treatment of the lesion should be performed, including extirpation and reinforcement of the tracheal wall.

References


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The Importance of Identifying the Association Between Metabolic Alkalosis and Respiratory Acidosis

Sobre la importancia de identificar la asociación de alcalosis metabólica con acidosis respiratoria

Dear Editor:

It has been well documented that metabolic alkalosis (MAlk) is a very frequent disorder that is usually associated with situations of chronic respiratory acidosis (RA). This should be of no surprise if we keep in mind the regularity with which these patients receive treatment with loop diuretics, thiazides or low-salt diets, which are common causes for this disorder. Nevertheless, the recognition of this association is very infrequent, despite the severe consequences derived from the increased hypoventilation entailed in the compensatory response of MAlk.1–3 Thus, in daily practice we repeatedly observe a tendency to automatically attribute any elevation in plasma bicarbonate to the compensatory mechanism of RA, regardless of the amount.

It has been perfectly established, on the other hand, that for the correct diagnosis of an acid–base disorder, it is necessary to have, in addition to the understanding of the patients symptoms and the filiation of the primary acid–base disorder, the detailed analysis of the compensatory mechanisms in order to estimate its coherence. In chronic RA, for example, increases in bicarbonate of 3.5 mmol/l are considered normal for every 10 mm Hg that PaCO2 increases.4 Therefore, any deviation either above or

Table 1

<table>
<thead>
<tr>
<th>Case 1</th>
<th>Admittance</th>
<th>Day 2</th>
<th>Day 6</th>
<th>Day 11</th>
<th>Day 13</th>
<th>Day 16</th>
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<tbody>
<tr>
<td>pH</td>
<td>7.44</td>
<td>7.48</td>
<td>7.49</td>
<td>7.49</td>
<td>7.43</td>
<td>7.37</td>
</tr>
<tr>
<td>PaO2 (mm Hg)</td>
<td>53</td>
<td>65.6</td>
<td>83.3</td>
<td>33.6</td>
<td>57</td>
<td>78.4</td>
</tr>
<tr>
<td>PaCO2 (mm Hg)</td>
<td>57.9</td>
<td>55.2</td>
<td>49.6</td>
<td>50.2</td>
<td>47.3</td>
<td>44.7</td>
</tr>
<tr>
<td>HCO3− predicted (mmol/l)</td>
<td>30.3</td>
<td>29.3</td>
<td>27.4</td>
<td>27.6</td>
<td>26.6</td>
<td>24.2</td>
</tr>
<tr>
<td>Treatment</td>
<td>Furomide, 120 mg/day intravenously</td>
<td>Furomide, 120 mg/day intravenously</td>
<td>Furomide, 120 mg/day intravenously</td>
<td>Furomide, 120 mg/day intravenously</td>
<td>Suspension furomide, Acetazolamide, 500 mg/day, orally and KCl</td>
<td>Suspension furomide, Acetazolamide, 500 mg/day, orally and KCl</td>
</tr>
<tr>
<td>1st Consultation</td>
<td>Day 30</td>
<td>Day 45</td>
<td>Day 48</td>
<td>Day 52</td>
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<td></td>
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<tr>
<td>pH</td>
<td>7.49</td>
<td>7.5</td>
<td>7.46</td>
<td>7.40</td>
<td>7.41</td>
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<td>PaO2 (mm Hg)</td>
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<td>58.70</td>
<td>44.50</td>
<td>67.30</td>
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<tr>
<td>PaCO2 (mm Hg)</td>
<td>47</td>
<td>51.60</td>
<td>63.10</td>
<td>43</td>
<td>42.4</td>
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<td>HCO3− (mmol/l)</td>
<td>35</td>
<td>37.80</td>
<td>44.20</td>
<td>26.70</td>
<td>25.4</td>
<td></td>
</tr>
<tr>
<td>HCO3− predicted (mmol/l)</td>
<td>26.4</td>
<td>28.10</td>
<td>32.10</td>
<td>24.10</td>
<td>24.5</td>
<td></td>
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<tr>
<td>Potassium (mmol/l)</td>
<td>2.9</td>
<td>3.3</td>
<td>3</td>
<td>3.7</td>
<td>4.1</td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>Furomide, 60 mg/day, orally</td>
<td>Furomide, 60 mg/day, orally</td>
<td>Furomide, 60 mg/day, orally</td>
<td>Suspension furomide, Acetazolamide, 500 mg/day, orally</td>
<td>Suspension furomide, Acetazolamide, 500 mg/day, orally</td>
<td></td>
</tr>
</tbody>
</table>


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failure that diuretics, especially the furosemide (the only relevant therapeutic modification) notably improved the situation. One of the patients was able to stop home oxygen therapy, which had been prescribed some months earlier (Table 1).

MAIk generally initiates with digestive loss (vomiting, nasogastric aspiration) or renal loss (diuretics) of hydrons (H⁺). As the hydrons come from the dissociation of H₂CO₃, for each mequiv. of H⁺ lost, another mequiv. of bicarbonate is generated. Given that the renal capacity for excreting the excess of bicarbonate is great, MAIk only perpetuates when certain circumstances coexist, such as a reduction in effective volemia, hypochloremia, hypokalemia or hyperaldosteronism, in which the renal reabsorption of bicarbonate is higher. The increase in plasma bicarbonate raises the pH, whose compensatory mechanism is hypoventilation that reduces PaO₂ and increases PaCO₂, which in turn compromises even more the respiratory situation in a patient with RA. The usual treatment used in MAIk (sodium chloride, potassium chloride, suspension of diuretics, etc.) may not be prudent in patients with chronic RA, especially if they present with edemas. It is in this context when ACZ is especially effective when used for some days. It is a mild diuretic that increases renal excretion of bicarbonate by the inhibition of carbonic anhydrase that, over the long-term, may cause AM. ACZ has already demonstrated its usefulness in hypercapnic respiratory failure in patients with COPD or with obesity-hypoventilation syndrome, even when there is no accompanying MAIk. It is, however, especially useful when said association is given, just as the clinical and blood gas evolution of our patients seems to endorse.

In short, we can affirm that MAIk frequently complicates and perpetuates situations of RA. Thus, we believe it necessary for the clinical services that are involved to analyze this problem and to implement pertinent actions. Lastly, it would be important to initiate controlled, randomized studies in order to more closely define the effectiveness of ACZ in this situation.

References


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