

Effect of Corticosteroids on Serum Potassium Levels in Patients with Obstructive Airway Disorders

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ABSTRACT

Corticosteroids, also called glucocorticoids or steroids, are hormones produced by the adrenal cortex, part of the adrenal glands. These hormones affect almost all body organs and are extremely important in maintaining homeostasis when secreted in normal amounts to have different functions. The goals of corticosteroids during treatment are to decrease exacerbation of symptoms and improve quality of life in patients with obstructive airway diseases. Thus corticosteroids play an important role in the therapy of obstructive airway diseases. Hypokalemia is one of the major adverse effects of corticosteroids. Serum potassium is the most specific indicator available for the diagnosis of hypokalemia. This review is designed to investigate the effects of corticosteroids on serum potassium levels in patients with obstructive airway disorders along with the route which caused more effects and the drug which caused more prominent effects.

Method: This is a prospective analytical study is going to be conducted in the department of Pulmonary Medicine, Pushpagiri Medical College Hospital, Thiruvalla. All patients who are willing to participate in the study are briefly explained about the study procedure. Approximately 60-65 patients will be taken into consideration. Blood sample (residual) of 3ml is required which is obtained from the laboratory and serum potassium level is estimated on admission and after 3 days. The patients are selected based on inclusion and exclusion criteria. The level of electrolyte can be analysed by using semi autoanalyzer. The results obtained from the study can be compared with the normal range of potassium in blood.

Keywords: Obstructive airway disorders, Potassium, Corticosteroids, Hypokalemia.

INTRODUCTION

Obstructive airway disease is a category of respiratory disease characterized by airway obstruction. Many obstructive diseases of the lung result from narrowing of small bronchi and larger bronchioles, often because of excessive contraction of smooth muscle itself. Types of obstructive lung diseases include asthma, bronchiectasis, bronchitis and chronic obstructive pulmonary diseases, cystic fibrosis

Corticosteroids, also called glucocorticoids or steroids, are hormones produced by the adrenal cortex, part of the adrenal glands. The term corticosteroids actually refer to all secretions of the adrenal cortex, but it is most often used to designate the glucocorticoids, which are important in metabolic, inflammatory, and immune processes. Glucocorticoids include cortisol, corticosterone and cortisone. Major exogenous glucocorticoids used are hydrocortisone, triamcinolone,

prednisolone, methylprednisolone, dexamethasone, deflazacort functions. Mineralocorticoids play a vital role in the maintenance of fluid and electrolyte balance. Aldosterone is the main mineralocorticoid and is responsible for approximately 90% of mineralocorticoid.

Corticosteroids are commonly used in the treatment of asthma because of their anti-inflammatory effects. In acute asthma or status asthmaticus high doses of systemic corticosteroids are given orally and iv respectively. In chronic asthma, inhaled corticosteroids are the drugs of first choice. Corticosteroids are more helpful in acute exacerbations than in stable Chronic obstructive pulmonary diseases (COPD). Inhaled corticosteroids produce minimal adverse effects. These are also used in arthrides, eye diseases, septic shock, thyroid storm, skin diseases, Inflammatory bowel diseases.

Potassium is a very important mineral for the proper function of all cells, tissues, and organs in the human body. Potassium is crucial to heart function and plays a key role in skeletal and smooth muscle contraction, making it important for normal digestive and muscular function.

Hypokalemia and muscle weakness can be seen after administration of glucocorticoids.

Hypokalemia is generally defined as a serum potassium level of less than 3.5 mEq/L (3.5 mmol/L). Severe hypokalemia is a level of less than 2.5 mEq/L. If potassium levels in blood is low; weakness, fatigue, lassitude, lack of energy, palpitation, abnormalities in ECG, cardiac arrhythmias occurs. Glucocorticoids, such as prednisone and hydrocortisone, increase potassium excretion nonspecifically through their effect on the filtration rate and distal sodium delivery. When given over the long term, these drugs reduce serum potassium only slightly (by 0.2 to 0.4 mmol per liter).

When serum potassium decreases to less than 2.5 mmol per liter, muscle necrosis can occur, and at serum concentrations of less than 2.0 mmol per liter, an ascending

paralysis can develop with eventual impairment of respiratory function. Potassium depletion and hypokalemia increase both systolic and diastolic blood pressure when sodium intake is not restricted, presumably by promoting renal sodium retention.

Hypokalemia is always the result of potassium depletion induced by abnormal losses of potassium. More rarely, hypokalemia occurs because of an abrupt shift of potassium from the extracellular compartment into cells.

REVIEW ARTICLES:

1) Said A. Omar ^[1] et al., (2000); conducted a study on "Effect of Prenatal Steroids on Potassium Balance in Extremely Low Birth Weight Neonates." (ELBW) Serum potassium (SK) concentration, potassium intake and output, and renal clearance were collected prospectively each day during the first week of life. Infants whose mothers received a full course of steroids before delivery (PNS group: $n = 16$) were compared with those infants whose mothers did not receive steroids (nonsteroid group [NSG]: $n = 14$). Potassium intake and excretion and serum and urine electrolytes were measured every 12 hours, and urine output was monitored every 2 to 3 hours. The peak SK was significantly lower in the PNS group than in the NSG group ($5.2 \pm .2$ mmol/L vs $6.2 \pm .4$ mmol/L). Moreover, the peak SK was higher than 6.5 mmol/L in 70% of the NSG infants and in none of the PNS group. Hyperkalemia occurred in the NSG infants within the first 2 days when urine output was significantly lower than in PNS infants. The conclusion of the study was that treatment with PNS prevents the nonoliguric hyperkalemia known to occur in ELBW neonates.

2) G Leitch ^[2] et al., (1976); conducted a study on "Effect of intravenous infusion of salbutamol on ventilator response to carbon dioxide and hypoxia and on heart rate and plasma potassium in normal men". Intravenous infusion of salbutamol 10 Lg/min was given in seven healthy subjects.

There was a significant increase in their ventilator responses to inhaled CO₂, in both hypoxia and hyperoxia. These changes in chemical control of breathing are unlikely to be significant when the drug is used in severe asthma but may benefit patients with acute exacerbations of chronic ventilatory failure. The infusion also increased heart rate, which was most pronounced when hypoxia was combined with hypercapnia. The infusion produced an average fall in plasma potassium from 3.99 to 3.10 mmol/l, which was associated with an increase in plasma glucose and serum insulin, suggesting that this arose from a shift of potassium from the extracellular to the intracellular space. Routine monitoring of plasma potassium and the electrocardiogram is indicated when an intravenous salbutamol infusion is used to treat severe asthma as the drug may predispose to cardiac dysrhythmias.

3) Weng-Sheng Tsai [3] et al.,(2004); conducted a case study on Life-Threatening Hypokalemia in an Asthmatic Patient Treated with High-Dose Hydrocortisone in a 66-year-old man who complained of generalized muscle weakness, shallow respiration, and palpitations after receiving high dose intravenous HC (total dose, 2400 mg over 4 days) to treat a severe asthma attack. During this therapy, there was a weight gain of 1.0 kg. An electrocardiogram revealed ventricular arrhythmia with frequent premature ventricular contractions. Hypokalemia was profound, with plasma potassium (K⁺) concentration of 1.7 mEq/L, and associated with renal potassium wasting, as evidenced by a transtubular potassium concentration gradient of 12; metabolic alkalosis (plasma HCO₃⁻, 37 mEq/L) was also present. When treated with spironolactone, KCl supplementation, and substitution of HC with prednisolone, his plasma K⁺ concentration rapidly normalized, metabolic alkalosis was corrected, and arrhythmia disappeared within 3 days. Because of unwanted mineralocorticoid side-effects, high-dose HC may cause life-threatening hypokalemia

in asthmatic patients. Because of these potential risks, plasma acid-base and electrolyte concentrations should be monitored frequently in any patient treated with high-dose HC.

4) J. M. Cruickshank [4] et al.,(1974);conducted a study of “Possible role of catecholamine’s, corticosteroids, and potassium in production of electrocardiographic abnormalities associated with subarachnoid haemorrhage”. 40 patients with subarachnoid haemorrhage were selected to correlate electrocardiographic abnormalities with urinary normetanephrine and metanephrine, plasma cortisol, and with serum and total body potassium levels. Urinary normetanephrine and metanephrine, and plasma cortisol values were significantly raised in 19 patients with abnormal electrocardiograms compared with the 8 patients with normal cardiograms. No significant differences in serum potassium levels in patients with either normal or abnormal electrocardiograms were revealed. 13 cases had total body exchangeable potassium measured about one week after subarachnoid haemorrhage, and abnormally low or low normal levels were observed in 9 of the 10 patients with abnormal or changing electrocardiograms. The conclusion of the study was the possible mode of action of catecholamines upon the heart with accent on the sensitizing effect of corticosteroids. This permissive role of corticosteroids probably stems from their ability to cause potassium deficiency, not reflected in serum levels, where a low myocardial intracellular potassium renders the heart susceptible to the necrotizing effect of catecholamines. It is, therefore, suggested that catecholamines, potentiated by corticosteroids and the associated potassium deficiency, are responsible for electrocardiographic abnormalities associated with subarachnoid haemorrhage.

5) Dr. Robert J. Hoffman [5] et al., (2001); conducted a study on “Clenbuterol Ingestion Causing Prolonged Tachycardia, Hypokalemia, and Hypophosphatemia with

Confirmation by Quantitative Levels. A poisoned patient, a 28-year-old woman, developed sustained sinus tachycardia at 140/min, hypokalemia (2.4 mEq/L, 2.4 mmol/L), hypophosphatemia (0.9 mg/dL, 0.29 mmol/L), and hypomagnesemia (1.52 mg/dL, 0.76 mmol/L) after ingesting a reportedly small quantity of clenbuterol. The patient received repeated doses of metoprolol to treat her cardiovascular stimulation and potassium chloride to treat her hypokalemia. She remained symptomatic for more than 20 hours after the ingestion

CONCLUSION

Corticosteroids are shown to cause a decrease in serum potassium levels in patients known to administer them. Thus regular monitoring of serum potassium levels can prevent hypokalemic symptoms and complications.

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