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Nursing Care in Sodium Imbalance Commonly Encountered in Neurosurgery Patients*

Opieka pielęgniarska nad chorymi z dolegliwościami neurochirurgicznymi w przypadkach nierównowagi sodu

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Abstract

Hormones released from pituitary, hypothalamus and endocrine system are affected in various neurological conditions including traumatic brain injuries, cranial surgeries, and brain tumors can impair the fluid and electrolyte balance of the body. The most frequent sodium imbalance types are the syndromes of inappropriate antidiuretic hormone secretion (SIADH) causing hyponatremia and diabetes insipidus (DI) causing hypernatremia. Hyponatremia related to SIADH and hypernatremia related to DI can cause deterioration of the general medical statuses of the patients and even death through the cerebral edema or cerebral infarcts or low blood pressure related to systemic volume loss and therefore reduce cerebral perfusion pressure, respectively. In this review, the nursing interventions are summarized that could guide the nurses for the early diagnosis and follow-up of the sodium imbalances which are frequently seen in patients staying in neurosurgery clinics. **(PNN 2013;2(1):37-42)**
Key words: Syndrome of ınappropriate antidiuretic hormone, *diabetes insipidus*, nursing care, neurosurgery, sodium imbalance

Streszczenie

W wielu stanach neurologicznych, takich jak urazowe uszkodzenie mózgu, zabiegi z zakresu chirurgii czaszki czy guzy nowotworowe mózgu, pod wpływem hormonów wydzielanych przez przysadkę mózgową, podwzgórze oraz systemy układu hormonalnego może zostać zachwiana równowaga płynów ustrojowych oraz równowaga elektrolityczna. Najczęściej spotykane przypadki nierównowagi sodu, to powodujący hiponatremię zespół niewłaściwego uwalniania hormonu antydiuretycznego (SIADH) oraz będący przyczyną hipernatremii diabetes insipitus (DI). Rozwijająca się z powodu SIADH hiponatremia może doprowadzić do obrzęku lub niedokrwienia mózgu; zaś spowodowana DI hipernatremia doprowadzić może do utraty objętości płynów ustrojowych, a w rezultacie do spadku ciśnienia krwi, związanego z tym spadku ciśnienia perfuzyjnego mózgu, a co za tym idzie do pogorszenia stanu klinicznego pacjenta, a nawet do utraty życia. W niniejszym opracowaniu streszczony zostanie opis wczesnego rozpoznawania nierównowagi sodu często występujący u pacjentów leczonych szpitalnie w klinikach neurochirurgicznych oraz opis czynności pielęgniarskich pomocnych pielęgniarkom pracującym w tej dziedzinie.
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Słowa kluczowe: zespół niewłaściwego uwalniania hormonu antydiuretycznego, moczówka prosta (*diabetes insipitus*), opieka pielęgniarska, neurochirurgia, nierównowaga sodu

Introduction

FLUID AND ELECTROLYTE balance and adequate oxygenation must be provided in order to maintain the functions of central nervous system (CNS) [1]. The fact that the hormones released from the pituitary gland, hypothalamus and endocrine system are affected by the various neurologic conditions such as head trauma, cranial surgery and brain tumors can disrupt the body's fluid and electrolyte balance. The most common complication especially after intracranial aneurysms and the surgery around the hypothalamus and the pituitary gland is sodium and water imbalances. The most common sodium imbalance types in neurosurgical patients are the syndromes of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt wasting syndrome causing hyponatremia [1-8] and diabetes insipidus (DI) causing hypernatremia [1,3,7-12]. In this review, the nursing interventions that could guide nurses in this area are summarized for early diagnosis and follow-up of SIADH and DI emerging as a result of antidiuretic hormone (ADH) imbalances, causing sodium imbalances and seen frequently in patients staying in neurosurgery clinics.

Syndrome of Inappropriate Antidiuretic Hormone Secretion

SIADH IS CHARACTERIZED BY THE inappropriate secretion of ADH even though there is no persistent hemodynamic and osmotic stimuli [1]. Intracranial causes of SIADH are listed as traumatic brain injury (TBI), infections (meningitis, encephalitis, abscess), tumors, CNS hemorrhage such as subarachnoid hemorrhage and subdural hematoma, cerebrovascular disease and CNS disease such as Guillain Barre Syndrome, Multiple sclerosis and hydrocephalus [1-3,6,7,13-15].

There have been many studies about development and causes of hyponatremia of neurosurgical patients. Moro et al. (2007) in their study of 298 patients with TBI observed a hyponatremia rate of 16.8% and concluded that there is a high incidence of hyponatremia after TBI. Lohani and Devkota (2011) found the incidence of hyponatremia 27.27% after TBI and reported that the most common cause of hyponatremia is SIADH. Similar results were reached by Agha et al. (2004) in their study over 102 TBI patients (developed hyponatremia due to SIADH). The literature rate of hyponatremia development after subarachnoid hemorrhage, which is considered as a different patient group, was reported between 10-30% [16]. Sherlock et al. (2006) in their retrospective study of hyponatremia after subarachnoid hemorrhage, found that 19.6% of the patients (62 patients) developed severe hyponatremia (plasma sodium <130 mmol / l) and hyponatremia of 69.2% (39/62) of this patient group was due to SIADH.

ADH produced at hypothalamus is stored at posterior pituitary. ADH, managed by the plasma volume and blood osmolarity, is released in response to a sensitive feedback mechanism. However, this system breaks down during SIADH and ADH is continuously released. Its level arises in the blood, thus water is reabsorbed at the kidney tubules and the volume of the plasma increases. The amount of water reabsorbed by kidneys is relatively higher than sodium. As a result of this, dilutional hyponatremia and renal natriuresis (excessive amounts of sodium in the urine) develop [1,13]. Despite an increase in the total body fluid, the blood volume stays at a normal level and peripheral edema doesn't develop [3,17]. CNS symptoms during SIADH, are observed due to hyponatremia. Hyponatremia causes an increase in intracellular fluid volume and thus cerebral edema and intracranial hypertension happen. This leads to poor neurological outcomes (cerebral ischemia and infarction). In the more advanced stages of hyponatremia, seizures, respiratory arrest, coma and brain stem herniation may occur. This situation could result in death [1,3-6,14,15,18]. The Figure 1 summarizes pathophysiology of SIADH.

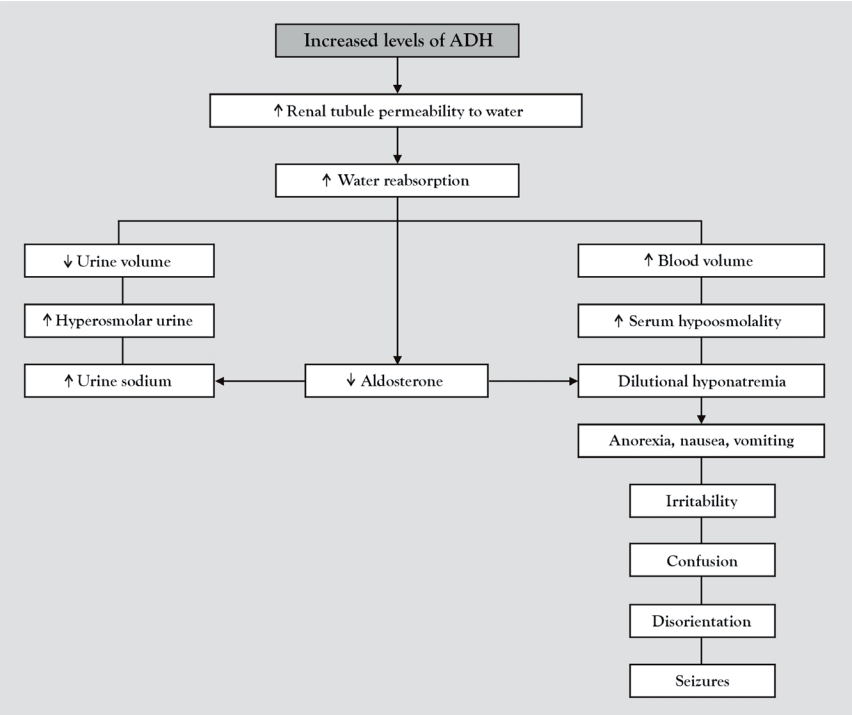


Figure 1. Pathophysiology of SIADH [1]

SIADH Nursing Care

SIADH is commonly seen in hospitalized patients during postoperative period or in intensive care unit. Therefore, patients with post-pituitary surgery (ADH in response to surgery may increase within 5-7 days), traumatic brain injuries and nervous system infection (meningitis) as well as ones using pharmacological agents to ADH stimulus should be continuously assessed for the risk of SIADH development [1]. The purpose of SIADH treatment/care is to bring the level of serum sodium back to normal values progressively by fluid restriction, prevent development of complications by frequent and continuous neurological assesment, and avoid cerebral edema by necessary interventions [1,3,18]. Nursing care in SIADH patients includes the following:

- Neurologic assesment are performed frequently, changes in consciousness level due to cerebral edema or raised intracranial pressure are evaluated, patient deterioration or well-being is closely monitored and carefully documented.
- In addition to neurologic assesment, patients at risk should be monitored for any signs and symptoms of SIADH. These signs and symptoms include: thirst, confusion, agitation, dyspnea, headache (headache gets worse when sodium level is under 100-110 mEq/L), fatigue, weakness, rise in body weight without edema, changes in consciousness level (from lethargy to coma), vomiting, muscle weakness and cramps, muscle twitching, convulsions and seizures (coma and death occur when sodium level is at or falls below 99-100 mEq/L).
- During patient's treatment (at mild cases), fluid restriction is applied to maintain water balance at 800-1000 ml over 24 hours; the reason of this restriction is explained to patient and family.
- To follow dilutional hyponatremia and fluid retention (low urine output), fluid intake and output should be monitored carefully.
- To determine patient's intravascular fluid volume loss, blood pressure and central venous pressure should be measured.
- To monitor rapid weight gain without edema, patient's weight with similar amounts of clothes (hospital clothes) should be measured daily at the same time of day.
- Urine specific gravity should be monitored closely (could be required hourly monitoring at serious cases).
- A sample is taken for urine osmolarity and serum sodium.
- If fluid restriction doesn't treat hyponatremia, 3% of sodium chloride solution is used. The solution should be given slowly; patient's condition should be reassessed frequently.
- Because fluid restriction causes dry mouth, mouth care should be given frequently.
- In the case of a decrease in serum sodium level, the patient should be monitored due to the possibility of seizures. If the patient has seizures, duration of seizures (1), type of seizure (focal or generalized) (2), duration of loss of consciousness (3), abnormal motor activity, (4), characteristics of post-seizure period (5) and used anticonvulsant drugs (6) should be monitored and recorded.
- Changes in patient's conditions should be monitored and reported to the other members of the health care team.
- Patient's response to the treatment and care interventions are evaluated.
- Patients at risk should be closely monitored for any signs and symptoms of hyponatremi and serum sodium levels should be checked daily or every 2 days.
- Due to increasing SIADH risk, all drugs (oral hypoglycemic drugs, diuretics, synthetic hormone replacements, etc.) should be monitored.
- Fluid retention is a predisposing factor for pressure ulcers at hospitalized patients. Frequent skin care should be done, nursing interventions to prevent pressure ulcers such as rotation in bed and positioning should be implemented.
- Patient should be educated about signs and symptoms of hyponatremia and SIADH [2,3,7, 13,14,19].

Diabetes Insipidus

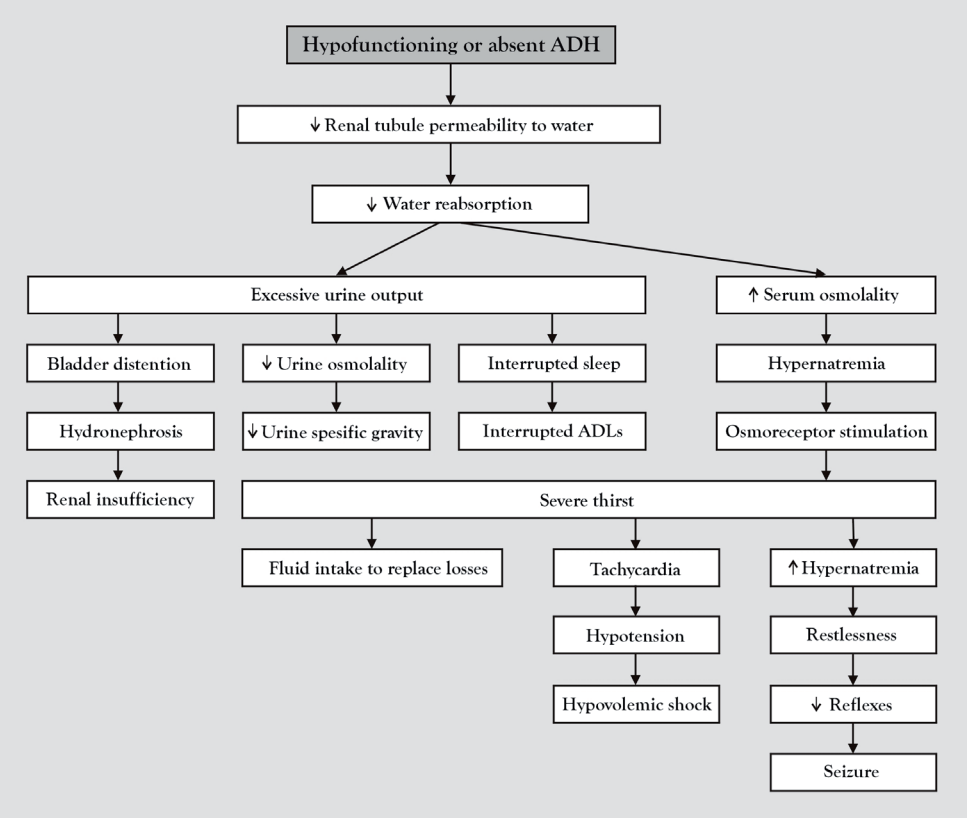
DI (neurogenic) is characterized by polydipsia (excessive thirst), polyuria (diluted, light-colored, excessive increase in the amount of urine), water loss due to dehydration (there is excess fluid loss in the body tissues and main electrolyte disturbances: sodium, potassium, and chloride), low urine specific gravity (1001-1005), serum hyperosmolarity and hypernatremia [1,3,7,11,13,15,17,20]. Intracranial causes of DI can be listed as TBI, brain tumors (especially the back of pituitary gland), intracranial aneurysm, inflammatory diseases of hypothalamus and pituitary gland and surgical procedures to these organs, intracranial hemorrhage, stroke, brainstem hypoxia, aneurysm, arteriovenous malformation, sinus thrombosis, vascular conditions such as intratalamic hemorrhage and infections (meningitis, encephalitis, abscess) [1,3,11,13,17].

Wong et al.(1998) found DI incidence of 3.7% in neurosurgery patients and reported that DI might occurr after subarachnoid hemorrhage (12/29), severe head trauma (11/29), pituitary surgery (5/29) and intracerebral hemorrhage (1/29). Sudhakar et al. (2004) reported DI as the most common complica-

tion with 23% rate in patients after transfenidoneal surgery. In a different study, the most common postoperative complication after the endoscopic endonasal adenomectomy was DI occurring in 18% to 31% of patients postoperatively [9]. Agha et al. (2004) in their study reported that 21.6% of the patients (22 patients) developed DI after TBI and 5 out of 22 patients were found to have permanent DI.

DI develops due to insufficient synthesis, transport or release of ADH when posterior pituitary gland is damaged by variety of reasons such as tumor, trauma or surgical intervention [1,3,7,11,13,20]. DI development due to trauma or neurosurgery follows a triphasic pattern:

- The first phase of triphasic DI lasts 4 to 7 days. Approximately 12-24 hours after surgery, ADH secretion suddenly stops, which causes polyuria and falling urine specific gravity.
- In the second phase, ischemic and degenerated neurons in neurohypophysis occur as a result of secretion of presynthesized ADH. Water is reabsorbed by kidney and hypo-osmolarity and hyponatremia develops in serum. Urine returns to normal.
- In the third phase, ADH is not released, either because the stored amount is exhausted or the damaged hypothalamus cannot produce more ADH. The development of this phase may be delayed. Depending on progressive neuronal degeneration DI may develop. The third phase, if not treated, can lead to permanent DI [1,9,13,20].



ADLs – Activities of daily living

Figure 2. Pathophysiology of DI [1]

In patients with DI, the distal tubular segments at kidneys cannot reabsorbe water due to lack of ADH, this leads to a production of large amounts of dilute urine. The amount may exceed the value of 15L/day. In addition, water and sodium loss from kidneys (due to loss of water) leads to an increase in osmolarity. Thirst mechanisms in the body are activated in case of excessive water loss to cause patients to drink the required amount of water such that serious reductions in body fluids are prevented. However, severe dehydration can develop rapidly at patients with blurred consciousness, confined to bed or who can drink limited water [1,7,15,17]. If DI is not treated, symptoms of hypovolemic shock can be seen [11]. Signs and symptoms of hypernatremia can occur when the amount of urine output is higher than fluid intake [11,20]. In these patients, hypernatremia causes blood pressure to fall as a result of systemic volume loss and thus decreases cerebral perfusion pressure. This can result in worsening of the clinical condition or even loss of patient's life [1]. Figure 2 summarizes pathophysiology of DI.

DI Nursing Care

The responsibility of nurses who provide care to patients with DI is the application of fluid replacement. If the patient is conscious and can receive adequate amounts of fluid, the patient can respond to raised water intake request, thus oral fluid loss can be compensated. However, if the patient is unconscious or not oriented, IV route should be used. The primary principle for DI treatment/care is to measure and document fluid intake and output carefully. The purpose of patient care with DI is to identify DI at early stage with continuous neurological assessment and patient monitoring, and prevent development of dehydration, hypovolemia and shock by applying proper nursing in-

terventions [1,3,13]. Nursing care to patients with DI includes the following procedures:

- Patients at risk should be monitored for any signs and symptoms of dehydration due to DI. These signs and symptoms include: feeling excessive thirst (if the patient is conscious and the thirst center is not affected), craving for iced water or cold drink, polydipsia, weight loss, constipation, polyuria, frequent urination, nocturia, dry skin, poor skin turgor, dry cracked lips, insufficient tear production, fever, orthostatic hypotension, weakness, dizziness and fatigue.
- The patient should be monitored for symptoms related with severity of hypernatremia. These signs and symptoms include: restlessness, confusion, stupor, coma, decreased deep tendon reflex, neuromuscular hyperactivity and seizure.
- If the patient is unconscious and disoriented, she/he is under the risk of dehydration and hypovolemic shock development. Patients should be monitored for hypovolemic shock symptoms. These symptoms are: changes in consciousness level, tachycardia, tachypnea and hypotension.
- To determine fluid balance or loss, fluid intake and output should be monitored carefully.
- The fluid replacement is applied in a way that the first half of the patient's fluid deficit is replaced over 12-24 hours with the remaining half given over the following 48-72 hours. During the fluid replacement, the flow rate should be monitored continuously.
- When the amount of urine is 200 ml/hour or more in the following 2-hour period of time, its color is too light (straw-colored) and urine specific gravity is 1005 or less, it should be reported to the physician (large amounts of fluid output can cause a quick increase in dehydration).
- Urine specific gravity should be monitored (hourly in severe cases).
- To identify abnormal values, samples should be taken for laboratory tests to measure serum electrolytes, BUN, creatinine, glucose, serum and urine osmolarity, for which the test results are evaluated and the treatments are directed by the attending physician.
- During fluid replacement, the solutions containing glucose are usually used. If there is excessive urine output, vasopressin can be added to the IV solution treatment by the physician's request and the infusion dose is continuously monitored.
- Patients who have received vasopressin replacement therapy intranasally should be monitored for presence of rhinitis or sinusitis (this reduces the drug absorption).
- The awake patient who gets excessively thirsty, should be encouraged for fluid intake.
- At the same time, the patient's weight with similar amounts of clothes (hospital clothes) should be measured daily and documented.
- Adequate rest is provided to the patient.
- To avoid fatigue and any injuries due to dizziness, safety measures should be taken.
- The treatment results, patient deterioration or well-being conditions are closely monitored.
- The patient and her/his family should be educated about DI signs and symptoms and how to monitor the patient's fluid intake and output.
- The patient should be warned that frequent urination and excessive thirst will interfere with sleep and other activities [1,3,7,11,13,19,20].

If the reason of DI is identified and treated properly, the process can be reversed, and the patient can recover from DI completely. If the reason cannot be determined or the process is irreversible, then permanent DI may develop and drug use may be required throughout the patient's life. For patients with permanent DI, patient education plan should be developed and implemented. In this training plan: (1) DI should be explained, (2) daily DI care program should be recommended, (3) the individual drug program should be reviewed (frequency, side effects, overdose), (4) complementary care should be planned and (5) the use of DI bracelet should be recommended [13,20]. In addition, weight monitoring, alcohol use and regular health check-ups should be taught to the patient [20].

Conclusions

Sodium imbalances are one of the serious comorbidities of neurosurgery patients. The absence of proper assesment and treatment/care can lead to serious clinical symptoms such as confusion, coma and even death [1-4,14]. Nurses in neurosurgery clinics should be able to identify the patients at risk, with regards to the possibilty of sodium imbalance (such as patients with TBI and CNS infections or brain tumor, aneurysm, pituitary surgery) at the early stages by continuous patient monitoring, not overlook signs and symptoms, and providing the necessary treatment and care, and be equipped with enough knowledge to support patients.

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