Unexplained hypokalaemia and metabolic alkalosis

D Devendra, P A Rowe

A 23 year old girl was admitted to the medical admission unit with a three month history of malaise and generalised muscle cramps. Her general practitioner performed routine biochemistry and had revealed a potassium concentration of 2.0 mmol/l. She was investigated four years before for a similar electrolyte disturbance which was found to be inconclusive. She was currently taking the combined oral contraceptive pill. There was no history of diarrhoea or vomiting. There was no family history of endocrine disease. On examination the patient appeared well, her body mass index was 19, and her blood pressure was 118/70 mm Hg. She was euvoalaemic clinically. She had no evidence of postural hypotension nor did she have any peripheral oedema. Her skin turgor was normal.

Biochemical investigations revealed a marked hypokalaemic metabolic alkalosis (see table 1). Full blood count, liver function tests, and serum calcium were within normal limits.

Table 1  Biochemical investigations

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium (mmol/l)</td>
<td>128</td>
</tr>
<tr>
<td>Potassium (mmol/l)</td>
<td>2.0</td>
</tr>
<tr>
<td>Chloride (mmol/l)</td>
<td>73</td>
</tr>
<tr>
<td>Bicarbonate (mmol/l)</td>
<td>40</td>
</tr>
<tr>
<td>Calcium (mmol/l)</td>
<td>2.32</td>
</tr>
<tr>
<td>Magnesium (mmol/l)</td>
<td>0.64</td>
</tr>
<tr>
<td>Urea (mmol/l)</td>
<td>2.8</td>
</tr>
<tr>
<td>Creatinine (µmol/l)</td>
<td>71</td>
</tr>
<tr>
<td>Urine osmolality (mosmol/l)</td>
<td>460</td>
</tr>
</tbody>
</table>

Despite aggressive oral potassium supplementation the patient remained hypokalaemic (potassium = 3.0 mmol/l).

Questions
(1) What are the causes of a hypokalaemic metabolic alkalosis?
(2) What further investigations may be warranted in this patient?
(3) What is the underlying diagnosis and what are the treatment options for this condition?
Self assessment questions

The urine chloride concentration was raised in than 20 mmol/l if the serum chloride is raised. With low serum chloride and conversely more chloride less than 20 mmol/l is seen in a patient Bartter’s or Gitelman’s syndrome. A urinary chloride concentration, on the other hand, the diuretic e surreptitious diuretic use is variable—high if losis. The value of urinary chloride with have been vomiting and have a metabolic alka-

QUESTION 2

A careful physical examination should be undertaken to look for signs of self induced vomiting which include calluses and scarring of the dorsum of the hand (Russell’s sign), dental erosions caused by chronic exposure to acid gastric secretions, and puffy cheeks resulting from hypertrophy of the salivary glands. A urine chloride estimation may help to contrib-

Box 1: Causes of hypokalaemic alkalosis

Potassium loss in gastrointestinal tract
- Vomiting.
- Laxative abuse.
- Infectious diarrhea.
- Tumours (for example, VIPoma).
- Enteric fistula.
- Cancer therapy (for example, radiation enteropathy).

Potassium loss in urine
- Mineralocorticoid excess: primary hyperaldosteronism, congenital adrenal hyperplasia (17α-hydroxylase defi-
ciency), renin secreting tumours, Cushing’s syndrome, renovascular hypertension.
- Apparent mineralocorticoid excess: Liddle’s syndrome, 11β-hydroxysteroid dehydrogenase deficiency.
- Impaired chloride associated sodium transport: Gitelman’s syndrome, Bartter’s syndrome.
- Drug induced: diuretics, fluoro-cortisone, licorice, aminoglycosides, high dose glucocorticoids.

QUESTION 3

Due to the high urine chloride concentration and normal diuretic screen, this suggests that the patient may have either Bartter’s or Gitelman’s syndrome. Because our patient presented in adulthood, was able to concen-

Final diagnosis

Gitelman’s syndrome.

Answers

QUESTION 1

The causes of hypokalaemic alkalosis can be divided into loss in the gastrointestinal tract or loss in the urine (see box 1).1

Table 2 Characteristics of Bartter’s syndrome and Gitelman’s syndrome

<table>
<thead>
<tr>
<th>Localisation of defect</th>
<th>Bartter’s syndrome</th>
<th>Gitelman’s syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of presentation</td>
<td>Ascending limb of Henle</td>
<td>Distal tubule</td>
</tr>
<tr>
<td>Biochemical differences</td>
<td>Prenatal, during infancy or early childhood</td>
<td>Late childhood or at adulthood</td>
</tr>
<tr>
<td>Serum magnesium may be decreased</td>
<td>Urinary excretion of calcium increased or normal</td>
<td>Serum magnesium decreased</td>
</tr>
<tr>
<td>Molecular differences</td>
<td>Na-K-2Cl transporter or apical K channel or basolateral Cl channel in thick ascending limb of Henle</td>
<td>Urinary calcium excretion reduced</td>
</tr>
<tr>
<td>Functional studies</td>
<td>Concentrating capacity severely impaired</td>
<td>Concentrating capacity normal or slightly impaired</td>
</tr>
</tbody>
</table>

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