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

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Patricia Carmona Soto, Miguel Congregado, Jesús Loscertales*

Servicio de Cirugía Torácica, Hospital Universitario Virgen Macarena, Sevilla, Spain

* Corresponding author.
E-mail address: jloscert@us.es (J. Loscertales).

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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 $PaCO_2$ increases.[4] Therefore, any deviation either above or

Table 1
Evolution of Patient Blood Gases.

	Admittance	Day 2	Day 6	Day 11	Day 13	Day 16
Case 1						
pH	7.44	7.48	7.49	7.49	7.43	7.37
PaO_2 (mm Hg)	53	65.6	83.3	33.6	57	78.4
$PaCO_2$ (mm Hg)	57.9	55.2	49.6	50.2	47.3	44.7
HCO_3^- (mmol/l)	38.2	40.9	37.7	38.1	27.5	25.1
HCO_3^- predicted (mmol/l)[a]	30.3	29.3	27.4	27.6	26.6	24.2
Potassium (mmol/l)		2.6		3	3.6	3.8
Treatment	Furosemide, 120 mg/day intravenously	Furosemide, 120 mg/day intravenously	Furosemide, 120 mg/day intravenously	Furosemide, 120 mg/day intravenously	Suspension furosemide. Acetazolamide, 500 mg/day, orally and KCl[b]	Suspension furosemide. Acetazolamide, 500 mg/day, orally and KCl[b]
	1st Consultation	Day 30	Day 45	Day 48	Day 52	
Case 2						
pH	7.49	7.5	7.46	7.40	7.41	
PaO_2 (mm Hg)	50.20	58.70	44.50	67.30	65	
$PaCO_2$ (mm Hg)	47	51.60	63.10	43	42.4	
HCO_3^- (mmol/l)	35	37.80	44.20	26.70	25.4	
HCO_3^- predicted (mmol/l)[a]	26.4	28.10	32.10	24.10	24.5	
Potassium (mmol/l)	2.9	3.3	3	3.7	4.1	
Treatment	Furosemide 60 mg/day, orally	Furosemide 60 mg/day, orally	Furosemide 60 mg/day, orally	Suspension furosemide. Acetazolamide, 500 mg/day, orally	Suspension furosemide. Acetazolamide, 500 mg/day, orally	

Normal values. Arterial blood: pH: 7.36–7.44; $PaCO_2$: 36–44 mm Hg; HCO_3^-: 22–26 mequiv./l. Venous blood: pH: 7.31–7.37; $PaCO_2$: 42–50 mm Hg; HCO_3^-: 23–27 mequiv./l.
[a] Calculation: for every 10 mm Hg of increase in $PaCO_2$, HCO_3^- increases 3.5 mmol/l.
[b] Potassium chloride.

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below predicted would indicate the coexistence of alkalosis—which aggravates hypoventilation—or of metabolic acidosis (MA).

To illustrate this, we feel it is interesting to report two cases of chronic RA coexisting with MAlk that we have treated recently and which reflect the worrisome reality of a much larger universe. The first patient presented with obesity-hypoventilation syndrome, and the second with chronic obstructive pulmonary disease (COPD), both with *cor pulmonale* in addition. The lack of recognition of the mixed disorder caused worsened symptoms and poorer blood gas analyses in the two cases. In both instances, treatment with 500 mg/day of acetazolamide (ACZ) for some days and the suspension of 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).

MAlk generally initiates with digestive loss (vomiting, nasogastric aspiration) or renal loss (diuretics) of hydrons (H^+). As the hydrons come from the dissociation of H_2CO_3, 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, MAlk 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 PaO_2 and increases $PaCO_2$, which in turn compromises even more the respiratory situation in a patient with RA. The usual treatment used in MAlk (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 MAlk.[5,6] 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 MAlk 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.

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José María Prieto de Paula,[a,*] Silvia Franco Hidalgo,[b] Laura Borge Gallardo,[a] Eduardo Mayor Toranzo[a]

[a] *Servicio de Medicina Interna, Hospital Clínico Universitario de Valladolid, Valladolid, Spain*
[b] *Servicio de Medicina Interna, Complejo Hospitalario de Palencia, Palencia, Spain*

* Corresponding author.
E-mail address: jmpripaula@yahoo.es (J.M. Prieto de Paula).

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