ACCOMPANIED by her husband, Mrs. J, 64, arrives in the ED via ambulance. At the scene, her husband reported that he woke at 0430 to find his wife confused. Mrs. J was last seen in her usual state of health at 2300 the night before, when she said goodnight to her husband.

Mrs. J’s husband states that she was recently diagnosed with heart failure (HF) and started on enalapril, metoprolol, and furosemide. He adds that because the furosemide was causing urinary frequency, she stopped taking it several days ago. He also says he’s unsure of her adherence with the other prescribed medications and states that her last weight was 144.5 lb (65.7 kg) 2 days ago. Her weight today is 149.5 pounds (67.9 kg). He also shared concerns over her nonadherence to her prescribed low sodium diet.

Mrs. J is confused to place and time, and complaining of a frontal headache (5/0-10) and nausea. Her vital signs are as follows: oral temperature, 97.9° F (36.6° C); heart rate, 115 beats/minute and regular; respirations, 28 regular and slightly labored; Spo₂, 98% on supplemental oxygen at 2 L/min via nasal cannula; and BP supine, 102/58 mm Hg. She’s placed on continuous cardiac monitoring, which shows sinus tachycardia.

Physical assessment findings include 1+ bilateral lower extremity pitting edema, bibasilar inspiratory crackles, and bilateral neck vein distension at 30 degrees.

Her lab results include: random plasma glucose, 89 mg/dL (normal, less than 140 mg/dL); cardiac troponin T, <0.01 ng/mL (normal, <0.2 ng/mL); serum sodium, 126 mEq/L (normal, 135-145 mEq/L); serum osmolality, 265 mOsm/kg (normal, 280-300 mOsm/kg); urine osmolality, 115 mOsm/kg (normal, 300-900 mOsm/kg); and urine sodium 18 mEq/L (normal, >20 mEq/L).1-3

Mrs. J’s ECG demonstrates sinus tachycardia without signs of ischemia or infarction. The chest X-ray shows
A matter of volume

Hyponatremia can present as a hypovolemic, euvoletic, or hypervolemic state. Hyponatremia can also present as a hypotonic hyponatremia resulting from an osmotic shift of water from the intracellular fluid compartment to the extracellular fluid (ECF) compartment, such as occurs with hyperglycemia. In this situation, the sodium in the ECF becomes diluted as water moves out of body cells in response to the osmotic effects of the elevated blood glucose level.

- **Hypovolemic hypotonic hyponatremia** is the most common type of hyponatremia. It occurs when water is used to replace the loss of isosmotic body fluids. Among the causes of hypovolemic hyponatremia is excessive diaphoresis in hot weather, particularly during heavy exercise, which leads to loss of sodium and water. Hyponatremia develops when water, rather than electrolyte-containing liquids, is used to replace fluids lost in diaphoresis. Isosmotic fluid loss, such as occurs in vomiting or diarrhea, doesn’t usually lower serum sodium levels unless these losses are replaced with disproportionate amounts of orally ingested or parenterally administered water.

- **Euvolemic (normovolemic) hypotonic hyponatremia** represents retention of water with dilution of sodium while maintaining the effective circulatory volume within a normal range. It’s usually the result of SIADH. The risk of euvoletic hyponatremia is increased during the postoperative period when ADH levels are often high, producing increased water reabsorption by the kidneys. The hyponatremia becomes exaggerated if electrolyte-free fluids (such as 5% glucose in water) are used for I.V. fluid replacement.

- **Hypervolemic hypotonic hyponatremia**, as in Mrs. J’s case, occurs in edematous states such as decompensated heart failure, advanced liver disease, and renal disease. Although the total body sodium is increased in heart failure, the baroreceptors often sense the effective circulatory volume as inadequate, resulting in fluid retention.


Hyponatremia can have many causes, including HF as in Mrs. J’s case, cardiomegaly and extensive bilateral interstitial markings.

Diagnosed with acute hypervolemic hyponatremia, Mrs. J is admitted to a cardiac step-down unit for further management.

Hyponatremia is classified according to serum antidiuretic hormone (ADH) release, especially if serum osmolality is low, and according to volume status (see A matter of volume). For the purposes of this article, the focus will be on volume status in acute hyponatremia, particularly the subordinate classifications of isotonic, hypertonic, and hypotonic hyponatremia.

**Etiology of hyponatremia**

Normally, the interplay of many body systems regulates water and sodium levels to maintain homeostasis (see Regulation of sodium balance). Hyponatremia, a clinical disorder in which the serum sodium level is less than 135 mEq/L, represents a relative excess of water in relation to sodium (see Pathophysiology of hyponatremia). It can be induced by a marked increase in water intake (primary polydipsia) and/or by impaired water excretion resulting from advanced renal failure or from persistent release of ADH. It’s reported in the literature as occurring in 12% to 30% of hospitalized patients. If hyponatremia is mild or moderate, presentation may be asymptomatic and it may be incidentally diagnosed; however, these patients may have subclinical mental or gait impairments.

Acute hyponatremia develops within 24 hours. Hyperacute hyponatremia develops in a few hours due to impaired water excretion and/or excess water intake, as may occur in long-distance runners or in users of the recreational drug ecstasy. Severe acute or hyperacute hyponatremia is associated with a substantial rise in morbidity and mortality, severe neurologic deficits, increased length of hospital stay, increased hospital costs, increased risk of ICU admission, increased 30-day hospital readmission rate, and a rise in discharges to a short- or long-term-care facility.

Hyponatremia can also be classified by severity as follows:

- **Mild hyponatremia**, a serum sodium concentration between 130 and 135 mEq/L.
- **Moderate hyponatremia**, a serum sodium concentration between 121 and 129 mEq/L. Mrs. J’s serum level was 126 mEq/L, indicating moderate hyponatremia.
- **Severe hyponatremia**, a serum sodium concentration of 120 mEq/L or less.

Hyponatremia can have many causes, including HF as in Mrs. J’s case, decompensated heart failure, advanced liver disease, and renal disease. Although the total body sodium is increased in heart failure, the baroreceptors often sense the effective circulatory volume as inadequate, resulting in fluid retention.
case. Other causes include cirrhosis, syndrome of inappropriate anti-diuretic hormone secretion (SIADH), primary polydipsia, hypothyroidism, adrenal insufficiency, and diuretic use. Causes of hypervolemic hypotonic hyponatremia, the focus of this article, include edematous states such as decompensated HF, advanced liver disease, and renal disease.

Even though hyponatremia is associated with various disorders, the incidence and prevalence is significantly higher (27% to 50%) in patients with certain comorbidities, such as decompensated HF, cirrhosis, and malignant brain tumors. Experts suggest that hyponatremia is an independent predictor of worse outcomes in patients with these primary diagnoses.

Clinical manifestations
Mild-to-moderate symptoms of hyponatremia, as in Mrs. J’s case, are relatively nonspecific and include fatigue, headache, confusion, nausea, vomiting, and gait disturbances. Signs and symptoms of severe hyponatremia include seizures, decreased mentation, coma, and respiratory arrest. The presentation and severity of signs and symptoms directly correspond to the degree and rate of serum sodium level decline.

Brain cells are especially sensitive to changes in serum osmolality and fluid and electrolyte shifts. Patients experiencing acute hyponatremia are at severe risk for neurologic complications such as seizures, coma, brain herniation, and death from cerebral edema induced by rapid water movement into the brain.

Pathophysiology of hyponatremia
In hyponatremia, water shifts out of the extracellular spaces and into cells. Hypernatremia, an excess of sodium, causes water to shift out of the cells. Serum sodium levels below 135 mEq/L indicate hyponatremia. Serum sodium levels above 145 mEq/L indicate hypernatremia.

Patient assessment and diagnosis
Diagnosis of hyponatremia includes a thorough evaluation of the patient’s presenting signs and symptoms and a detailed review of the patient’s health history and current drug therapy if applicable. It’s also critical to determine the patient’s extracellular fluid volume status, time of symptom onset, and duration of low sodium levels, if possible.

Because Mrs. J’s serum glucose level (89 mg/dL) was within a normal range, the possibility of glucose causing hypertonic hyponatremia was ruled out. Simultaneously, serum and urine specimens were evaluated to quantify Mrs. J’s sodium and osmolality levels, urine to serum sodium ratios, uric acid and urea levels, and serum creatinine level (which can be used to estimate glomerular filtration rate, pH, other electrolyte levels, and fractional excretion of sodium).

Following determination of volume status, duration of the sodium decline, signs and symptoms, and etiology of the hyponatremia, treatment options are considered. The rate of sodium decline not only marks the severity of clinical manifestations but also directly guides the rate at which hyponatremia is corrected.

In all three classifications of hypotonic hyponatremia (hypovolemic, hypervolemic, and euvolemic or normovolemic), treatment guidelines vary based upon whether the patient is symptomatic or asymptomatic. In the initial stages of treatment, nurses must continue monitoring both serum and urine lab results at least every 6 to 8 hours for a minimum of 24 to 48 hours to identify trends so that treatment can be adjusted accordingly. The following is a generalized review of treatment based on current guidelines.

Symptomatic or not?
Treatment decisions are guided by the patient’s volume status, duration and degree of hyponatremia,
and the presence and severity of clinical signs and symptoms. An asymptomatic patient may be treated as follows.\textsuperscript{11}

- **Hypovolemic hyponatremia:** Isotonic saline (0.9\% sodium chloride) should be administered to replace intravascular volume and treat the cause of vasopressin release. If hypovolemia is secondary to diuretic use, repletion of potassium may also be indicated. If treatment triggers a large water diuresis and hyponatremia is corrected too rapidly, the patient may be given a hypotonic fluid such as 5\% dextrose and 0.45\% sodium chloride solution.

- **Hypervolemic hyponatremia:** Patients with hyponatremia who are hypervolemic are treated with sodium and fluid restriction, loop diuretics, and correction of the underlying cause. A nonpeptide vasopressin receptor antagonist (vaptan) may be indicated in some cases.

- **Euvolemic (normovolemic) hyponatremia:** The usual treatment of choice is free water restriction, based on the patient’s renal diluting capacity. Administration of hypertonic saline isn’t appropriate in these patients.

When treating symptomatic patients (such as those experiencing seizures or severe neurologic deficits), administer hypertonic saline solution as prescribed. Normal (isotonic) saline solution (0.9\% sodium chloride) can exacerbate hyponatremia in patients with SIADH, who may excrete the sodium and retain the water. Another option is to combine I.V. administration of 0.9\% sodium chloride solution and diuresis with a loop diuretic such as furosemide to elevate the serum sodium concentration. This approach may be indicated for patients with high urine osmolality because loop diuretics reduce urine osmolality. Loop diuretics also increase free water excretion, reducing the risk of fluid overload.

In patients with normovolemic hypotonic hyponatremia, free water restriction may be indicated.

**Nursing considerations**

Nursing responsibilities include monitoring vital signs, neurologic status, fluid status, and serial electrolyte results. Assess the patient for signs of complications, especially seizures and osmotic demyelination syndrome, a severe and potentially irreversible complication (see Understanding osmotic demyelination syndrome). Due to the high osmolarity of hypertonic saline solution, it should be administered only via a central venous access device.\textsuperscript{5,8,9}

**Following Mrs. J**

Mrs. J is admitted to a cardiac step-down unit for treatment of acute hypervolemic hypotonic hyponatremia
Regulation of sodium balance

Sodium is the most plentiful electrolyte in the extracellular fluid (ECF) compartment, with a concentration ranging from 135 to 145 mEq/L. Sodium doesn’t readily cross the cell membrane, so only a small amount (10 to 15 mEq/L) is found in the intracellular fluid compartment. As the major cation in the ECF compartment, Na⁺ and its attendant Cl⁻ and HCO₃⁻ anions account for 90% to 95% of the osmotic activity in the ECF. Thus, serum osmolality usually varies with changes in serum sodium concentration.

Derived from dietary sources, sodium normally enters the body through the gastrointestinal (GI) tract. Although the body’s need for sodium usually can be met by as little as 500 mg/day, dietary intake frequently exceeds that amount.

Most sodium losses occur through the kidneys, which are extremely efficient in regulating sodium output. When sodium intake is limited or conservation of sodium is needed, the kidneys can reabsorb almost all the sodium that’s been filtered in the glomerulus, resulting in essentially sodium-free urine.

Less than 10% of sodium intake is usually lost through the GI tract and skin. Sodium losses increase with conditions such as vomiting, diarrhea, and GI suction. Sodium leaves the skin by way of the sweat glands, which secrete a hypotonic solution containing both sodium and chloride. Although sodium losses due to diaphoresis are usually negligible, they can increase greatly during heavy exercise and periods of exposure to a hot environment.


and her signs and symptoms resolve. A 1-week follow-up appointment is scheduled with her cardiologist.

REFERENCES

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The author has disclosed no financial relationships related to this article.

DOI-10.1097/01.NURSE.0000522006.83149.20

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