Sir: Essential thrombocythaemia can be associated with arterial and venous thrombosis.1 The association with lateral sinus thrombosis and intracranial hypertension has not previously been described in this disease. We report such a case in a patient who also had evidence of portal vein thrombosis.

A 31-year-old man presented with intermittent epigastric pain. Marked splenomegaly and a microcytic hypochromic anaemia with a haemoglobin of 10 g% were noted. Bone marrow examination suggested early myeloproliferative disease. Eight months later he had a haematemesis and endoscopy showed oesophageal varices. White cell count then was 5·0 x 10^9/l with normal differential and a platelet count of 442 x 10^9/l. An ultrasound examination of the liver showed a highly echogenic area in the region of the porta hepatitis. CT scan of the abdomen showed gross splenomegaly and the portal vein did not increase in density after contrast injection supporting a diagnosis of portal vein thrombosis. Four months later, during an admission for injection of oesophageal varices he developed diarrhoea and vomiting. Salmonella was isolated and he was treated with ampicillin and metronidazole. Seven days later he complained of an initially predominantly right sided headache and photophobia. Neurological examination was normal. A CT scan of the brain with enhancement was normal. A lumbar puncture in the recumbent position showed a CSF pressure of 25 cm of H2O, (normal up to 18 cm of H2O), normal protein, cell content and glucose. At this time his platelet count which had been persistently over 500 x 10^9/ml had reached 792 x 10^9/ml. Generalised headaches increased by cough persisted and further lumbar punctures over the ensuing two months showed pressures of 28 and 24 cm of H2O. After each of these the headache was temporarily relieved. During this time he was treated with acetzolamide. Digital subtraction angiography with intravenous injection of contrast showed adequate filling of arterial cerebral vessels but a filling defect due to a thrombus in the left lateral sinus (ng). A further bone marrow aspirate and trephine showed active haemopoiesis with increased megakaryocytes and increased reticulin stain. The appearances were considered characteristic of essential thrombocythaemia (Prof J Barrett). A single dose of 50 mg oral busulphan was administered. The platelet count has remained between 150 and 300 x 10^9/ml and no recurrence of his symptoms has been observed for three months.

The two venous thromboses within a year were probably secondary to essential thrombocythaemia. One previously reported case of intracranial hypertension in this condition was interpreted as "benign" or "pseudo-tumour cerebri".2 Other recorded neuro-logical complications include stroke, transient ischaemic attacks, confusional states, migraine, epilepsy, polyneuritis and radioculomypelopathy.3 5 We suggest that the lateral sinus thrombosis shown by digital subtraction angiography was the cause of the observed intracranial hypertension. Dehydration may have also been involved in the pathogenesis of this thrombosis as vomiting

Lateral sinu thrombosis and intracranial hypertension in essential thrombocythaemia


21 Lateral oblique view of the skull during digital subtraction angiography (intra venous study). The arrows indicate a clot shown as a filling defect within the left lateral sinus.

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and diarrhoea preceded the onset of headache by a week.

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calised to the right iliac fossa. He was apyrexial. Examination of the abdomen revealed a distended caecum and a tender, loaded sigmoid colon. The bowel sounds were increased. A diagnosis of constipation was made and the patient managed conservatively. The dose of atropine was reduced and the symptoms settled. He remained well for a further 3 months when he was admitted with a 6 hour history of generalised, colic-like abdominal pain. This was accompanied by non-bloody diarrhoea and vomiting. On questioning he reported recent episodic diplopia and weakness. At the time of admission the dose of pyridostigmine was 685 mg daily in divided doses. On examination he was sweating but apyrexial. The pulse was 60 beats per minute and his pupils were constricted and reacting briskly. Abdominal examination revealed generalised tenderness. There was no guarding and the bowel sounds were increased. The symptoms and signs were thought to be consistent with a cholinergic excess and he was managed with intravenous atropine. The pain resolved and the bowel sounds returned to normal. Eight hours later he developed further abdominal pain which was localised to the right iliac fossa and he passed fresh blood per rectum. The temperature rose to 37.5°C. Examination revealed rebound tenderness in the right iliac fossa. A radiograph of the abdomen demonstrated dilated small bowel loops.

The haemoglobin concentration was 14.9 gm/dl, leucocyte count 10,400, serum amylase 65 international units, sodium 144 mmol/l, potassium 4.1 mmol/l, chloride 106 mmol/l, bicarbonate 27 mmol/l and urea 3.9 mmol/l.

A laparotomy was performed and revealed a 10 cm caeco-colic intussusception which was easily reduced. The bowel was viable and no polyp was present. The remainder of the laparotomy was normal. An appendicectomy was performed and the caecum fixed to the posterior abdominal wall. After operation the patient required temporary ventilatory support in the Intensive Therapy Unit, but his post-operative course was otherwise uneventful. He was re-established on pyridostigmine at a slightly reduced dose, and continued on atropine.

At follow up in the Out-patient Department he remained well with no further episodes of abdominal pain.

Intestinal intussusception is rare in adults and is usually associated with an intestinal polyp.1 We present a case of caeco-colic intussusception occurring in an adult in the absence of a polyp.

Pyridostigmine, by virtue of its parasym-

References


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Caeco-colic intussusception in a patient with myasthenia gravis

Sir: A 33-year-old Caucasian male presented with a 4 week history of dysphagia, fluid regurgitation, weakness and dysarthria. These symptoms resolved following the intravenous administration of 10 mg of edrophonium. A diagnosis of myasthenia gravis was made and the patient treated initially with pyridostigmine 90 mg 4 times daily and atropine 0.6 mg twice daily. He underwent plasmapheresis and thymectomy. Histology of the thymus tissue was normal. Assay for acetylcholine receptor antibodies was positive.

Management of the myasthenia gravis was difficult and required frequent alterations in the dose of pyridostigmine. He required, in addition, prednisolone 25–50 mg daily and azathioprine 50 mg twice daily.

Six months after his initial presentation he attended with abdominal pain which he lo-
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