Hydrochloric Acid Induced Hyperkalemia

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Significant disturbances of potassium homeostasis may cause considerable morbidity and mortality and the prompt recognition and appropriate treatment of these disturbances after drinking inorganic acid could be life-saving. The administration of the mineral acid HCl to experimental animals causes an elevation in the plasma potassium concentration, which has been attributed to shift of the potassium from the intracellular to the extracellular space. The authors report upon a case of hyperkalemia due to the drinking inorganic acid (0.4N HCl) in 51 years-old male, and provide a literature review of hyperpotassemia in inorganic acid-related acidosis

Key Words : Hyperkalemia, HCl drinking

Introduction

It is well known that hyperpotassemia in acidosis is caused by a K^+ shift from the intracellular to the extracellular space; the reverse of alkalosis. Animal experiments showed that the serum potassium concentration increases during acidosis and decreases in alkalosis regardless of changes in the total body potassium concentration. However, in humans, it was reported that the serum potassium concentration increases by 0.5-1.2 mEq/L when the extracellualr pH decreases by 0.1 during acidosis correction, and decreases by 0.4-1.0 mEq/L for the same pH change during alkalosis correction[1)]. In vitro experiments using muscle samples showed similar changes in extracellular potassium flux[2)]. These relationships do not apply to metabolic acidosis due to organic acid (i.e., lactic acidosis, ketone acidosis) overproduction; i.e. the acidosis caused by organic acid does not cause a significant increase in the serum potassium concentration.[3-6)] The mechanisms driving these changes are not well known, but may be related to organic anion in ketoacidosis, and involve hydroxybutyrate at the H^+-K^+ pump level[3,7)]. However, the serum potassium concentration increases significantly in metabolic acidosis caused by inorganic acid (HCl) overproduction because only 60% of the H^+ overproduced in the extracellular fluid can be buffered.[8)] Then, because of the influx of extracellular Cl^- is prohibited, the intracellular electric stability must be sustained by the efflux of intracellular K^+ and Na^+. Recent investigations also suggest that a change of H^+/K^+ ratio induces the change in the serum potassium concentration automatically to maintain the ratio of H^+/K^+ inside and outside cell similarly.[9)] However, others have claimed that the accurate relationship between serum pH and potassium concentration is not straightforward, and thus, it is not clear whether the ratio of H^+/K^+ inside and outside the cell is modulated by simple interaction.[10)] We report a case of hyperpotassemia occurring in a person who drank inorganic acid unintentionally, and provide a review of the literature.

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Case Report

Patient: 51-old-year, male

Chief complaint: oral cavity pain

Present illness: Accidental drinking of 400 mL of 10% HCl (0.4N) 3 hours before admission to the emergency room.

Past history: He received an operation for a small intestine perforation due to a traffic accident 10 years previously, and has been taking an oral hypoglycemic agent for 10 years due to diabetes.

Physical examination: His mental state was clear, but he was nervous. The oral cavity showed swollen and rotten teeth. His breathing sounds were clear, and his heart sound was free of murmurs, though the heart rate was 110 bpm and irregular beats.

Laboratory findings: On admission, the laboratory results showed Hg 16.5 g/dL, WBC 11,000/mm^3, platelet 86,000/mm^3, BUN 30 mg/dL, serum creatinine 2.4 mg/dL, Ca^{2+} 8.2 mg/dL and SGOT 84 IU/L, SGPT 56 IU/L. Serum electrolyte results showed Na^+ 139 mEq/L, K^+ 6.0 mEq/L, Cl^- 119 mEq/L and anion gap 12.3 mEq/L. Blood gas analysis showed pH 7.0, $PaCO_2$ 25.1 mmHg, HCO_3^- 7.7 mEq/L, PaO_2 110 mmHg, and SaO_2 96.1%, suggestive of severe metabolic acidosis and hyperpotassemia.

Treatment and prognosis: On admission, antibiotics (Cefuroxime 1,500 mg, Amikacin 250 mg) were administrated to prevent secondary infection due to esophageal damage, and methylprednisolone 250 mg was also injected. On the 8th day after admission when the patient showed signs of massive lower GI bleeding, visceral angiography (superior mesenteric artery of the celiac trunk) was performed to search for the bleeding focus. However, there was no positive result. On the 20th day after admission, hematemesis was not controlled. The patient received hemigastrectomy with gastrojejunostomy and gastroduodenal artery ligation. However, the patient died of shock on the 34th day.

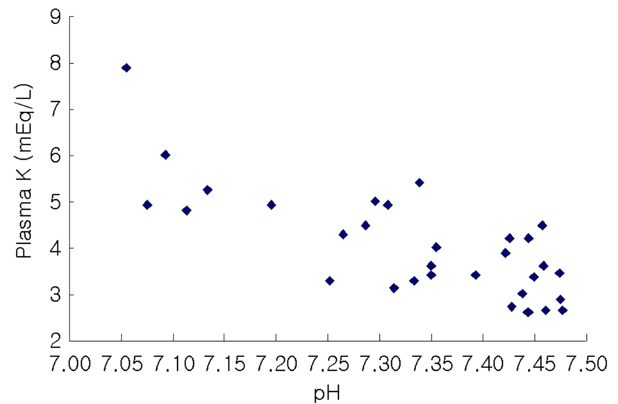

Fig. 1. Changes in plasma K^+ concentration according to blood pH during hospital stay.

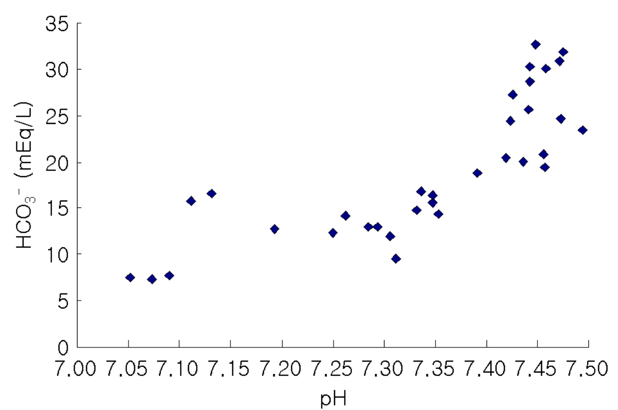

Fig. 2. The relationship between blood pH and plasma HCO_3^- concentration during hospital stay.

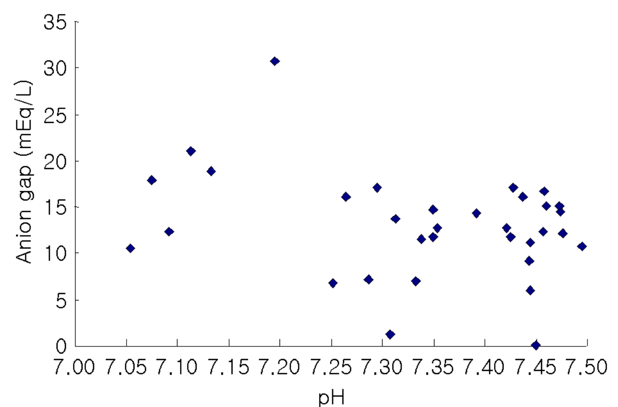

Fig. 3. Changes in anion gap according to blood pH level during hospital stay.

The changes of K^+, HCO_3^-, Cl^-, and anion gap according to serum pH: Fig. 1-4 show the changes of K^+, HCO_3^-, Cl^-, and anion gap according to serum pH during the 34 days from admission to death, and suggests the following; 1) the lower the

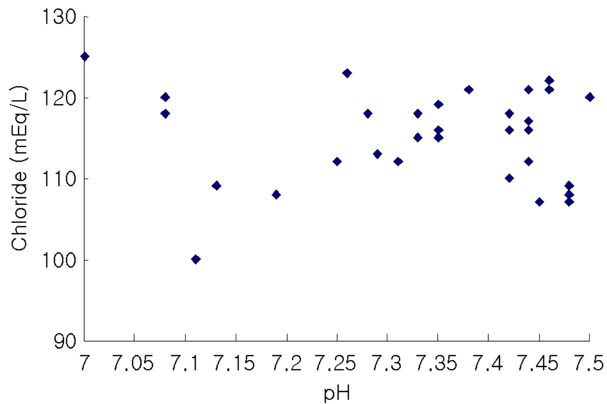

Fig. 4. Changes in plasma chloride concentration according to blood pH during hospital stay.

pH, the higher the serum potassium (Fig. 1); 2) the more severe the acidosis, the lower is the serum HCO_3^- concentration (Fig. 2); 3) the lower the serum pH, the higher the serum anion gap (Fig. 3); and 4) the lower the serum pH, the higher the serum Cl^- concentration (Fig. 4) – indicating hyperchloremic metabolic acidosis.

Discussion

When measured in the nephrectomized cat 2 hours after HCl, NH_4Cl, acetic acid, or lactic acid administration, serum potassium concentrations showed a significant increase after HCl and NH_4Cl administration, no change after acetic acid and a decrease after lactic acid administration. Moreover, there was a larger amount of K^+ efflux to the extracellular fluid after NH_4Cl administraion than after HCl administration[11]. As K^+ shift in acidosis is known to be directed towards unbuffered compartments, and the area not buffered in acidosis caused by organic acid is the extracellular fluid[12]. However, there is no net K^+ flux, because intrinsic or extrinsic organic acid can penetrate the cell wall and produce similar levels of acidosis in inside and outside the cell[13]. In experiments that investigated the effect of organic and inorganic acid on K^+ flux in rat diaphragm muscle, HCl was found to influence intracellular K^+ flux, but lactic and hydroxybutyric acid did not[2,13]. Theoretically, if lactate, acetate, and hydroxybutyrate ions could penetrate the cell wall with H^+, there would be induced change in the electric charge as occurs with HCl penetration (i.e. H^+ penetrates the cell wall in the absence of Cl^- flux)[13]. In a recent experiment, normal saline, HCl, NH_4Cl, lactic acid, hydroxybutyric acid and methylmalonic acid were administered into an anesthetized dog for 1-3 hours. The results revealed that when inorganic acid was administrated the serum potassium concentration increased significantly, and that there was a negative relationship between serum pH and the serum potassium concentration[14]. However, the serum potassium concentration showed no change in the case of organic acid administration.

It should be pointed out that there is a difference between experimental states and clinical conditions, in that a larger amount of acid is produced within the cell by lactic acidosis. If the lactate ion can penetrate the cell wall freely, then the acid cannot be viewed as a major factor in production of a potassium flux. It has been revealed in studies of normal volunteers that the serum potassium concentration tends to increase slightly above baseline after NH_4Cl and $CaCl_2$ administration, and also to increases significantly after NH_4Cl.

Significant acidosis in humans usually occurs due to organic acid accumulation, but until now, few cases showing serum potassium concentration elevation in acidosis have been reported. Unfortunately, the relationship between serum pH and the serum potassium concentration is not simple in clinical conditions as many other factors, such as potassium deficiency, renal failure, and oliguria, are involved. Cl^-, SO_4^{2-} and HCO_3^- are unable to penetrate the cell wall, but were reported to increase the potassium concentration during respiratory acidosis and uremic acidosis only [15]. Hyperchloremic acidosis-related hypopotassemia is associated with fecal potassium loss, due to for example, diarrhea or urinary potassium loss caused by

type 1 or 4 renal tubular acidosis (RTA). But in type 4, RTA hyperpotassemia is one of the characteristics and is caused by hypoaldosteronemia.

Lactic acidosis is the most common type of metabolic acidosis in clinical medicine. Though some reports found that serum potassium concentrations were maintained or increased during lactic acidosis, animal experiments showed that hyperpotassemia does not occur in lactic acidosis[13]. The possibility has been suggested that the intracellular K^+ needn't move extracelluarly because the lactate ion can penetrate the cell freely, despite controversies about lactate acid's effect on K^+ flux compared to organic acid[12]. Though, this has not been demonstrated experimentally, it is viewed as being a likely explanation. Grand mal (seizure), when considered as a attractive model of temporary lactic acidosis in the human, evoked lactic acidosis for 1 hour, but no significant change in the serum potassium concentration[5]. This was the first trial that undertook to investigate the relationship between the extracellular potassium concentration and lactic acidosis in the absence of human complications. When associated with metabolic abnormalities, lactic acidosis combined with tissue hypoxia produced a loss of organic acid and hyperpotassemia due to increased intracellular K^+ loss. In this case, hyperkalemia was developed by K^+ shift from intracellular to extracellular space due to hydrochloric acid.

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