

Case Report

Baking soda induced severe metabolic alkalosis in a haemodialysis patient

Yalcin Solak, Kultigin Turkmen, Huseyin Atalay and Suleyman Turk

Department of Nephrology, Meram School of Medicine, Selcuk University, Turkey

Correspondence and offprint requests to: Yalcin Solak; E-mail: yalcinsolakmd@gmail.com

Abstract

Metabolic alkalosis is a rare occurrence in hemodialysis population compared to metabolic acidosis unless some precipitating factors such as nasogastric suction, vomiting and alkali ingestion or infusion are present. When metabolic alkalosis develops, it may cause serious clinical consequences among them are sleep apnea, resistant hypertension, dysrhythmia and seizures. Here, we present a 54-year-old female hemodialysis patient who developed a severe metabolic alkalosis due to baking soda ingestion to relieve dyspepsia. She had sleep apnea, volume overload and uncontrolled hypertension due to metabolic alkalosis. Metabolic alkalosis was corrected and the patient's clinical condition was relieved with negative-bicarbonate hemodialysis

Keywords: baking soda; haemodialysis; metabolic alkalosis

Background

Metabolic acidosis is commonly encountered in end-stage renal disease (ESRD) patients due to inability of kidneys to excrete daily acid load arising from metabolic reactions in the body. However, metabolic alkalosis is a rare condition in this patient population unless there is nasogastric suction, severe vomiting or alkali infusion [1]. We present a maintenance haemodialysis patient with severe metabolic alkalosis due to baking soda ingestion.

Case report

A 54-year-old female patient who had type 2 diabetes mellitus, hypothyroidism, congestive heart failure and ESRD was admitted to our nephrology clinic. She had complaints of increasing shortness of breath, orthopnoea, fatigue and generalized pruritus. Her complaints also included daytime sleepiness, sudden awakening at night without witnessed apnoea. She had been on haemodialysis three times weekly for 3 years. At admission, she appeared non-distressed,

blood pressure was 160/80 mmHg, heart rate was regular 78 bpm, she was afebrile and had 3/6 systolic ejection murmur on mitral region. There were diminished breath sounds at the lung bases; she had pedal oedema +/+ bilaterally. She had 5 kg above her dry weight. Thyroid gland was normally palpable. The urine output was ~100 ml/day.

Initial biochemistry revealed BUN: 117 mg/dl, Cre: 5.9 mg/dl, Na: 141 mEq/l, K: 3.3 mg/dl, Cl: 83 mEq/l, albumin: 2.9 g/dl, TSH: 29.3 mU/l and free T4: 0.54 pmol/dl. On complete blood count, haemoglobin was 9.7 g/dl, WBC 8700/mm³ and platelets 160 000/mm³. Arterial blood gas (ABG) analysis showed pH: 7.637, HCO₃: 45 mmol/l, PaCO₂: 43 mmHg, PaO₂: 53 mmHg and oxygene saturation: 93%.

Her medications included insulin glargine, L-thyroxine, quetiapine, sertraline, enalapril, furosemide and bisoprolol.

The patient had severe metabolic alkalosis. However, there were no conditions that could lead to metabolic alkalosis, i.e. vomiting, nasogastric suction or alkali administration. On further inquiry, she revealed that she had been taking baking soda occasionally for ~6 years in order to relieve dyspepsia. During the last month, she increased the amount of ingestion to 4–5 packs per day due to severe dyspeptic complaints. She underwent upper endoscopy for dyspeptic complaints a few months prior to current admission, and the procedure revealed chronic gastritis.

We attributed the presence of metabolic alkalosis to oral alkali intake, because she did not have sufficient renal function to excrete excess alkali load. She was not hospitalized previously for any reason at our institution, and routine ABG analysis was not done at her dialysis centre. Thus, we do not exactly know her acid–base status prior to this admission. We performed haemodialysis with a haemodialysis solution whose bicarbonate concentration was 8 mEq/l below standard bicarbonate value, i.e. 26 mEq/l. We also monitored ABG values. After three consecutive haemodialysis sessions with this bicarbonate value, subsequent ABG analysis revealed pH: 7.338, HCO₃: 19 mmol/l, PaCO₂: 37 mmHg and PaO₂: 74 mmHg. L-thyroxine dose was increased to attain euthyroid state. Her presenting symptoms were relieved at follow-up with the normalization of volume status, metabolic alkalosis and hypothyroidism.

Discussion

When their functions are normal, kidneys have the capability of excreting large amounts of alkali load [2]. Despite the necessity of renal impairment for developing and sustaining metabolic alkalosis, ongoing excessive amounts of alkali intake especially if not accompanied by sufficient fluid intake may lead to hypokalaemic metabolic alkalosis even in persons with normal renal function. Some authors reported severe metabolic alkalosis in patients with normal renal function due to ingestion of large amounts of baking soda or antacid drugs [3,4]. Over-the-counter medications such as calcium carbonate and sodium bicarbonate have long been used by patients to relieve dyspeptic complaints. These medications have been taken as part of pica as well. Ward and Kutner reported pica behaviour in 16% of the chronic haemodialysis population [5].

Excessive bicarbonate ingestion places patients at risk for a variety of metabolic derangements including metabolic alkalosis, hypokalaemia, hypernatraemia and even hypoxia. The clinical presentation is highly variable but can include seizures, dysrhythmias and cardiopulmonary arrest [4].

Severe metabolic alkalosis can also cause accelerated hypertension, volume overload, secondary carbon dioxide retention and sleep apnoea syndrome [6]. The latter condition is very prevalent among maintenance haemodialysis patients. One of the presumed reasons of this high prevalence is metabolic alkalosis depressing central ventilatory drive [7].

Treatment of metabolic alkalosis in an HD patient is straightforward; stopping the offending medication, making standard or, in severe cases like ours, negative bicarbonate haemodialysis (up to -8 mEq/l). Standard bicarbonate HD solutions contain 34 mEq sodium bicarbonate, and even normal bicarbonate HD can correct the metabolic alkalosis provided that further alkali ingestion be stopped. In our patient, prolonged intake of large amounts of baking soda to relieve dyspeptic complaints led to severe metabolic alkalosis. She had a native arteriovenous fistula, and no access problems were evident. Her haemodialysis was adequate when evaluated by Kt/V values. As one can expect, supraphysiologic bicarbonate concentrations in routinely used dialysis solutions can predispose patients to mild alkalosis especially when frequent dialysis is administered.

In addition, if a patient takes alkali-containing substances in considerable amounts, this can exceed the capacity of haemodialysis to eliminate the excess alkali load and the daily down titration of bicarbonate by endogenous acid. Some authors reported that even patients with normal renal function could develop metabolic alkalosis after ingestion of large amounts of alkali ingestion [3,4]. Her presenting symptoms were mainly due to volume overload, metabolic alkalosis and secondary carbon dioxide retention due to severe metabolic alkalosis. Concomitant congestive heart failure and lack of residual renal function rendered her susceptible to volume overload arising from intake of sodium bicarbonate in baking soda.

Although much less prevalent than metabolic acidosis in maintenance HD patients, metabolic alkalosis may cause significant morbidity and mortality and should be kept in mind if the patient has sleep apnoea, resistant hypertension, volume overload, muscle weakness and cramps, dysrhythmia and seizures. If metabolic alkalosis is detected in an HD patient with lack of an apparent causative factor, the patient should be questioned further in terms of over-the-counter alkali-containing medication use.

Conflict of interest statement. None declared.

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Received for publication: 14.1.09; Accepted in revised form: 8.4.09