Near-Drowning in the Dead Sea: A Retrospective Observational Analysis of 69 Patients

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Abstract

Background: The Dead Sea in Israel has a very high mineral content. Near-drowning in the Dead Sea is expected to result in severe electrolyte abnormalities and respiratory failure. Previous limited studies reported a high mortality rate.

Objective: To evaluate the clinical and biochemical manifestations and disease outcome of near-drowning in the Dead Sea.

Methods: Data were abstracted from the archives of Soroka University Medical Center. The cohort comprised 69 patients who nearly drowned in the Dead Sea.

Results: The median age of the patients was 68 years (range 21–84). There were two major manifestations of near-drowning in the Dead Sea: electrolyte imbalance and acute lung injury. Serum calcium, magnesium and phosphorus (but not sodium, potassium and chloride) were abnormal in most patients. Median serum electrolyte levels (and range) on admission were 10.9 mg/dl (9–24) for calcium, 4.3 mg/dl (1–30) for magnesium, and 4.1 mg/dl (2–9) for phosphorus. These levels quickly normalized with forced diuresis within 24 hours. Acute lung injury – namely, hypoxic bilateral pneumonitis – occurred in 29 patients. Mechanical ventilation was required in 11 patients. Sixty-five patients recovered fully, while the remaining 4 had minor sequelae.

Conclusions: Near-drowning in the Dead Sea is a syndrome of severe electrolyte abnormalities and lung injury. Early treatment with forced diuresis and supportive care results in prompt recovery.

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The Dead Sea is a terminal lake of the Jordan Rift Valley, formed some 150 million years ago and presently situated 400 m below sea level [1]. The lake has a uniquely high concentration of sodium, chloride, calcium, magnesium, phosphorus and other minerals. For example, the NaCl concentration at the surface is over 275 g/L. These high concentrations of solutes are caused by the high rate of water evaporation, a consequence of high surface temperatures and low humidity in the area [2]. Owing to the buoyancy of the water in the Dead Sea, it is very difficult to drown [3–7]. However, momentary submersion may lead to ingestion and aspiration of its water, a syndrome called Near-Drowning in the Dead Sea [3]. Even small amounts of water ingested can cause severe electrolyte imbalance. Aspiration of the water may lead to respiratory failure within 10 minutes of submersion, according to a canine model [7]. Earlier studies suggested that near-drowning in the Dead Sea is a syndrome with a high mortality rate (19–50%) [3,4,8]. The present study was conducted to reassess the clinical spectrum of this syndrome and to evaluate current trends in management and outcome.

Materials and Methods

Given its proximity, the Soroka University Medical Center receives all patients with near-drowning in the Dead Sea. During 1990–2001, 69 patients who had nearly drowned in the Dead Sea were admitted to the medical or intensive care wards of this hospital. Data were retrieved from the medical archives by one author using a standardized abstraction form. These data included: demographic characteristics, past medical history and medication; clinical examination, electrocardiograph and chest X-ray results on admission, as well as differential cell count, blood electrolytes (sodium, potassium, calcium, magnesium and phosphorus levels) and blood gases on admission and after 24 hours. Statistical differences were calculated by Student's paired *t*-test. Details of patient management, complications and outcome were noted. The data chart was reviewed by both authors together and then analyzed.

Results

Clinical findings on admission

The medical records of 34 men and 35 women were reviewed. The age range of the patients was 21–84 years with a mean ± SD of 66 ± 13.5; the median age was 68 years. The clinical and laboratory

Table 1. Clinical and laboratory findings on admission

Consciousness	Fully conscious	61 (88)
No. (%)	Stupor	2 (3)
	Unconscious	6 (9)
Respiratory support	Supplemental	58 (84)
No. (%)	oxygen	11 (16)
	Mechanical ventilation	
Temperature (°C)	Range	34–39
	Median	36.6
Pulse (per min)	Range	60–126
	Median	88
Blood pressure (mm Hg)	Range	90/49–225/115
	Median	145/80
Respiratory (per min)	Range	12–32
	Median	18
Lung auscultation	Clear	14 (20)
No. (%)	Wheezes/rales	40 (58)
	Crackles	14 (20)
ECG	Sinus rhythm	64 (93)
No. (%)	Normal QTc	65 (94)
Chest X-ray	Infiltrate	20 (29)
No. (%)	Congestion	7 (10)

findings on admission are summarized in Table 1. Most patients were fully conscious on arrival to hospital. Six patients were unconscious and required intubation. The indication for ventilatory support was acute hypoxemic respiratory failure. On admission most patients (85.5%) were normothermic, though 4 patients (6%) had a temperature above 38°C and 6 (9%) had a temperature below 36°C. Tachypnea (respiratory rate > 20) was noted in 26 patients (38%), and tachycardia (pulse > 100) in 22 patients (32%). On arrival several patients were hypertensive: 38 patients (55%) had systolic blood pressure above 140 mmHg and 24 (35%) had diastolic blood pressure above 100 mmHg. The majority of patients had abnormal findings on lung auscultation; 40 (58%) had rales and/or wheezes and 14 (20%) had crackles. Chest X-ray on admission revealed that 20 patients (29%) had an infiltrate; 6 patients had bilateral pulmonary infiltrates that could have been interpreted as pulmonary edema.

An ECG recording was available for 67 patients. Normal sinus rhythm was noted in 97%, while atrial fibrillation was observed in 3 patients, 2 of whom were known to have chronic atrial fibrillation. One patient had atrial flutter, which spontaneously converted to a normal sinus rhythm. One patient had a first-degree atrioventricular block.

Laboratory blood tests

Laboratory blood analysis was done on arrival and included differential cell count and blood chemistry [Table 2]. The levels of calcium, magnesium and phosphorus were high on admission, but by 24 hours these values had decreased and in many cases were in the normal range. Sodium, potassium and chloride levels were mostly within normal range and were not re-measured at 24 hours. Arterial blood gases on admission revealed a normal pH (median 7.36) with mild hypoxemia in most patients.

Course and treatment

Pulmonary complications dominated the course in many patients. Pneumonia (infectious, aspiration) occurred in 26 patients (38%), while 3 patients developed acute respiratory distress syndrome. Respiratory support included nasal oxygen in 49 patients (71%) and mechanical venti1ation in 11 (16%). The rate of mechanical ventilation was higher in earlier reports [3], approaching 25% (11/44). Eight of the 11 patients requiring artificial ventilation were intubated at the scene. Eleven patients (16%) did not require any respiratory support.

Antibiotic treatment was administered to 42 patients (61%). The main indication for antibiotic treatment was fever associated with pulmonary infiltrates. Blood cultures were negative in all; sputum cultures were not sent. Most of the patients received amoxycillin-clavulanic acid. Nine patients (13%) underwent gastric lavage. Forced diuresis was used in 67 patients (97%); this included an intravenous fluid infusion and intravenous diuretics (furosamide). Two patients required hemodialysis for severe hypercalcemia and hypermagnesemia accompanied by cardiac arrhythmia. Corticosteroids were administered to 21 patients (30.4%). The neurologic complications included a seizure in one patient who had had a cerebrovascular accident in the past. One patient sustained hypoxic encephalopathy. Two patients had persistent hypoxemia requiring prolonged oxygen administration, while one had mild hypoxic encephalopathy. One patient required prolonged ventilatory support. The median length of hospitalization was 3 days. Outcome was generally favorable. There were no mortalities and only four patients sustained residual dysfunction.

Table 2. Laboratory values upon admission and after 24 hours

Variable Range	Upon admission (range/median, mean ± SD)	24 hours after admission (range/median, mean ± SD)	% with abnormal values
Hemoglobin 12–16 g/dl	9.7–20.8/14.8 14.8 ± 2.08 (n=68)		23.5
Hematocrit 37–47%	31–63/45 44.8 ± 6.45 (n=64)		33
White blood cells 5–11 x 1,000/mm^3	5.2–29.0/13.6 14.5 ± 5.4 (n=67)		58
Urea 10–36 mg/dl	9–149/38 43 ± 18.7 (n=69)		46
Sodium 135–145 mEq/L	129–156/142 141.5 ± 5.9 (n=69)		23
Potassium 3.5–5 mEq/L	3–5/4.2 4.2 ± 0.54 (n=69)		12
Chloride 98–106 mEq/L	96–127/108 108.9 ± 7.5 (n=45)		49
Calcium 8.6–10.2 mg/dl	9–24/11 12 ± 2.9 (n=69)	8–17/9 10 ± 1.5* (n=58)	55*** 28****
Magnesium 1.7–2.3 mg/dl	1–30/4 5 ± 4.1 (n=69)	1–12/2 3 ± 1.5* (n=56)	71*** 50****
Phosphorus 2.7–4.5 mg/dl	2–9/4 4.3 ± 1.5 (n=61)	2–6/3.5 3.7 ± 1.1* (n=38)	31*** 21****
PO_2 69–116	38–182/79 88 ± 31.2** (n=58)		50
PCO_2 32–42	20–47/37 37 ± 6 (n=58)		26

* $P < 0.01$ compared with pre-treatment values.
** Some of the blood gasses were drawn under oxygen treatment.
*** Upon admission.
**** 24 hours after admission.

Discussion

Near-drowning in the Dead Sea is a medical emergency. It presents with two major manifestations: acute lung injury and electrolyte imbalance. Knowledge of the condition and its treatment are essential to save the patients' lives.

Hypoxic bilateral pneumonitis characterizes the acute lung injury. Superimposed infection may complicate the course. The

lung injury tends to develop within as little as 4 hours, but sometimes later [9]. The presumed mechanism for this acute lung injury is direct chemical irritation of the aspirated salt-rich water. Although most patients need mild supportive measures, severe lung injury may develop requiring mechanical ventilation. In our series, most patients required oxygen and several were intubated due to acute hypoxemic respiratory failure. The fact that only 11% of patients required invasive ventilation in the current series as opposed to 25% in a previous report [3] is important. This difference may be attributed to attenuation of lung injury due to improved early therapy. Most of our patients recovered after a few days of therapy and were quickly weaned off the ventilator.

Corticosteroids were used to treat pulmonary injury in some of our patients, as reported also by others [8]. The role of corticosteroids in this setting is controversial. There are no studies supporting this practice and no clear indication could be determined. This therapy was more commonly used in the 1990s for treating pulmonary injuries associated with near-drowning. It is unclear though whether this therapy affected their course and outcome.

The electrolyte imbalance observed in victims of near-drowning in the Dead Sea is due to the uniquely high mineral concentrations in its water. Swallowing rather than aspiration of salty water in the Dead Sea is considered to be the main pathogenic factor in causing acute combined hypercalcemia and hypermagnesemia [6]. The kidneys are evidently incapable of excreting this solute overload without serum electrolyte imbalances occurring first [4]. As observed with our patients and with patients in previous studies, hypercalcemia and hypermagnesemia are the most striking electrolyte abnormalities seen after submersion in the Dead Sea [5,6]. Extreme hypermagnesemia could be fatal, but the accompanying hypercalcemia has a counteracting protective effect [5]. Hypermagnesemia can cause blockade of neuromuscular transmission, conduction abnormalities and depression of the sympathetic ganglia [5]. None of the patients was intubated due to weakness, a possible consequence of hypermagnesemia. Hypercalcemia can cause central nervous system disturbances, neuromuscular dysfunction and cardiac arrhythmia [4]. All the effects of magnesium are partially antagonized by calcium. Hypermagnesemia causes prolongation of the QT interval, while hypercalcemia causes the opposite effect. These opposing effects may explain the normal QT interval observed in our patients [5].

Most of our patients were treated with forced diuresis. In most of the cases the Emergency Medical Service initiated this treatment at the scene of the near-drowning. There were no specific indications for this therapy or a pre-defined threshold. This approach was introduced gradually and empirically. Nonetheless, it appears to have contributed significantly to the marked reduction in mortality observed in our series.

Gastric lavage was used in nine patients in the early 1990s. There was no difference in clinical characteristics and outcome of patients in whom gastric lavage was not performed. The indication for gastric lavage could not be determined from the information available in the charts. One might assume that an attempt was made to diminish absorption of solutes swallowed. However, given the rapidity with which water and electrolytes are absorbed, it is unlikely that this intervention could help. Therefore, we do not recommend this practice.

Two patients in this study were treated with dialysis for severe hypercalcemia and hypermagnesemia complicated by cardiac arrhythmia. Following treatment there was a significant drop in calcium and magnesium levels and resolution of the cardiac arrhythmia. No specific anti-arrhythmic treatment was added. It is worth noting that sodium and potassium concentrations did not rise in the blood of these patients, reflecting the ability of the kidneys to excrete these cations very quickly.

A review of ECGs from 37 victims of near-drowning in the Dead Sea notes a tendency towards prolongation of the PR interval followed by a prominent U wave [6]. Only one patient had first-degree atrioventricular block. Three patients in that study had a bout of ventricular tachycardia while the QTc intervals were within normal limits. In our study only three patients were found to have a shortened QTc. A likely explanation is that hypermagnesemia normalizes the QT interval (which is usually shortened by isolated hypercalcemia) in patients with combined hypercalcemia and hypermagnesemia.

In three previous studies, the mortality rate was rather high, 19%, 50% and 28% respectively [3,4,8]. Mortality was caused by respiratory failure and/or pneumonia in some cases and by intractable arrhythmia, apparently induced by severe electrolyte imbalances, in others. In comparison, our results were excellent. Due to the lack of reliable data it is impossible to compare between the severity of illness of the patients in our series and those previously reported.

In conclusion, it is awareness of this condition and the immediate aggressive treatment of the electrolyte and pulmonary complications that is of paramount importance. This approach prevents mortality and residual morbidity.

References

1. The New Encyclopedia Britannica. 15th edn. Vol. 3, 1887:936–7.
2. Leiberman A, Kraus M. Inner ear dysfunction due to hypertonic sea water. *Isr J Med Sci* 1988;24:255–6.
3. Porath A, Mosseri M, Harman I, Ovsyshcher I, Stone D. Dead Sea water poisoning. *Ann Emerg Med* 1989;18:187–91.
4. Yagil Y, Stalnikowicz R, Michaeli J, Mogel P. Near drowning in the Dead Sea. *Arch Intern Med* 1985;145:50–3.
5. Oren S, Rapoport J, Zlotnic M. Extreme hypermagnesemia due to ingestion of Dead Sea water. *Nephron* 1987;47:199–201.
6. Mosseri M, Porath A, Ovsyshcher I, Stone D. Electrocardiographic manifestations of combined hypercalcemia and hypermagnesemia. *J Electro* 1990;23(3):235–41.
7. Bark H, Porath A, Gueta V, Heimer D. Physiological changes in respiration associated with near drowning in the Dead Sea: a canine model. *Isr J Med Sci* 1990;26(4):183–7.
8. Alkan ML, Gesztes T, Kotev S, Ben-Ari J. Near drowning in the Dead Sea. *Isr J Med Sci* 1977;13:290–4.
9. Thanel F. Near drowning – rescuing patients through education as well as treatment. *Postgrad Med Symp* 1998;103(6):141–53.

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