Intractable metabolic acidosis in a patient with colovesical fistula

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Abstract

A 58-year-old female presented with urosepsis and faecaluria secondary to a colovesical fistula of diverticular aetiology. A plan was made for surgical repair of the fistula. Preoperatively the patient developed a hyperchloraemic metabolic acidosis, with hyperkalaemia and hyponatraemia. Renal function was normal, and a short synachten test ruled out Addison’s disease. Postoperatively her acid-base physiology normalised in the absence of medical management, demonstrating that surgical intervention was responsible for resolution of the patient’s metabolic acidosis. The mechanisms by which colovesical pathophysiology causes hyperchloraemic metabolic acidosis are discussed. Although diverticular disease is the most common cause of colovesical fistulae, this is the first report of such fistulae causing metabolic acidosis.

Acid-base disturbance is a known complication of urinary diversion into the gastrointestinal tract.\(^1\)\(^-\)\(^3\) Although diverticular disease is the most common cause of colovesical fistulae, no reports exist of such fistulae causing metabolic acidosis.

We present the first case of a patient with metabolic acidosis secondary to a colovesical fistula of diverticular aetiology.

Case report

A 58-year-old female was admitted with persistent urosepsis and faecaluria. Abdominal CT demonstrated a colovesical fistula at the level of the sigmoid colon (Figure 1). Flexible sigmoidoscopy was limited to 25 cm due to a tight stricture. Flexible cystoscopy and biopsy demonstrated the absence of urinary tract obstruction, ruled out bladder neoplasm and confirmed CT findings of a communication between bladder and bowel.

With no evidence of malignancy, no prior abdominal radiotherapy and in the absence of Crohn’s disease, the fistula was deemed to be diverticular in aetiology. The patient was prescribed prophylactic trimethoprim and prepared for a sigmoid colectomy.

Preoperatively the patient developed hyperkalaemia (5.8 mmol/L) and hyponatraemia (128 mmol/L), in the context of normal renal function (creatinine 50 mmol/L, urea 4.4 mmol/L). An arterial blood gas (ABG) demonstrated a hyperchloraemic metabolic acidosis (pH 7.28, chloride 110 mmol/L, base excess 13.4 mmol/L, anion gap 15.6 mmol/L, lactate 0.5 mmol, glucose 5.6 mmol/L). A short synacthen test excluded Addison’s disease.

A repeat ABG 24 hours prior to the operation demonstrated a recalcitrant metabolic acidosis (pH 7.25, base excess –14.8mmol/L, anion gap 11.7mmol/L). One litre of 1.26% bicarbonate solution was infused and plasma pH normalised to 7.36.
The patient proceeded to theatre for a sigmoid colectomy. During the operation the transverse colon was freed from the dome of the bladder at a point of dense adhesion within which a 5 mm hole was found and repaired. A second larger colovesical fistula (15 mm × 10 mm) was found at the sigmoid colon. This was resected and an end colostomy formed along with repair of the bladder defects.

In the postoperative period the patient’s acid-base physiology reversed to a mild metabolic alkalosis (pH 7.48, base excess 0.6 mmol/L). Since this acid-base status persisted and normalised in the absence of bicarbonate treatment we can be confident that repair of the colovesical fistula was responsible for resolution of the patient’s metabolic acidosis.

**Figure 1. Sequential CT segments showing the colovesical fistula**

![Sequential CT segments showing the colovesical fistula](image)

Bl (Bladder), Si (Sigmoid colon).

**Discussion**

Two mechanisms contribute to metabolic acidosis in colovesical fistula pathophysiology. Firstly, urinary chloride delivered through the fistula is absorbed in exchange for bicarbonate by a chloride/bicarbonate transporter found in colonic epithelial cells. Bicarbonate is then lost in the stool. Secondly, urinary urea is delivered through the fistula and broken down by colonic bacterial ureases into ammonium ions. The ammonium ions are reabsorbed via a colonic sodium-hydrogen antiporter, as ammonium takes the place of sodium. In summary, the metabolic acidosis occurs due to a net gain of ammonium and chloride ions in association with loss of bicarbonate. This phenomenon is dependent on urine tracking from bladder to bowel, an atypical presentation of enterovesical fistula which usually sees predominant flow in the opposite direction.
We recommend that patients with colovesical fistulae have their acid-base balance monitored preoperatively and corrected appropriately to prevent intraoperative complications.

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