Teaching Point
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A 77 year-old haemodialysis patient with unexpected alkalosis

Sibylle von Vietinghoff, Friedrich C. Luft and Ralph Kettritz

Medical Faculty of the Charité, HELIOS Klinikum, Berlin, Germany

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Introduction

Haemodialysis patients do not commonly acquire metabolic alkalosis. Metabolic acidosis is more common. Profound vomiting and massive gastric drainage are possible causes [1]. Dialysis patients do not have kidneys that can excrete bicarbonate. Exogenous ingestion of bicarbonate in the form of bromoseltzer and other pica forms have been described [2]. A dialysis patient gave himself 1500 mmol of sodium bicarbonate because he thought he might have hyperkalaemia [3]. Luckily he did not employ insulin to treat his perceived but non-existent problem. We recently encountered metabolic alkalosis in one of our dialysis patients. We were initially puzzled and then embarrassed to find the cause.

A 77-year-old haemodialysis patient was admitted because of progressive dyspnoea for 1 week. Two years earlier, she was diagnosed as having anticytoplasmic antibodies (ANCAs) directed against myeloperoxidase (MPO) resulting in rapidly progressive glomerulonephritis and chronic renal failure. Her condition had been treated with corticosteroids and cyclophosphamide pulse therapy. Treatment had resulted in stabilization of the extrarenal disease manifestations, but not in improvement of her renal function. She had been anuric since.

On admission, she weighed 45 kg and was 160 cm tall. Her respiratory rate was 24/min and laboured. She required accessory muscles for respiration. There were bilateral wheezes over both lung fields. Her heart was not enlarged and her systolic murmur was unchanged from earlier examinations. There was no gallop rhythm. Her abdominal examination was not remarkable and there was no peripheral oedema.

Correspondence and offprint requests to: Ralph Kettritz, MD, Franz Volhard Clinic, Wiltbergstrasse 50, D-13125 Berlin, Germany. Email: kettritz@fvk-berlin.de

Cutaneous or other extrapulmonary findings of active vasculitis were absent.

Her haemoglobin was 10.5 g/dl and the haematocrit was 32 vol/%. Her pH was 7.44, PaCO₂ 39 mmHg, PaO₂ 66 mmHg and HCO₃ 26 mmol/l. MPO ANCAs were >400 U/l on the initial enzyme-linked immunosorbent assay (ELISA). The chest roentgenogram showed partial consolidation bilaterally confirmed by computed tomography (CT) shown in Figure 1. We treated the patient with high-dose pulsed prednisone 500 mg for three consecutive days and pulses of 500 mg/m² cyclophosphamide. Because of her severe hypoxaemia, we elected to conduct plasmapheresis with fresh frozen plasma to remove the MPO ANCAs. To our surprise, after three consecutive exchanges, a blood gas determination on room air in our patient disclosed metabolic alkalosis. We repeated the determinations during the course of plasmapheresis treatments and subsequent haemodialysis as shown in Table 1.
Questions

What had we done to this patient? How should we ameliorate this iatrogenic problem?

Answers

The fresh frozen plasma packaged at our blood bank is anticoagulated with sodium citrate at 14 g/dl. The formula for sodium citrate is C₆H₅O₇Na₃. Metabolism to CO₂ and H₂O also yields 3NaHCO₃. Metabolic alkalosis has been described after plasmapheresis for Goodpasture’s syndrome, systemic lupus erythematosus and thrombocytopenic purpura [4]. Since our patient was anuric, she was unable to excrete any generated bicarbonate. Citrate anticoagulation in dialysis patients can also result in metabolic alkalosis [5]. Moreover, increased aluminium levels have been observed when the material is administered in glass containers rather than plastic bags. Citrate chelates the aluminium present in glass bottles or vials. Finally, citrate anticoagulation can cause serious hypocalcaemia [6]. Our patient’s ionized calcium values indeed fell during the course of plasmapheresis from 1.5 to 1.3 mmol/l. However, the patient remained asymptomatic. Additional plasmaphereses were performed with 5% albumin containing no sodium citrate. Furthermore, the subsequent haemodialysis was performed with a dialysate bicarbonate concentration of 27 mmol/l. The values with this treatment are given in Table 2.

The recent MEPEX (methyl prednisolone or plasma exchange for severe renal vasculitis) trial showed that patients with ANCA vasculitis who had severe renal involvement benefit from plasmapheresis [7]. Those who were oliguric seemed to benefit the most. These new data from a randomized study suggest that plasmapheresis, in addition to immunosuppressive drugs, will be used more frequently to treat patients with ANCA vasculitis with decreased renal function. Furthermore, clinicians perform plasmapheresis for diffuse pulmonary haemorrhage in patients with small-vessel vasculitis. We interpreted the CT in our patient as highly suggestive of intrapulmonary haemorrhage. We felt that in the face of severe alveolar bleeding and decreasing PaO₂, fresh frozen plasma containing significant amounts of coagulation factors was more appropriate than albumin. With plasmapheresis, high-dose prednisone and cyclophosphamide, the roentgenographic picture improved remarkably and our patient made an uneventful recovery. However, a combination of albumin together with fresh frozen plasma might have been more appropriate, particularly in an anuric patient who was unable to eliminate the excess bicarbonate generated from the citrate anticoagulant used in the fresh frozen plasma preparations.

Teaching points

Be aware that:

1. Each molecule of citrate generates three molecules of bicarbonate.
2. Dialysis patients have no renal function with which to excrete bicarbonate. Thus, they are at risk for developing metabolic alkalosis, particularly as a result of plasmapheresis.

Conflict of interest statement. None declared.

References


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Table 1. Blood gas values during the initial plasmaphereses (PPH)

<table>
<thead>
<tr>
<th>Time</th>
<th>pH</th>
<th>PaCO₂</th>
<th>PaO₂</th>
<th>HCO₃</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before PPH</td>
<td>7.41</td>
<td>53</td>
<td>54</td>
<td>33</td>
</tr>
<tr>
<td>1/2 PPH</td>
<td>7.41</td>
<td>53</td>
<td>52</td>
<td>33</td>
</tr>
<tr>
<td>After PPH</td>
<td>7.44</td>
<td>54</td>
<td>50</td>
<td>35</td>
</tr>
<tr>
<td>After haemodialysis</td>
<td>7.37</td>
<td>62</td>
<td>84</td>
<td>35</td>
</tr>
</tbody>
</table>

All values were obtained on room air with the exception of the value after subsequent haemodialysis, which was with supplementary 21/min O₂. The patient was asymptomatic. Gas tensions are in mmHg. HCO₃ is in mmol/l. After an additional haemodialysis with antecedent plasmapheresis, her HCO₃ decreased to 29mmol/l.

Table 2. Blood gas values during plasmaphereses (PPH) with 5% albumin containing no sodium citrate

<table>
<thead>
<tr>
<th>Time</th>
<th>pH</th>
<th>PaCO₂</th>
<th>PaO₂</th>
<th>HCO₃</th>
</tr>
</thead>
<tbody>
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<td>Before PPH</td>
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<td>75</td>
<td>29</td>
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<tr>
<td>1/2 PPH</td>
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<td>39</td>
<td>82</td>
<td>21</td>
</tr>
<tr>
<td>After PPH</td>
<td>7.37</td>
<td>50</td>
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<td>After haemodialysis</td>
<td>7.41</td>
<td>42</td>
<td>74</td>
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</tr>
</tbody>
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All values were obtained on room air. Gas tensions are in mmHg and HCO₃ is in mmol/l.