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Metabolic Acidosis Caused by Laxatives in a Patient with Duchenne Muscular Dystrophy Receiving Non-Invasive Mechanical Ventilation☆

Acidosis metabólica por laxantes en paciente con distrofia muscular de Duchenne y ventilación mecánica no invasiva

Dear Editor,

Patients with Duchenne's muscular dystrophy (DMD) have a high incidence of constipation, and the chronic use of laxatives is an important component of their treatment. It has been postulated that the underlying cause of constipation in these patients is the functional deterioration of the smooth muscles of the gastrointestinal tract, which can cause gastric dilation, slowing of gastric and ileal emptying, and even intestinal pseudo-occlusion. Pathological and functional smooth muscle abnormalities appear to be derived from dystrophin deficiency, the locus-encoded protein associated with DMD.[1] Gastric or intestinal dilation may be aggravated specifically in patients with hypercapnic respiratory failure, due to the ingestion of air during non-invasive ventilation (NIV).[2] It is well known that treatment with laxatives can lead to the development of metabolic acidosis,[3,4] an electrolyte disturbance that could have respiratory consequences in patients with chronic hypoventilation.

We report the case of a 37-year-old DMD patient with chronic hypercapnic respiratory failure, who required NIV 12 h a day. A stable phase lung function study showed: FEV_1/FVC 94%, FVC 26%, FEV_1 30%, MIP 12%, MEP 8%, peak cough flow 149 L/min, pH 7.37, pO_2 86.1 mmHg, pCO_2 49.2 mmHg, HCO_3 27.9 mEq/L. Routine outpatient monitoring revealed iron deficiency anemia, so oral iron was added to his usual treatment (betahistine, dihydrochloride, and paracetamol). The patient's chronic constipation worsened, so magnesium hydroxide [$Mg(OH)_2$] was started as a laxative. Successive follow-ups detected a worsening of the acid-base balance with the appearance of metabolic acidosis (see Table 1) and an increase in ventilatory support needs of up to 18–20 h a day. The patient's targeted clinical history showed no focus of infection. Blood gases also showed a decrease in pCO_2 and bicarbonate (HCO_3). Correct renal function and the presence of a normal anion gap (sodium 139 mmol/L, chlorine 102 mmol/L) were confirmed. Given the suspicion of metabolic acidosis caused by the use of magnesium hydroxide, this compound was replaced with bisacodyl and lactitol. Subsequent blood gas monitoring showed the correction of acidosis, presenting results similar to the baseline status. Two years later, the patient reinitiated magnesium hydroxide treatment for persistent constipation, resulting in a new episode of metabolic acidosis (see Table 1).

The chronification or worsening of metabolic acidosis can have several consequences, including cardiac contractility changes with decreased cardiac output, increased incidence of arrhythmias, arterial and venous vasodilation, increased pulmonary vascular resistance, metabolic demands, insulin resistance, anaerobiosis due to reduced adenosine-triphosphate synthesis (ATP), hyperkalemia, and alterations in the level of consciousness.[5,6]

Specifically, magnesium hydroxide is an osmotic laxative that can disturb the acid–base balance by the loss of bicarbonate, and the higher the loss, the greater the degree of acidosis.[3,7,8] The initial response to bicarbonate loss is decreased pCO_2 due to the stimulation of ventilation, producing hyperventilation to normalize pH. On average, the loss of 1 mEq/L of HCO_3 is compensated by a 1.2 mmHg drop in pCO_2,[9] but this response cannot be maintained, whether metabolic acidosis becomes chronic or if the patient is unable to respond to the respiratory demand, and may result in muscle fatigue, altered ventilation mechanics and increased NIV needs. Based on this mechanism, the use of acidifying drugs as part of the treatment of residual hypercapnia in patients receiving home mechanical ventilation has been proposed.[10] However, in patients with neuromuscular diseases, the compensatory hyperventilation mechanism could lead, as in this case, to an increase in the work of breathing.

Table 1
Course of Blood Gases.

	Stable Phase	Treatment With $Mg(OH)_2$	Discontinuation of $Mg(OH)_2$	Reintroduction of $Mg(OH)_2$
pH	7.37	7.33	7.35	7.32
pCO_2 (mmHg)	49.2	45.4	47.5	43
pCO_2 (mmHg)	86.1	93.3	87.4	108
HCO_3 (mEq/L)	27.9	22.5	25.6	21.5
Base excess (mEq/L)	3	−1.7	0.7	−3.7

HCO_3: bicarbonate; $Mg(OH)_2$: magnesium hydroxide; pCO_2: partial pressure of carbon dioxide; pO_2: partial pressure of oxygen.

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It is therefore important to be cautious when using osmotic laxatives in patients with neuromuscular pathology and chronic respiratory failure receiving treatment with home mechanical ventilation, as the consequences could be particularly severe.

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Evaluation of the Response to the Radiofrequency Treatment of a Pulmonary Nodule by Contrast-enhanced Ultrasound (CEUS)☆

Evaluación de la respuesta al tratamiento con radiofrecuencia de un nódulo pulmonar mediante ecografía con contraste (CEUS)

Dear Editor,

Computed tomography (CT)-guided radiofrequency ablation (RFA) is a minimally invasive technique used to treat solid tumors that offers a reliable alternative to surgery in oligometastatic lung disease[1] and in early stage primary lung cancer.[2] A major challenge in treating lung lesions with RFA has been the reliable evaluation of treatment response after the procedure. Unlike surgical resection, in which a histopathological analysis is performed post-procedure, the treated tumor is left *in situ* during RFA, so direct histopathological verification is not possible. The options currently available for post-procedure follow-up are CT,[3] positron emission tomography (PET), and dual-modality imaging (PET-CT).[4] CT and PET are unable to detect microscopic tumor foci and are less than optimal in the detection of early recurrence.[5] Contrast-enhanced ultrasound (CEUS) is a technique that has been used for years in the immediate follow-up of the ablative treatment of focal lesions, especially in liver and kidney disease.[6,7] Given the history of CEUS as a technique in evaluating the response of lesions treated with RFA in other parts of the anatomy, it is possible that in subpleural lesions accessible by ultrasound examination, CEUS may have the potential to be a complementary tool to CT and PET-CT in the evaluation of lung lesions treated by RFA.

We present the case of a 73-year-old man who was diagnosed in August 2010 with cutaneous squamous cell carcinoma of the basal cells after years of skin lesions on the cheek and shoulder. Treatment involved surgical resection of the affected areas, and subsequent locoregional recurrences were also treated surgically. The patient then presented with a new recurrence involving 2 subpleural solid pulmonary nodules in the right lower lobe and another lesion in the left lower lobe. Palliative chemotherapy was administered, composed of carboplatin, 5-fluorouracil, and cetuximab, and complementary treatment with taxol. Subsequent radiological controls showed stable disease until November 2016, when an increase in the size of the lesions was reported (Fig. 1A). The multidisciplinary committee of our center decided to perform CT-guided RFA on the 2 lesions located in the right lower lobe and to apply radiation therapy to the contralateral lesion.

On January 30th, 2017, CT-guided RFA was performed on the 2 nodules located in the right lower lobe. One lesion was treated with a 3 cm umbrella needle (Fig. 1B), which resulted in mild pneumothorax and halo after treatment, and the other was treated with a 2 cm straight needle. In the immediate follow-up CT, right pneumotho-

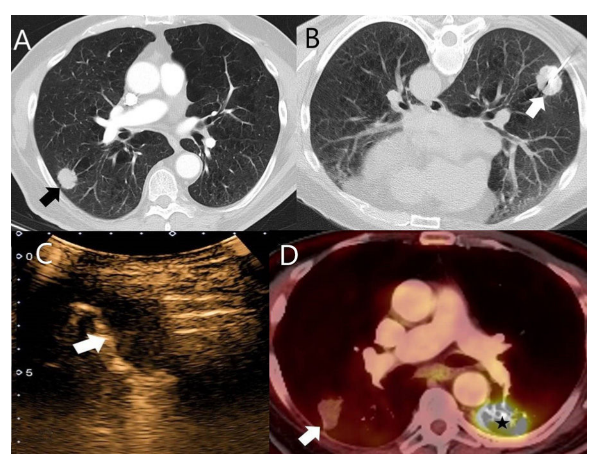

Fig. 1. (A) Chest CT with iodinated IV contrast: lung window. A solid subpleural nodule (arrow) is seen in a patient with metastatic disease of cutaneous squamous cell carcinoma of the basal cells. This node was treated using CT-guided RFA. (B) Axial slice with patient in a supine position during CT-guided RFA. An umbrella needle (arrow) was used. (C) Pulmonary CEUS of the same lesion. No contrast uptake is observed throughout the procedure, indicating a complete response to ablative treatment. (D) Chest PET-CT of the same patient 1 year after ablative treatment, showing a decrease in the size of the treated lesion and no FDG uptake (arrow), consistent with a complete response. A pathological increase in FDG was observed in the left lower lobe lesion treated with radiation therapy (star).

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