



## CASE REPORT

# Water Intoxication and Hyponatremia due Religious Ritual “Ruqayyah”: Case Report

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## Abstract

**Introduction:** This paper discusses a patient who developed severe acute hyponatremia due to consuming an excessive amount of water (19 liters) over 4 hours during a religious ritual called “Ruqayyah.” Understanding this case can contribute to raising awareness and preventing similar occurrences in the future.

**Case presentation:** A 29-year-old woman, who had been undergoing psychotherapy for depression for the past month, was brought to the emergency department by emergency medical services after experiencing a new-onset generalized tonic-clonic seizure. Patients consumed approximately 19 liters of fluid over a period of 4 hours due to religious beliefs. The diagnosis of water intoxication with severe hyponatremia (Na = 109 mmol/L) was made. Optimum fluid and electrolyte management achieved, and the patient discharged with a good neurological outcome.

**Conclusion:** The presented case offers a compelling narrative that intersects cultural practices, medical emergencies, and the importance of culturally sensitive healthcare delivery. Therefore, healthcare providers must be aware of the potential risks associated with certain rituals and be prepared to address any medical emergencies that may arise.

## Keywords

Water intoxication, Hyponatremia, Osmotic demyelination syndrome, Spirituality

procedures such as colonoscopy preparation and uroflowmetry, military training camps, child abuse, hazing activities, and ingestion of 3,4-Methylenedioxy methamphetamine (MDMA) [1].

The acute onset of hyponatremia, marked by a decrease in sodium levels within 48 hours, represents a medical emergency with potentially serious consequences, as evidenced by a case mortality rate of 17.9% [1].

A serum sodium level below 125 mmol/L is categorized as severe hyponatremia and can lead to irreversible clinical outcomes like coma, rhabdomyolysis, or even death. Neurological damage can be difficult to promptly diagnose due to the nonspecific nature of symptoms such as headache, nausea, neurological deficits, seizures, coma, and, ultimately, death caused by cerebral edema [2].

In our case report, we found that the religious ritual known as “Ruqayyah” led to water intoxication. During this ritual, participants drink blessed water guided by a “Shaikh,” who prepares the water and oversees the ritual process. Signs and symptoms of water intoxication and hyponatremia might be subtly overlooked by participants, who may rationalize them as part of a ritualistic process.

This paper discusses a patient who developed severe acute hyponatremia due to consuming an excessive amount of water (19 liters) over 4 hours during a religious ritual called “Ruqayyah.” Understanding this case can contribute to raising awareness and preventing similar occurrences in the future.

## Introduction

Water intoxication, resulting from excessive oral fluid intake, is a significant cause of symptomatic hyponatremia, leading to a range of complications that can be fatal in various situations. These include psychogenic polydipsia, endurance sports, medical

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## Background of the Religious Ritual “Ruqayyah”

The religious ritual “Ruqayyah” originates from Islamic tradition and serves as a means of seeking spiritual healing and protection from harm. Participants engage in reciting specific prayers and verses from the Quran, along with making supplications, to seek relief from various afflictions, both physical and spiritual. Central to the ritual is the use of water infused with these prayers, believed to possess potent healing properties and the ability to ward off evil. Participants consume this blessed water or use it in ritual ablutions, trusting in its cleansing and purifying qualities. “Ruqayyah” holds deep cultural and religious significance for many individuals and communities, underscoring the convergence of faith and healing practices. Understanding the historical context and importance of this ritual is essential for comprehending its role in the lives of its practitioners [3].

## Case Presentation

A 29-year-old woman, who had been undergoing psychotherapy for depression for the past month, was brought to the emergency department by emergency medical services after experiencing a new-onset of seizure.

On the same day, the family brought in a “Shaikh” to perform “Ruqayyah” on her as a treatment for depression. The “Shaikh” began administering a large quantity of boiled water to the patient. In total, the patient consumed approximately 19 liters of fluid (equivalent to 2 boxes of water, each containing 48 bottles, with each bottle holding 200 ml) over a period of 4 hours. Throughout this time, the patient experienced persistent vomiting and had one episode of diarrhea. Additionally, the patient suffered a single seizure episode, characterized by a generalized tonic-clonic attack, with upward rolling of the eyes and frothing at the mouth, lasting approximately 10 minutes before subsiding.

Upon arrival at the hospital, the patient exhibited a decreased level of consciousness, with a Glasgow Coma Scale score of 4 out of 15. Her vital signs were as follows: blood pressure 114/59 mmHg, oxygen saturation 98%, pulse rate 110 bpm, temperature 36.6 °C, respiratory rate 22 breaths per minute, and glucose level 7.8 mmol/L. The patient’s pupils were non-reactive to light, and she was observed biting her tongue.

Shortly after arriving at the hospital, the patient experienced a tonic-clonic seizure. Her venous blood gas (VBG) revealed a low sodium level of 109 mmol/L. As hypertonic saline was not readily available, the patient received an intravenous (IV) bolus of midazolam 2 mg and 4.8% sodium bicarbonate 100 mg IV. Although the tonic-clonic seizure ceased, the patient continued to exhibit tongue biting and unreactive pupils. Five minutes later, another tonic-clonic seizure occurred,

prompting the administration of an additional 4.8% sodium bicarbonate 100 mg IV and phenytoin 20 mg/kg IV. A repeat VBG showed an increase in sodium to 112 mmol/L, with a pH of 7.25, potassium level of 3.5 mmol/L, lactate level of 6.9 mmol/L, and bicarbonate level of 17.2 mmol/L.

The patient’s pupils showed slight reactivity to light, but she continued to exhibit tongue biting. Meanwhile, 3% hypertonic saline 100 mg became available from the pharmacy and was infused over 20 minutes. Following the infusion, the patient’s Glasgow Coma Scale (GCS) improved to 7 out of 15. A repeat venous blood gas (VBG) showed sodium levels at 113 mmol/L, pH at 7.19, potassium at 2.9 mmol/L, lactate at 7.9 mmol/L, and bicarbonate at 16.5 mmol/L. A computed tomography (CT) scan was performed, revealing no remarkable findings.

Internal medicine and intensive care unit (ICU) doctors were consulted, and the patient was closely monitored. The following day, the patient’s Glasgow Coma Scale (GCS) improved to 15 out of 15, with intact neurological examination findings. Laboratory results as shown below (Table 1).

## Outcome

Patient GCS improved gradually from 3 to 15, her electrolyte including sodium normalized in the second day of admission. No history of further seizure or decreased level of consciousness. The patient responded well to treatment without any complications and was discharged on the third day of admission with a good neurological and functional state.

## Discussion

The presented case offers a compelling narrative that intersects cultural practices, medical emergencies, and the importance of culturally sensitive healthcare delivery. Several points of interest emerge from this case, each highlighting critical aspects of patient care and healthcare system responsiveness.

Firstly, the case underscores the significance of understanding cultural and religious practices in healthcare delivery. The ritual of “Ruqayyah,” deeply rooted in Islamic traditions, serves as a poignant example of how cultural beliefs and practices can influence health-seeking behaviors. Healthcare providers must recognize and respect the cultural nuances surrounding patients’ beliefs and practices, as they may impact medical decision-making and treatment adherence.

Spirituality plays a significant role in shaping physical, emotional, and social well-being [4]. It is deeply intertwined with the human experience of health, healing, illness, and dying [5]. Understanding the spiritual needs of patients can offer healthcare providers a deeper insight into their holistic care [6]. Ultimately, spirituality and religiosity can positively

**Table 1:** Laboratory results.

Time point	Clinical features	Investigations	Managements
At ED presentation	<ul style="list-style-type: none"> <li>GCS 3/15</li> <li>Vitals: Tachycardia, otherwise normal vital signs</li> <li>Nonreactive pupils</li> <li>Stiff jaw and tongue biting</li> <li>Chest: Crepitation over the left lung</li> <li>Cardiac: Normal S1, S2 no added sound</li> <li>Abdomen is soft and lax</li> </ul>	<ul style="list-style-type: none"> <li>VBG: (ph: 7.25, Na 109 mmol/L, K 3.5 mmol/L, lactate 6.5 mmol/L, pCO<sub>2</sub>: 48. HCO<sub>3</sub> 18 mmol/L)</li> <li>Random Blood Glucose: 7.2 mmol/L</li> </ul>	<ul style="list-style-type: none"> <li>Patient was connected to monitor</li> <li>IV lines were established</li> </ul>
10 minutes after ED presentation	<ul style="list-style-type: none"> <li>Seizure attack (tonic-clonic) non-reactive pupil, jaw stiffness</li> </ul>	<ul style="list-style-type: none"> <li>Labs sent: Complete blood count (CBC), Renal function panel, electrolytes</li> </ul>	<ul style="list-style-type: none"> <li>Midazolam 2 mg IV bolus</li> <li>100 ml of 4.8% sodium bicarbonate IV bolus</li> </ul>
25 minutes after ED presentation	<ul style="list-style-type: none"> <li>Clinical seizure was continued</li> <li>Patient face was puffy</li> </ul>		<ul style="list-style-type: none"> <li>Repeated dose of 100 ml of 4.8% sodium bicarbonate IV bolus</li> </ul>
35 minutes after ED presentation	<ul style="list-style-type: none"> <li>Subclinical seizure, persistent non-reactive pupil, jaw stiffness</li> </ul>	VBG: (ph: 7.25, Na 112 mmol/L, K 3.5 mmol/L, lactate 6.9 mmol/L, HCO <sub>3</sub> 17.2 mmol/L)	<ul style="list-style-type: none"> <li>Started phenytoin 20 mg/kg IV</li> </ul>
45 minutes after ED presentation	<ul style="list-style-type: none"> <li>Continued subclinical seizure</li> </ul>	VBG: (ph: 7.19, Na 113 mmol/L, K 2.9 mmol/L, lactate 7.9 mmol/L, HCO <sub>3</sub> 16.5 mmol/L)	<ul style="list-style-type: none"> <li>3% hypertonic saline 100 ml arrived from the pharmacy and infused over 20 minutes</li> </ul>
1 hour and 5 minutes after ED presentation	<ul style="list-style-type: none"> <li>GCS 7/15</li> <li>Pupils were reactive to the light</li> </ul>	VBG: (ph: 7.37, Na 116 mmol/L, K 2.6 mmol/L, lactate incalculable, HCO <sub>3</sub> 22 mmol/L)	<ul style="list-style-type: none"> <li>Monitoring was continued</li> </ul>
2 hour after ED presentation until admission	<ul style="list-style-type: none"> <li>Desaturation on room air, fluctuating O<sub>2</sub> saturation (75-87%)</li> </ul>	<p>Chest X-ray: Multifocal airspace opacity in right lung</p> <p>CT brain: Unremarkable</p> <p>The presentation labs resulted:</p> <p>CBC: (WBCs 19.9 10<sup>3</sup>/uL, absolute neutrophils 17.5 10<sup>3</sup>/uL, RBCs 3.58 10<sup>6</sup>/uL, hemoglobin 11.3 g/dl, platelet 210 10<sup>3</sup>/uL)</p> <p>Renal function test: (Na: 110 mmol/L, k: 3.1 mmol/L, corrected calcium 1.88 mmol/L, phosphorus 0.42 mmol/L, magnesium 0.45 mmol/L, creatinine 26 micro-mol/L, urea 2.5 mmol/L)</p>	<ul style="list-style-type: none"> <li>Nasal cannula</li> <li>Simple face mask</li> <li>Non rebreather</li> <li>High flow nasal cannula</li> <li>Electrolyte was sent every 8 hours and corrected accordingly</li> <li>VBG and ECG was sent every 2 hours</li> <li>Piperacillin-tazobactam 4.5 mg IV started for the patient</li> <li>Internal medicine consultation</li> <li>Psychiatric consultation</li> </ul>
Day 1 after ED presentation	<ul style="list-style-type: none"> <li>GCS 15/15</li> <li>No seizure attack</li> <li>Neurological examination intact</li> <li>Abdomen is soft and lax</li> <li>Chest: Crepitation over the left lung</li> <li>Cardiovascular: Normal S1, S2, no added sound</li> </ul>	<p>CBC: (WBCs 10.9 10<sup>3</sup>/uL, absolute neutrophils 10.1 10<sup>3</sup>/uL, RBCs 2.13 10<sup>6</sup>/uL, hemoglobin 7.4 g/dl, platelet 94 10<sup>3</sup>/uL)</p> <p>Renal function test: (Na: 122 mmol/L, k: 4.6 mmol/L, corrected calcium 2.08 mmol/L, phosphorus 0.73 mmol/L, magnesium 0.36 mmol/L, creatinine 32 micromol/L, urea 1.4 mmol/L)</p> <p>plasma osmolality 255 mOsm/kg</p>	<ul style="list-style-type: none"> <li>Free water 200 ml orally every 6 hours</li> <li>High flow nasal cannula was weaned</li> <li>Piperacillin-tazobactam continued</li> <li>Chest X-ray repeated (the result was similar to the previous)</li> </ul>

Day 2 after ED presentation	<ul style="list-style-type: none"> <li>• Patient was doing well, oriented to place Time and person.</li> <li>• GCS 15/15</li> <li>• Patient was clinically and Vitally stable</li> </ul>	<p>CBC: (WBCs 7.63 10<sup>3</sup>/uL, absolute neutrophils 6.02 10<sup>3</sup>/uL, RBCs 4.07 10<sup>6</sup>/uL, hemoglobin 12.7 g/dl, platelet 192 10<sup>3</sup>/uL)</p> <p>Renal function test: (Na: 140 mmol/L, k: 4.1 mmol/L, corrected calcium 2.07 mmol/L, phosphorus 0.53 mmol/L, magnesium 1.03 mmol/L, creatinine 62 micro-mol/L, urea 2.1 mmol/L)</p> <p>plasma osmolality 295 mOsm/kg</p> <p>Chest X-ray: Interval improvement in aeration in both lungs</p>	<ul style="list-style-type: none"> <li>• Electrolyte corrected</li> <li>• Piperacillin-tazobactam continued</li> </ul>
Day 3 after ED presentation	<ul style="list-style-type: none"> <li>• Patient was doing well, oriented to place and person.</li> <li>• GCS 15/15.</li> <li>• Patient was clinically and vitally stable.</li> </ul>	<p>CBC: (WBCs 5.67 10<sup>3</sup>/uL, absolute neutrophils 3.25 10<sup>3</sup>/uL, RBCs 3.74 10<sup>6</sup>/uL, hemoglobin 11.6 g/dl, platelet 179 10<sup>3</sup>/uL)</p> <p>Renal function test: (Na: 143 mmol/L, k: 4.3 mmol/L, corrected calcium 2.07 mmol/L, phosphorus 0.77 mmol/L, magnesium 0.73 mmol/L, creatinine 39 micro-mol/L urea 2.8 mmol/L)</p> <p>plasma osmolality 311 mOsm/kg</p>	<ul style="list-style-type: none"> <li>• Patient discharged with good neurological outcome</li> <li>• Discharged on augmentin for 7 days</li> </ul>

impact health preservation and contribute to mental well-being, although maintaining a balance is crucial [7].

Secondly, the case emphasizes the potential dangers of excessive fluid intake, particularly in the context of religious rituals. The patient's consumption of 19 liters of water over a span of four hours led to severe acute hyponatremia, precipitating life-threatening complications such as seizures and altered mental status. This highlights the critical need for patient education regarding the risks of water intoxication and the importance of moderation, even within the context of religious practices. While a moderate daily water intake of 1.5-2 liters is often recommended for individuals with regular renal and endocrine function, exceeding the body's capacity to excrete water can result in "water intoxication," a rare but serious condition [8,9]. Water intoxication is a well-recognized cause of symptomatic hyponatremia which can be complicated by severe life-threatening and neurological consequences [10].

Furthermore, the case underscores the importance of prompt recognition and management of medical emergencies. The rapid onset of severe hyponatremia necessitated immediate intervention to stabilize the patient and prevent further deterioration. Interdisciplinary collaboration among emergency medical services, emergency department staff, and specialists in internal medicine and intensive care was essential in delivering appropriate care.

In this case, the patient believed in this religious ritual so-called "Ruqayyah" and followed "shaikh" orders until

developing severe symptomatic hyponatremia with significant complications (status epilepticus).

All patients with severe symptomatic hyponatremia should receive treatment in a hospital setting with close monitoring due to the heightened risk of complications [11-13]. Seizures in patients with severe hyponatremia should be promptly managed according to standard seizure protocols, with antiepileptic medications administered parenterally. Hypoxic episodes resulting from seizures are well-known risk factors for osmotic demyelination and neurological damage due to exacerbation of cerebral edema [14].

The primary treatment for symptomatic hyponatremia is 3% hypertonic saline, with various administration rates recommended. According to US guidelines, a bolus of 100 mL over 10 minutes, repeated up to three times as needed for severe symptoms, is advised. For moderate symptoms, a rate of 0.5 to 2 mL/kg per hour is suggested. European guidelines propose a bolus of 150 mL over 20 minutes, with 2-3 repeated doses for severe symptoms, and a single bolus of 150 mL over 20 minutes for moderate symptoms [15,16].

8.4% sodium bicarbonate may provide a solution to the logistical problem of 3% hypertonic saline and can be used as an alternative treatment for hyponatremia. While 100-150 mL boluses of 3% sodium chloride are commonly used for severe symptomatic hyponatremia. As sodium bicarbonate, available in 8.4% ampoules, can also be utilized to raise serum sodium levels [17].



An ampule of sodium bicarbonate 8.4% (50 mL) contains 50 mEq of sodium, comparable to 51.3 mEq of sodium found in 100 mL of 3% sodium chloride [18,19]. The dose of sodium bicarbonate needed is empirical and can be unpredictable; initially, 150 mEq [17]. However, the use of isotonic saline remains a primary choice for treating symptomatic or severe hyponatremia.

To avoid unwanted adverse effects of the treatment which include osmotic demyelination syndrome (ODS), This can be accomplished by knowing the chronicity of hyponatremia and weighing the risks and benefits [20].

The American Expert Panel Recommendations endorse a serum sodium (SNa) correction limit of 10-12 mEq/L in any 24-hour and 18 mEq/L in any 48-hour period for patients at average risk of ODS, and 8 mEq/L in any 24-hour period for patients at high risk of ODS [15]. In The European Clinical Practice Guidelines recommended that correction of hyponatremia be limited to 10 mmol/L in the first day and 8 mmol/L for every subsequent day thereafter [16]. Some investigators have proposed an even more conservative limit of 6 to 8 mmol/L per day [21].

Basis of what we know today, correction of a sodium  $\leq 120$  mmol/L by  $> 10$  mmol/L within 24 hours or by  $> 18$  mmol/L within 48 hours should be avoided [20].

In one case series by Ayus, et al. (2015), a correction up to 12 to 14 mmol/L in the first 24 to 48h was found to be safe without evidence of ODS [22]. However, certain patient conditions may increase the risk of developing ODS, including serum sodium levels  $< 105$  mmol/L, hypokalemia, alcoholism, malnutrition, and advanced liver disease [23,24]. For patients at high risk of ODS, a limit up to 8 mmol/L is advocated by the US guidelines [25].

Current recommendations suggest a target rise in serum sodium concentration in patients with chronic hyponatremia (More than 48 hours) stratified by the risk of developing ODS. They suggest a target maximum rise of 4-8 mmol/L per day in patients with low risk of ODS, with a target maximum limit not to exceed 10-12 mmol/L in any 24 hours or 18 mmol/L in any 48 hours. For those at high risk of ODS, they suggest a lower maximum target rise of 4-6 mmol/L per day, with a maximum target limit not to exceed 8 mmol/L in any 24-hour period. Factors that place a patient at high risk of developing ODS with correction of chronic hyponatremia include starting serum sodium concentration  $\leq 105$  mmol/L, hypokalaemia, alcoholism, malnutrition, and advanced liver disease [15].

In true acute hyponatremia (where the decrease in plasma sodium has been documented to be in the prior 24-48 hours), the rate of correction need not be restricted as tightly as in chronic hyponatremia as there is a lower risk of osmotic demyelination. However, if there is any uncertainty as to the rapidity of onset of

hyponatraemia (chronic versus acute), then the target limits for correction of chronic hyponatremia should be adhered to [26].

In 2023 a large multicenter cohort study on 22,858 patients with hyponatremia was conducted, showing that rapid correction of serum sodium is common ( $n = 3632$  [17.7%]), but ODS was rare ( $n = 12$  [0.05%]). with a conclusion of "future studies with a higher number of patients with ODS are needed to understand potential causal factors for ODS" efficiently [27].

In this study, the patient's sodium levels increased by 13 mmol/L within the first 24 hours, no additional sodium products were given to the patient after the initial treatment in the emergency department. The patient was discharged with a favorable neurological outcome.

Additionally, the case prompts consideration of long-term outcomes and follow-up care. While the patient demonstrated neurological recovery and normalization of electrolyte levels during the hospitalization, ongoing monitoring for potential long-term sequelae of acute hyponatremia is imperative. Long-term follow-up and neurological assessment are essential to ensure optimal recovery and detect any lingering effects of medical emergency.

## Conclusion

Acute hyponatremia can be life threatening leading to significant neurological symptoms and complications if not promptly addressed. Identifying hyponatremic cases in the emergency department is crucial while dealing with them depends on the severity of symptoms and the underlying causes. Although hyponatremia in mentally competent patients is a rare yet serious complication that should not be neglected. Hence, education and awareness of symptoms must be considered.

The case highlights the importance of cultural competence and sensitivity in healthcare delivery. Understanding the religious and cultural practices of diverse patient populations is essential to provide appropriate care and to avoid inadvertent harm. Healthcare providers must be aware of the potential risks associated with certain rituals and be prepared to address any medical emergencies that may arise.

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