Case Report

Hyperkalaemia as a complication of ureteroileostomy: a case report and literature review

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Introduction

Ureteral diversion in which the ureter is implanted into either the sigmoid colon or a short loop of ileum is associated with multiple metabolic complications [1,2]. Ureterosigmoidostomy commonly leads to metabolic acidosis due to the presence of colonic anion exchange pumps that reabsorb luminal chloride as bicarbonate is secreted across the sigmoid colon. In such cases, reabsorption of urinary ammonium that contacts the sigmoid colon may also contribute to metabolic acidosis [1]. In addition, sigmoid loops usually lead to hypokalaemia due to colonic potassium secretion. However, if the intestinal conduit in contact with ureteral contents is jejunum, hyperkalaemia may occur, presumably due to absorption of urinary potassium by the jejunum. This infrequent complication of ureteral diversion has been rarely reported but should be considered in cases of hyperkalaemia in patients with ureteroileostomies. In such cases, jejunal contact with the ureteral drainage likely is occurring and resulting in hyperkalaemia via jejunal absorption of potassium. We report an illustrative case in which enteral feeding repeatedly resulted in hyperkalaemia in a patient who underwent urinary diversion with a conduit assumed to be implanted in the low ileum.

Case

A 77-year-old man with advanced rectal carcinoma underwent pelvic exconeration, cystectomy and ileal conduit followed by radiation treatment ~6 years ago. He was hospitalized recently with a 1-week history of abdominal pain, fever, bleeding per rectum and obstipation. Colonoscopy revealed ischaemic colitis. He was treated conservatively and discharged after 5 days. He presented 6 weeks later with abdominal pain, nausea and vomiting due to bowel obstruction. He was also noted to have a colonic to urinary tract fistula. Exploratory laparotomy was performed with extensive lysis of adhesions, creation of loop ileostomy and revision of the urinary conduit. His prior surgery and radiation as well as multiple adhesions complicated the procedure and made bowel segment identification difficult. Nephrology consultation was called 6 days postoperatively for hyperkalaemia.

On physical examination, he appeared thin, chronically ill and complained of decreased energy. His blood pressure was 108/63 mmHg, heart rate 56 and temperature 37.5°C. The abdomen was soft, mildly distended and non-tender. He had a PEG tube in place. The ileoconduit was intact and in place. His extremities were well perfused. His medications included codeine on PRN basis, Famotidine 20 mg BID, Heparin 5000 units SQ q 8 h and Metoprolol 25 mg BID. The blood chemistry test results and urine test results at the time of nephrology consultation are shown in Table 1. The calculated Trans Tubular Potassium Gradient (TTKG) was 13.4 indicating normal aldosterone effect. The patient’s urine output was ~3 L/day.

Postoperatively, the patient received total parenteral nutrition (TPN) and maintained normal serum electrolytes (Figure 1). On postoperative day 5, enteral feeding was begun and his plasma potassium rose to 5 mmol/L. At one point during his hospital stay, the enteral feeding was discontinued because of nausea and vomiting and TPN was resumed for 4 days. His electrolytes, including potassium, were normal during these 4 days (Figure 1). At the time of nephrology consultation, the patient was on enteral feeding.

Table 1. Blood and urine chemistry at the time of consultation

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na</td>
<td>133 mmol/L</td>
</tr>
<tr>
<td>K</td>
<td>5.0 mmol/L; peak level 5.3; normal for lab 3.5–4.5 mmol/L</td>
</tr>
<tr>
<td>Cl</td>
<td>99 mmol/L</td>
</tr>
<tr>
<td>CO2</td>
<td>30 mmol/L</td>
</tr>
<tr>
<td>BUN</td>
<td>23 mg/dL</td>
</tr>
<tr>
<td>Scr</td>
<td>1.0 mg/dL</td>
</tr>
<tr>
<td>Ca</td>
<td>8.4 mg/dL</td>
</tr>
<tr>
<td>Mg</td>
<td>1.9 mg/dL</td>
</tr>
<tr>
<td>PO4</td>
<td>3.6 mg/dL</td>
</tr>
<tr>
<td>Urine Na</td>
<td>37 mmol/L</td>
</tr>
<tr>
<td>Urine K</td>
<td>92.3 mmol/L</td>
</tr>
<tr>
<td>Urine Cr</td>
<td>66.7 mg/dL</td>
</tr>
<tr>
<td>Urine Osm</td>
<td>380 mOsm/kg</td>
</tr>
<tr>
<td>Urine Glu</td>
<td>Neg</td>
</tr>
</tbody>
</table>

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ing jejunum is presumed to occur as a result of jejunal hyperkalaemia only with higher potassium enteral feeding segment used as the conduit. The patient’s clinical course of not exclude the possibility of a very high ileal or jejunal the implanted urinary conduit was in the ileum but could revision of the urinary conduit. The surgeon believed that extensive lysis of adhesions, creation of loop ileostomy and urinary conduit surgery involved exploratory laparatomy, 6 years ago as well as his past radiation treatment. The previous pelvic exoneration, cystectomy and ileal conduit conduit was unknown; the procedure was complicated by the exact bowel segment used for insertion of the urinary insertion of the urinary conduit was unrevealing. Discussion report of our patient in an attempt to delineate the anatomic causes of hyperkalaemia are listed in Table 2.

Hyperkalaemia rarely occurs in normal subjects due to multiple mechanisms that prevent the accumulation of potassium in the extracellular fluid. These mechanisms include the shifting of potassium in and out of the cellular compartment and increased urinary excretion of potassium. Thus, increasing potassium intake does not cause hyperkalaemia unless it occurs very acutely or in the setting of impaired potassium excretion (e.g. acute kidney injury or failure). Transient elevation in the serum potassium level can occur as a result of an increased release of cellular potassium or decreased cellular potassium entry, but persistent hyperkalaemia requires an impairment in urinary potassium excretion. Causes of hyperkalaemia are listed in Table 2.

Urinary diversion procedures using jejunal conduits are less common than sigmoid or ileal conduits and have been noted to result in hyperkalaemia although the number of reported cases are few [3–8]. A review of the operative report of our patient in an attempt to delineate the anatomic insertion of the urinary conduit was unrevealing. Discussion with the surgeon performing the procedure suggested that the exact bowel segment used for insertion of the urinary conduit was unknown; the procedure was complicated by previous pelvic exoneration, cystectomy and ileal conduit 6 years ago as well as his past radiation treatment. The urinary conduit surgery involved exploratory laparotomy, extensive lysis of adhesions, creation of loop ileostomy and revision of the urinary conduit. The surgeon believed that the implanted urinary conduit was in the ileum but could not exclude the possibility of a very high ileal or jejunal segment used as the conduit. The patient’s clinical course of hyperkalaemia only with higher potassium enteral feeding suggests that the conduit may be in the jejunum (Figure 1).

Hyperkalaemia in the setting of urinary conduits using jejunum is presumed to occur as a result of jejunal

Discussion

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Hyperkalaemia in the setting of urinary conduits using jejunum is presumed to occur as a result of jejunal

Table 2. Causes of hyperkalaemia

<table>
<thead>
<tr>
<th>Deceased potassium entry into cells or increased potassium release from cell</th>
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<tbody>
<tr>
<td>(1) Metabolic acidosis</td>
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<tr>
<td>(2) Insulin deficiency and hyperglycaemia</td>
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<td>(3) Hyperosmolality, like mannitol induced</td>
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<td>(4) β-Adrenergic blockade</td>
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<td>(5) Increased tissue breakdown/catabolism such as rhabdomyolysis, crush injury, trauma, cytotoxic or radiation therapy and hypothermia</td>
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<td>(6) Exercise</td>
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<tr>
<td>(7) Pseudohyperkalaemia, like with extracorporeal haemolysis</td>
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<tr>
<td>(8) Medications like digitalis overdose, succinylcholine, calcineurin inhibitors (has other mechanism by inducing hyponatraemic hypoadrenosteronism and direct interference with the effect of aldosterone on the potassium-secreting cells), diazoxide, minoxidil and several volatile anesthetics like isofluran.</td>
</tr>
<tr>
<td>(9) Genetic disorder such as hyperkalaemic periodic paralysis</td>
</tr>
</tbody>
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Reduced potassium excretion in the urine

(1) Diminished distal delivery of sodium and water, usually associated with a substantial decline in GFR
(a) Advanced kidney disease, especially with decreased urine output
(b) Decreased effective circulating volume as in heart failure and cirrhosis
(2) Hypoaldosteronism
(a) Hyponatraemic hypoaldosteronism, could be caused also by medications like NSAIDs and B-blockers.
(b) Primary adrenal insufficiency
(c) Medications
(i) ACEI and ARB, Heparin, Triamteren and Pentamidine that lower aldosterone release or effect
(ii) NSAIDs that act in part by removing the stimulatory effect of renal prostaglandins on the release of renin
(iii) Potassium-sparing diuretics that directly block sodium reabsorption and potassium secretion in the collecting tubules
(3) Type 1 renal tubular acidosis with hyperkalaemia, when the primary defect is impaired sodium reabsorption in the cortical collecting tubule decreasing the lumen electronegative charge and hence reducing both hydrogen and potassium secretion
(4) Gordon’s syndrome, enhanced chloride reabsorption (chloride shunt)—when sodium being reabsorbed with chloride, rather than in exchange with potassium and hydrogen

Ureterojejunostomy

Following a urinary diversion procedure in which the ureters are inserted into the jejunum and presumably absorption of urinary potassium by the jejunum
of jejunal potassium absorption is unclear and although relatively rare [3–6] does occur. Since our patient became hyperkalaemic only with higher K enteral feeding (Figure 1), jejunal potassium absorption is clearly implicated. Subsequently, our patient was treated with a low-potassium enteral diet 50 mEq/day and his potassium level remained around 4.5 mmol/L.

Summary

Although jejunal potassium absorption is recognized, it is rarely reported and may be misinterpreted as a complication of ileal diversion [7,8]. Given the differences in ileal and jejunal potassium handling and the difficulty of surgically identifying exact intestinal segments in the setting of multiple past procedures and adhesions, post-procedure hyperkalaemia should raise suspicion that the urinary conduit was planted in the high ileum or jejunum. Controlling potassium intake in such cases is the foundation of treatment.

Conflicting of interest statement. None declared.

References


Received for publication: 17.8.07
Accepted in revised form: 1.2.08