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CASE REPORT

# Symptomatic Hypotonic Hyponatremia Presenting at High Altitude

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We present a case of altered mental status and seizure that occurred at an altitude known to cause high altitude-related illnesses. Based on the presenting symptoms, the patient was initially transferred to the hospital with a presumptive diagnosis of high altitude cerebral edema. On review of imaging and laboratory data, she was found to be experiencing symptomatic hypotonic hyponatremia. This case presented an interesting diagnostic challenge and underscores the importance of maintaining a broad differential diagnosis when evaluating a patient with altered mental status from an alpine setting.

*Key words:* hyponatremia, exertional associated hyponatremia (EAH), syndrome of inappropriate antidiuretic hormone (SIADH), hypertonic saline (HTS), high altitude cerebral edema, search and rescue

## Introduction

Recreation in our national parks is an increasingly popular way for people to spend their free time and experience the natural beauty of our nation.[1] Unfortunately, traumatic injuries, environmental illness, and exacerbation of underlying medical conditions are not uncommon in wilderness settings. Although trauma-related complaints comprise the majority of presenting illness in mountainous wilderness settings, altered mental status and seizure may occur at high altitude,[2] and individual case fatality for medical complaints including altitude illness is disproportionally high.[3] Providers who care for these patients need to consider the spectrum of disease that is encompassed by wilderness and environmental medicine, although not excluding uncommon presentations of common conditions.

## Case Report

### PREHOSPITAL CLINICAL HISTORY

A previously healthy 29-year-old woman presented to our medical center after a 3-day hiking and camping trip in the Sierra Nevada Mountains of central California in midsummer. Roughly 48 hours before her presentation at the hospital, the patient drove from sea level to an altitude of 2400 m (7800 feet). Once at this altitude she participated in multiple strenuous hikes above 2500 m (8000 feet). In the 24 hours immediately preceding her presentation at the hospital, the patient started to complain of a headache, became progressively weaker, and began to experience nausea, vomiting, and diarrhea. She had 5 episodes of emesis. By midday she required assistance ambulating back to her camp as a result of fatigue and dizziness. Although it is unclear whether she experienced ataxia, she was noted to have fallen from a height of 0.6 m (2 feet) while attempting to squat to urinate. Once back at camp she rested in her tent and continued to be anorexic and progressively lethargic.

At 2100 hours she experienced a witnessed seizure with tonic-clonic activity followed by a sustained state of altered mental status while resting in her tent. A cell phone call reached the National Park authorities, and Parkmedics were dispatched to the scene. By the time of arrival at 0215 hours, Parkmedics found the patient obtunded and vomiting.

Parkmedic assessment at the scene demonstrated a patient who was not protecting her airway owing to a decreased mental status and who was at high risk of aspiration of emesis. She responded to pain with eye opening, verbal groans, and localizing to pain (E2V2M5 = 9), representing a Glasgow Coma Score (GCS) of 9. Her airway was secured in the field with the placement of a King LT-D, a sterile, single-use supraglottic airway commonly known as a "Kingtube." Her respiratory rate before securing her airway was even and unlabored at a normal rate. Her breath sounds were equal without rales.

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Initial heart rate was 78 beats/min and blood pressure was 110/60 mm Hg. Her finger-stick glucose reading was 169 mg/dL. Pupils were equal at 4 mm and reactive to light, with gross neurological examination showing movement of all 4 extremities to pain and an absence of facial droop. Limited skin examination was remarkable for the absence of rash, skin mottling, or external signs of trauma, and she was noted to be pink, warm, and dry.

According to her traveling companions, she had a history of gradual-onset malaise and flulike symptoms with ascent above 2500 m (8000 feet) and a chief complaint of headache unresponsive to aspirin and ibuprofen self-administered 3 times (1300, 1500, 1700 hours) before seizure onset at 2100 hours. She had no known preexisting medical conditions or medication use. She did not have a history of alcohol or substance abuse.

The Parkmedic established Base Hospital contact with the Medical Control for Sequoia Kings Canyon National Park (SEKI) through the park's dispatch at 0320 hours. Orders from the Base Hospital Physician included administering 1 L normal saline bolus, 4 mg of ondansetron intravenously (IV), serial neurological examinations, and a recommendation to litter-carry the patient to an elevation below 2500 m (8000 feet). The exact elevation of the patient was only known at that time to be over 2500 m (8000 feet), and the precise altitude was not known. The backcountry evacuation to a suitable landing zone by litter-carry was estimated to take approximately 3 hours. Immediate transport by air ambulance was precluded by the darkness of night. The litter team rendezvoused with flight paramedics at the trailhead at 0730 hours, and the patient was evacuated by helicopter to Community Regional Medical Center, which is at an elevation of 216 m (415 feet).

## HOSPITAL PRESENTATION

The air ambulance landed at 0845 hours with the patient. En route, the patient's supraglottic airway adjunct had been exchanged for an endotracheal tube using rapid-sequence intubation by the prehospital flight nursing crew. On arrival to the hospital, the patient's vital signs were stable with a heart rate of 91 beats/min and blood pressure of 123/78 mm Hg, and her finger-stick glucose reading remained normal at 133 mg/dL. She was mechanically ventilated with oxygen saturation at 100% on 60% inspired oxygen. Portable chest x-ray demonstrated patchy air space opacities in the left upper and lower lobes and right upper lobe. She was administered 500 mL normal saline IV and 10 mg of dexamethasone IV, and had imaging and laboratory studies initiated. The patient was admitted to the medical intensive care unit with a battery of laboratory and imaging tests pending.

Among the initial tests conducted were a computed topography (CT) scan of the head and serum blood work including chemistry panel, liver function studies, complete blood count, and urinalysis. Further testing in the emergency department included a magnetic resonance image (MRI) of the brain and lumbar puncture. Intravenous saline at 100 mL/h was included as part of the patient's initial treatment. Additionally, early consultations with the neurology and neurosurgical services were requested, and an electroencephalogram (EEG) was performed several hours after her initial hospital arrival. The emergency medicine and intensivist teams considered a broad differential including meningitis, encephalitis, electrolyte abnormalities, accidental or intentional toxidrome, Addisonian crisis, myxedema coma, primary epilepsy, nonconvulsive status epilepticus, spontaneous intracranial hemorrhage, acute mountain sickness, and high altitude cerebral edema. The initial clinical impression was high altitude cerebral edema.

The head CT suggested mild edema of the basilar structures. The initial blood work revealed a mild leukocytosis at $13.4 \times 10^3/\mu L$, ketones in the urine, and a hypotonic hyponatremia with initial serum sodium at 122 mEq/L and osmolality at 268 mOsm/kg. Blood urea nitrogen (BUN) level was 6 mg/dL (normal range, 6–20 mg/dL) and initial creatinine was 0.5 mg/dL (normal range, 0.5–1.1 mg/dL). Random urine sodium several hours after arrival was elevated at 13 mmol/L (maximally dilute level $<5$ mmol/L), and measured urine osmolality was 65 mOsm/kg (normal range, 35–1400 mOsm/kg). Liver function studies were normal. The brain MRI, including fluid-attenuated inversion recovery (FLAIR) images, did not show any signs of cerebral edema or other disease. The EEG revealed mild slowing without seizure activity. The results of the lumbar puncture, including opening pressure, were normal. A repeat chemistry panel drawn 6 hours after admission revealed the serum sodium had increased to 131 mEq/L.

During the next 48 hours the serum sodium was closely monitored, and the patient was treated with alternating IV normal saline and 5% dextrose in water to increase her serum sodium by 10 mEq/L in each 24-hour period. After 48 hours in the medical intensive care unit, the patient was successfully extubated without complication, and by the morning of the third hospital day her serum sodium was 141 mEq/L. She was alert and oriented without any neurological deficit. She was discharged into the care of her family. The final diagnosis was symptomatic hypotonic hyponatremia.

## Discussion

This case brings up several areas of discussion pertinent to those directing prehospital care, receiving and