Severe metabolic alkalosis: a case report

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Abstract

A 45-year-old man who was admitted with nausea, vomiting, and abdominal pain was found to have severe metabolic alkalosis, with a Paco2 of 11.4 kPa (85.5 mm Hg), Pao2 of 5.8 kPa (43.5 mm Hg), pH of 7.61, and plasma bicarbonate concentration of 82.0 mmol/l. He was treated with oxygen, intravenous physiological saline, and phenytoin and improved within 48 hours. Radiographs showed gastric outlet obstruction secondary to peptic ulcer, which was treated by surgery.

Though severe, the rise in carbon dioxide concentration in this patient was probably lifesaving. The Paco2 was therefore allowed to fall gradually as the alkalosis was treated. The return of both Paco2 and plasma bicarbonate values to normal in parallel suggests that hyperventilation compensated for the metabolic alkalosis and emphasises the importance of conservative treatment in cases of metabolic alkalosis.

Introduction

We report a patient with severe metabolic alkalosis, out of the range of most acid-base nomograms. The metabolic alkalosis was due to vomiting secondary to gastric outlet obstruction. The initial arterial plasma bicarbonate concentration was 82 mmol(litEq)/l, but it returned to normal within 48 hours of the start of treatment.

Case report

A 45-year-old man was admitted with a two-day history of nausea, vomiting, and abdominal pain. He had a history of alcohol withdrawal seizures. He had symptoms of peptic ulcer and an upper gastro-

References


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intestinal x-ray examination six years earlier had shown a scarred duodenal bulb and acute duodenal ulcer. He was taking hydrochlorothiazide for hypertension. There was no history of chronic obstructive pulmonary disease.

On admission the patient was stuporous and had dry skin. His reflexes were normal. The blood pressure was 130/70 mm Hg, pulse rate 92/min, respiratory rate 12/min, and rectal temperature 38.3°C. Packed cell volume was 49.8%, and white blood cell count 15.3 x 10¹¹/l. Blood urea nitrogen was 10 mmol/l (28 mg/100 ml). Serum amylase activity was normal. Serum sodium was 142 mmol/l (Eq/l), potassium 2.0 mmol/l (Eq/l), and chloride 50 mmol/l (Eq/l). Arterial carbon dioxide tension (Paco2) was 11.4 kPa (85.5 mm Hg) (at 37°C) and oxygen tension (Pao2) 5.8 kPa (43.5 mm Hg), pH 7.61, and the calculated plasma bicarbonate concentration was 82.6 mmol/l (Eq/l). Initially he was treated with naloxone without response. Oxygen, intravenous physiological saline, potassium chloride, and phenytoin were administered. He improved within the next 48 hours (see table) but developed aspiration pneumonitis while in the intensive care unit.

### Acid-base variables during patient's stay in hospital (values corrected to patient's body temperature)

<table>
<thead>
<tr>
<th>Time</th>
<th>Admission</th>
<th>6 hours later</th>
<th>12 hours later</th>
<th>24 hours later</th>
<th>48 hours later</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIO₂</td>
<td>0.21</td>
<td>0.40</td>
<td>0.35</td>
<td>0.35</td>
<td>0.21</td>
</tr>
<tr>
<td>Paco₂ (kPa)</td>
<td>8.9</td>
<td>14</td>
<td>10.9</td>
<td>12.7</td>
<td>10.5</td>
</tr>
<tr>
<td>Paco₂ (mm Hg)</td>
<td>62.4</td>
<td>77</td>
<td>84</td>
<td>93</td>
<td>84</td>
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<tr>
<td>pH</td>
<td>7.59</td>
<td>7.56</td>
<td>7.47</td>
<td>7.43</td>
<td>7.43</td>
</tr>
<tr>
<td>HCO₃ (mmol/l)</td>
<td>82</td>
<td>65</td>
<td>95</td>
<td>35</td>
<td>23</td>
</tr>
</tbody>
</table>

FIO₂ = Fractional concentration of inspired oxygen.

Convection: ST to traditional units—Paco₂ and Paco₂; 1 kPa = 7.5 mm Hg.

Two days after admission his serum sodium was 146 mmol/l, potassium 3.8 mmol/l, and chloride 102 mmol/l. Blood urea nitrogen was 5.7 mmol/l (16 mg/100 ml) and serum creatinine 80 mmol/l (0.9 mg/100 ml). Radiographs of the upper gastrointestinal tract showed gastric outlet obstruction secondary to peptic ulcer, for which he underwent surgery. His pulmonary function tests showed a forced vital capacity (FVC) of 4.01 l (80%, of predicted), forced expiratory volume in one second (FEV₁) of 3.39 l (90%, of predicted), FEV₁ as a percentage of VC of 84.4%, and maximum mid-expiratory flow rate of 3.98 l/sec (108% of predicted).

**Arterial blood gas analysis**—Arterial blood gases were analysed by appropriate electrodes at 37°C model 813, Instrumentation Laboratory, Lexington, Mass.) Pco₂ and Po₂ electrodes were calibrated against gases of known concentration as well as human blood tonometered with the same gases. The pH electrode was calibrated with precision buffers. The patient's arterial blood gas values were corrected to his body temperature and plasma bicarbonate was calculated from the Henderson-Hasselbalch equation by using a Severinghaus blood gas calculator.

### Discussion

This patient suffered from the most severe metabolic alkalosis ever reported as a result of vomiting secondary to gastric outlet obstruction. Hydrochlorothiazide worsened the process. Hypoventilation, which prevented a lethal alkaline pH, was at least partly due to the depression of central and peripheral chemoreceptors by the alkalasia. The patient was not taking any drugs which would depress respiration, and he showed no response to naloxone.

Although grand mal seizure may lead to carbon dioxide retention and might have further augmented the rise in Paco₂, its contribution to hyperventilation in this patient was uncertain. A severe grand mal seizure is often associated with lactic acidosis resulting in an increased anion gap (Na⁺ + K⁺) — (Cl⁻ + HCO₃⁻). This patient had a low anion gap initially. Nevertheless, if our patient had seizure-induced carbon dioxide retention, the contribution of such a sharp increase in carbon dioxide concentration to the rise in plasma bicarbonate concentration was minimal. Furthermore, chronic obstructive lung disease and carbon dioxide retention on that basis were ruled out by his history and by normal pulmonary function studies. The initial alveolar-arterial difference for oxygen tension, (A–A) Do₂, was also normal.

It has been suggested that compensatory carbon dioxide retention in metabolic alkalosis is limited since the impending hypoxaemia and concomitant hypokalaemia prevent hypoventilation. Many patients with severe metabolic alkalosis and pronounced compensatory carbon dioxide retention have been hypoxaemic, however, and it is now well-known that the carotid body is less sensitive to hypoxaemia in the presence of alkalasia. This patient initially had a Paco₂ of 5.8 kPa (43.5 mm Hg).

We have shown that percentage regulation of hydrogen in the arterial blood of normal subjects with steady state metabolic alkalosis is independent of changes in plasma potassium concentrations. Other reported cases, moreover, have shown severe carbon dioxide retention compensatory to metabolic alkalosis with potassium depletion and hypokalaemia. This patient's initial plasma potassium concentration was 2.0 mmol/l. Therefore, it does not follow that hypokalaemia would necessarily limit hypoventilation. On the contrary, respiratory muscle weakness secondary to hypokalaemia might itself have aggravated hypoventilation, although this probably did not contribute to carbon dioxide retention in our patient since his reflexes were intact.

Several recent studies have attempted to define more fully the normal ventilatory response to metabolic alkalosis. For a bicarbonate of 82 mmol/l our regression equation predicts a Paco₂ of 10.4 kPa (75 mm Hg), while that of van Ypersele de Strihou predicts a Paco₂ of 12 kPa (90 mm Hg). This patient's Paco₂ of 11.4 kPa (85.5 mm Hg) was therefore, predicted reasonably accurately given the need to extrapolate beyond the data used to generate either regression equation.

Although very severe, the rise in carbon dioxide concentration in our patient was potentially lifesaving. It was therefore important to allow the Pco₂ to fall gradually in response to treating the metabolic alkalosis. This gradual return of Paco₂ to normal concomitant with the return of plasma bicarbonate values to normal also suggests that hypoventilation was compensating for metabolic alkalosis in this patient. Moreover, the rapid recovery of our patient underscores the importance of conservative treatment (volume expansion, potassium, and oxygen) even in the most severe cases of metabolic alkalosis; the normal kidney has a large capacity for excreting bicarbonate.

### References


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Many attempt to cure a cold, by getting drunk. But this, to say no worse of it, is a very hazardous experiment. No doubt it may sometimes succeed, by suddenly restoring the perpiration; but when there is any degree of inflammation, which is frequently the case, strong liquors, instead of removing the malady, will increase it. By this means a common cold may be converted into an inflammatory fever.

(Buchan's Domestic Medicine, 1786.)