Alerts, Notices, and Case Reports

Severe Hypernatremia From Sea Water Ingestion During Near-Drowning in a Hurricane

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INGESTION OF SEAWATER, whether voluntary[1] or accidental,[2,3] and its clinical sequelae have not been amply reported in the literature. Although seawater ingestion has been directly observed in animal drowning experiments[4] and is believed to occur during human drowning,[2,3,5] it has not been reported to play a significant role in producing serum electrolyte abnormalities in near-drowning victims. Because of the high concentration of sodium in seawater (approximately 350 to 500 mmol per liter[10,11]), ingestion of this fluid and subsequent absorption of electrolytes can lead to hypernatremia. This condition is caused not only by the addition to the body of proportionately more sodium than water, but by water loss from solute diuresis or osmotic diarrhea.

The near-drowning patient whose case is reported in this paper experienced unusual circumstances as the result of a shipwreck during the height of a hurricane; the patient's immersion and involuntary seawater ingestion led to the development of severe hypernatremia. This case is interesting not only because of the patient's harrowing ordeal, but also because it illustrates the physiologic changes and clinical symptoms that can occur in a victim of near-drowning when significant quantities of seawater are ingested rather than aspirated. To my knowledge, the patient's serum sodium levels are the highest reported in the literature on near-drowning.

Report of a case

A 35-year-old fisherman was rescued by the United States Coast Guard on September 16, 1995, and flown to the emergency department at the US naval hospital in Puerto Rico. He had been found clinging to floating debris on the open sea following the passage of

Hurricane Marilyn through the US Virgin Islands.[6] The patient had remained on board a fishing vessel that was moored in an unprotected harbor during the storm. On his recovery we learned that he had been forced to abandon his vessel at night after it broke from its anchorage, struck a reef, and was swamped by waves. He remained in the water (surface temperature 28°C [82.4°F]) for the next 11 hours (from about 9 PM on September 15 to about 8 AM September 16), during which time he was repeatedly submerged by breaking ocean swells (estimated to be more than 20 feet high). He also experienced sustained winds of 109 miles per hour, wind gusts up to 129 miles per hour, and the eye of the passing hurricane (written communication, Lieutenant Commander J.R. Jarvis USN, Officer in Charge, US Naval Atlantic Meteorology and Oceanography Detachment, Puerto Rico, December 1997). While submerged he involuntarily inhaled, "choked" on, and swallowed mouthfuls of water after reaching his breath-holding breaking point. Despite turbulent conditions, his life jacket kept him afloat during most of this episode.

The patient's medical, surgical, and family history were unremarkable, and he had not been taking medications. Immediately before his ordeal he was in good health, had not been deprived of water, and had no symptoms of thirst, polyuria, lassitude, or gastroenteritis. When the patient was specifically questioned after recovery, he recalled episodes of micturition while in the water.

On examination, the patient's rectal temperature 35.7°C (96.3°F); pulse rate while lying flat 120 beats per minute; blood pressure 90/50 mm of mercury; respiratory rate 30 breaths per minute; and weight 61 kg (136 lb). He was a thin man in moderate distress who spoke incoherently and was disoriented as to time and place. Examination of his head showed no evidence of trauma. His neck veins were flat, and his mucous membranes were dry. His gag reflex was normal. Fine crackles were auscultated at both lung bases. Cardiac examination revealed tachycardia without gallop or murmur. His neurologic examination revealed no focal deficits. Except for a few superficial abrasions on his right knee, the remainder of his physical exam was normal.

Values for serum chemistries and arterial blood gas samples that were collected before therapy are listed in Table 1 (Day 1). The patient's complete blood count revealed a hematocrit level of 40% and a leukocyte count of 24.4 (24,400 mm³). His urinalysis revealed a pH of 5 and a specific gravity of 1.025. Urine electrolyte studies could not be performed. A serum screening for illicit drugs was negative. Thyroid function studies were normal. The results of the patient's blood cultures were normal. A chest radiograph revealed mild hypoinflation but was otherwise normal. An electrocardiogram showed sinus tachycardia with a heart rate of 120 beats per minute and left ventricular hypertrophy with repolarization changes.

TABLE 1.—Serum Electrolyte and Arterial Blood Analysis*

	Day 1						Day 2			Day 3
Local Time*	0918	0952	1330	1600	1800	2100	0520	0900	1800	1000
Time Hospitalized*	00:00	00:34	03:38	06:42	08:42	11:42	20:02	23:42	32:42	44:42
Weight (kg)	61						62			
Laboratory test*										
Sodium (mmol/L)	175	174	164	159	154	147	142	142	143	142
Potassium (mmol/L)	5.1	5.0	4.3	3.9	4.1	3.9	3.4	3.4	4.6	4.3
Chloride (mmol/L)	148	147	138	132	128	119	115	115	114	112
HCO_3^- (mmol/L)	15	15	12	12	12	14	19	19	21	24
Creatinine (mg/dl)	1.1	1.0	1.0	1.1	1.1	1.2	1.1	1.1	1.4	1.2
Urea nitrogen (mg/dl)	15	15	14	13	13	12	8	8	16	12
Glucose (mg/dl)	112									
Calcium (mg/dl)	8.3	8.8								
Phosphorus (mg/dl)	1.9	2.7								
Magnesium (mg/dl)	3.3	2.0								
Arterial Blood Gas										
pH	7.19**		7.2**				7.3**		7.4**	
Partial pressure of CO_2 (mmHg)	45		32				29		34	
Partial pressure of O_2 (mmHg)	125		269				78		90	

*To convert the values for creatinine to micromoles per liter, multiply by 88.4. To convert the values for urea nitrogen to millimoles per liter, multiply by 0.357. To convert values for glucose to millimoles per liter, multiply by 0.05551. To convert values for calcium to millimoles per liter, multiply by 0.25. To convert values for phosphorus to millimoles per liter, multiply by 0.3229. To convert values for magnesium to millimoles per liter, multiply by 0.4114.
*Atlantic Standard Time.
*Time in hours and minutes from presentation to the emergency medicine department.
*Laboratory test data performed obtained using a Dimension XX AR clinical chemical analyzer. PN 715914.001; E. I. Du Pont de Nemours & Co., Inc., Wilmington, DE.
**Breathing 6L/min humidified oxygen by face mask.
**Breathing room air.

Initial therapy included high-flow supplemental oxygen administered through a face mask, application of warming blankets, and intravenous injection of a bolus of approximately 500 ml of isotonic saline. After the results of the patient's serum electrolyte levels became available, his intravenous fluids were changed to 5% dextrose in water. Because of concern that overly rapid correction of the patient's hypernatremia might lead to cerebral edema,[7] it was decided to correct the water deficit slowly over the next two days, while measuring serum electrolyte levels at frequent intervals and keeping the patient under close observation.

A water solution containing 5% dextrose was slowly infused. The patient's urine output during the first four hours of his hospitalization was 750 ml. Within 8 hours after presentation his sensorium cleared, he began to complain of severe thirst, and he experienced several episodes of diarrhea. Our attempts to limit his intake of water were unsuccessful. During brief periods when the patient was not under direct observation by the nursing staff, he was found voraciously drinking unknown quantities of water from a nearby sink. Total input volume of water for hospital day 1 could not be accurately measured. Total measured urine output was 2210 ml for hospital day 1. On the morning of hospital day 2 he was

breathing normally, had normal mental activity, and was without complaint. His weight was 63 kg (140 lb), serum sodium concentration 142 mmol per liter (a decrease of 33 mmol per liter in 24 hours), and his leukocyte count had returned to normal. A repeated chest radiograph was unremarkable. His diet was increased and restrictions on oral water intake were relaxed. By the evening of hospital day 2, the only abnormal laboratory test parameter that persisted was mild hyperchloremia. On hospital day 3 he was discharged from the hospital, at which point he was able to walk and tolerate a full diet.

Two days following his discharge from the hospital, the patient traveled to mainland United States and remained there. Although further direct contact with the patient has not been made, acquaintances who have been in contact with him have stated that he has reported feeling well and has returned to work.

Discussion

Ingestion of hypertonic sodium solutions is a classic, uncommon cause of acute hypernatremia.[8] Even more unusual is the occurrence of severe hypernatremia in cases of water near-drowning at sea.[9] Despite the high

sodium concentration in ocean waters (approximately 350 to 500 mmol per liter[10,11]), the electrolyte values reported in retrospective series of patients suffering from drowning and near-drowning in this fluid have usually been in the normal range.[5,10–13] This is probably because submersion, and the subsequent chain of events—including struggle, aspiration, asphyxia, and hypoxemia (with closed or open glottis[3])—occur immediately and are interrupted in a matter of minutes by either rescue or death. Typically, there is neither time nor opportunity for electrolytes to accumulate in the extracellular fluid compartment (ECFC). The case of near-drowning reported here is unusual in that recurrent submersions resulted in ingestion and accumulation of the solute over a period of many hours.

When seawater is aspirated it causes impairment of alveolar gas exchange, which results in hypoxemia and chest abnormalities secondary to pulmonary edema and atelectasis.[5,7–14] The marked hypertonicity of the aspirate results in rapid fluid shifts: protein rich fluid from plasma moves into the lungs, and some solutes of seawater move into the plasma.[14] Although inhalation of salt spray whipped up by high winds was inevitable in this patient given the circumstances, the lack of radiograph findings coupled with his relatively mild degree of hypoxemia indicate that little seawater was actually aspirated—certainly not enough to have a significant effect on his volume status or serum electrolyte concentration.[10]

When seawater is swallowed, relatively poorly absorbed, osmotically active solutes such as magnesium, phosphates,[10] and particulate matter,[5] as well sodium and chloride, are added to the luminal content of the gastrointestinal tract. As a result, water translocates into the bowel, causing diarrhea, while sodium and chloride ions move along their concentration gradients from the lumen of the bowel to the ECFC. Seawater ingestion probably caused the diarrhea that occurred in this patient and also could have contributed to his hypovolemia.

Another factor contributing to the patient's hypernatremia and hypovolemia was polyuria. The high urine output observed during the initial hours of his hospitalization revealed that diuresis was occurring despite the apparent presence of hypovolemia. The polyuria was likely secondary to the combined physiologic effects of head-out water immersion,[15] mild hypothermia,[16] and solute diuresis induced by the ingestion of hypertonic seawater.[17,18] Because the sum of the concentrations of sodium plus potassium in seawater ($SW_{[Na]} + SW_{[K]}$) is higher than the sum of the concentrations of these electrolytes in human urine ($U_{[Na]} + U_{[K]}$), ingestion of seawater results in the addition to the body of more sodium plus potassium than water, or a positive tonicity balance.[18] In this setting, production of urine that is relatively more diluted than ingested fluid causes simultaneous accumulation of solute and loss of free water to and from the ECFC. Moreover, if urine volume plus stool water volume are greater than the volume of seawater ingested, the total amount of water in the body will decrease. This process results in a self-perpetuating cycle of sodium

gain and water loss that continues until interrupted by the cessation of seawater ingestion and/or the administration of appropriate volumes of free water.[18]

The patient's severe electrolyte imbalance and clinical presentation resulted primarily from seawater ingestion and consequent water loss from the gastrointestinal tract and kidneys, rather than from the aspiration syndrome typically seen in cases of near-drowning. I base this conclusion on the patient's normal chest radiograph and lack of pulmonary complaints,[10–14] as well as on previous reports of seawater ingestion, which also involve polyuria despite hypovolemia[17,18]; postresuscitation diarrhea[1]; hypernatremia[3,9]; hypermagnesemia[2,14]; and persistence of hyperchloremia after free water replacement.[1,5,9,14] Although higher serum sodium values have been observed in dehydrated Cuban refugee rafters who voluntarily drank seawater (personal communication, K. Candiotti MD, Jackson Memorial Hospital, Miami, Florida, March 1997), I believe the patient presented in this paper had the highest serum sodium level reported in the literature after involuntary ingestion of seawater during an episode of near-drowning. In the three days following his severe hypernatremia he did not appear to be adversely affected by the precipitous decrease in his serum sodium level, although the rate of change far exceeded the threshold above which patients are considered to be at risk for cerebral edema.[7] A longer follow-up period might be required to definitively prove a lack of significant central nervous system sequelae in this patient. Unfortunately, a longer follow-up period was not possible in this case, although reports from the patient's acquaintances indicate he is doing quite well and has returned to the activities of daily living.

The case reported here demonstrates that severe symptomatic hypernatremia can occur as a result of near-drowning episodes when significant amounts of seawater are swallowed rather than aspirated. The effects of seawater ingestion may be important components of the abnormalities observed in near-drowning victims who experience recurrent involuntary submersion in rough seas. Accordingly, controlled administration of free water, whether orally or intravenously, should be a part of the patient's resuscitative regimen.

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Acknowledgment

Long-Term Remission of HIV-Associated Thrombocytopenia Parallels Ongoing Suppression of Viral Replication

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THROMBOCYTOPENIA MAY be found at any stage of human immunodeficiency virus (HIV) infection.[1] Its prevalence increases as HIV disease advances, with ranges reported of 20% to 45% from the early to the late stages of infection.[2] The mechanisms producing HIV-associated thrombocytopenia are likely multifactorial, with evidence for both decreased platelet production and increased immune system destruction.[3–9] Pharmacotherapy against HIV may result in thrombocytopenia,[2,10] although zidovudine, the most-studied antiretroviral agent in this clinical setting, is more likely to cause leukopenia and anemia.[11]

Treatment options for symptomatic or life-threatening HIV-associated thrombocytopenia have relied on the use of zidovudine alone or in combination with prednisone followed by splenectomy for refractory cases.[2,12–14] Corticosteroids produce a response in most HIV-infected persons but, after tapering, fail to induce a

lasting remission. In addition, concerns exist over their potential for further immunosuppression.[2] Intravenous immune globulin therapy is variably effective because of its rapid onset of action but short-lived benefits.[2,15] The use of interferon alfa 2b may improve platelet counts, but it requires indefinite therapy at high costs and has poor patient acceptability.[2,16] One study found a short course of high-dose dexamethasone able to induce a remission in a small number of patients.[17]

The literature on antiretroviral therapy for HIV-associated thrombocytopenia has been limited to the use of zidovudine monotherapy. This use has been hampered by a previous inability to document its efficacy against HIV, relying on insensitive surrogate markers such as p24 antigenemia.[2,7,18–20] Therapeutics against HIV infection have evolved to a more aggressive approach that uses combinations of antiretrovirals while monitoring viral load to assess the treatment response.[21–27] We report the successful management of a case of HIV-associated thrombocytopenia in which long-term remission was maintained with dual nucleoside analog therapy alone in association with the ongoing suppression of viral replication.

Report of a Case

The patient, a 42-year-old homosexual man, was seen at an urgent care facility because of epistaxis and ease of bruising over a three- to four-week period. A complete cell count was done and found to be normal except for a low platelet count of 15.0×10^9 per liter (15,000 per mm^3). Hospital admission was immediately arranged under the care of a hematologist.

The patient's medical history was remarkable only for a previous bout of viral hepatitis, type unknown, and occasional bouts of allergic rhinitis. Basically healthy, he used antihistamines for sinus relief and ibuprofen on occasion for pain and only drank socially. The patient engaged in risky sexual activity and reported a negative HIV test taken four years ago.

A physical examination revealed normal vital signs. Pertinent findings were limited to the skin, which showed multiple petechiae and large ecchymotic lesions on both lower legs and the right forearm. No manifestations of HIV infection or related opportunistic infections were noted.

The patient was given intravenous immune globulin (IVIG), 800 mg per kg over 12 hours on two consecutive days for presumptive treatment of immune thrombocytopenic purpura (ITP). Per his request, an enzyme-linked immunosorbent assay test to screen for HIV infection was obtained with a positive result confirmed by Western blot analysis. A CD4 lymphocyte count revealed 364×10^9 per liter (364,000 cells per mm^3). The patient was released on the fourth day with a platelet count of 91×10^9 per liter.

In follow-up two weeks later, the patient's platelet count had fallen to 12×10^9 per liter. He again received IVIG, a single dose of 400 mg per kg, as an outpatient