Letters

and diarrhoea preceded the onset of headache by a week.

DEBORAH MITCHELL

JACQUELINE FISHER

D IRVING*

BG GAZZARD

RJ GUILLOFF

*Departments of Neurology and Gastroenterology,
Westminster Hospital,
Horseferry Road,
London SW1P 2A UK*

**Department of Radiology,
Lewisham Hospital,
London, UK*

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Accepted 6 July 1985

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-

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 parasymp-

athetic activity, increases gastro-intestinal motility and can produce abdominal pain. We have been unable to find any previous reports of bowel intussusception as a complication of anti-cholinesterase treatment, but feel that this drug may have contributed to the formation of an intussusception in this case. Indeed, upon initial assessment, it was felt that the patients symptoms were due to increased gastro-intestinal motility and they resolved, albeit temporarily, after the administration of intravenous atropine. The fact that the patient was also on long-term corticosteroids may have masked abdominal signs. We should like to draw attention to this unusual complication of anti-cholinesterase treatment.

We thank Dr. David Maxwell Park for permission to report a patient under his care.

NEIL MURRAY

CHRISTOPHER BAYNES

*Departments of Medicine and Neurology,
Southend General Hospital,
Prittlewell Chase,
Westcliffe-on-Sea,
Essex, SS0 0RY, UK*

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Accepted 12 June 1985