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Fatal Water Intoxication in a Case of Psychogenic Polydipsia

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ABSTRACT: The term "water intoxication" is used to describe a condition of agitation, delirium, convulsion, and coma brought on by excessive intake of water, resulting in severe hyponatremia. Psychogenic polydipsia (compulsive water drinking) has until recently been considered a relatively benign process. Since 1974, however, three fatal cases of water intoxication, resulting from psychogenic polydipsia, have been reported. All three individuals died while hospitalized, thereby permitting performance of blood electrolyte determinations and documentation of the associated electrolyte imbalance. In the authors' case, there was a well-documented prior episode of water intoxication in which serum electrolytes showed a pattern typical of this entity. Death, however, occurred at home, thus preventing valid serum electrolyte determinations to be performed. Analysis of the vitreous humor revealed a severe hyponatremia, thus substantiating the diagnosis of fatal water intoxication. This case, once again, points out the usefulness of electrolyte analyses on the vitreous humor as an aid to establishing a cause of death.

KEY WORDS: pathology and biology, psychogenic polydipsia, death

Until fairly recently, psychogenic polydipsia (compulsive water drinking) was considered a fairly benign process. Since 1974, however, three fatal cases of water intoxication, resulting from psychogenic polydipsia, have been reported [*1,2*]. All three individuals died while hospitalized, thereby permitting performance of blood electrolyte determinations and documentation of the associated electrolyte imbalance. We are reporting a fourth case, differing from the previous reports in that the individual was found dead at home, thus preventing performance of valid blood electrolyte determinations. Electrolyte examinations of the vitreous humor, however, revealed a severe hyponatremia, confirmatory evidence of the diagnosis of water intoxication.

Case Report

At 6:30 p.m. on 21 July, a 54-year-old white female was found dead at home, face up in bed. She had been seen alive 2 h before then. The husband stated that she had been released from Parkland Memorial Hospital the day before. He did not know why she had

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been hospitalized. The body was transported to the Dallas County Medical Examiner's Office for determination of the cause and manner of death.

Prior to the autopsy, the deceased's medical records had been obtained from Parkland Memorial Hospital and reviewed. These indicated that she had been admitted on 14 July, after "passing out" and "possibly seizing." She was a mental patient with a long history of psychosis, for which she had been hospitalized on a number of occasions. According to the husband, she had run out of her usual medications (haloperidol and trihexyphenidyl) several days before admission. She became increasingly nervous and began to drink large quantities of water. On the morning of admission, she became more agitated and confused, and defecated in bed. On the way to the bathroom, she passed out, collapsing on the floor. Her face "turned black" and then she became very pale. The husband called the Dallas Fire Department Mobile Intensive Care Unit, which transported her to Parkland Memorial Hospital.

On admission, she was a semicomatose woman, thrashing about and in obvious respiratory distress. Blood pressure was 90/60 mm Hg without tilt; heart rate, 90/min and regular; respiratory rate, 32/min; and temperature, 37.6°C (99.6°F) rectally. Examination of the head and neck was unremarkable. On examination of the chest, there was decreased resonance to percussion bilaterally with diffuse rales throughout both lung fields, but with no increased breath sounds. Cardiac examination revealed decreased heart sounds without murmurs, rubs, or gallop rhythm. The PMI (point of maximum impulse of the heart) was not palpable. The abdomen was soft without masses or organomegaly, and bowel sounds were normal. Neurological examination revealed negative Doll's eyes. Caloric stimulation tests were normal. Cranial nerves III through XII appeared intact. The deep tendon reflexes were 2+/4+ bilaterally. Motor and sensory innervation appeared intact. There were no Babinski reflexes or Hoffmann's signs present. The remainder of the physical examination was unremarkable.

An initial arterial blood gas test revealed a pH of 7.19 with an arterial oxygen tension PO_2 of 55 mm Hg and an arterial carbon dioxide tension PCO_2 of 24 mm Hg. These improved with oxygen (40% by Ventimask®) to a pH of 7.9, PO_2 of 79 mm Hg, and PCO_2 of 38 mm Hg. Blood electrolyte tests at admission demonstrated a serum sodium level of less than 110 meq/litre and a potassium of 3.6 meq/litre. Additional blood studies indicated a total protein of 6.0 g/dl; albumin, 4.3 g/dl; calcium, 8.2 mg/dl; alkaline phosphatase, 92 mU/ml; and serum glutamic oxalacetic transaminase, 78 mU/ml. Serum lactate on admission was 13.3 mg/dl. A chest X-ray showed patchy interstitial infiltrates and a normal-sized heart. An electrocardiogram gave normal results. Although the initial impression had been a possible overdose, a drug screen of the blood for alcohol and acidic, basic, and neutral drugs revealed only a blood salicylate concentration of 5 mg/dl. The admitting diagnoses were (1) pathologic water-drinking with severe hyponatremia, (2) metabolic acidosis, (3) hypotension, and (4) possible adult respiratory distress syndrome.

Following administration of fluids and hypertonic saline, the sodium level was 127 meq/litre; potassium, 3.6 meq/litre; and CO_2, 27 meq/litre. Eighteen hours after admission, a repeat test showed the serum sodium level was 135 meq/litre with potassium, 3.8 meq/litre; chloride, 104 meq/litre; and CO_2 content, 23 meq/litre. The blood pressure rose to 120/80 mm Hg. By 24 h after admission, she could be roused but was clearly disoriented. Free water intake was restricted. Four days later, blood electrolyte levels and arterial blood gas values of oxygen were all within normal ranges and her chest X-ray had cleared. She was discharged on 20 July with a diagnosis of psychogenic water intoxication.

Before the autopsy the medical examiner had all the aforementioned clinical information concerning the deceased's prior admission and course in the hospital. A complete autopsy was performed and there were no significant findings, either grossly or microscopically. Complete toxicologic analyses for alcohol and acidic, basic, and neutral drugs were performed. The only drug detected was chlorpromazine at a blood concentration of 0.01 mg/dl.

At the time of autopsy, vitreous humor was removed for electrolyte studies. Analysis revealed a sodium level of 115 meq/litre; chloride, 105 meq/litre; and potassium, 7.6 meq/litre. The vitreous glucose level was less than 25 mg/dl and the creatinine level was 0.4 mg/dl.

Discussion

The term "water intoxication" is used to describe a condition of agitation, delirium, convulsions, and coma brought on by excessive intake of water, resulting in severe hyponatremia. Severe water intoxication can and does occur in individuals with psychogenic polydipsia who do not have any predisposing illness.

Until recently, patients with psychogenic polydipsia had not been considered in serious danger, as this entity had been presumed to be readily reversible. In 1974, however, Raskind [*1*] reported the first fatality. The patient, a 56-year-old woman, began to drink excessive amounts of water while hospitalized for an attempted suicidal drug overdose. On admission to the hospital, her blood electrolyte levels were within normal limits. Following development of symptoms of water intoxication, her serum electrolyte levels were sodium, 111 meq/litre; potassium, 2.6 meq/litre; and chloride, 73 meq/litre. The urine electrolyte tests showed a sodium level of 44 meq/litre; potassium, 14 meq/litre; and chloride, 39 meq/litre. The urine specific gravity was 1.007. Toxicological screen of the urine and blood was negative. The patient died the following day. An autopsy revealed only cerebral edema. One complicating factor in this case was the fact that the patient had received thioridazine and hydroflumethiazide, two drugs with recognized antidiuretic hormone-releasing properties.

In the two fatal cases of water intoxication reported by Rendell et al [*2*], neither patient had received drugs with potential antidiuretic hormone effects. Their first case was a comatose, 29-year-old female, who eight days before her death had been discharged from the hospital following admission for water intoxication. Blood electrolyte levels at the time of her first hospitalization were sodium, 127 meq/litre; potassium, 3.4 meq/litre; and chloride, 84 meq/litre. Laboratory values on the second admission were sodium, 102 meq/litre; potassium, 2.6 meq/litre; and chloride, 68 meq/litre. She never regained consciousness and died 24 h after admission. The autopsy revealed only pulmonary edema.

The second case was that of a 52-year-old female. When admitted, she was somnolent but could be roused. Blood electrolyte levels on admission were sodium, 94 meq/litre; potassium, 5.3 meq/litre; and chloride, 58 meq/litre. The patient became progressively more lethargic and by the third day was comatose. She died three weeks after admission. No postmortem examination was performed.

In the authors' case, there was a well-documented prior episode of water intoxication, in which serum electrolyte levels showed the typical pattern of that entity. The death of this patient differed from the previously reported cases in that valid serum electrolyte determinations could not be performed, as death had occurred at home. Serum electrolyte levels determined on postmortem blood do not accurately reflect antemortem levels. However, determinations of sodium and chloride levels in the vitreous humor do accurately reflect the antemortem electrolyte status, if decomposition has not occurred. In our case, the previous medical history in conjunction with a lack of autopsy and toxicologic findings strongly suggested fatal water intoxication. It was, however, necessary to have some objective evidence to document this impression. The finding of hyponatremia in the vitreous humor substantiated the impression of water intoxication.

This case, once again, points out the usefulness of electrolyte analyses on the vitreous humor as an aid to establishing a cause of death. In all cases in which the cause of death is obscure, such a determination should be performed.

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