



HYPOPHOSPHATEMIA AS A FORGOTTEN CAUSE OF RESPIRATORY INSUFFICIENCY

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Objective:

INTRODUCTION

Hypophosphatemia is a common laboratory abnormality that occurs in 70% of patients in the intensive care units. It may lead to a multitude of symptoms, including cardiac and respiratory failure. After NIV on wards for management of COPD, asthma exacerbations and severe infections (particularly *Legionella*), hypophosphatemia became more often presented to pulmonologists.

The mechanism is considered to be decreased availability of phosphate-containing energy sources.



Depletion of 2,3- diphosphoglycerate shifts the oxygen dissociation curve to the left, decreasing oxygen delivery to peripheral tissue. This might be especially relevant in patients with chronic pulmonary disease; these patients may have higher 2,3-DPG levels to compensate for hypoxemia.

In metabolic acidosis renal excretion of phosphate is increased.

In respiratory alkalosis redistribution across the cell membrane occurs, what is the most common cause of hypophosphatemia in ICU patients.

CASE REPORT

We present a case of 48- years old woman with a history of diabetes, obesity, recurrent urticaria and smoking (20 p/y). COPD was diagnosed at her age of 32- years. She was hospitalized 3-4 times a year due to exacerbations, regardless of the season, few times in ICU. Permanently was treated with LABA+LAMA+ICS. She was hospitalized at our department under diagnosis pneumonia and COPD exacerbation. We realized that she was unrecognized severe asthma for 15 years. She has blood eosinophilia(380 cells/mcL peripherall blood) and normal AAT level (1.43 g/L). Spirometry have shown severe obstructive disorder but lung diffusing capacity was normal. CT scan excluded empysema as we could expect for patient with COPD and long-term smoking.



Patient was treated with antibiotics, corticosteroids, bronchodilators, low molecular heparin. Venipuncture was made because of polycythemia. Serum phosphate was found very low: 0.37 mmol/L. Because the serum phosphate concentration was less than 0.40 mmol/L, it was indicated administration of potassium phosphate in a dose of 10 ml+100 ml saline over 10 h for three days, after which the level was normalized. Right diagnose with adequate and prompt treatment led to satisfactory recovery of our patient, so her FEV1 at discharge from hospital was 65% of predicted.

CONCLUSION

Serum phosphate levels are not routinely measured in patients with respiratory insufficiency. Due to high prevalence of respiratory failure in asthma and COPD exacerbations, laboratory monitoring is recommended so hypophosphatemia should be early recognized and treated.

It is important to investigate in future whether hypophosphatemia causes higher mortality in itself or rather is associated with a higher severity of illness.