Severe Hypercalcemia and Polyuria in a Near-Drowning Victim

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A 73-year-old man was admitted because of near-drowning in a hot springs bath. Transient severe hypercalcemia and polyuria were seen during the first hospital day. It seemed that the hypercalcemia was due to acute intoxication from calcium contained in the water of the spring absorbed mainly through the alveoli. To our knowledge, this is the first case of acute hypercalcemia complicating a near-drowning in a hot spring. Analysis of serum and urine electrolytes during the polyuric phase revealed saline diuresis, which was probably due to interference by the hypercalcemia of the reabsorption of sodium and free water.

Key words: osmotic diuresis, hot spring, calcium, free water, sodium

Introduction

Care of disturbed cardiopulmonary function and the brain is the mainstay of critical medicine of near-drowning victims (1). The character of the water in which the near-drowning occurs is now considered to be not so important, except for the differentiation between fresh and salt water (1). Electrolyte disturbances rarely need treatment (1). Life-threatening electrolyte disturbance has been reported in very unusual circumstances, such as in the Dead Sea (2, 3) and a special industrial setting (4). We treated a case of near-drowning in a hot spring presenting severe hypercalcemia and polyuria.

Case Report

A 73-year-old male near-drowning victim was admitted to our hospital. He underwent ureterolithotomy in 1983, and diabetes mellitus and hypertension were diagnosed in 1990. But he received no medication including vitamins. He had been well until December 4, 1993, when he nearly drowned in a hot spring, Wakura Spa, after eating dinner with some sake. He fainted in the bathtub, and was found lying on his face in the water. How long he was in the water was not known. When he was pulled out of the water, he did not respond at all to any stimulation, and did not appear to breathe. Mouth-to-mouth breathing was performed by a man for a few minutes. Then, he rebreathed and awoke while vomiting some fluid. Soon he was transferred to our hospital by ambulance at ten thirty p.m., when he vomited some fluid and the meal. He was conscious and oriented. His body temperature was 35.7°C, the pulse was 120, and the systolic blood pressure was 220 mmHg. The skin was cyanotic and wet. The respiratory sounds were diminished, and inspiratory crackling rales were heard on both sides. An anteroposterior radiograph of the chest, electrocardiogram, and blood tests were performed just after his arrival in the emergency room. The chest radiograph revealed bilateral diffuse coalescing densities. The electrocardiograph revealed sinus tachycardia and non-specific ST-segment depression. The QT interval corrected for heart rate was 0.40 second. Laboratory data of the blood revealed white blood count of 12,100/mm³, hematocrit (Ht) of 46.4%, sodium (Na) of 147 mEq/l, chlorine (Cl) of 110.7 mEq/l, potassium (K) of 3.69 mEq/l, creatinine of 1.1 mg/dl, blood urea nitrogen of 27.7 mg/dl, asparate aminotransferase of 23 IU/l (normal range: 5-25), alanine aminotransferase of 15 IU/l (normal range: 5-40), lactate dehydrogenase of 561 IU (normal range: 180-400), creatine kinase of 98 IU/l (normal range: 20-180), blood glucose of 267 mg/dl. Arterial blood gas analysis while breathing 2 l/min oxygen by face mask revealed pH of 7.229, PCO₂ of 42.2 mmHg, PO₂ of 42.2 mmHg, and HCO₃⁻ of 15.7 mmol/l. Indwelling venous and urinary catheters were inserted and computed tomography of the brain, which revealed diffuse cerebral atrophy but no hemorrhage or infarction, was performed.

At eleven, thirty minutes after the arrival, he was admitted to our intensive care unit, when he vomited the meal and bilious fluid. Thus, little of the hot spring water was thought to have...
been ingested into the intestine. Expectorated sputum was scanty throughout the course. A pulmonary arterial catheter was inserted and cardio-circulatory data at one a.m., two hours and a half after the arrival, revealed a cardiac index of 4.24 l/min/m², mean pulmonary capillary wedge pressure of 5 mmHg, and mean right atrial pressure of 3 mmHg. Diffuse coalescing densities on the chest radiograph at that time (Fig. 1A) were almost the same as those seen on the previous radiograph and appeared to indicate inhaled water rather than congestive heart failure. Arterial blood gas analysis while breathing 6 l/min and 70% oxygen by the Venturi mask at one and two a.m. revealed pH of 7.3 and 7.336, PCO₂ of 38 and 37.5 mmHg, PO₂ of 62.9 and 68.6, and HCO₃ of 18.8 and 20.4 mmol/l, respectively. No bicarbonate was given. Intermittent subcutaneous regular insulin therapy rapidly lowered the blood sugar to around 200 mg/dl. During the two-hour period after his arrival, nifedipine was administered sublingually three times, and after that no antihypertensive agent was needed. Hydrocortisone sodium succinate, 300 mg, was intravenously administered at three and seven hours after admission to prevent pulmonary edema.

He exhibited polyuria, voiding 1,200 ml of urine during the first hour after admission. No diuretic agent was administered at any time during the course. During the first seven hours after admission, he voided about 3,800 ml of urine (Fig. 2). On the other hand, only 1,100 ml of fluid was administered intravenously during the same period (Fig. 2) for fear of inducing congestive heart failure. He was not permitted oral intake for fear of aspiration. He complained of severe thirst. At six a.m., seven hours after the admission, blood tests and chest radiograph were again performed. Ht increased to 52.3%, and mean atrial pressure decreased to 2 mmHg. Other laboratory data revealed blood glucose of 259 mg/dl, serum osmolarity of 292 mosm/l. Arterial blood gas analysis revealed pH of 7.386, PCO₂ of 38.7 mmHg, PO₂ of 83.5 mmHg, and HCO₃ of 23.6 mmol/l on the same oxygen supply. The chest radiograph (Fig. 1B) revealed that the diffuse coalescing densities were diminishing. It seemed that pulmonary edema did not appear and the inhaled water was being absorbed. If the sum of the intravenously-administered fluid (1,100 ml) and the absorbed spring water had been equal to or more than the volume of urine (about 3,800 ml) during the seven-hour period after admission, the volume of absorbed water would have been 2,700 ml or more. Because fever was low grade and oxygen was fully humidified, insensi-
ble water loss was thought to have been very small. The clinical course appeared too mild for us to regard that such a critical volume of water was inhaled into the lungs (4). Rather, it seemed that an excessive output of urine was voided compared to water input and caused the patient to be dehydrated. Because pulmonary edema did not occur, it did not seem that water redistribution making the near-drowning victim dehydrated (1) occurred.

Then we examined the cause of polyuria. Laboratory data at seven hours after admission revealed total serum calcium (Ca) of 18.1 mg/dl and serum inorganic phosphorus of 4.4 mg/dl. Retrospective examination of serum ionized Ca autoanalyzed concomitantly with blood gas analysis revealed that hypercalcemia had been already present on admission (Fig. 3). Analysis of the 2,600 ml of urine from the second to seventh hour revealed osmolarity of 319 mosmol/l, glucose of 24 mosmol/l, Na of 119 meq/l, urea nitrogen of 42.5 mosmol/l, Cl of 118 meq/l, and Ca of 4.0 mosmol/l. Osmotic diuresis probably associated with hypercalcemia rather than glycosuria was considered to be the cause of the polyuria. Because mild resolution of hypercalcemia occurred before glucocorticoid therapy in spite of progressive dehydration, it did not seem that the dehydrated state induced by drinking and bathing solely caused the hypercalcemia. No additional glucocorticoid as administered. Diuretic agents, theophylline, and calcitonin were not administered. Hypercalcemia was not explained solely by dehydration induced by drinking and bathing, as previously stated. No agents, such as diuretics or theophylline, which contribute to hypercalcemia were administered after admission. It seemed that the hypercalcemia was due to absorption of Ca from the inhaled water. Daily content of urinary excretion of Ca was about 691.2 mg on the first hospital day, 124 mg on the second, and 74 mg on the tenth. These data suggested that a considerable amount of Ca was loaded before admission and excreted after admission.

**Discussion**

The patient's hypercalcemia was of an acute nature, rapidly resolved, and did not recur. There was no history or clinical evidence of neoplastic disease, milk-alkali syndrome, intoxication of vitamin D, or hyperparathyroidism. The course of the hypercalcemia was not explained solely by dehydration induced by drinking and bathing, as previously stated. No agents, such as diuretics or theophylline, which contribute to hypercalcemia were administered after admission. It seemed that the hypercalcemia was due to absorption of Ca highly contained in the inhaled water through the alveoli. The high urinary excretion of Ca found just after admission was consistent with this speculation. It seemed that intestinal absorption of Ca played only a little part, because the patient recurrently vomited the gastric content including the meals taken before he nearly drowned. Moreover, the maximum point of hypercalcemia by intestinal absorption is reported to appear later than in the present case according to the literature (3, 5).

We re-examined water-electrolyte balance after admission (Fig. 2). During the first seven hours, 1,100 ml of fluid, containing Na of 102 meq and Ca of 27 mg, was administered intravenously. Urine volume was 1,200 ml with unknown contents during the first hour plus 2,600 ml containing Na of 309 meq and Ca of 421.2 mg during the next six hours. Then the amount of negative Na-balance was more than Na of 207 meq, which was equal to Na contained in 3.7 l of the spring water. It did not seem that such a critical volume had been inhaled by the lungs, as previously stated. On the other hand, the amount of negative Ca-balance was Ca of 394 mg, which was equal to Ca
Near-Drowning and Hypercalcemia

-contained in 3.7 dl of the spring water. Total content of urinary Ca-excretion from the second to twenty-fifth hour after admission was about 691.2 mg, which was equal to Ca contained in 6.4 dl of the spring water. Because we do not know at all the rate of absorption of Ca through alveoli, we could not know the exact amount of inhaled water. But it may be said that the amount of the inhaled water was considerably smaller than the critical volume. In fact, during the twenty-five hours after admission, the total volume of urine was about 6,050 ml, and the total volume of fluid administered intravenously was 4,800 ml. Oral intake was nil. Insensible water loss was small, as previously stated. The negative balance, 1,250 ml, may be close to the amount of absorbed fluid. The negative Na balance probably represented excessive saline diuresis.

Polyuria in the present case was analyzed in Fig. 4. It demonstrates the relationship between serum Ca, urinary volume, and urinary excretion of Na. Just following admission, the following were present: poluria, marked Na excretion, a high level of osmolal clearance (C osm), and a low level of reabsorption of free-water (T^c H_2O) in spite of the dehydrated state of the patient. As the level of serum Ca normalized, urinary volume and Na excretion decreased and T^c H_2O increased. We could find only one clinical report (5) of hypercalcemia-induced polyuria in a near-drowning victim. The data in that report also seemed to indicate saline diuresis. In some reports (6, 7) of experimental acute hypercalcemia, hypercalcemia was said to interfere with reabsorption of Na and free-water. Such a phenomenon probably occurred in the present case. We could not find any clinical report which describes how such acute and severe hypercalcemia as that of the present case influenced renal function.

To our knowledge, hypercalcemia complicating near-drowning has been reported only in patients who nearly drowned in the Dead Sea (2, 3) and in a special industrial setting (5). Serum Ca is not always included in routine laboratory studies in emergency rooms. But this case suggests that we should sometimes consider hypercalcemia in near-drowning victims in hot springs, which are numerous in Japan. Hypercalcemia is clinically important because it may cause lethal arrhythmia or mental disorder (2, 3, 8), both of which are usually considered to be due to hypoxia, heart failure, disturbance of K, or cerebral ischemia (1). In the present case, hypertension, nausea, and vomiting seemed to be related to hypercalcemia. But no other clinical problems including electrocardiographic abnormality were seen. In near-drowning victims in the Dead Sea, concomitant hypermagnesemia was said to neutralize the toxic effect of hypercalcemia (2). Unfortunately, serum magnesium was not checked until the third hospital day in the present case; it was found to be normal. It did not seem that hypermagnesemia occurred in the present case, because the content of magnesium was low in the hot water. In one case report (9), in which hypercalcemia as high as 22 mg/dl accidentally occurred by intravenous hyperalimentation, no clinical disorder including electrocardiographic disturbance was encountered. Undoubtedly, many other clinical conditions influence the toxic effect of acute hypercalcemia. When it is not known what type of fluid is aspirated or when polyuria of unknown etiology is encountered in a near-drowning victim, the serum Ca should be analyzed.

References