



NEGLECTED ASPECTS OF LONG-TERM OXYGEN THERAPY IN SEVERE COPD PATIENTS - CASE REPORT

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Objective:

Introduction: Long-term oxygen therapy (LTOT) is the cornerstone mode of treatment in patients with severe chronic obstructive pulmonary disease (COPD) associated with resting hypoxaemia. When appropriately prescribed and correctly used, LTOT has clearly been shown to increase survival and improve quality of life. Despite all the benefits of LTOT there are some often neglected aspects that adversely affect life quality of patients, these are e.g. homebound patients, physical inactivity, severe and prolonged vitamin D deficiency, osteomalacia and osteoporosis.

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Case report: We present a clinical case of a 70-years old male patient with a history of severe COPD (GOLD 4, D), bullous emphysema, global respiratory insufficiency and chronic dilatative cardiomyopathy. COPD was diagnosed in 2008. Patient is using LTOT via a nasal cannula at a flow rate of 1-2L/min since 2010. He has frequent exacerbations (1-2 times a year) that require hospitalization. This time he was admitted to the hospital for exacerbation of dyspnea, productive cough and peripheral edema. On physical examination, he was moderately dyspnoeic and tachypnoeic, afebrile and less mobile. Chest plain radiograph showed a hyperinflated chest and bilateral pneumonia. Initial arterial blood gas showed respiratory acidosis and laboratory test showed microcytic anaemia, leukocytosis and elevated CRP. As part of diagnostic processing, significant hypophosphatemia and hypocalcaemia were observed and the patient was examined by an endocrinologist who stated that the electrolyte disturbance was probably due to vitamin D deficiency. To confirm this, additional laboratory processing was performed. Fortunately, at this time patient did not express any clinical symptoms of osteomalacia. He was treated with parenteral antibiotics, received standard bronchodilator therapy, diuretic, iron preparations, vitamin D preparations and calcium carbonate. Due to respiratory failure he was briefly ventilated with NIV. The applied treatment measures improved the general condition and respiratory status of the patient. Inflammatory parameters and radiological finding were also improved. In a stable condition, he was discharged home with a recommendation to use LTOT 1-2 L/min, bronchodilator therapy, vitamin D preparations and calcium carbonate.

Conclusion: We aimed to show the importance in raising awareness of vitamin D deficiency in homebound COPD patients. Vitamin D deficiency can precipitate or exacerbate osteopenia and osteoporosis, cause osteomalacia and muscle weakness, and increase the risk of fracture. It is important to detect and treat vitamin D deficiency in still asymptomatic patients and thus prevent the development of complications.