Imaging Findings in Patients with Near-drowning Incidents in the Dead Sea and Their Correlation with Clinical Outcomes

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ABSTRACT **Background:** The Dead Sea is a hypersaline lake with unique mineral concentrations. Ingestion and aspiration of its water can lead to severe electrolyte imbalances and chemical pneumonitis.

Objectives: To evaluate imaging findings from Dead Sea near-drowning victims and to correlate these findings with clinical outcomes.

Methods: We conducted a retrospective study of patients (age > 18 years) admitted to a tertiary medical center between 2004 and 2024 following Dead Sea near-drowning. Imaging studies were categorized by a radiology expert into normal, interstitial changes, unilateral consolidation, or bilateral consolidation. Clinical outcomes, including mortality and intensive care unit (ICU) admission, were analyzed across the groups.

Results: Of the 150 patients identified, 68.7% (n=103) had normal chest X-rays. Abnormal findings included bilateral consolidation 12.7% (n=19), unilateral consolidation 9.3% (n=14), and interstitial changes 9.3% (n=14). Patients with bilateral consolidation had significantly higher peak serum magnesium (median 10.5 vs. 3.1 mg/dl in the normal group; $P < 0.001$) and peak calcium (median 13.1 vs. 10.3 mg/dl; $P < 0.001$). Bilateral consolidation was a strong predictor of severity: 26.3% in-hospital mortality rate compared to 1% in the group with normal X-rays ($P < 0.001$). ICU admission was required for 78.9% of patients with bilateral consolidation and 71.4% with interstitial changes ($P < 0.001$).

Conclusions: Radiologic findings in Dead Sea near-drownings are predictive of clinical trajectory. Bilateral alveolar consolidation is an indication of massive mineral aspiration, severe hypermagnesemia, and high mortality. Early imaging is essential for risk stratification and for determining the level of care required.

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KEY WORDS: chemical pneumonitis, chest X-ray, Dead Sea, hypermagnesemia, near-drowning

The Dead Sea holds the title of the deepest hypersaline lake in the world. With a salinity of 342 g/kg, it ranks among the saltiest bodies of water globally [1-3]. For thousands of years, the Dead Sea has drawn visitors from across the Mediterranean Basin and was one of the first health resorts in history.

The water composition of the Dead Sea is unique, comprised of 14.4% calcium chloride, 4.4% potassium chloride, 50.8% magnesium chloride, and 30.4% sodium chloride. The Dead Sea has a notably low concentration of sulfate ions and the highest concentration of bromide ions found in any body of water on Earth [4].

Due to the water's buoyancy, drowning in the Dead Sea is highly unlikely. However, brief submersion can lead to the ingestion and aspiration of its water, resulting in a condition known as near-drowning in the Dead Sea. Even small amounts of ingested water can cause significant electrolyte imbalances, and aspiration may lead to respiratory failure within 10 minutes of submersion, according to studies using canine models [5-10].

Previous research has indicated that the near-drowning syndrome in the Dead Sea has a high mortality rate, ranging from 19% to 50% [6,7,11]. Fatalities have been attributed to respiratory failure, pneumonia, and severe arrhythmias caused by electrolyte imbalances [6,7,11]. The most recent study on this syndrome was conducted in 2003 and reported generally favorable outcomes [5].

However, these earlier studies involved a limited number of patients, and there was no analysis of the nature of lung injuries and their effects on outcomes. For this study, we evaluated the characteristics and frequency of lung injuries in patients experiencing near-drowning in the Dead Sea and to assess their impact on overall outcomes.

PATIENTS AND METHODS

STUDY POPULATION

We retrospectively reviewed medical records of all patients admitted to the Soroka University Medical Center (SUMC) with a diagnosis of near-drowning syndrome in the Dead Sea from 2004 to 2024. SUMC, a 1150-bed tertiary medical facility in Southern Israel, caters to a population of approximately 1.2 million individuals. Our inclusion criteria were patients over 18 years of age who had been hospitalized with the diagnosis of near-drowning syndrome (ICD 9 code 994) [Supplemental Figure 1, available in the online version only]. The study was approved by the ethics committee at SUMC (Approval No. SOR-0217-24). Due to the retrospective nature of the study, a waiver of informed consent was granted.

DATA COLLECTION

An internal medicine specialist reviewed each computerized medical record, while a radiology expert evaluated all imaging studies, including X-rays and computed tomography (CT) scans. Patients were categorized into groups based on their lung injury status: no lung injury (normal study), interstitial lung injury, unilateral alveolar consolidation, and bilateral alveolar consolidation [Supplemental Figure 2, which is available in the online version only].

Demographic information, medical history, laboratory results, and clinical outcomes were extracted from the computerized hospital database. Initial laboratory tests recorded on admission to the emergency department and at 24 hours were analyzed. Patients were assigned an identification number, which was de-identified before data analysis. A radiology specialist examined all imaging studies within the hospital's computerized picture archiving and communication system. Clinical outcomes included in-hospital mortality, the need for admission to the intensive care unit (ICU), and the length of hospital stay.

STATISTICAL ANALYSIS

Statistical analyses were performed using IBM Statistical Package for the Social Sciences statistics software, version 25 (SPSS, IBM Corp, Armonk, NY, USA). The distribution of continuous variables was evaluated for normality using the Shapiro-Wilk test. Accordingly, continuous data are expressed as median and interquartile range (IQR)

Table 1. Clinical characteristics

Clinical characteristics	Normal chest X-ray (n=103)	Interstitial changes (n=14)	Unilateral consolidation (n=14)	Bilateral consolidation (n=19)	*P*-value
Age, years, median (IQR)	71 (49–77)	73 (62–79)	71 (60–77)	74 (61–79)	0.509
Male, n (%)	48 (46.6)	10 (71.4)	4 (28.6)	9 (47.4)	0.154
Ischemic heart disease, n (%)	2 (1.9)	2 (14.3)	none	1 (5.3)	0.088
Hypertension, n (%)	7 (6.8)	2 (14.3)	none	1 (5.3)	0.502
Diabetes mellitus, n (%)	17 (16.5)	4 (28.6)	1 (7.1)	3 (15.8)	0.2
COPD, n (%)	12 (11.7)	3 (21.4)	1 (7.1)	none	0.235
Charlson Comorbidity Index, median, IQR	3 (1–4)	1 (1–3)	3 (1.7–3.3)	3 (2–5)	< 0.001
Stupor or coma, n (%)	1 (1.0)	2 (14.3)	3 (21.4)	3 (15.8)	< 0.001
Mechanical ventilation, n (%)	12 (11.7)	3 (21.4)	5 (35.7)	8 (42.1)	0.005
First systolic blood pressure, mmHg, median (IQR)	148 (130–164)	127 (113–134)	156 (141–199)	142 (133–156)	0.054
First diastolic blood pressure, mmHg, median (IQR)	85 (76–97)	83 (81–86)	85 (79–104)	83 (75–98)	0.891
First heart rate, median (IQR)	85 (74–97)	96 (91–118)	73 (61–102)	91 (60–109)	0.166
First SaO_2, %, median (IQR)	97 (95–99)	95 (79–96)	89 (84–98)	86 (78–87)	0.007
First temperature, °C, median (IQR)	36.6 (36.4–36.8)	36.6 (36.4–36.6)	36.5 (36.4–36.6)	36.4 (36.4–36.4)	0.891

P-values represent the global comparison across all four groups using the Kruskal-Wallis test for continuous variables and Fisher's exact test for categorical variables
COPD = chronic obstructive pulmonary disease, IQR = interquartile range

and were compared across radiological groups using the Kruskal-Wallis test. Post-hoc pairwise comparisons were performed using the Mann-Whitney U test with Bonferroni adjustment to account for multiple testing. Categorical variables are presented as frequencies and percentages. Proportions were compared using the chi-square test or Fisher's exact test, as appropriate based on expected cell frequencies. Specifically, Fisher's exact test was applied for clinical outcomes where subgroup sizes were limited. All tests were two-sided, and a *P*-value of < 0.05 was considered statistically significant. The study was conducted in accordance with the STROBE statement for the reporting of observational studies.

RESULTS

RADIOLOGIC DISTRIBUTION AND CLINICAL PRESENTATION

Of the 150 patients identified between 2004 and 2024, the majority (n=103, 68.7%) had normal chest X-rays on admission. Abnormal imaging findings included bilateral alveolar consolidation (n=19, 12.7%), unilateral consolidation (n=14, 9.3%), and interstitial changes (n=14, 9.3%). In addition, five patients had a pleural effusion. Patients with bilateral consolidation were significantly more likely to present in a state of stupor or coma (15.8%) compared to those with normal imaging (1.0%; $P < 0.001$). Baseline oxygen saturation (SaO_2) was lowest in the bilateral consolidation group (median 86%, IQR 78–87) compared to the group with normal chest X-rays (median 97% IQR 95–99; $P = 0.007$) [Table 1].

CT was performed in a small subset of patients (n=8, 5.3%) using various protocols. Due to the limited sample size and lack of standardization, these data were not included in the formal statistical analysis. Qualitatively, CT findings were consistent with the initial chest X-ray categorizations.

LABORATORY FINDINGS AND ELECTROLYTE IMBALANCES

Significant electrolyte disturbances were observed across all groups but were most profound in patients with bilateral consolidation.

Magnesium and calcium

On admission, serum magnesium was highest in the bilateral consolidation group (median 10.2; IQR 5.1–14.6 mg/dl), which also showed the highest peak magnesium during hospitalization (median 10.5; IQR 5.1–14.6 mg/dl; $P < 0.001$) [Table 2, Table 3]. Similarly, peak serum calcium was significantly higher in the bilateral consolidation group (median 13.1; IQR 11.9–15.6 mg/dl) compared to those with normal CXR (median 10.3 (IQR 9.8–11.6) mg/dl; $P < 0.001$) [Table 3].

Table 2. Laboratory data on admission

Parameters	Normal chest X-ray (n=103)	Interstitial changes (n=14)	Unilateral consolidation (n=14)	Bilateral consolidation (n=19)	*P*-value
Platelets in ED, median (IQR)	240 (202–297)	220 (180–297)	258 (209–335)	273 (231–358)	0.8
Hemoglobin in ED	14.9 (13.7–16.1)	15.9 (14.2–16.9)	15.3 (14.8–16.7)	14.4 (13.9–15.4)	0.3
WBC in ED, median (IQR)	11.1 (8.5–15.1)	12.8 (9.1–19.5)	11.9 (10.0–18.8)	12.4 (9.4–15.3)	0.4
First pH, median (IQR)	7.31 (7.26–7.36)	7.25 (7.17–7.31)	7.29 (7.24–7.31)	7.21 (7.17–7.28)	< 0.001
First PO_2, median (IQR)	32 (24–47)	34 (27–58)	35 (28–61)	54 (42–75)	0.02
First PCO_2, median (IQR)	50 (42–57)	49 (36–57)	50 (43–57)	49 (45–57)	0.7
First serum phosphorus, median (IQR)	3.9 (3.1–4.6)	5.1 (3.9–5.3)	4.0 (3.6–5.8)	3.9 (3.1–4.6)	< 0.001
First serum chloride, median (IQR)	106 (103–108)	110 (107–120)	109 (108–114)	115 (109–121)	< 0.001
First serum potassium, median (IQR)	4.2 (4–4.5)	4.3 (4.1–4.5)	4.2 (4.1–4.4)	4.3 (4–4.6)	0.9
First serum sodium, median (IQR)	141 (139–142)	142 (140–143)	142 (140–142)	140 (138–144)	0.5
First serum magnesium, median (IQR	3 (2.3–4.5)	5.7 (3.6–11.3)	4.7 (4–5.4)	10.2 (5.1–14.6)	< 0.001
First serum calcium, median (IQR)	10.3 (9.7–11.6)	11.8 (10.9–13.9)	10.9 (10.6–12.2)	13.1 (11.9–15.6)	< 0.001
First serum creatinine, median (IQR)	0.85 (0.71–1.01)	1.10 (0.82–1.18)	0.72 (0.66–0.93)	1.01 (0.94–1.27)	0.002
First serum urea, median (IQR)	36 (29–45)	46 (30–51)	43 (31–49)	47.6 (43.6–55.9)	0.001

P-values represent the global comparison across all four groups using the Kruskal-Wallis test
ED = emergency department, IQR = interquartile range, WBC = white blood cells

Table 3. Laboratory data at hospitalization

Parameters	Normal chest X-ray (n=103)	Interstitial changes (n=14)	Unilateral consolidation (n=14)	Bilateral consolidation (n=19)	*P*-value
Max serum phosphorus at hospitalization, median (IQR)	4.0 (3.4–4.8)	5.1 (3.9–5.3)	4 (3.7–5.3)	6 (4.5–6.6)	< 0.001
Min serum phosphorus at hospitalization, median (IQR)	2.9 (2.3–3.5)	3.4 (2.8–4.0)	3.1 (2.2–3.4)	2.1 (1.7–3.3)	0.03
Max serum chloride at hospitalization, median (IQR)	107 (104–109)	111 (107–119)	111 (109–118)	119 (110–126)	< 0.001
Max serum potassium at hospitalization, median (IQR)	4.3 (4.1–4.5)	4.6 (4.4–5.3)	4.5 (4.3–4.7)	4.6 (4.3–5)	0.01
Min serum potassium at hospitalization, median (IQR)	4.1 (3.7–4.4)	3.6 (3.3–3.9)	4.2 (3.7–4.4)	3.5 (3.1–3.7)	< 0.001
Max serum sodium at hospitalization, median (IQR)	140 (138–143)	140 (138–143)	142 (140–145)	146 (141–152)	< 0.001
Min serum sodium at hospitalization, median (IQR)	138 (136–140)	139 (137–140)	138 (136–141)	138 (136–139)	0.4
Max serum magnesium at hospitalization, median (IQR)	3.1 (2.4–4.5)	5.7 (3.6–11.3)	4.7 (4–5.4)	10.5 (5.1–14.6)	< 0.001
Min serum magnesium at hospitalization, median (IQR)	2.1 (2.0–2.4)	2.2 (2.0–2.5)	2.3 (2.0–2.6)	1.9 (1.7–2.3)	0.3
Max serum calcium at hospitalization, median (IQR)	10.3 (9.8–11.6)	11.8 (10.9–13.9)	10.9 (10.6–12.2)	13.1 (11.9–15.6)	< 0.001
Min serum calcium at hospitalization, median (IQR)	9.3 (9–9.6)	8.8 (8.3–9.1)	9.1 (8.3–9.9)	8.9 (8.2–9.4)	0.004
Max serum creatinine at hospitalization, median (IQR)	0.98 (0.7–1.2)	0.79 (0.7–1.0)	1.0 (0.7–1.5)	1.2 (1.1–1.6)	< 0.001
Min serum creatinine at hospitalization, median (IQR)	0.8 (0.6–0.9)	0.7 (0.5–0.8)	0.7 (0.6–1.0)	0.8 (0.6–1.0)	0.6
Max serum urea at hospitalization, median (IQR)	45 (34–61)	34 (24–52)	50 (35–77)	73 (51–97)	< 0.001
Min serum urea at hospitalization, median (IQR)	36 (29–47)	28 (23–39)	36 (28–49)	41 (36–55)	0.2
Max platelets at hospitalization, median (IQR)	239 (191–285)	248 (210–298)	277 (236–347)	258 (186–475)	0.4
Min PLT at hospitalization, median (IQR)	210 (168–224)	226 (192–252)	214 (161–254)	133 (90–169)	<0 .001
Max Hemoglobin at hospitalization, median (IQR)	14.1 (13.1–15.1)	15.6 (14.0–16.9)	13.8 (12.2–15.5)	14.7 (13.3–15.5)	0.2
Min Hemoglobin at hospitalization, median (IQR)	13.2 (12.5–14.2)	12.3 (10.8–13.9)	12.4 (10.7–13.1)	10.2 (9.5–12.4)	< 0.001
Max WBC at hospitalization, median (IQR)	12.9 (9.5–18.4)	13 (7.9–16.7)	16.3 (11.6–23.2)	17.6 (12–25)	0.07
Min WBC at hospitalization, median (IQR)	11.0 (8.7–15.3)	10.7 (6.8–13.4)	11.3 (6.6–14.0)	8.6 (8.3–13.6)	0.2

P-values represent the global comparison across all four groups using the Kruskal-Wallis test
IQR = interquartile range, Max = maximum, Min= minimum, WBC = white blood cells

Table 4. Outcomes

Outcomes	Normal chest X-ray (n=103)	Interstitial changes (n=14)	Unilateral consolidation (n=14)	Bilateral consolidation (n=19)	*P*-value
In-hospital mortality, n (%)	1 (1)	none	none	5 (26.3)	< 0.001
30-day mortality, n (%)	1 (1)	none	none	5 (26.3)	< 0.001
Length of hospital stay, days, median (IQR)	1 (1–2)	3 (1–5)	2 (1–4)	2 (1–4)	< 0.001
Admission to ICU, n (%)	13 (12.6)	10 (71.4)	6 (42.9)	15 (78.9)	< 0.001
ICU hospitalization days, median (IQR)	1 (1–2)	2 (1–6)	1 (1–7)	3 (1–10)	< 0.001

P-values represent the global comparison across all four groups using the Kruskal-Wallis test for continuous variables and Fisher's Exact test for categorical variables
ICU = intensive care unit, IQR = interquartile range

Acid-base balance

A significant decrease in admission pH was noted in the interstitial and bilateral groups (median 7.25; IQR 7.17–7.3, and 7.21; IQR 7.17–7.28, respectively) compared to the normal group (median 7.31; IQR 7.26–7.36); $P < 0.001$) [Table 2].

Renal function

Peak serum creatinine (median 1.2; IQR 1.1–1.6 mg/dl) and urea (median 73; IQR 51–97 mg/d) were significantly higher in patients with bilateral consolidation ($P < 0.001$) [Table 3].

CLINICAL OUTCOMES

Imaging findings were strongly correlated with clinical severity and survival.

Mortality

In-hospital and 30-day mortality was 26.3% (n=5) in the bilateral consolidation group, significantly higher than the 2.1% (n=1) in the normal chest X-ray group ($P = 0.001$). No deaths occurred in the unilateral or interstitial groups [Table 4].

ICU hospitalization

Admission to the ICU was required for 78.9% of patients with bilateral consolidation and 71.4% of those with interstitial changes, compared to only 12.6% of those with normal CXR ($P < 0.001$).

Length of hospital stay

Patients with interstitial changes had the longest median hospital stay (3 days; $P < 0.001$) [Table 4].

DISCUSSION

This study demonstrates that while most near-drowning incidents in the Dead Sea result in normal imaging and favorable outcomes, the presence of bilateral alveolar consolidation is a potent predictor of mortality and severe systemic toxicity.

THE PATHOPHYSIOLOGY OF DEAD SEA ASPIRATION

The unique chemical composition of the Dead Sea–specifically the extreme concentrations of magnesium, calcium, and bromide–creates a chemical pneumonitis distinct from fresh or saltwater drowning [1-3]. Our data show that bilateral consolidation is not merely a lung injury but a marker for massive salt ingestion and aspiration, confirmed by the significantly higher peak levels of magnesium and calcium in this group. These electrolytes are known to cause cardiac arrhythmias and neuromuscular depression, which likely contributed to the high mortality rate (26.3%) observed in these patients. Furthermore, notable hypermagnesemia was already evident at admission [Table 2], indicating rapid mineral absorption and mitigating potential testing-related time bias. Physiologically, an interstitial pattern likely represents an early osmotic fluid shift, whereas bilateral consolidation may suggest more massive aspiration leading to alveolar flooding and potential surfactant dysfunction.

IMAGING AS A PROGNOSTIC TOOL

Our findings suggest that early chest imaging is critical for risk stratification. Patients with interstitial changes or unilateral consolidation, while often requiring ICU admission for monitoring (71.4% and 42.9%, respectively), did not experience mortality in our cohort. In contrast, bilateral alveolar consolidation appeared to represent a critical threshold of aspiration that correlated with acute kidney injury (higher urea/creatinine) and severe respiratory failure [Table 3,Table 4].

COMPARISON TO THE LITERATURE

Compared to the 2003 study by Saidel-Odes et al. [5], which reported generally favorable outcomes, our larger 20-year cohort highlights a persistent high-risk subgroup. The mortality rate in our bilateral consolidation group remained high, aligning with earlier studies from the 1980s that cited rates up to 50% [6,7,11]. This finding suggests that despite improvements in intensive care and mechanical ventilation, the initial chemical insult of Dead Sea water remains life-threatening.

Last, geographical and temporal variations warrant consideration. SUMC primarily serves the southern basin of the Dead Sea, which consists of industrial evaporation ponds with higher mineral concentrations than the northern basin. A literature review yielded no published clinical or radiological data from hospitals serving the northern basin (e.g., in Jerusalem) for comparison. In addition, salinity has progressively increased over the 20-year study period [12]. Consequently, our findings specifically reflect the clinical profile of the hypersaline southern environment.

STRENGTHS

The primary strength of this study is the extended 20-year observation period, which provides one of the largest cohorts of Dead Sea near-drowning incidents to date. By utilizing a single-center approach at a major tertiary medi-

cal center (SUMC), which serves as the primary referral point for the Dead Sea region, we ensured consistency in clinical protocols and laboratory measurements. Furthermore, the specialized radiologic review of all imaging studies allowed for a more granular understanding of how specific patterns of lung injury, rather than just the presence of injury, correlate with systemic electrolyte toxicity and survival.

LIMITATIONS

Despite these strengths, several limitations must be acknowledged:

- Retrospective design: As a retrospective medical record review, the study is subject to inherent documentation biases. Some pre-hospital data, such as the exact volume of water ingested or the precise duration of submersion, were not consistently available.
- Sample size of subgroups: While the total population is large for this specific syndrome, the number of patients in the bilateral consolidation and interstitial subgroups was relatively small, which may limit the statistical power for certain secondary outcomes.
- Selection bias: Our data are limited to patients who reached the hospital alive. Cases of immediate drowning at the scene were not captured, potentially leading to an underestimation of the total mortality associated with Dead Sea submersion.
- Long-term follow-up: While we tracked 30-day mortality, we lack data on long-term pulmonary function or neurological outcomes in survivors of severe hypermagnesemia.

CONCLUSIONS

Imaging findings serve as a vital surrogate for the severity of Dead Sea water aspiration and ingestion. Bilateral alveolar consolidation on a chest X-ray is a red-flag finding associated with profound hypermagnesemia, hypercalcemia, and a tenfold increase in mortality compared to patients with normal imaging. We recommend that any patient presenting with abnormal imaging after a Dead Sea submersion be admitted to an intensive care environment for aggressive electrolyte monitoring and respiratory support.

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Capsule

Human vaccine responses regulated by parallel cytokine pathways

Chen et al. identified two largely parallel cytokine signaling pathways that independently contribute to the generation of protective immune responses. One pathway primarily promoted activation and expansion of T-cell responses, whereas the second preferentially supported B-cell activation, antibody production, and germinal center development. Rather than functioning sequentially, these pathways operated simultaneously and together determined the magnitude and quality of vaccine-induced immunity. Importantly, individuals exhibiting stronger activation of both pathways developed more robust humoral and cellular immune responses. The study also demonstrated substantial inter-individual variability in cytokine signatures, providing a biological explanation for differences in vaccine responsiveness among healthy individuals.

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