



Hyponatremia Caused by Polydipsia

Lyne Chamberlain, RN, MSN, CNS, CCRN-CMC, CCNS

Hyponatremia (serum sodium <135 mEq/L) is the most common electrolyte disorder. The severity of symptoms is related to how rapidly the condition develops and the degree of cerebral edema that results from the low serum level of sodium. Hypertonic saline and the new vasopressin receptor antagonists are highly effective treatments for severe symptomatic hyponatremia, yet they can result in severe neurological complications if sodium levels are restored too quickly. Hyponatremia is classified as hypovolemic, euvolemic, and hypervolemic. Treatments include administration of high-risk medications and fluid restriction to restore fluid and electrolyte balance and relieve cerebral effects. Nursing care to ensure safe outcomes involves multidisciplinary collaboration, close monitoring of serum sodium levels and intake and output, and assessment for neurological changes. (*Critical Care Nurse*. 2012;32[3]:e11-e20)

H yponatremia (serum sodium level <135 mEq/L) is the most common electrolyte disorder in hospitalized patients.¹ In a study² of 120 000 patients, the prevalence of hyponatremia was 42%, and more than 25% of the patients had the abnormality at the time of admission to the hospital. The severity of neurological symptoms due to sodium deficiencies is related to the degree of cerebral edema caused by electrolyte and fluid imbalances and is correlated with the rate at which hyponatremia develops² and the extent of the hyponatremia. Most patients do not have signs or symptoms unless the serum sodium level is less than

129 mEq/L, when nausea, vomiting, fatigue, irritability, disorientation, and headache may develop,³ although elderly patients with even mild hyponatremia may have cognitive problems and experience unsteadiness and/or falls.² Elderly patients with hyponatremia also have prolonged reaction times and significantly more mental errors than do patients of a similar age who have normal serum sodium levels.⁴ A serum sodium level less than 125 mEq/L is a strong independent predictor of inpatient mortality for critical care patients.⁵ Critical care nurses must monitor electrolytes and take prompt actions to avoid marked decreases in sodium levels.

The standard treatments for hyponatremia are often not well

tolerated and are potentially dangerous (see Figure). Hyponatremia creates relative hypo-osmolality, resulting in an osmolar gradient across the membranes of cells of the blood-brain barrier and other tissues. This gradient causes water to move into the cells, resulting in cellular overhydration.⁷ If sodium levels are adjusted too quickly, an irreversible neurological catastrophe can occur. Osmotic demyelination syndrome (ODS), also termed osmotic myelinolysis and formerly termed central pontine myelinolysis, and/or other neurological problems may develop because of rapid osmotic shifts of water out of the brain cells when sodium levels increase too quickly, and the damage might not be apparent for at least 4 days.⁹ ODS is due to a loss of oligodendrocytes and myelin in central and extrapontine sites; it rarely occurs if the serum sodium level remains greater than 120 mEq/L and when the onset of hyponatremia is rapid.³

Classifications of Hyponatremia

Hyponatremia is classified first as acute (<48 hours) or chronic (at least 48 hours) and then according to the severity of signs and symptoms and fluid balance (Table 1).

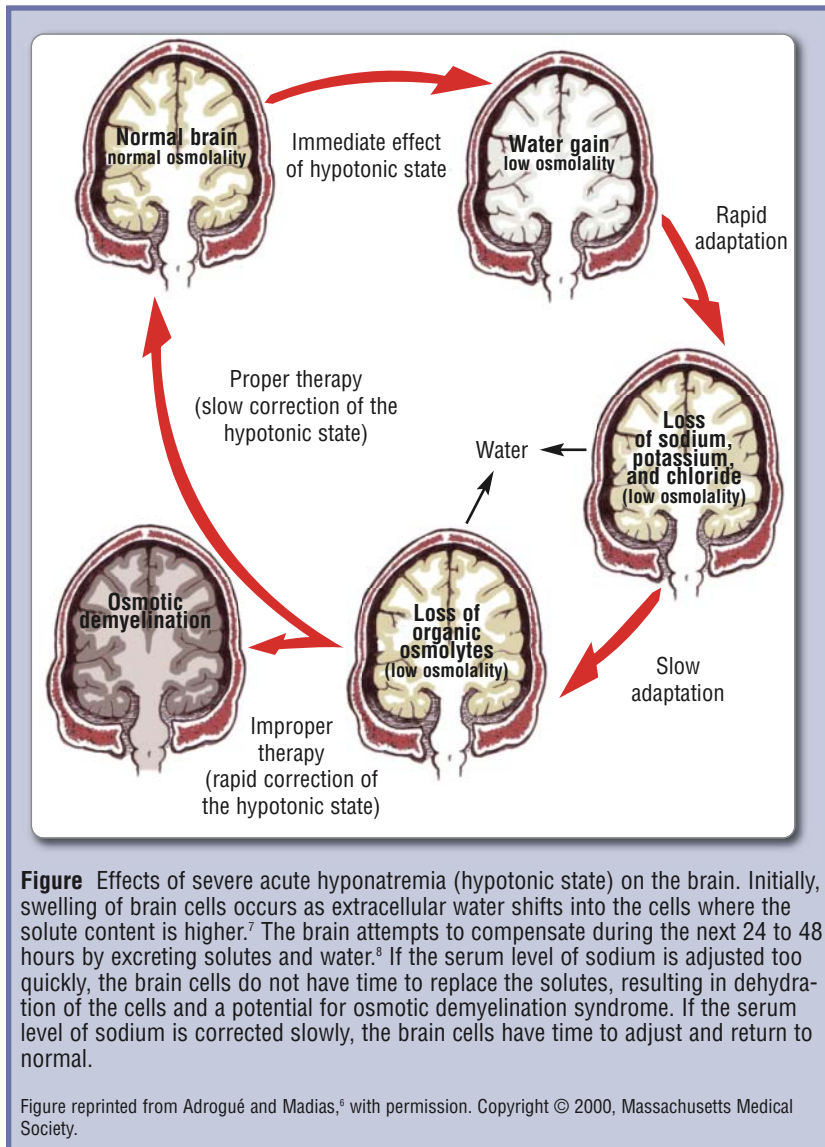


Figure Effects of severe acute hyponatremia (hypotonic state) on the brain. Initially, swelling of brain cells occurs as extracellular water shifts into the cells where the solute content is higher.⁷ The brain attempts to compensate during the next 24 to 48 hours by excreting solutes and water.⁸ If the serum level of sodium is adjusted too quickly, the brain cells do not have time to replace the solutes, resulting in dehydration of the cells and a potential for osmotic demyelination syndrome. If the serum level of sodium is corrected slowly, the brain cells have time to adjust and return to normal.

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Fluid balance, determined by serum osmolality and physical examination of edema, is categorized as hypovolemic, euvoletic, or hypervolemic.¹⁰

Among patients with hyponatremia, 50% have no physical indications of fluid overload, and the

hyponatremia is classified as euvoletic²; syndrome of inappropriate antidiuretic hormone secretion (SIADH) is the most common cause of euvoletic hyponatremia in critical care patients.² SIADH occurs in response to multiple causes that

result in excess release of vasopressin (antidiuretic hormone or arginine vasopressin) from the posterior part of the pituitary gland. The released antidiuretic hormone adheres to renal receptors, causing water to be reabsorbed by the kidneys and leading to increased intravascular volume.¹¹ The excess volume causes a dilution of sodium and a hypo-osmolar state.⁷ Natriuresis (renal sodium excretion) occurs with SIADH, even in the presence of hyponatremia, and this excretion prevents the formation of edema. Urine osmolality is greater than 100 mOsm/kg in SIADH, indicating an inability of the kidneys to maximally dilute urine to correct hyponatremia.

Hypovolemic hyponatremia is the result of fluid shifts that create intravascular dehydration, such as the shift associated with severe hyperglycemia.¹⁰ Elevated glucose levels in diabetic ketoacidosis or a hyperosmolar hyperglycemic state dilute the serum level of sodium via an osmotic effect¹¹ (Table 2 gives the formula for calculating the actual sodium level for patients with diabetic ketoacidosis or hyperosmolar hyperglycemic state). Other common causes of hypovolemic hyponatremia are use of diuretics, diarrhea, excessive diaphoresis,⁹ and cerebral salt wasting.¹ Cerebral salt wasting, which can occur after a neurological injury, stimulates vasopressin secretion, leading to hyponatremia. Cerebral salt wasting may be mistaken for SIADH, but in the former, release of vasopressin is stimulated by volume depletion.¹

Hypervolemic hyponatremia, the type of most concern in critical care, is the result of excess fluid causing sodium dilution¹⁰; about 25% of

Author

Lyne Chamberlain is a clinical nurse specialist in cardiology at Dr P. Phillips Hospital, Orlando, Florida.

Corresponding author: Lyne Chamberlain, RN, MSN, CNS, CCRN-CMC, CCNS, 9400 Turkey Lake Road, FL 32819 (e-mail: lyne.chamberlain@orlandohealth.com).

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Table 1 Hyponatremia (serum level of sodium <135 mEq/L): classifications, causes, and treatments^a

Characteristic	Dilutional		
	Hypervolemic	Euvolemic	Hypovolemic
% of patients	~25	~50	~25
Total body water	Very high	High	Low
Serum level of sodium	High	Normal	Very low
Signs	Peripheral edema, ascites in patients with heart failure, liver disease, renal disease, or jugular venous distention	No edema	Hypotension, tachycardia
Plasma osmolality, mOsm/kg	<280	<280	≥280
Urine osmolality, mOsm/L	>100	>100	>500
Urine sodium level, mEq/L	<20 or >20	>20	<10 or >20
Common causes	>20 mEq/L urinary sodium: acute or chronic renal failure, fluid overload, psychogenic polydipsia; <20 mEq/L urinary sodium: heart failure, cirrhosis, or nephritic syndrome	Syndrome of inappropriate antidiuretic hormone secretion, adrenal insufficiency, hypothyroidism	<10 mEq/L urinary sodium: extrarenal loss such as diarrhea, vomiting, diuretics, severe hyperglycemia; >20 mEq/L urinary sodium: renal sodium loss such as cerebral salt wasting or diuretics
Usual treatments	3% saline, diuretics with caution, vasopressin receptor blockers		Fluid restriction, 0.9% saline, discontinuation of diuretics, electrolyte replacement

^a Acute cases are <48 hours; chronic cases are 48 hours or longer.

Table 2 Calculation of actual serum level of sodium in patients with hyperglycemia^a

For every 100 mg/dL of glucose >100 mg/dL, add 1.6 mEq/L to serum level of sodium.

For example, if serum level of glucose is 800 mg/dL and serum level of sodium is 128 mEq/L:

$$800 \text{ mg/dL} - 100 = 700 \text{ mg/dL}$$

$$\frac{700 \text{ mg/dL}}{100} = 7$$

$$7 \times 1.6 \text{ mEq/L} = 11.2 \text{ mEq/L}$$

$$128 \text{ mEq/L serum sodium} + 11.2 \text{ mEq/L corrective factor} = 139.2 \text{ mEq/L corrected serum level of sodium}$$

^a Based on information from Goh.¹² To convert glucose level to millimoles per liter, multiply by 0.0555.

patients with hyponatremia have the hypervolemic type.² Hypervolemic hyponatremia can be differentiated from euvolemic hyponatremia on the basis of physical signs of fluid overload, such as jugular venous distention, pulmonary edema, and/or

pitting edema. The most common cause of hypervolemic hyponatremia is increased reabsorption of sodium by proximal nephrons³ (as in heart failure, hypothyroidism, nephritic syndrome, and cirrhosis). Another cause of dilutional hyponatremia is

primary polydipsia, or water intoxication due to excessive fluid intake, a common condition in patients with schizophrenia.¹ In psychogenic polydipsia, the volume of fluid intake may overwhelm normal renal functioning, creating a dilutional hyponatremia that often self-corrects with diuresis.¹ Psychogenic polydipsia has an incidence of 4% to 20% in psychiatric patients.¹³ The actual mechanism is unknown, but one possibility is a malfunction of the thirst center in the hypothalamus and dysregulation of secretion of antidiuretic hormone due to a change in the hypothalamic-pituitary feedback loop.¹⁴ Many psychiatric medications cause xerostomia (dry mouth),¹⁵ which may also promote polydipsia.¹³

Several medications influence sodium excretion: diuretics, carbamazepine and other antiseizure medications, amiodarone, theophylline, selective serotonin reuptake inhibitors (antidepressants), vasopressin, and phenothiazine antipsychotics.¹⁶ Diuretics can result in hypovolemia, but most of the other medications that affect sodium excretion may cause SIADH, which can be reversed by discontinuing the medication.¹⁶

Treatment Goals

The initial goals in treatment of symptomatic hyponatremia are to reduce the cellular edema in the brain and improve the serum sodium concentration.¹¹ However, in order to prevent ODS, sodium concentrations should be adjusted only to the level required to maintain normal respiration and prevent seizures.¹¹ Treatment of hyponatremia is based on volume status, severity of neurological signs and symptoms (correlated with the degree of hyponatremia), onset of the hyponatremia (acute vs chronic), and consideration of the risks and benefits of the specific treatments (Table 1). Patients are at risk for ODS if the sodium concentration is corrected too quickly, especially patients with chronic hyponatremia. Sodium levels should be monitored every 2 to 4 hours until signs and symptoms abate and then every 4 to 8 hours to ensure that the serum sodium level is not adjusted too quickly.⁹

Traditional treatment options include fluid restriction (usually <1000 mL/d),¹ infusion of isotonic (0.9%) or hypertonic saline, medications to reduce fluid retention, and—for patients with hypovolemia—

discontinuation of diuretics.⁹ Hypertonic saline promotes osmosis of fluid from intracellular spaces into the vasculature so that the kidneys can excrete the excess fluid as urine. Isotonic saline suppresses vasopressin, a situation that permits rapid excretion of excess water, for most cases of hyponatremia that are not related to SIADH. A new drug class, vasopressin receptor antagonists (vaptans), now offers an alternative treatment for euvolemic and hypervolemic hyponatremia that directly targets excess arginine vasopressin, which is most often the reason for water disequilibrium¹⁰ and hyponatremia in hospitalized patients (see Sidebar, “Vasopressin Receptor Blockers”).

The hyponatremia treatment guidelines¹ published in 2007 suggest that chronic hyponatremia should be corrected to less than 10 to 12 mEq/L over 24 hours and to less than 18 mEq/L over 48 hours to prevent neurological complications but that the goal for the first 24 hours should be 8 mEq/L to allow for absolute safety. Severe symptomatic hyponatremia, generally an acute sodium level less than 120 mEq/L, is a medical emergency⁹ because the osmotic movement of water into the cells of the brain can cause seizures or coma.¹⁰ Hypertonic saline or a vasopressin receptor antagonist may be prescribed initially in these critical cases to increase sodium levels by 1 to 2 mEq/L per hour⁷ until the serum sodium is 125 mEq/L or signs and symptoms subside or until the serum sodium concentration has increased by 25 mEq/L in the first 48 hours of treatment.¹ A more rapid corrective rate of 3 to 5 mEq/L per hour can be used for up

to 2 hours if imminent brainstem herniation is suspected.¹⁰ Loop diuretics can be given concurrently with hypertonic saline for patients with hypervolemia,¹⁰ but the combination must be used cautiously to avoid a too rapid reversal of the hyponatremia. Table 3 shows calculations for determining rates of administration for 3% saline.

Euvolemic hyponatremia, most commonly due to SIADH, is primarily treated with fluid restriction.⁹ Normal saline is not corrective in patients with euvolemic hyponatremia, so hypertonic saline or vaptans are necessary if more aggressive treatment is needed.¹ The goal of treatment is to maintain fluid intake of 500 mL less than urine output, but fluid restriction is difficult in critical care patients because of nutrition and treatment needs, and patients often do not comply with the restriction in other settings.²³ Standard treatment of hypovolemic hyponatremia includes fluid restriction, electrolyte replacement, and discontinuation of diuretics. Ideally, fluid balance and sodium deficits are replaced simultaneously, with the primary focus on fluid for the first 48 hours and then slow sodium replacement in the next 48 hours.⁹ An increase of 0.5 mEq/L per hour in the serum level of sodium may be sufficient unless signs and symptoms are severe. In severe cases, a higher rate may be targeted until signs and symptoms resolve or the sodium level is greater than 120 mEq/L.⁹ Vaptans are contraindicated in hypovolemia because they cause profound renal aquaresis.¹⁸

Hypervolemic hyponatremia requires a reduction of fluid volume. Hypertonic saline and/or intravenous

Vasopressin Receptor Blockers

A new drug class, vasopressin receptor antagonists, now offers an alternative treatment for hyponatremia that directly targets excess arginine vasopressin (AVP), the most common reason for water disequilibrium¹⁰ and hyponatremia in hospitalized patients.

The renin-angiotensin-aldosterone system is the primary regulator of sodium metabolism, and AVP is the primary regulator of water metabolism¹⁰; the primary function of vasopressins is to regulate vascular tone and control water excretion from the kidneys.¹⁷ Despite the regulatory function of AVP, too much of the vasopressin can cause severe hyponatremia from fluid overload.⁵ AVP acts on membrane receptors in the vasculature (V_{1a} receptors), the collecting ducts of the kidneys (V_2 receptors), and the pituitary gland (V_3 receptors). Binding of AVP to V_{1a} receptors produces vasoconstriction, and possibly platelet aggregation and hypercoagulation.⁵ Binding to V_3 receptors stimulates release of β -endorphins and adrenocorticotropic hormones.⁵ The V_2 receptors and their antagonists are crucial components in hyponatremia and influence the treatment of this abnormality.⁹ The V_2 receptors mediate the antidiuretic effect of AVP to stimulate water reabsorption and retention.^{5,10} Without vasopressin, the collecting ducts remain impermeable to water reabsorption, and dilute urine is produced.¹⁷

Vasopressin receptor antagonists prevent the binding of AVP to the V_2 receptors. Blocking of the receptors results in aquaresis, so the kidneys excrete water and reabsorb sodium.¹⁸ Conivaptan (Vaprisol), the first vasopressin receptor antagonist to receive approval from the Food and Drug Administration for treatment of hypervolemic and euvolemic hyponatremia in the United States, was approved in 2005 for intravenous use only.¹⁹ Conivaptan must be administered in a hospital, and can be given for up to 4 days.²⁰ Tolvaptan (SAMSCA) was approved by the Food and Drug Administration in 2009; it is indicated for clinically severe euvolemic or hypervolemic hyponatremia (serum level of sodium ≤ 125 mEq/L) that has not responded to fluid restriction.²¹ Oral tolvaptan has been approved for the treatment of hospitalized patients with syndrome of inappropriate antidiuretic hormone secretion, heart failure, and cirrhosis,²¹ and a recent study indicated continued long-term efficacy for outpatient treatment.²⁰ Conivaptan binds to both V_{1a} and V_2 AVP receptors,²² whereas tolvaptan is selective for the V_2 receptor. Another AVP receptor antagonist, lixivaptan, has not yet been approved by the Food and Drug Administration for treatment of hyponatremia.

Several studies have indicated that the new vasopressin receptor antagonists are more effective than fluid restriction in improving serum sodium levels.²³⁻²⁶ However, none of the studies provided evidence that the high-cost vaptans are safer than traditional therapies. Conivaptan must be given intravenously in a large vein, and the injection site must be changed every 24 to 48 hours. Oral tolvaptan costs approximately \$250 per tablet.²⁰

Vaptans are strong inhibitors of CYP3A4, a substrate of P450 cytochromes, and therefore are contraindicated for concurrent administration with other CYP3A4 inhibitors to avoid drug interactions.¹⁹ A CYP3A4 inhibitor given with other CYP3A4 inhibitors may increase the concentration of conivaptan or the coadministered drug.¹⁹ Common CYP3A4 inhibitors include amiodarone, diltiazem, verapamil, macrolides, omeprazole, and antifungals,²⁷ which are often prescribed for patients who would be given vaptans (ie, critical care patients, elderly patients, and patients with heart failure or liver cirrhosis).

loop diuretics or vasopressin receptor blockers are effective. The new vaptans create aquaresis, in which water is excreted and electrolytes are retained. Renal damage, which may occur with loop diuretics, has not been detected with the vaptans. Currently, 2 vaptans have been approved by the Food and Drug Administration. Conivaptan (Vaprisol) is for intravenous administration only, and tolvaptan (SAMSCA) is for oral administration (see Sidebar for more information on vasopressin receptor blockers). With both vaptans, patients must be monitored because sodium levels can be corrected too quickly, creating the potential for ODS (see Sidebar, "Osmotic Demyelination Syndrome").

Nursing Care

The nursing care of patients with hyponatremia begins with an understanding of the relationship between sodium and fluid balance and the role of vasopressin, recognition of a patient's volume status, onset of the sodium deficit (acute or chronic), severity of signs and symptoms, and degree of the sodium deficit.

Critical care patients being treated for hyponatremia require diligent monitoring. Depending on the severity of the condition and the selected treatments, serum sodium levels should be measured every 1 to 2 hours during initial treatment and then at least every 4 hours until signs and symptoms resolve.²⁸ Levels should be measured every 1 to 2 hours while hypertonic saline is being infused.²⁰ The serum concentration of sodium, especially for patients with chronic

Table 3 Dosage calculation for 3% hypertonic saline^a

Determine sodium deficiency

Determine desired serum level of sodium—often 120 mEq/L
Subtract current serum level of sodium
Multiply by 0.6 for total body water per kilogram of body weight
Multiply by patient's weight in kilograms

For example:

120 mEq/L desired serum level of sodium
-112 mEq/L current serum level of sodium

8 mEq/L sodium deficiency
X 0.6 total body water/kg body weight

4.8 adjusted mEq/L sodium deficiency
X 60 kg

288 mEq needed to replace sodium deficiency

$288 \div 513^b = 0.561$ L of 3% saline or 561 mL

$\frac{561 \text{ mL}}{24 \text{ hours}} = 23 \text{ mL/h}$

Limit correction of serum level of sodium to <10 mEq in the first 24 hours.

Note: If patient has no urinary output, 1 mEq/L per hour of 3% saline will increase serum level of sodium by 1 mEq/L per hour.

Serum level of sodium must be monitored diligently; because the actual rate of change is highly variable because the hypertonic saline affects the amount of excretion of electrolyte-free water.

^a Based on information from Decaux and Soupart.²⁸

^b 3% saline has 513 mEq sodium per liter.

hyponatremia, must be adjusted slowly to prevent the development of ODS (Table 1). A key nursing responsibility is to ensure that laboratory tests are ordered, blood samples are obtained, and results of tests are reported in a timely manner. Results should be reported to the ordering physician per written parameters or hospital policy. Generally, the goal of serum sodium adjustment is for a maximum of 8 mEq/L, and definitely less than 12 mEq/L, in the first 24 hours of treatment, and less than 18 mEq/L in the first 48 hours. Strict intake and output are necessary because the kidneys excrete both water and electrolytes.

Table 3 shows the calculations to determine the sodium deficiency and dosage of hypertonic saline for severe euvolemic or hypervolemic hyponatremia. Hospitals may have

a policy to guide administration of hypertonic saline to ensure patient safety. Hypertonic saline should be administered cautiously, and safety features should be followed, such as using independent double verification and an intravenous pump that provides a drug library and limits for high-risk medications and tubing that prevents free flow of fluids.²⁹

Patients who are symptomatic have neurological signs and symptoms of brain cellular edema, so assessments should include determining the progress of the neurological condition, such as level of consciousness, mental status, and reflexes (Table 4). Safety precautions are important. Severe hyponatremia can lead to seizures or coma, so side rails may need to be padded, and nurses should be prepared to provide suctioning and airway support.

Patients who are ambulatory may be weaker than normal, and precautions should be taken to prevent falls.

Oral care is important because patients are likely to have a dry mouth, exacerbated by fluid restrictions. Fluid restriction may be difficult when critical care patients receive their nutrition and medications in liquid forms. Collaboration with nutritionists and pharmacists can help maximize concentrations of medications and limit the volume of tube feedings or intravenous fluids, although this intervention may require a central catheter to accommodate the more concentrated solutions and to reduce the risk of infiltration.

Case Study

Abby (a pseudonym) was a 40-year-old man with paranoid schizophrenia who lived in a group home. He came to the emergency department at 10:30 AM because he was “getting a kidney stone.” Vital signs were as follows: body temperature 37°C (98.6°F), pulse rate 104/min, respirations 16/min, blood pressure 140/88 mm Hg, and oxygen saturation 95% on room air. Abby weighed 142 kg (312 lb) and was 1.8 m (6 ft) tall. He appeared bloated and had some edema in his hands and ankles. His initial score on the Glasgow Coma Scale (GCS) was 15. He was placed in a room near the nurses’ station in the emergency department.

Abby was initially cooperative, but during a short period, he became increasingly labile, paranoid, and delusional. He paced in his room or sat on the bed and rapidly rocked back and forth. He refused to have laboratory tests and would not speak with the emergency department

Osmotic Demyelination Syndrome

Osmotic demyelination syndrome (ODS) is a rare and poorly understood neurological disaster that has been associated with rapid correction of serum levels of sodium in patients with hyponatremia but now has been diagnosed in patients with normal levels of sodium.⁸ Liver transplant recipients, patients with alcoholism, and patients who are malnourished are also at risk.⁸ The current notion is that ODS is related to the inability of the brain to counter rapid extracellular changes in osmolality, resulting in dehydration of brain cells and axonal damage in the pontine or extrapontine area.⁸ One idea is that the shrinkage of brain cells in an attempt to correct the balance between intracellular and extracellular fluid leads to axonal shear damage and to disruption of the blood-brain barrier, allowing inflammatory mediators into the central nervous system, where the mediators may cause demyelination.⁸ Signs and symptoms of ODS may include the following^{8,20}:

Changes in mental status	Rapid development of quadriparesis
Dysphagia	Dysarthria
Reduced attention span	Reduced speed of processing information
Loss of memory	Ataxia and Parkinson-like symptoms

ifested within a few days of treatment. Diagnosis is based on evidence of demyelination in the pontine and/or extrapontine areas on magnetic resonance images²⁰ and corresponding clinical neurological signs and symptoms. Currently, most patients with ODS are thought to be clinically asymptomatic.⁸ For patients who have signs and symptoms, recovery varies; some patients require lifelong custodial care and others experience marked improvement.

Table 4 Signs and symptoms of hyponatremia^a

- No signs or symptoms
- Difficulty concentrating
- Anorexia
- Headaches
- Irritability
- Restlessness
- Unstable gait and/or falls
- Nausea and/or vomiting
- Fatigue
- Disorientation and/or confusion
- Seizures
- Coma
- Respiratory arrest

^a Based on information from *MedlinePlus Medical Encyclopedia*.³⁰

staff. He refused to let a physician examine him. Abby was given ziprasidone hydrochloride (Geodon) 20 mg intramuscularly after striking at a nurse. Within moments of receiving the medication, Abby experienced a generalized seizure that lasted 3 minutes; during the seizure he was incontinent of urine and bit his tongue. He was given lorazepam (Ativan) 2 mg intravenously to prevent further seizures and to sedate him to allow computed tomography of the brain. Blood specimens for a chemical profile, complete blood cell count, and drug

screen were obtained immediately after the seizure. Table 5 gives the initial laboratory values.

Abby's GCS score decreased to 8 from the initial 15, and he vomited. He was intubated to protect his airway, and mechanical ventilation was started. A nasogastric tube was placed for low continuous suctioning. Once the laboratory results indicated hypervolemic hyponatremia (serum sodium level 115 mEq/L, urea nitrogen 52 mg/dL [to convert to millimoles per liter, multiply by 0.357], serum osmolality 238 mOsm/kg), an immediate nephrology consultation was requested to manage the electrolyte and fluid imbalances. An indwelling urinary catheter was inserted to monitor urinary output, and an arterial catheter was used to monitor blood pressure. A central vascular access device was placed, and central venous pressure was monitored for fluid balance. The nephrologist ordered an infusion of 3% saline at 30 mL/h, which was to continue until the serum concentration of sodium reached 125 mEq/L. The rate of 30 mL/h was based on the hospital's policy not to exceed an infusion rate of 30 to 40 mL/h for 3% hypertonic saline and a maximum increase of 8 mEq/L in the serum sodium level in the first 24 hours.

Upon transfer to the intensive care unit, Abby was agitated and thrashing in the bed. He localized painful stimuli, and he did not follow commands. His score on the Riker Sedation-Agitation Scale (SAS; Table 6) was 7, indicating a dangerous level of agitation.

Propofol (Diprivan) was administered at 5 µg/kg per minute. Vital signs remained stable. Pupils were

Table 5 Results of laboratory tests

Test	Initial results	Normal values
Sodium, mEq/L	115	136-144
Potassium, mmol/L	2.4	3.6-5.1
Chloride, mmol/L	92	101-111
Carbon dioxide, mmol/L	26	22-32
Urea nitrogen, mg/dL	52	8-20
Creatinine, mg/dL	1.0	0.4-1.0
Urea nitrogen to creatinine ratio	26	7.3-21.7
Glucose, mg/dL	97	5-100
Anion gap	5	2-12
Calcium, mg/dL	8.0	8.9-10.3
Serum osmolality, mOsm/kg	238	280-300
White blood cell count, $\times 10^3/\mu\text{L}$	8.1	4.1-10.4
Red blood cell count, $\times 10^6/\mu\text{L}$	4.5	3.5-5.0
Hemoglobin, g/dL	9.1	11.8-15.1
Hematocrit, %	27.3	34.0-44.0
Platelets, $\times 10^3/\mu\text{L}$	195	145-355
Urinary sodium, mOsm/L	28	10-20
Urine osmolality, mOsm/L	250	300-900

SI conversion factors: To convert urea nitrogen to millimoles per liter, multiply by 0.357; creatine to micromoles per liter, multiply by 76.25; glucose to millimoles per liter, multiply by 0.555; calcium to millimoles per liter, multiply by 0.25.

equal and reactive. Central venous pressure was 25 mm Hg, markedly greater than the normal 5 to 10 mm Hg. Breath sounds were clear. Abby had 2+ bilateral pedal edema.

During the first 24 hours of hospitalization, Abby excreted more than 10000 mL of urine. Although specimens for assays of serum sodium levels were obtained every 4 hours, 16 hours after the initial value was obtained, Abby's serum sodium level had increased to 130 mEq/L. The physician was notified immediately, and the hypertonic saline was replaced with normal saline 150 mL/h in an attempt to allow the sodium concentration to return to normal levels more slowly.

Throughout day 2 in the intensive care unit, the sodium levels continued

to increase, but at a much slower rate. Just 24 hours after admission, the serum sodium concentration was normal at 135 mEq/L. Abby's weight was down to 133 kg (293 lb),

a loss of 9 kg (20 lb). Attempts were made to minimize sedation to better assess neurological status, but Abby was agitated, with an SAS score of 6, and he required frequent doses of lorazepam and haloperidol (Haldol) and continued use of restraints simply to keep him in the bed. Ziprasidone hydrochloride was not given again because it can cause seizures. Unfortunately, Abby had no family or close friends who might have been able to calm him and allow a reduction in the number of medications. A psychiatric consultation offered no additional solutions. Abby's vital signs remained stable, and his hemodynamic values were normalizing. Abby was turned every 2 hours and given passive range-of-motion exercises. Chest radiographs showed that his lungs remained clear.

On day 3 in the intensive care unit, Abby was less agitated, with an SAS score of 5, and his neurological and respiratory conditions allowed extubation. After extubation, Abby's GCS score was 14. Slowly Abby improved during the next several hours until his GCS score returned to 15 and his SAS

Table 6 Riker Sedation-Agitation Scale^a

Score	Description	Examples
7	Dangerous agitation	Trying to get out of bed, pulling out tubes, thrashing
6	Very agitated	May require physical restraint, unable to calm with verbal instructions
5	Agitated	Mild agitation or anxiety but calms with verbal instructions
4	Calm and cooperative	Arouses easily and follows commands
3	Sedated	Difficult to arouse, but does arouse to verbal or physical stimuli, able to follow simple commands
2	Very sedated	Does not follow commands but arouses to physical stimulation
1	Unarousable	Little or no response to noxious stimuli

^a Based on information from Riker et al.³¹

remained at 5. Previous medical records indicated that Abby's baseline behavior matched his pacing his room and repeatedly asking the same questions with rapid speech. A nurse helped calm Abby by simply assuring him that he had space to pace without dislodging his intravenous catheter and sitting with him. By the end of the day 4, Abby was transferred to the progressive care unit. Eventually, he was able to tell us that he did not want to get another kidney stone, so he drank large amounts of water in a short time "to flush out my system." Thus, Abby's hyponatremia was the result of self-induced water intoxication (psychogenic polydipsia), and he had stopped taking his medications several months ago, a situation that led to the delusions about kidney stones. Often hyponatremia-induced psychogenic polydipsia self-corrects, but no history was initially available for Abby and the seizures and reduced level of consciousness required prompt treatment. Concerns continued about the possibility of neurological damage from the rapid improvement in the serum concentration of sodium because signs and symptoms may not appear for 4 or more days. Abby was monitored, and at the end of hospitalization day 6, he was transferred to the behavioral health unit for stabilization of antipsychotic medications and behavior. Several days

later, Abby was discharged back to his group home, still showing no deterioration in neurological status.

Discussion

Abby's treatment required the collaboration of nurses, specialty physicians, pharmacists, respiratory therapists, laboratory technologists, and chaplains. Abby was a challenging patient because his schizophrenic behavior both contributed to and delayed detection of the hyponatremia and the reason for the hypervolemic hyponatremia. Hyponatremia, with treatments dependent on a multitude of variables, is a complex condition fraught with potential life-threatening and/or disabling consequences. The rapid diuresis and resulting quick increase in Abby's serum sodium level placed him at risk for neurological complications such as ODS, although this syndrome is more common in patients with chronic hyponatremia. Nurses caring for patients with hyponatremia must monitor the patients' urine output and sodium levels and adhere rigorously to treatment orders to ensure a slow adjustment in the serum concentration of sodium and maximize the opportunity for a safe recovery. Once the hyponatremia was resolved, Abby's schizophrenic behaviors made it difficult to determine his mental status and neurological condition. For example, the health care staff could not tell whether Abby was choosing not to follow commands or that he did not understand the commands, a finding that might indicate neurological damage. Fortunately, Abby had no residual neurological problems. Once his psychiatric condition was stabilized with medications, he was able to return to his

group home, where his fluid intake would be monitored.

Conclusions

Hyponatremia is a common electrolyte disorder and has several causes. Nurses must recognize the degree of sodium deficiency, the fluid status, and whether the hyponatremia developed acutely or chronically because this information correlates with the severity of signs and symptoms and determines the best treatment options. Correction of hyponatremia requires collaboration of the entire health care team to ensure patient safety. The hyponatremia treatment guidelines¹ suggest that chronic hyponatremia be corrected to less than 10 to 12 mEq/L over 24 hours and less than 18 mEq/L over 48 hours to prevent neurological complications, but the goal for the first 24 hours should be 8 mEq/L to allow for absolute safety. Severe symptomatic hyponatremia, generally with an acute serum sodium level less than 120 mEq/L, is a medical emergency,⁹ because the osmotic movement of water into the cells of the brain can cause seizures or coma.¹⁰

Hypertonic saline, or intravenous vasopressin receptor blockers, when ordered, should be administered with meticulous care via a pump with safety features. Strict intake and output, careful administration of medications, neurological assessments, and assays of serum sodium levels every 1 to 4 hours are the basic components of care for patients with severe hyponatremia. **CCN**

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To learn more about hyponatremia, read "Central Neurogenic Diabetes Insipidus, Syndrome of Inappropriate Secretion of Antidiuretic Hormone, and Cerebral Salt-Wasting Syndrome in Traumatic Brain Injury" by John and Day in *Critical Care Nurse*, April 2012;32:e1-e7. Available at www.ccnonline.org.

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