A Quick Reference on Hyponatremia

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KEYWORDS

• Hyponatremia • Sodium • Diabetes mellitus • Electrolytes • SIADH

KEY POINTS

- Many disorders that cause hyponatremia are easily identified from the history, physical examination, and a minimum database that includes serum biochemistry as well as urinalysis.
- Patients with diabetes mellitus can be have hyperosmolar or normoosmolar hyponatremia.
- Hypovolemia causes a decrease in effective circulating volume, triggering antidiuretic hormone secretion and free water retention, and develops after gastrointestinal losses, renal losses, hemorrhagic shock, hypoadrenocorticism, and other causes of hypovolemia.

SUMMARY OF SODIUM DISTRIBUTION AND HYPONATREMIA

- Most sodium is located in extracellular fluid (ECF). A low intracellular sodium concentration is maintained by the activity of the cell membrane sodium-potassium-ATPase (Na-K-ATPase).[1]
- Serum sodium concentration is a reflection of the amount of sodium relative to the volume of water in the body and not a reflection of total body sodium content.[1]
- Hyponatremic patients may have decreased, increased, or normal total body sodium content.
  - Adjustments in water balance via thirst and vasopressin (antidiuretic hormone [ADH]) secretion maintain normal serum osmolality and serum sodium concentration.
  - Adjustments in sodium balance maintain normal ECF volume by decreasing or increasing renal sodium excretion. Mechanisms include the effects of glomerulotubular balance, aldosterone, atrial natriuretic peptide, and renal hemodynamic factors.

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- Expansion of ECF volume increases sodium excretion, whereas ECF volume contraction decreases sodium excretion.
- Sodium and its attendant anions account for approximately 95% of the osmotically active particles in ECF. Hyponatremia usually is associated with hypoosmolality.[2]

CAUSES OF HYPONATREMIA

Hyponatremia is a common electrolyte disorder in critically ill and hospitalized patients.[2] Serum sodium concentration should be measured in patients at high risk for hyponatremia, which include dogs and cats with abnormal mentation or behavior; those having seizures; and those with dehydration, polyuria, polydipsia, vomiting, diarrhea, cardiac diseases, and pleural or peritoneal effusion.

Cerebral edema and water intoxication are clinical features that may occur if hyponatremia develops faster than the brain's defense mechanisms can work. Neurologic signs include focal and diffuse deficits, occasionally with seizures. The more rapid the development of hyponatremia, the more severe the neurologic signs are likely to be.

Cause of hyponatremia can be further divided into 3 categories, based on osmolality and volume status (**Table 1**).

Most hyponatremic patients are in the hypovolemic, hypo-osmolar category, with the notable exception of patients with diabetes mellitus (discussed later).[2] In a large study investigating causes of hyponatremia in dogs and cats, it was shown that, in dogs, gastrointestinal fluid loss was the most common factor (55.8%), followed by third-space fluid loss (21.7%) and fluid shift from intracellular fluid (ICF) to ECF because of moderate to severe hyperglycemia (13.4%).[2] In cats, the most common pathophysiologic factors potentially contributing to the development of hyponatremia were urologic diseases (34.8%), followed by gastrointestinal fluid loss (28.3%), and third-space fluid loss (26.2%).[2] Most animals had more than 1 pathophysiologic factor potentially contributing to hyponatremia.

Hypovolemia causes a decrease in effective circulating volume, triggering ADH secretion and free water retention. Hypovolemia develops after gastrointestinal losses (eg, vomiting, diarrhea), renal losses, hemorrhagic shock, hypoadrenocorticism, and other causes of hypovolemia. Hypovolemic animals may have clinical signs such as tachycardia, pale mucous membranes, prolonged capillary refill time, decreased pulse quality, and impaired consciousness. Patients with interstitial dehydration (low ECF volume) may have decreased skin turgor, dry mucous membranes, sunken eyes, increased packed cell volume and total protein concentration, and high urine specific gravity.

Hypervolemic patients may be presented with ascites, jugular distention, peripheral edema, or pulmonary edema. Patients in congestive heart failure (CHF) or advanced liver failure with ascites also have decreased effective circulating volume, despite excessive fluid retention and hyponatremia. In these syndromes, decreased effective circulating volume or stroke volume secondary to myocardial or valvular disease (eg, CHF) also triggers ADH release and thirst, leading to hyponatremia despite hypervolemia. The same is true in patients with liver failure, in which portal hypertension leads to ascites and third-space fluid accumulation as well as decreased venous return caused by low splanchnic flow, which stimulates ADH release and water retention.

Patients with diabetes mellitus can have hyperosmolar or normoosmolar hyponatremia. In diabetes mellitus, hyperosmolality resulting from the high blood glucose concentration induces a shift of water from ICF to ECF and results in dilution of the serum sodium concentration. This dilution is the primary reason that hyponatremia occurs in diabetes