

Bronchospasm secondary to Hypocalcemia in a patient with chronic obstructive pulmonary disease: A case report

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Abstract

Iatrogenic hypoparathyroidism and hypocalcemia are frequent complications in patient's undergone head and neck surgery. Symptoms of hypocalcemia; such as bronchospasm should be differentiated from bronchial obstructive symptoms of chronic obstructive pulmonary disease (COPD). We present a case with COPD and hypoparathyroidism resulted in hypocalcemic bronchospasm. Symptoms of the patient was resisted during bronchodilator therapy and only resolved by emergency treatment of hypocalcemia.

Keywords: hypoparathyroidism, hypocalcemia, bronchospasm, chronic obstructive pulmonary disease

1. Introduction

Iatrogenic hypoparathyroidism and hypocalcemia are frequent complications in patient's undergone head and neck surgery. Iatrogenic hypoparathyroidism is the most common form of acquired hypoparathyroidism [1, 2]. Decreased parathyroid hormone (PTH) levels leads to increased renal calcium loss and reduced intestinal calcium absorption via decreased Vitamin D levels [3]. It develops by removal, devascularization or damage to the parathyroid glands during surgery [4]. It may cause transient or permanent hypocalcemia. Postoperative hypoparathyroidism is usually transient and recover in a couple of weeks or months [5]. Permanent hypocalcemia is defined as insufficient parathyroid hormone levels to maintain calcium homeostasis 6 months after surgery [4].

Clinical symptoms of hypocalcemia vary on the depth and development rate of hypocalcemia [3]. Most of the mild cases are asymptomatic. Many of the signs and symptoms occur by the increase in neuromuscular irritability. Characteristic symptoms include numbness and tingling at upper and lower extremities and perioral region and carpopedal spasm [3]. Tetany is the most typical symptom of severe hypocalcemia which may occur latent or spontaneously. Chevostek and Trousseau signs are both indicators of latent tetany, however, trousseau sign is more specific for hypocalcemia than chevostek sign [6]. In severe cases, neurologic disorders including confusion, disorientation, and delirium; cardiac disturbances such as prolonged QT interval, arrhythmias and congestive heart failure, and laryngo-bronchospasms which may lead to respiratory failure [7].

An acute bronchospasm case in a patient with hypocalcemia caused by iatrogenic hypoparathyroidism mimicking acute exacerbation of chronic obstructive pulmonary disease (COPD) is presented.

2. Case Presentation

A 71 year old man was admitted to the emergency department with shortness of breath and palpitations. He had been diagnosed as COPD 20 years ago and he described similar complaints in the past which resolved with additional doses of his inhaled

medications. Despite application of additional inhalers, his symptoms were not reduced during last 24 hours. On admission, his blood pressure was 105/60 mmHg, heart rate was 118 beats per minute and rhythmic, body temperature was 36.3 centigrade degree and oxygen saturation was 88%. Chest examination revealed bilateral diffuse ronchi. He diagnosed as acute exacerbation of COPD and consulted to a pulmonologist at emergency room. Severe bronchospasm was persisted despite use of intensive inhaled steroids, beta-2 agonists and anticholinergics and systemic corticosteroids.

After detection of hypocalcemia in blood biochemistry, patient was consulted to an internist. Medical inquiry and physical examination repeated. Patient had a tracheostomy, since total laryngectomy operation 8 years ago due to larynx cancer. He was been on medication of L-thyroxin 150 mcg and calcium plus vitamin D daily for treatment of postoperative hypothyroidism and hypoparathyroidism. He confessed that he stopped using calcium plus vitamin D for the last one year. On physical examination, in addition to the bilateral rhonchi, chevostek and trousseau signs were elicited.

Bronchospasm due to hypocalcemia was the diagnosis after detection of a corrected calcium (Ca) level of 4.8 mg/dL in blood biochemistry of the blood samples. Emergency treatment of hypocalcemia was started and patient hospitalized. 2 vials of 10% calcium gluconate infused intravenously in 100 5% dextrose solution in 10 minutes. Bronchospasm was resolved with emergency treatment. Then, eight vials of 10% calcium gluconate infused intravenously in 1000 cc of 0.9% NaCl solution in 24 hours. Corrected Calcium level was risen to 6.96mg/dl in repeated analyze. Other serum biochemical test results were as follows; urea: 73 mg/dl (12-42 mg/dL), creatinine: 0.81 mg/dL (0.72-1.25 mg / dl), Ca: 6 mg/dl (8.8-10 mg/dl), albumin: 2.8 mg/dL (3.4-4.8g/dl), corrected Ca: 6.96 mg/dL, magnesium: 1,41 mg/dL (1.8-2.6 mg/dL), phosphorus: 4.2 mg/dL (2.5-4.5 mg/dL), parathyroid hormone (PTH):2.6 pg/ml (12-88 pg/ml), 25-Hydroxy Vitamin D: 10.3 ng/mL (20-52 ng/ml) and serum alkaline phosphatase: 57 IU/L (40-150 IU/L). Thyroid function tests were within the normal range,

therefore, oral L-thyroxin continued with the same dosage. Calcitriol 1 mcg and calcium carbonate 2g daily has been prescribed for maintenance therapy. Serum calcium reached to normal level of 8.5 mg/dl on 2nd day. The patient was discharged with appropriate recommendations.

3. Discussion

We reported here an iatrogenic hypoparathyroidism case presented with acute symptoms of hypocalcemia mimicking acute exacerbation of COPD.

Iatrogenic hypoparathyroidism is a well-known entity after head and neck surgeries especially in patients undergone total laryngectomy. Supplementation of active vitamin D (Calcitriol) and oral calcium tablets is recommended to maintain calcium homeostasis for such patients. In present case, patient had been stopped to use usual vitamin D and oral calcium supplements which lead to hypocalcemia in over a year period. Hypocalcemia usually occur acutely over minutes to hours, as in post-parathyroidectomy period, or chronically over weeks to months, as during chronic kidney disease and discontinuation of supplementation in hyperparathyroidism^[8]. Patients usually can tolerate severe hypocalcemia in chronic hypocalcemia^[9]. However, our patient developed acute symptoms in a hypocalcemia developed in chronic fashion. Similar to our case, Diniotis *et al.*, reported severe hypocalcemia required treatment with intravenous calcium gluconate in a patient undergone laryngectomy for larynx cancer 4 years before his admission^[10]. Manifestations of increased neuromuscular irritability related to hypocalcemia are tetany, convulsions, laryngospasm and bronchospasm^[8]. Accordingly, our patient presented with bronchospasm in presented case. Hypocalcemic symptoms relate to the absolute level of serum calcium and to the rate of change in the serum calcium concentration. Therefore, in chronic hypocalcemia, patients should be asymptomatic even with extremely low calcium concentrations. In fact, our patient was maybe asymptomatic for hypocalcemia during 1 year period of calcitriol and calcium free period. Nevertheless, contributing factors should aggravated symptoms of hypocalcemia.

Bronchospasm is an acute symptom of acute hypocalcemia, as a result of neuromuscular irritability^[8]. The patient presented was also suffered from bronchospasm resistant to bronchodilator therapy but calcium infusion.

Another explanation for acute hypocalcemia symptoms in such a relatively chronic hypoparathyroidism case could be that additional drugs that may contribute a more severe hypocalcemia. Steroids decrease serum calcium levels and therefore they are used in treatment of hypercalcemia. A study in literature reported hypocalcemia in a patient treated with corticosteroids^[11]. Moreover, a study in literature concluded that hypocalcemia after parathyroidectomy could be reversed by adrenalectomy^[12]. Our patient was irregularly used inhaled steroid, however, he began to use it in a more frequent dosage than usual recently because of subjective sense of dyspnea for a couple of days before admission to emergency department. Therefore, increased inhaled steroids might contribute acute hypocalcemia symptoms in present iatrogenic chronic hypoparathyroidism case.

Patient presented was also increased the dosage and rate of inhaled beta mimetics due to the same reason of steroids. Increased catecholamines may reduce serum magnesium levels which may cause or exaggerate hypocalcemia^[13, 14]. Moreover, Whyte *et al.*, found that Salbutamol decreased the serum levels

of magnesium^[15]. Our patient was extremely used such medicines for COPD recently.

4. Conclusion

In conclusion, we suggest to consider hypocalcemia induced bronchospasm in COPD patients during acute attack, especially when patient's medical history was relevant with head and neck surgery.

5. References

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