Appendicovesical fistula presenting as hypokalaemic hyperchloraemic metabolic acidosis: a case report

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ABSTRACT

A 52-year-old man was admitted with diarrhoea and faecaluria and referred recurrent urinary tract infections for over 20 years. He also reported a two-week hospital admission more than 20 years ago for right iliac fossa pain, which was managed conservatively. Computed tomography showed a fistulous tract extending from the bladder with an unclear connection to the bowel. Cystoscopy confirmed the presence of a vesical fistula and biopsy of the tract confirmed colonic mucosa. Flexible sigmoidoscopy was negative. A cystogram was requested as an outpatient procedure and the patient was discharged after antibiotic treatment. A few days after discharge the patient was readmitted as an emergency to critical care for severe hyperchloraemic hypokalaemic acidosis and a Glasgow Coma Score of 6/15. He was intubated and ventilated and his metabolic derangement was treated. As soon as his conditions improved, he underwent emergency laparotomy, which revealed the presence of a fistula between the caecal fundus and the bladder. The fistula was repaired and the patient recovered swiftly and completely and was discharged on postoperative day 5. At 12-month follow up the patient was completely symptoms-free, his bowel habits were normal and he has not had any urinary infection. Appendicovesical fistula is a rare and potentially lethal condition due to its metabolic consequences. Past history of right iliac fossa pain treated conservatively, diarrhoea and recurrent urinary tract infection must raise suspicion.

KEYWORDS

Appendiculovesical fistula – Metabolic acidosis – Acute appendicitis

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Background

Appendicovesical fistula arising as a consequence of acute appendicitis is a rare condition. Only few cases are reported in the literature.¹ The prompt surgical treatment for acute appendicitis probably contributes to its rarity. In this case, we discuss hypokalaemic hyperchloraemic metabolic acidosis as a potential complication and biochemical indicator of an appendicovesical fistula. To date, there have only been three case reports associating this metabolic derangement with appendicovesical fistula.²

Case history

A 52-year-old man was admitted complaining of generalised weakness and strangury, pneumaturia and faecaluria for over 20 years. He also reported continuous diarrhoea over the same period. His past medical history is significant of a two-week hospital admission more than 20 years ago with right iliac fossa pain, which was managed conservatively. A clinical examination showed no abnormalities. Urine analysis showed pyuria and cultures grew mixed

organisms. His electrolytes on admission showed low potassium and inflammatory markers were positive. He was treated with oral antibiotics and intravenous fluid and potassium replacement. Computed tomography (CT) showed a thick-walled urinary bladder and a fistulous tract extending from the right and superior aspect of the posterior bladder wall with an unclear connection to the bowel. Cystoscopy showed a fistulous connection on the posterior wall of the bladder secreting faecal matter, and biopsies taken from that site illustrated colonic-type mucosa with chronic active inflammation, suggesting a colovesical fistula. A flexible sigmoidoscopy was normal.

The patient's metabolic derangement normalised and he was discharged on loperamide. A cystogram was requested on an urgent outpatient basis but on the eighth day he was found collapsed at home and was readmitted with acidotic breathing and a Glasgow Coma Score of 6/15. An arterial blood gas showed severe hypokalaemic hyperchloraemic acidosis. He was intubated immediately and transferred to critical care. He was managed with aggressive fluid resuscitation and intravenous bicarbonate and potassium replacement.

After five days the metabolic derangement was normalised and the patient was taken to the operating theatre for a laparotomy, to treat the suspect colovesical fistula and to prevent further urinary tract infection. A fistula was actually found between the caecum at the base of the appendix and the right posterior-superior aspect of the bladder. The appendix itself could not be identified. The sigmoid was normal. The fistula was divided and both the caecum and the bladder were repaired. The hard tissue between the caecum and the bladder was removed and histology showed residual intestinal mucosa within fibrous tissue, but no specific feature was identified. The patient made a quick and complete postoperative recovery, his metabolic profile was consistently normal, and he was discharged on postoperative day 5.

At 12-month follow up, the patient was completely asymptomatic. His bowel habits were normal, with solid stools. Since the operation he has not had any urinary tract infection and his blood tests were normal.

Discussion and conclusion

Appendicovesical fistulae occur more commonly in men, as in women the uterus is interposed between the bladder and appendix, and in the age group 10–40 years, mostly as a consequence of an acute appendicitis that was not operated promptly,³ or an appendiceal tumour or Crohn's disease. Cases have been described of possibly iatrogenic appendicovesical fistula. Patients present complaining of recurrent symptoms of urinary tract infection. The symptoms are usually vague and diagnosis is hence delayed. Some cases, including the one described here, have taken more than 10 years to diagnose.¹ Diagnostic investigations include CT with contrast, cystoscopy, cystography, barium enema and colonoscopy.

Sometimes it is difficult to outline the fistula track and conventionally diagnosis has been made during surgery. Colonoscopy has been widely used to investigate possible cases of colovesical fistula but false negative results are very frequent. In our case, CT raised the suspicion of a sigmoidovesical fistula, although a clear communication was not visible, but flexible sigmoidoscopy was negative.

To the best of our knowledge, only three cases have been published so far describing the finding of hypokalaemic hyperchloraemic metabolic acidosis with appendicovesical fistulae, but there is no in depth study of this metabolic derangement in this condition.² Hypokalaemic hyperchloraemic metabolic acidosis is commonly observed after urinary diversion procedures and is believed to be due to (a) increased urinary chloride absorption across the bowel mucosa in exchange for bicarbonate and hence

bicarbonate loss in feces; (b) increased urinary NH₄⁺ absorption across the bowel mucosa and therefore increase in plasma acid load; and (c) recurrent pyelonephritis resulting in renal tubular dysfunction and urinary potassium wasting. In addition, potassium and bicarbonate are secreted in the bowel lumen in exchange for urinary sodium and chloride absorption. With low plasma potassium, renal tubular concentrating function deteriorates and polyuria ensues, thus starting a vicious circle.⁴

In our patient's case, we believe that the fistula tract was narrow, allowing more urine to reflux into the bowel than faecal material moving into the bladder. Moreover, the pressure difference between the bladder and the caecum favoured this passage. Differently, sigmoidovesical fistulae bear a reverse flow, as the pressure within the diverticular sigmoid may be higher than the pressure in the bladder. In those patients, the more frequent symptom is recurrent infections, with faecaluria and no diarrhoea. In our case, the fistula was causing urinary diversion and hence resulted in the ensuing metabolic derangements. It is known that continent urinary diversion procedures have a higher risk of metabolic derangement in contrast to the incontinent procedures, and this relates to the amount of time the urine is in contact with the bowel mucosa, so they represent an ideal model to understand the metabolic consequences of appendicovesical fistulae. In our case, the patient was initially discharged on loperamide and this may have increased the urine transit time in the bowel and hence resulted in the subsequent acute presentation with severe acidosis. The treatment of appendicovesical fistula is surgical, by laparotomy, laparoscopy or robotic surgery.⁵

In conclusion, appendicovesical fistula is a rare condition that requires a high index of suspicion. Past medical history that suggests previous acute appendicitis managed conservatively and recurrent urinary tract infections should draw attention. Hypokalaemic hyperchloraemic metabolic acidosis should be recognised as a potential complication of this condition.

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