CASE REPORT

Is constipation a trivial matter?

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Accepted 17 January 2016

SUMMARY

Constipation is a common symptom of patients presenting to the emergency department. If poorly managed, it can have significant consequences. A 50-year-old man was admitted to the emergency department following cardiac arrest. His medical history included a diagnosis of cerebral palsy and multiple admissions to hospital with constipation. Clinical examination revealed a grossly distended and tense abdomen. Circulatory supply to the lower limbs was acutely impaired. CT scan revealed massive dilation of the large bowel due to faecal loading with compromised circulation to the lower limbs. Despite aggressive attempts at resuscitation, the patient continued to deteriorate and passed away. On reflection, a diagnosis of abdominal compartment syndrome secondary to severe constipation was made. This case highlights the vigilance required when managing what is often felt to be a trivial complaint and the considerable consequences when there is a failure to do so.

BACKGROUND

Abdominal compartment syndrome (ACS) refers to organ dysfunction caused by intra-abdominal hypertension (IAH). IAH has multiple possible causes and is often under-recognised by clinicians.

It is arbitrarily defined in research as pressure >12 mm Hg and can be acute, subacute or chronic in nature. ACS is defined within research as sustained intra-abdominal pressure greater than 20 mm Hg associated with new organ dysfunction. For clinical purposes it is defined as IAH-induced new organ dysfunction.¹

We present a case that highlights ACS as a rare and life-threatening complication of an extremely common condition, namely chronic severe constipation.

CASE PRESENTATION

A 50-year-old man with a background of cerebral palsy and chronic constipation (figure 1) presented to the emergency department following an out of hospital asystolic cardiac arrest. He was a nursing home resident and dependent on carers for activities of daily living. Following immediate cardiopulmonary resuscitation at the scene, return of spontaneous circulation (ROSC) was obtained after 15 min.

On arrival to the department, he was intubated but breathing spontaneously, saturating at 100% on 90% FiO₂, with a blood pressure of 80/30 and sinus tachycardia on ECG at 100 bpm. His Glasgow Coma Scale score (GCS) was 4/15, E1 V1 M3. On presentation, marked abdominal distension was evident. His abdomen was stony hard and dull

to percussion, with taut and mottled overlying skin. Peripheral examination revealed bilateral ischaemic lower limbs. All pulses were absent and his right leg was pale and cold with mottled skin.

INVESTIGATIONS

On arrival to the emergency department, the patient's venous blood gas showed lactic acidosis (pH 7.03, pO₂ 7.3, pCO₂ 5.3, lactate 12.5 mmol/L) and hyperkalaemia (6.7 mmol/L). This was treated initially with fluid resuscitation, calcium gluconate and insulin dextrose. Venous bloods on admission showed an acute kidney injury (AKI) with a creatinine level of 147 µmol/L from a baseline of 60 µmol/L, and his liver function tests were consistent with hepatic dysfunction (total bilirubin 26 µmol/L, alkaline phosphatase 166 U/L, albumin 33 g/L). Recent bloods tests prior to admission had shown normal renal and hepatic function.

Once stabilised, the patient was transferred for a CT chest, abdomen and pelvis scan, which showed massive dilation of the large bowel due to faecal loading, measuring up to 20 cm (figure 2). There was mass effect on the distal aorta (figure 3), with no contrast opacification seen beyond the right common iliac artery bifurcation. Neither bowel perforation nor vascular aneurysm was identified on the CT scan. There was significant diaphragmatic splinting with compression of the lungs and heart (figure 4). CT scan ruled out a diagnosis of pulmonary embolism.

Figure 1 Abdominal plain radiograph, 1 month prior to current admission, showing severe faecal loading.

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To cite: Starnes DS, Lawrence H, Navaratnam R. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2015-213085

Figure 2 Sagittal CT image showing massive dilation of the large bowel.

Repeat arterial blood gas an hour and a half after admission showed worsening acidosis (pH 6.84, lactate 12.2 mmol/L) and worsening hyperkalaemia (K 9.1 mmol/L). ECG showed changes consistent with hyperkalaemia but not with acute myocardial infarction.

DIFFERENTIAL DIAGNOSIS

The differential diagnoses considered initially on presentation included bowel perforation, obstruction, or a vascular event

Figure 3 Transverse CT image showing compression of the aorta.

Figure 4 Coronal CT image illustrating mass effect of the dilated large bowel on the heart and lungs.

such as a ruptured abdominal aortic aneurysm (AAA). This was due to the patient's history of sudden cardiovascular collapse, his tense and distended abdomen and his ischaemic legs on examination. He had clear metabolic disturbance in his blood gas analysis and evidence of end organ dysfunction due to his AKI, hepatic dysfunction and his low GCS.

The CT scan revealed the extent of the faecal loading and intra-abdominal compression and showed evidence of neither bowel perforation nor ruptured AAA. This led us to the conclusion of ACS causing this patient's new organ dysfunction.

TREATMENT

Treatment consisted of post-ROSC cardiovascular resuscitation and management for hyperkalaemia. A surgical opinion was sought and manual evacuation carried out in order to clear the obstruction and re-perfuse the legs and organs. Some evacuation of faeces was possible, and this successfully re-perfused the patient's legs and abdominal skin. The patient remained haemodynamically unstable, however, with worsening lactic acidosis and hyperkalaemia. Operative management was considered but, after multidisciplinary discussion between intensive care and surgical consultants, it was decided that the patient was, unfortunately, too unstable for this. This was considered a catastrophic event that, sadly, was not survivable, and therefore it was decided to keep the patient comfortable with supportive management only.

OUTCOME AND FOLLOW-UP

The patient, regrettably, continued to decline despite supportive management and passed away within 5 h of admission. The cause of death was listed as ACS secondary to severe constipation.

Review of previous abdominal radiographs showed faecal loading for the past 6 years, which had been treated conservatively with laxatives in the community, with prior hospital admissions.

DISCUSSION

ACS is represented by a constellation of pulmonary, cardiovascular and renal compromise arising from pathologically elevated intra-abdominal pressure. Current consensus defines ACS as existing when the intra-abdominal pressure is persistently elevated above 20 mm Hg.²

The pathology underlying its development is multifactorial and, as such, three categories for its aetiology exist. The first attributes its development directly to intra-abdominal pathology,

the second arises due to extra-abdominal pathology resulting in the accumulation of intra-abdominal fluid, and the third is a consequence of cirrhosis or ascites and their related disease processes.² Ideally, intra-abdominal pressures should be measured. If this is not possible, signs and symptoms in extreme cases include a tensely distended abdomen, oliguria, hypoxia and cardiac instability. As was the case in our patient, absence of femoral pulses and signs of lower limb ischaemia can be present.³ This was likely due to compression of the aorta, either secondary to the elevated intra-abdominal pressure or from direct, extrinsic mechanical compression of the aorta itself. It may be expected that the inferior vena cava would have become compressed prior to the presentation resulting in signs of caval compression, for example, bilateral lower limb oedema or venous engorgement. Such signs, although not present in our case, may herald the onset of an ACS before its presentation as a medical emergency. Untreated, mortality rates are significant in ACS.⁴

The literature review has highlighted very few cases of ACS secondary to constipation. Cases reported in the literature are those of children. Flageole *et al*⁵ describe the case of an 11-year-old boy presenting with intractable constipation and significant haemodynamic compromise. Birkhahn and Gaeta⁶ report a case secondary to a congenital megacolon. Images were presented by Gorecki *et al*⁷ of intractable constipation that resulted in the development of abdominal compartment syndrome. To date, ours represents the first reported case of ACS secondary to constipation in an adult patient.

It has been proposed that the patient featured here may have suffered from a megacolon. It is well documented that patients suffering with cerebral palsy suffer a multitude of gastrointestinal disorders, with chronic constipation and the development of a megacolon being one.⁸ It has been suggested that the intricate neurological control of the digestive tract is disrupted during its development, resulting in such conditions.⁹ These often manifest in childhood but, with the increasing life expectancy of patients with cerebral palsy, they are increasingly seen in the adult population.¹⁰

In previously reported cases, surgical intervention was required in order to relieve the intra-abdominal hypertension. Unfortunately, in our case, the extent of the multiorgan compromise was such that medical management was unsuccessful and surgical options were not feasible due to the patient's unstable haemodynamic state. It was noted that an attempt at manual evacuation did result in an improvement in lower limb perfusion, but deterioration in the patient's condition. It is postulated he suffered a reperfusion injury. At an earlier stage, the

involvement of a neurogastroenterologist may have been of benefit in this case, to provide insight into effective medical management of this condition prior to it developing into a medical emergency.

This unique case adds weight to the need for aggressive management of constipation, as inadequate management can result in the development of life-threatening conditions such as ACS.

Learning points

- ▶ Severe constipation does warrant prompt and at times aggressive management.
- ▶ Abdominal compartment syndrome (ACS) should be considered in cases of extreme abdominal distension.
- ▶ Signs of inferior vena cava compression may point to the onset of possible ACS.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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