Hypercapnia Does Not Always Require Mechanical Ventilation

To the editor: Noninvasive mechanical ventilation is a technique used in the treatment of hypercapnic respiratory insufficiency.\(^1\) In recent years we have witnessed extended use of this type of ventilatory support but it must be remembered that not all patients with hypercapnia are candidates for this therapeutic modality.

Several times over the past months we have been asked to review noninvasive mechanical ventilation treatment for patients with general respiratory insufficiency secondary to what had been diagnosed as obstructive sleep apnea syndrome, obesity, hypoventilation syndrome, or chronic obstructive pulmonary disease. After taking a case history, assessing complementary tests, and revising treatment we observed that some patients in fact presented metabolic alkalosis with hypokalemia.

Metabolic alkalosis is a relatively common clinical condition, frequently brought about by the use of loop or thiazide diuretics or by the loss of gastric secretions through vomiting or nasogastric suction. The rise in plasma bicarbonate concentration can be attributable to a loss of renal or gastrointestinal hydrogen ions, intracellular shift of ions (which occurs with hypokalemia), administration of alkalis, or volume depletion (through massive diuresis, vomiting, or nasogastric suction in the case of achlorhydria, loss through sweat in the case of cystic fibrosis, or diarrhea secondary to laxative abuse or villous adenoma). Another factor is sometimes involved (volume and/or potassium depletion in most cases) to increase the net reabsorption of bicarbonate and impede the excretion of the excess in urine. The accumulation of bicarbonate produces an increase in the pH (alkalosis). The respiratory system tries to compensate through hypoventilation, reducing PaO₂ and increasing PaCO₂. Hypoventilation must be considered as a compensatory factor rather than a primary disorder in these cases.^{2,3}

The treatment of this metabolic disorder must be directed at the underlying cause. This includes treatment for volume depletion (sodium chloride), hypokalemia (potassium chloride instead of potassic citrate or aspartate), and hypochloremia. If the alkalosis is secondary to the use of loop or thiazide diuretics, these must be substituted for potassium-saving diuretics. If continuous gastric drainage is needed, a proton pump inhibitor should be administered to reduce acid loss. ^{2,3}

In edematous conditions, alkalosis is caused by excessive use of diuretics and accompanying secondary hyperaldosteronism. In these cases sodium chloride cannot be administered nor diuretic treatment suspended, so it is essential to correct the potassium deficit with potassium chloride, assess the association of acetazolamide (inhibitor of carbonic anhydrase) or potassium-saving

LETTERS TO THE EDITOR

diuretics, and, in patients with advanced renal insufficiency, consider dialysis treatment. If these measures do not produce a response, infusion of hydrochloric acid should be considered if the symptoms are severe (including hypoventilation) with PaCO₂ of over 60 mm Hg and pH over 7.55. Conditions accompanied by hypermineralocorticoidism, hypervolemia, and hypertension can be treated with potassium-saving diuretics, dexamethasone, potassium chloride, and prostaglandin inhibitors.^{2,3}

In conclusion, it should be remembered that not all patients with hypoxemia and hypercapnia should be treated with noninvasive mechanical ventilation. The presence of metabolic alkalosis should be considered as it necessitates the correction of hydroelectrolyte alterations in order to control compensatory hypoventilation.

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