

UNUSUAL CAUSE OF HYPOKALAEMIA AND PARALYSIS IN AN ELDERLY PATIENT WITH BRONCHIAL ASTHMA

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DOI: 10.5455/ijmsph.2013.030720132

Received Date: 03.03.2013

Accepted Date: 03.07.2013

ABSTRACT

Asthma is a syndrome characterized by airflow obstruction that varies markedly, both spontaneously and with treatment. There are so many adverse effects of drugs which are used in the management of bronchial asthma in elderly. We report a case of sudden onset paralysis of both lower limbs after treatment of asthma with nebulized β_2 -agonists. The inappropriate and continuous use of such drugs may cause the hypokalaemic paralysis though lifesaving many times. Hypokalaemia is a common adverse effect of β_2 -agonists but hypokalaemic paralysis of both lower limbs is a rare entity.

Key-Words: Asthma; β_2 -Agonists; Hypokalaemia; Paralysis

Introduction

It has been suggested that sodium and potassium levels influence the responsiveness of the airway smooth muscles.^[1,2] The interest in the electrolyte disturbance in asthma patients has so far been focused on the serum potassium levels which are especially linked to the therapy with β_2 -agonists.^[3-5] Tremors, tachycardia, palpitations, and anxiety are the well-known side effects of such treatments.^[6] The mortality rate in patients with asthma is still rising and has been partly attributed to the adverse effects of the β_2 -agonists which are administered for asthma management.^[7] The use of β_2 -agonists may lead to hypokalaemia. The inappropriate and continuous use of such drugs may also cause hypokalaemic paralysis.

The monitoring of the electrolytes may be warranted in elderly asthmatic patients to decrease the mortality.

Case Report

An 80 year old elderly male who is a known case of bronchial asthma presented to the emergency room with the history of sudden onset of weakness of both the lower limbs since one day and initially has taken treatment from a chest physician for worsening symptoms of

breathlessness and chronic cough for the last one week. The patient had gone to bed at 10 pm with no weakness and awoke in the early morning to go to toilet he noticed the buckling of the knee joints. The weakness was bilateral and involved both the proximal muscles and hips as well as the distal extremities. He had mild pain in both thighs intermittently. He had no history of swallowing difficulty and was able to move his neck and facial muscles. There was no history of either sensory symptoms or bladder symptoms however there was history of not passed stools since one day. No prior history of similar complaints in the past. Patient had denied any recent diarrhoea, backache, radicular pain or any change in the mentation. Drug history revealed that patient had taken salbutamol nebulisation every 4th hourly for initial two days and every 6th hourly for next five days. He had also taken BID dose of beclomethasone inhalation since one week. Apart from this no other medications were used.

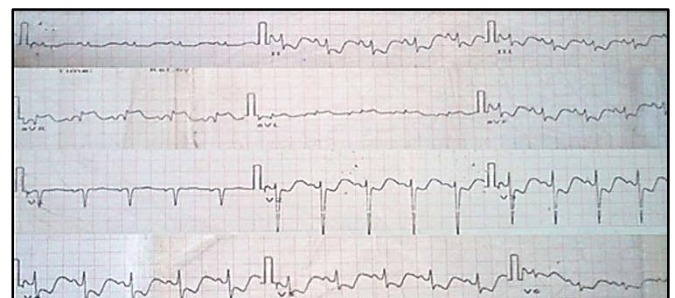


Figure-1: ECG showing Prominent U Waves and ST-T changes due to Hypokalaemia

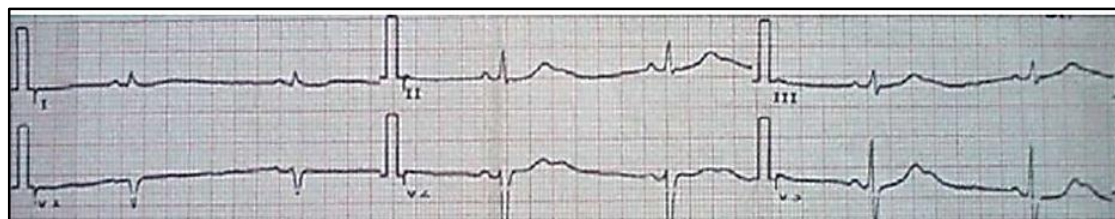


Figure-2: Normalization of ECG after Correction of Hypokalaemia

Patient denied use of alcohol or significant changes in diet or activity levels.

On physical examination, the patient's heart rate was 110/minute and blood pressure was 140/80 mmHg. He had BMI of 21.1 kg/m². No jugular venous distension, cyanosis or clubbing. Cardiac examination revealed sinus tachycardia with a regular rhythm and no murmurs. Examination of the lungs revealed scattered ronchi and abdomen examination showed absent bowel sounds. There were no deformities or edema of the extremities. Neurologic exam revealed flaccid paralysis of both lower limbs which involved the proximal and distal muscles of the hips and ankles. Sensation was intact but deep tendon reflexes were slightly diminished. Cranial nerve function was grossly intact.

Routine blood chemistry, liver enzymes and complete blood count were normal except for a potassium level of 2.5 (Normal - 3.5–5 mmol/L). Electrocardiogram revealed (Figure 1). Patient was initiated on intravenous potassium replacement, the patient's neurologic symptoms almost completely improved over two days. Repeat electrocardiogram revealed a normal sinus rhythm and rate (Figure 2). Follow up studies were performed to determine the etiology of the patient's hypokalaemia. Urine sodium and potassium, and serum aldosterone and renin levels were measured to rule out adrenal involvement and were found to be normal. Thyroid stimulating hormone (TSH), triiodothyronine (T3) and thyroxine (T4) levels were normal.

Discussion

Weakness is a common, albeit non-specific, presentation in both the emergency and outpatient setting. Although the differential diagnosis for the complaint of weakness is

extensive (Table 1), the focus is considerably narrowed when a patient presents with a demonstrable decrease in muscle strength on physical exam. A perturbation of sodium and calcium ion channels results in low potassium levels and muscle dysfunction.^[8] As this is primarily a problem with muscle contraction rather than nerve conduction, tendon reflexes may be decreased or absent but sensation is generally intact. Although the serum potassium level is often alarmingly low, other electrolytes are usually normal. Indeed, total body potassium is actually normal with the change in the serum level reflecting a shift of potassium into cells.^[9]

Table-1: Causes of Acute Weakness

Neurologic	Infectious
Stroke	Polio
Post-seizure paralysis	Diphtheria
Myasthenia gravis	Botulism
Cataplexy	Metabolic
Multiple sclerosis	Porphyria
Inflammatory	Alcohol/Opiates
Polymyositis	Electrolyte disorders
Dermatomyositis	

For acute asthma, repeated doses of nebulized β_2 -agonists and to a lesser extent, IV aminophylline, is the mainstay therapies which are used to relieve bronchospasm and airway obstruction.^[10,11] The decreased serum potassium levels may occur due to the active inhibition of potassium secretion in the cortical collecting tubule, which is possibly caused by the stimulation of the membrane sodium potassium-dependent adenosine triphosphatase that results in the hyperpolarization of the cellular membrane potential.^[12] The earliest electrocardiogram (ECG) change associated with hypokalemia is a decrease in the T-wave amplitude. As potassium levels decline further, ST-segment depression and T-wave inversions are seen, while the PR interval can be prolonged along with an increase in the amplitude of the P wave. The U wave is described as a positive deflection after the T wave, often best

seen in the mid-precordial leads (e.g., V2 and V3).^[13] So, the use of such therapies will increase the derangement of the existing abnormal electrolyte levels. Consequently, this may pose potential cardiac and respiratory hazards in the form of myocardial depression, ventricular arrhythmia^[12] and respiratory muscle fatigue, which may consequently increase the incidence of fatal asthma^[13]. It is likely that these complications may occur especially in the presence of hypoxia or acidosis, or in asthmatic patients with pre-existing cardiovascular disease.^[14] ECG Changes after hypokalaemia correction Figure 2. Therefore, the measurement of the serum electrolyte levels before and during the management of asthma with bronchodilators may reduce such risks, if they are corrected.

Conclusion

This patient presented with sudden onset paralysis and on analysis found that the treatment of asthma with β_2 -agonists has caused hypokalaemia. The inappropriate and continuous use of such drugs may also cause the hypokalaemic paralysis of the limbs and respiratory muscles. Whenever repeated doses of nebulized β_2 agonists are essential the monitoring of the electrolytes with immediate correction may be warranted in elderly asthmatic to decrease the mortality.

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Cite this article as: Rachaiah JM, Rachaiah NM. Unusual cause of hypokalemia and paralysis in an elderly patient with bronchial asthma. *Int J Med Sci Public Health* 2013; 2:1114-1116.

Source of Support: Nil

Conflict of interest: None declared