Acute Colonic Pseudo-Obstruction In An Elderly Alcoholic Man

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Citation

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Abstract

Acute colonic pseudo-obstruction or Ogilvie's syndrome is a rare clinical disorder of unknown aetiology which responds well to conservative measures. Occasionally pharmacotherapy in the form of cholinesterase inhibitors or colonoscopic decompression may be required. Acute colonic pseudo-obstruction has not been reported previously to present with delirium in the non post-operative setting.

INTRODUCTION

Acute colonic pseudo-obstruction is a rare disorder characterised by clinical and radiological features of acute large bowel obstruction in the absence of obvious colonic disease or mechanical obstruction.1 The precise mechanism by which colonic dilatation occurs in patients with acute pseudo-obstruction is unknown. Acute pseudo-obstruction is associated with conditions such as trauma, sepsis, neurological disorders, abdominal and spinal surgery and alcohol abuse.2 We report a case of an elderly man presenting with delirium secondary to acute colonic pseudo-obstruction on a background of longstanding alcohol abuse.

CASE REPORT

A 73-year-old man who lives alone presented to hospital with confusion and agitation. His past medical history was significant for a longstanding history of alcoholism consuming 2-3 litres of beer a day for 15 years and for the absence of prior surgery. He had ceased alcohol intake a year ago. Blood tests detected hypokalaemia (2.9 mmol/l). The rest of his laboratory investigations were unremarkable. His heart and lung examination was normal. On abdominal examination he had a markedly distended and tympanic non-tender abdomen with diminished bowel sounds. His abdominal x-ray showed the presence of dilated colonic loops and air throughout all colonic segments. His caecal diameter was recorded at 12 cm. The diagnosis of acute colonic pseudo-obstruction was made.

During admission the patient developed increasing abdominal distension with associated generalised tenderness. Abdominal x-ray revealed an increase in his caecal diameter to 15 cm in the absence of intraperitoneal free air (Figure 1 and 2). Initial conservative management included potassium replacement, the withholding of enteral feeding, instituting intravenous fluids, nasogastric suction and positioning the patient prone.

After 24 hours of failure of passage of faeces/flatus, pharmacological measures were trialled. Neostigmine 2.5 mg was administered intravenously under cardiac monitoring without a clinical response. Subsequent colonoscopic decompression revealed a grossly dilated colon with small submucosal ischaemic haemorrhages seen in the mid transverse colon. Colonoscopic decompression produced a dramatic reduction in colonic diameter and abdominal girth, with prompt passage of air and faecal matter.

The patient gradually recovered from his delirium and remained free of further abdominal distension and pain.

Figure 1

Figure 1: Abdominal X-ray supine: A massively air-filled dilated large bowel consistent with pseudo-obstruction

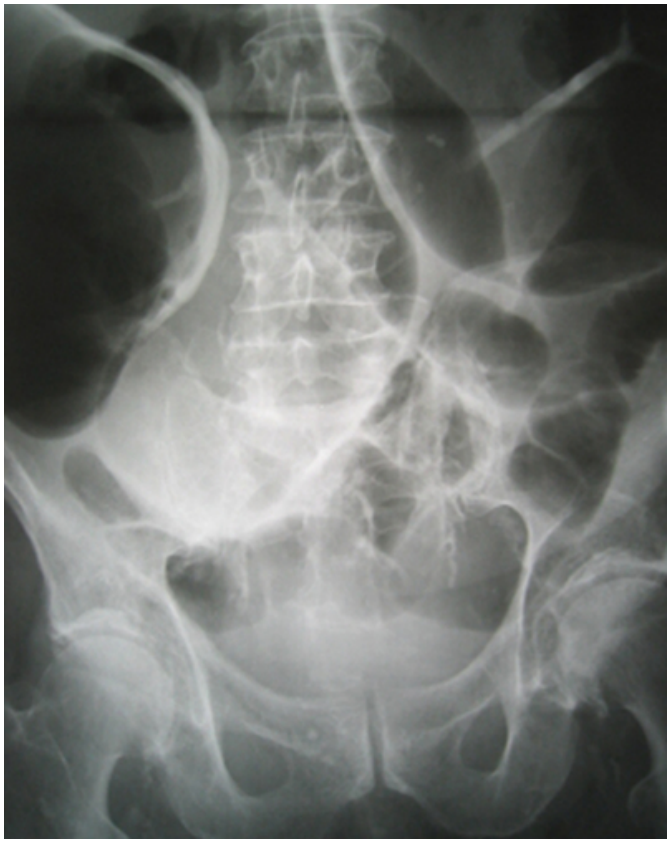
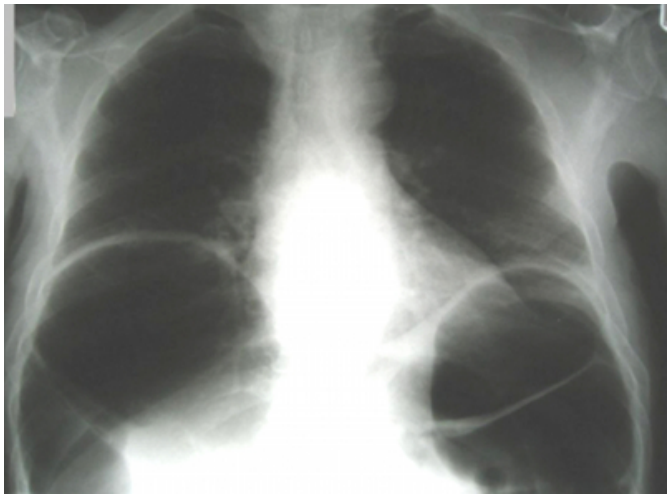


Figure 2

Figure 2: Chest X-ray erect: Elevation of the diaphragm bilaterally due to the pseudo-obstruction with no free intraperitoneal air



DISCUSSION

We described a case of a geriatric patient presenting with delirium caused by an acute colonic pseudo-obstruction. The symptoms of acute colonic pseudo-obstruction vary. Mild to moderate abdominal pain is present in the majority of cases and nausea and vomiting, constipation and even diarrhoea can be clinical manifestations.2 Abdominal distension is always present and often progressive. Delirium to date has not been reported as an initial presenting feature in the non post-operative setting.

The pathophysiology of acute colonic pseudo-obstruction is unclear but is thought to relate to an autonomic imbalance either with increased sympathetic tone or parasympathetic suppression resulting in decreased motility.3 It is more common in elderly patients and associated with underlying diseases such as trauma, infection, cardiac disease, spinal surgery, neurological disorders and may occur in patients with a history of alcohol abuse.2 Alcohol use may contribute to the development of colonic pseudo-obstruction via a toxic enteric neuropathy.4

Mechanical obstruction must be ruled out when making the diagnosis of acute colonic pseudo-obstruction. There is an argument for the use of a barium contrast enema in this respect although in the appropriate clinical context plain abdominal radiographs with classic findings are seldom incorrect.5 Plain abdominal radiographs show marked colonic distension with the finding of air throughout all colonic segments including the rectosigmoid.

The majority of patients with acute pseudo-obstruction resolve with conservative measures. These include treating concomitant disease such as infections, maintaining the patient on intravenous fluids, withholding enteral feeds, positioning manoeuvres, correcting electrolyte imbalances, discontinuing any offending drugs especially opiates and anticholinergics, and consideration of a nasogastric/rectal tube.6

Intravenous neostigmine has been demonstrated to be an effective pharmacological agent in the treatment of acute pseudo-obstruction when conservative measures have failed.7 Neostigmine increases the availability of acetylcholine at neuro-neuronal synapses and neuromuscular junctions in the enteric nervous system and this effect is accompanied by enhanced smooth muscle contractions.8 The administration of neostigmine must be carried out under appropriate cardiac monitoring, as bradyarrhythmias are a recognised complication. Atropine should always be immediately available.

Colonoscopic decompression is indicated in patients such as ours who fail neostigmine or who have contraindications to

neostigmine use. Surgical referral is indicated once pharmacological and colonoscopic measures are unsuccessful or if there is evidence of significant ischaemia or perforation.6

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