Syndrome of Inappropriate Antidiuretic Hormone Secretion in Malignancy: Review and Implications for Nursing Management

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Hyponatremia is a common fluid and electrolyte disturbance in adults with cancer. Although a number of etiologies are associated with hyponatremia, the syndrome of inappropriate antidiuretic hormone (SIADH) secretion is one of the most common underlying causes. Early symptoms often associated with SIADH are subtle but, if left untreated, may progress to life-threatening seizures, coma, and death. Because oncology nurses have frequent and ongoing contact with patients, they are in an ideal position to recognize patients who are at increased risk for SIADH and those who present with early symptoms. Beginning signs and symptoms are mild and can be mistakenly attributed to other causes. This article reviews the pathophysiology of SIADH, associated risk factors, signs and symptoms, diagnosis, treatment, and nursing care.

Key Words: inappropriate ADH syndrome; hyponatremia; carcinoma, small cell

Hyponatremia is a common fluid and electrolyte disturbance in adults with cancer. Hyponatremia has many causes, including the primary tumor, metastasis, diagnostic procedures, and therapeutic interventions, or it can result as a secondary complication (Berghmans, 1996; McDonald & Dubrose, 1993). Although the syndrome of inappropriate antidiuretic hormone (SIADH) secretion is a rare paraneoplastic syndrome (occurring in 1%–2% of adults with cancer), it is a common underlying etiology for hyponatremia (Poe & Taylor, 1989). In fact, several studies have identified that SIADH is among the most common reasons for hyponatremia and accounts for up to one-third of cases (Anderson, Chung, Kluge, & Schrier, 1985; Berghmans, Paesmans, & Body, 2000; Miller, Hecker, Friedlander, & Carter, 1996). Therefore, oncology nurses must be knowledgeable about this syndrome. This article provides a review of the pathophysiology, risk factors, signs and symptoms, diagnosis, treatment, and appropriate nursing management of patients with SIADH.

Pathophysiology

Sodium and water balance is tightly regulated in narrow physiologic ranges. Four mechanisms are involved in the regulation of sodium and water (Terpstra & Terpstra, 2000). The first mechanism is the secretion and regulation of antidiuretic hormone (ADH) from the hypothalamus-neurohypophyseal system. ADH is produced in special neurosecretory cells in the supraoptic and paraventricular nuclei of the posterior hypothalamus (Keenan, 1999; Terpstra & Terpstra). ADH is stored and released by the posterior pituitary gland (Batcheller, 1994). The production and release of ADH is regulated by receptors located in the kidneys, heart, and brain. Normally, ADH is secreted in response to increased serum osmolality and decreased plasma volume (Finley, 1998a). The release of ADH is inhibited by low plasma volume or an increased circulating blood volume. When serum osmolality reaches 295 mOsm/kg, arginine vasopressin (AVP), the biologic active form of ADH, is released (Haapoja, 2000; Metheny, 1982; Terpstra & Terpstra).

The second mechanism of action occurs in the kidneys (Terpstra & Terpstra, 2000). ADH acts on the V_2 receptors, which are located in the collecting ducts (Robertson, 2001). The ensuing reaction from the ADH and V_2 complex causes water channels to be inserted into the apical cell membrane, making the cell permeable to water (Haapoja, 2000; Robertson). This promotes water reabsorption and decreases urine output (Haapoja; Poe & Taylor, 1989).

The third mechanism of action occurs in the cardiovascular system. The body is able to sense shifts in the circulating blood volume and blood pressure by the stretch receptors in the left atrium and baroceptors in the aortic arch and carotid sinus (Terpstra & Terpstra, 2000). These receptors release atrial natriuretic peptide (ANP) in response to increased atrial pressure (Smeltzer & Bare, 2002). ANP acts on the distal tubule and collecting ducts to decrease water and sodium chloride reabsorption (Guyton & Hall, 1996).

The fourth mechanism is the stimulation of the limbic system. ADH production is increased by limbic stimulation (Terpstra & Terpstra, 2000). The limbic system is an interconnected complex of basal brain function, and its control center is the hypothalamus (Guyton & Hall, 1996). One major function of the limbic system is to control behavior, but it also controls many internal functions, including osmolality of body fluids (Guyton & Hall). Stimulation of the limbic system, by

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