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Rapid Self-infusion of Tap Water

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Intravenous self-infusion of tap water has never been reported in the literature. We present a 24-year-old healthy man who self-administered 2.5 L of tap water over 2 hours and developed acute illness including fever, change of mental status, acute hemolysis, low-grade disseminated intravascular coagulation, and acute kidney injury.

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Case Report

INTRODUCTION

Rapid intravenous hypo-osmolar fluid infusion is almost exclusively an iatrogenic event. Instances of inadvertent hypotonic fluid infusion resulting in osmolar injury and hemolysis has been reported in patients receiving copious volumes of distilled water bladder irrigation,[1] continuous veno-venous hemodialysis using distilled water as replacement fluid,[2] and intravenous human albumin diluted with sterile water.[3] An analogous condition such as fresh water drowning has only been described to any significant degree in animal models.[4] In humans, there was a single study of 10 individuals who received slow intravenous water infusion (20 mL/h) reported 4 decades ago. It was noted that 7 of the participants developed hemoglobinuria and an acute decrease in inulin clearance. All of the study subjects had reversal of the effects within 20 minutes of stopping the infusion.[5] Rapid tap water infusion (>1 L/h) in humans has never been previously described. We present a 24-year-old healthy man who self-administered 2.5 L of tap water over 2 hours.

CASE REPORT

A 24-year-old man, previously healthy, presented to the emergency department with complaints of shaking, chills, paresthesia and "feeling loopy" for 2 to 3 hours. He developed nausea, vomiting, and diarrhea approximately 8 hours after dinner at home the previous night. When he was not able to tolerate oral fluids he resorted to self-administration of intravenous fluids. He had been in the military where he learned the technique of intravenous fluid administration. He had an old intravenous infusion kit of 500 mL 0.9% saline at home. He proceeded with self-infusion of the saline followed by the refilling of the empty intravenous bag 5 times and self-infusing a total of about 2.5 L of tap water over a 2-hour period. Immediately following the infusion, he began to feel dizzy with a sensation of losing balance ("felt like I was drunk"). Shortly after, he developed double vision, palpitations, numbness of the fingertips and toes, and "lost track of events that followed." He urinated several times during the course of these events and his spouse noted a distinct reddish discoloration of his urine. He felt a sense of "impending doom."

On arrival to the local emergency department, approximately 10 hours after the tap water infusion, his heart rate was 73 per minute (regular), blood pressure was 140/90 mm Hg, respiratory rate 16 per minute, and temperature was 38.6°C. He had mild generalized abdominal tenderness and the remaining examination was unremarkable. His laboratory studies are summarized in the Table (all laboratory values were measured and not calculated).

He was admitted to the medical intensive care unit for close monitoring. He received intravenous isotonic fluids (approximately 12 L of 0.9% saline and 3 L of 0.45% saline with 75 mEq/L of sodium bicarbonate). His electrolyte abnormalities were gradually corrected over the next 24 hours.

The Patient's Laboratory Studies

Laboratory Studies	On Admission	Before Discharge
Blood		
Hemoglobin, g/dL	12.8	11.7
Total leukocyte count, × 10^9/L	7.1 (3.5 to 10.5)	…
Platelet count, × 10^9/L	163 (150 to 450)	…
Reticulocyte, %	1.3 (0.60 to 1.83)	…
Haptoglobin, mg/dL	41 (30 to 200)	…
Lactate dehydrogenase, U/L	668 (122 to 222)	558
D-dimer, DDU	1915 (0 to 250)	…
Fibrinogen, mg/dL	268 (175 to 430)	…
International normalized ratio	1.3 (0.8 to 1.2)	…
Activated partial thromboplastin time, sec	28 (28 to 38)	…
Sodium, mmol/L	135 (135 to 145)	139
Potassium, mmol/L	3.8 (3.6 to 5.2)	4.0
Chloride, mmol/L	101 (100 to 108)	106
Bicarbonate, mmol/L	27 (22 to 29)	24
Blood urea nitrogen, mg/dL	21 (8 to 24)	8
Creatinine, mg/dL	1.5 (0.8 to 1.3)	1.1
Ionized Calcium, mg/dL	4.59 (4.65 to 5.30)	…
Phosphorus, mg/dL	1 (2.5 to 4.5)	3.6
Magnesium, mg/dL	1.3 (1.7 to 2.3)	1.9
Total bilirubin, mg/dL	4.2 (0.1 to 1.0)	0.5
Direct bilirubin, mg/dL	1.2 (0.0 to 0.3)	…
Aspartate aminotransferase, U/L	67 (8 to 48)	53
Alanine aminotransferase, U/L	42 (7 to 55)	…
Alkaline phosphatase, U/L	49 (45 to 115)	…
Serum osmolality, mOsm/kg		288 (275 to 295)
Urine		
Osmolality, mOsm/kg	123 (150 to 1150)	485
pH	5.7 (5.0 to 8.0)	6.3
Hemoglobin	Large (negative)	Small
Myoglobin	Negative	…
Microscopy	Normal	Normal
Spot Sodium, mmol/L	27	…
Fractional excretion of sodium, %	< 1	…

He received sodium phosphate, 30 mmol, and magnesium sulphate, 2 gm, intravenously and the levels stabilized on the second day of hospital stay. His urine output was brisk in the first 3 hospital days (at least 3.8 L [partial collection], 4.0 L, and 5.2 L, respectively) and subsequently normalized to 1 L/d to 1.5 L/d. His kidney function improved with a serum creatinine concentration of 1.2 mg/dL on hospital day 3, and his serum osmolality (288 mOsm/kg) and circulating D-dimer concentration also normalized. He was discharged on hospital day 4 free of symptoms.

DISCUSSION

We present a rare case of 2.5 L tap water infusion over 2 hours in a previously healthy person with a possible viral illness-like presentation. Rapid intravenous water infusion acutely dilutes extracellular osmolalities and tonicity, triggering an acute shutdown of arginine vasopressin secretion which, in the presence of normal kidney function, is expected to maximally dilute urine and cause aquaresis, resulting in a clinical presentation equivalent to transient central diabetes insipidus. Our patient presented with a brisk increase in urine output associated with a reduced urine osmolality of 123 mOsm/kg, although the degree of dilution was limited possibly due to the concurrent acute kidney injury.

Acute reduction in extracellular fluid tonicity is known to cause hemolysis. Hemolyzed erythrocytes release heme and heme-containing degradative products that are nephrotoxic through mechanisms of (1) vasoconstriction resulting in acute reduction

in renal blood flow, (2) direct renal tubular cell toxicity associated with activation of reactive oxygen species, and (3) tubular obstruction due to binding of heme/heme degradative products with luminal Tamm-Horsfall proteins.[6]

In addition to acute reduction in the extracellular osmolality, causing aquaresis and intravascular hemolysis, tap water infusion can also be associated with adverse effects from endotoxin and chlorine or chloramine exposure. Tap water has been shown to contain 1 ng/mL to 3 ng/mL of endotoxin.[7] In one study, a 1.9°C increase in body temperature was produced with infusion of approximately 3 ng of *Salmonella typhosa* endotoxin per kilogram of body weight.[8] Febrile illness was also observed in distilled water infusion at a much slow rate.[5] Our patient did present with fever, which could have been linked to systemic reaction to endotoxin, although febrile illness could also be a nonspecific reaction to hemolysis. Chlorine and chloramine are routinely used as disinfectants in the treatment of municipal water supply.[9] Hemolysis (possibly related to oxidative damage to the erythrocytes) has been described in dialysis patients with acute chloramine exposure in settings where the dialysis units use town water as dialysis water supply.[10,11] Given the rapid infusion and large volume tap water exposure, our patient may have experienced a similar direct chorine or chloramine exposure, contributing to the acute hemolysis. Although our patient did not show elevated peripheral reticulocytes, it is not entirely surprising. In a case series reported by Liesveld and coworkers involving 109 patients with acute autoimmune hemolysis, 20% of the cases exhibited an initial reticulocyte count less than 4%, and 37% had an initial reticulocyte production index less than 2 times of the basal state. In most cases, the low initial reticulocyte counts represent a lag in marrow responsiveness to hemolytic stress.[12]

The initial laboratory studies from our patient also revealed a mild degree of hypomagnesemia and hypophosphatemia, which were clinically asymptomatic. Such laboratory abnormalities were not previously described as a part of water-infusion related manifestations. We speculate that these abnormalities were associated with the large urine output leading to urinary loss of magnesium and phosphorous. This assumption is supported by the observation that the abnormalities were readily corrected by a small amount of supplementation, inconsistent with chronic or intracellular deficiency of these elements or loss due to renal tubular damage.

Another laboratory abnormality was the mildly elevated circulating D-dimer. The etiology for its occurrence is not clear. We suspect the acute change in circulating osmolality may have caused acute activation in the coagulation system–analogous to a low-grade or incipient disseminated intravascular coagulation, possibly caused by exposure to endotoxin or chloramine in this case. While toxic effects of acute chloramine exposure on coagulation pathway have not been described specifically, the response to endotoxin (bacterial origin) and the pathways leading to disseminated intravascular coagulation have been described previously.[13]

In summary, we describe a rare case of rapid intravenous tap water infusion causing acute systemic illness in a previously healthy individual. It is valuable for clinicians to be familiar with the manifestations associated with such occurrence. Although fulminant and multisystemic, the acute illness was completely resolved with supportive care.

CONFLICT OF INTEREST

None declared.

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