal trials have been conducted. Other immunosuppressants have been reported effective. Some patients with hypothyroidism have responded completely to levothyroxine.6

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Acute abdomen from a Meckel lipoma

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In adults but not children, small-bowel intussusception is usually traceable to a physical lesion. We report a causation that does not seem to have been described before.

CASE HISTORY

A man aged 28 came to hospital after 24 hours of profuse rectal bleeding (bright red blood) associated with colicky central abdominal pain, vomiting and diarrhoea. Over the preceding twelve months he had been troubled by intermittent crampy abdominal pains about which he had not sought medical advice. There was no medical or family history of note. On examination he was pale, sweaty and in obvious discomfort, and an ovoid mass 10 cm in maximum...
diameter was palpable in the peri-umbilical region. The abdomen was generally tender but clinical peritonism was absent. His haemoglobin was 7.5 g/dL. Plain radiographs of the chest and abdomen were unremarkable but CT revealed a complex mass, consistent with the palpable lesion, involving small bowel and proximal large bowel. Radiological features were suggestive of an ileocolic intussusception, although no definite lead-point or causal lesion was identifiable (Figure 1). At laparotomy the presence of an enterocolic intussusception was confirmed, and when it was reduced by gentle manual traction the lead-point was identified as a small jejunal lesion that had travelled via the ileocaecal valve into the ascending colon. A standard resection with primary anastomosis was performed to remove the abnormal and intussuscepted region of small bowel. Pathological examination of the resected specimen revealed an inverted Meckel’s diverticulum that contained a lipomatous lesion about 30 mm in diameter, arising from its tip (Figure 2).

COMMENT

Intussusception is seen mainly in childhood, when it is idiopathic in 95% of cases. In the rare adult cases a causal lesion is identified in as many as 90%. Symptoms of intussusception in the adult are usually protracted, over weeks or months, and include abdominal pain, nausea, vomiting, rectal bleeding, altered bowel habit and weight loss. The paediatric triad of acute abdominal pain, a palpable sausage-shaped abdominal mass and ‘redcurrant jelly’ stools is seldom observed in adults though it can arise in acute-on-chronic cases such as we report here. On CT, the bowel-in-bowel or egg-in-cup sign is diagnostic, and occasionally the underlying lesion is identifiable. Usually this proves to be benign—a neoplasm, lymphoid hyperplasia, or an adhesion. Although an inverted Meckel’s diverticulum as a causal lesion has been described several times, we have found no previous report of an association with a lipoma as the lead-point.

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