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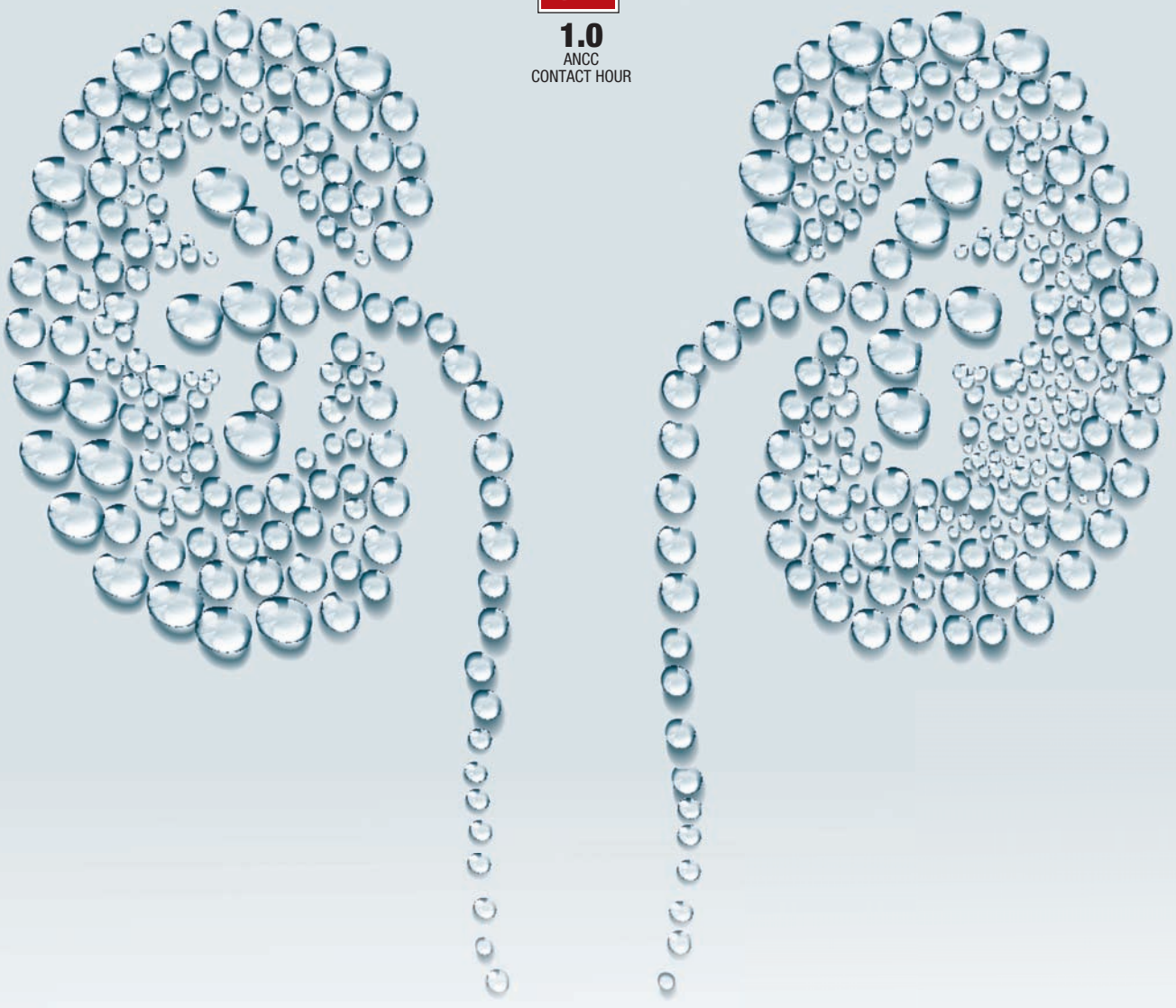

Diabetes insipidus

A matter of fluids

Nurses in all clinical areas, from pediatrics to geriatrics, may encounter this relatively rare disease. Knowing how to identify, monitor, and treat it can help save patients from potentially life-threatening complications.

By Amanda Perkins, DNP, RN

Diabetes insipidus (DI) is a rare condition affecting approximately 1 out of 25,000 people. Characterized by the passage of large amounts of dilute urine, increased thirst, and an increased likelihood of dehydration, this disorder is seen across the lifespan, equally among men and women. Diabetes mellitus (DM) and DI are neither the same condition, nor are they related. Although they both share the word *diabetes*, they are two very different disorders. In patients with DM, blood glucose levels are elevated; this isn't the case in individuals with DI.

This article provides a description of DI, including the different types, signs

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and symptoms, diagnosis, treatment, and nursing care of patients with the disorder.

The body's role in fluid balance

Relying on a variety of factors, including thirst, the kidneys, and the hormone vasopressin (also known as antidiuretic hormone [ADH]), the maintenance of fluid balance in the body is essential. Vasopressin plays a significant role in the regulation of urination and, in turn, fluid and electrolyte balance. In addition to being produced by the hypothalamus, vasopressin is also stored in and secreted by the pituitary gland.

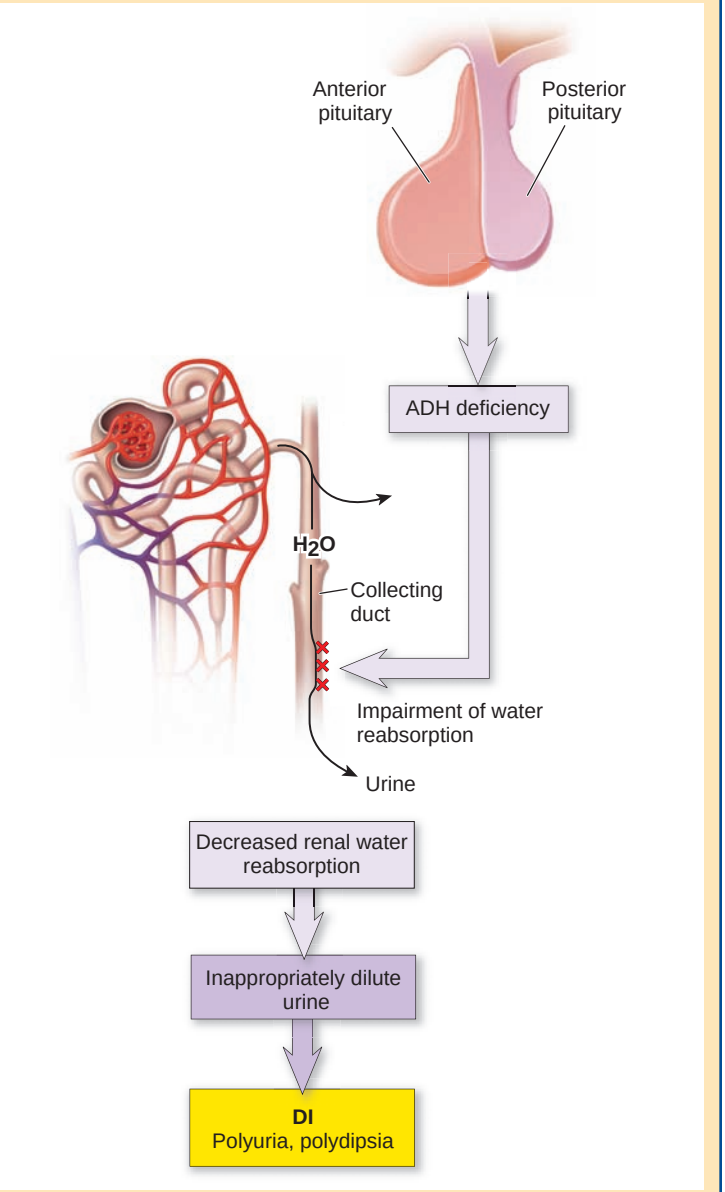
When the body senses that it has a low fluid level, vasopressin is released to tell the kidneys to absorb a smaller amount of fluid from the bloodstream, effectively increasing the amount of fluid in the body. In cases of dehydration or instances in which a person has hypotension, increased amounts of ADH are released to increase the amount of fluid in the body.

On the flip side, when the body senses that it has a high fluid volume, the release of vasopressin is either decreased or stopped completely, resulting in an increased loss of fluid in the form of urine. Basically speaking, vasopressin or ADH controls the amount of water that's released by the kidneys as urine.

Four types

When DI is present, the kidneys are unable to maintain the delicate fluid balance within the body (see *The mechanism of DI*). An individual with DI makes and passes too much urine, which can lead to significant problems including, but not limited to, dehydration and fluid and electrolyte imbalances. The urine of a person with DI is said to be insipid, which means dilute and odorless. Normally, an individual will have a urine output of approximately 1 to 2 L per day, whereas the individual with DI may pass anywhere from 3 to 20 L per day. Additionally, these individuals may urinate as often as every 15 to 20 minutes throughout the course of a day.

The mechanism of DI



Source: Braun C, Anderson C. *Applied Pathophysiology: A Conceptual Approach to the Mechanisms of Disease.* 3rd ed. Philadelphia, PA: Wolters Kluwer Health; 2017.

There are four types of DI: central, nephrogenic, dipsogenic, and gestational.

Central DI

Central DI is caused by damage to the hypothalamus or pituitary gland due to surgery, infection, inflammation, a

tumor, or a head injury. To review, the hypothalamus makes vasopressin, a hormone responsible for increasing or decreasing urine output, and the pituitary gland stores and releases vasopressin. Damage to these areas of the brain will lead to difficulty with the production, storage, and release of vasopressin. With central DI, the body has lower-than-normal amounts of vasopressin, which can lead to too much fluid being removed from the body in the form of urine. In many cases, fluid is rapidly lost from the body, leading to dehydration.

Nephrogenic DI

Nephrogenic DI is caused by kidneys that are unable to concentrate urine. The kidneys don't respond appropriately to vasopressin, resulting in too much fluid being removed from the body. In individuals with this condition, urine production can be as high as 12 L per day, with urination occurring as often as every hour. Children with nephrogenic DI have a congenital form, whereas adults present with an acquired form.

Causes of nephrogenic DI include an inherited gene, gene mutations, chronic kidney disease, medications, decreased serum potassium levels, increased serum calcium levels, and/or a blockage in the urinary tract. Lithium is the drug most commonly associated with the condition. In addition, the antiviral medication foscarnet and the antipsychotic drug clozapine have been associated with nephrogenic DI. Glucocorticoids and alcohol use may also lead to its development.

Medications associated with nephrogenic DI can cause damage to kidney cells, which leads to the cells not responding to vasopressin. Individuals taking lithium should have kidney function tests every 3 months. As is the case with lithium, hypercalcemia can lead to kidney damage, resulting in nephrogenic DI. Hypokalemia may alter the way that the kidneys concentrate urine, leading to large amounts of dilute urine.

Dipsogenic DI

Dipsogenic DI is caused by a defect of the thirst mechanism within the hypothalamus. Individuals with this type of DI are very thirsty, and this increased thirst correlates with an increase in fluid intake. When fluid intake is increased, we see a suppression in vasopressin secretion and increased urinary output. This condition typically has the following three phases: polydipsia and polyuria, hyponatremia, and water intoxication. The signs and symptoms of water intoxication are nausea, vomiting, delirium, ataxia, seizures, and coma.

Disorders associated with dipsogenic DI include chronic meningitis, granulomatous disease (immune system disorder in which phagocytes don't work properly), multiple sclerosis, and mental health disorders. When dipsogenic DI is caused by a mental health disorder, it may be referred to as psychogenic polydipsia or compulsive drinking. Schizophrenia, compulsive behaviors, affective disorders, psychosis, personality disorders, and stress have been associated with psychogenic polydipsia. Although seen in a variety of mental health disorders, this condition is most commonly seen in patients with schizophrenia. In some cases, the medications used to treat psychiatric disorders can cause dry mouth, leading to an increased intake of fluids. Note that this type of DI can lead to hyponatremia instead of hypernatremia. When hyponatremia develops, it can worsen mental health symptoms for the patient.

Gestational DI

Gestational DI typically occurs during the third trimester of pregnancy, affecting 2 to 6 women out of 100,000. The most common signs and symptoms of gestational DI are polyuria and polydipsia, both of which are common in pregnancy, making diagnosis a challenge in many

cases. The risk of gestational DI rises with an increased placental mass, such as occurs with multiple fetuses. Potential causes of gestational DI are hemolysis with elevated liver enzymes and low platelet count, or HELLP syndrome; preeclampsia; and acute fatty liver of pregnancy. Gestational DI may also be caused because prostaglandin produced during pregnancy can reduce the kidney's sensitivity to vasopressin.

With this disorder, the enzyme vasopressinase made in the placenta can break down the mother's vasopressin. During pregnancy, trophoblasts (cells that develop into a major part of the placenta) produce large amounts of the enzyme vasopressinase, which inactivates ADH. Interestingly, vasopressinase activity increases 40- to 50-fold during pregnancy. The liver's ability to degrade this enzyme increases fourfold and the release of ADH also increases. Although gestational DI can be problematic, this condition is usually mild with minimal symptoms and resolves spontaneously 2 to 3 weeks after delivery.

Signs and symptoms

A variety of signs and symptoms are associated with DI. Some of the more common signs and symptoms include:

- polyuria
- polydipsia
- dehydration
- hypernatremia
- dizziness
- weakness
- nocturia
- fatigue
- hypotension
- tachycardia
- decreased level of consciousness (LOC).

As discussed previously, a key feature of DI is the production of large amounts of urine that's dilute and light in color. We know that when an individual passes large amounts of urine, he or she is at risk for becoming dehydrated. The body prevents this risk by sending signals that the person is thirsty. Polydipsia as a result of thirst is another key feature of DI; it can be so significant that the patient drinks as much as 20 L per day. The polyuria and polydipsia that occur with DI occur both day and night, which is a distinguishing feature. Due to diuresis, patients with DI can develop hypernatremia, which can ultimately lead to confusion. Dehydration and hypernatremia may lead to the development of weakness, fatigue, hypotension, tachycardia, and a decreased LOC. In addition to dehydration, you'll see increased serum osmolality or concentrated blood. Individuals with DI are also at risk for hypovolemic shock (see *A closer look at hypovolemic shock*).

Young children may experience severe dehydration, vomiting, constipation, fever, irritability, sleep disturbances, poor growth, excessive crying, and failure to thrive. Older children may wet the bed, have a decreased appetite, and be fatigued. Children may also develop hypernatremia with inappropriately dilute urine. Children with DI should be monitored closely because they have an increased risk of poor outcomes associated with fluid loss.

DI can significantly impact an individual's quality of life, negatively affecting school, work, and social life. In many

A closer look at hypovolemic shock

In severe cases, DI can lead to hypovolemic shock—a loss of fluid so severe that the heart is unable to pump enough blood to perfuse the body. Hypovolemic shock is a medical emergency and must be identified and treated early. Signs and symptoms include:

- hypotension
- decreased temperature
- rapid, thready pulse
- tachypnea
- pallor
- cool, clammy skin
- decreased urine output
- confusion
- weakness
- anxiety
- agitation
- loss of consciousness.

cases, these individuals report that they feel generally unwell and struggle with feelings of fatigue, irritability, and poor concentration. Ask patients about their quality of life and provide assistance and support as needed.

Diagnosis

When caring for a patient with suspected DI, an accurate diagnosis is essential because the treatment plan will differ depending on the type of DI identified. The diagnosis of DI is made based on medical history, family history, a physical exam, urinalysis, blood tests, fluid deprivation tests, and MRI. When completing the nursing history, ask about medications used because some medications have adverse reactions that mimic DI and others can cause kidney damage, which increases the risk of DI development. An MRI may be able to detect problems with the hypothalamus or pituitary gland. Additional testing may be ordered to rule out other diseases. Be aware that gestational DI may be underdiagnosed due to an overlap of symptoms, such as polyuria and polydipsia, which are seen in both DI and during a typical pregnancy.

The diagnostic steps for DI are confirmation of polyuria, determination of the type of DI, and determination of the underlying etiology. When checking the patient for polyuria, identify and rule out other conditions that can present with similar symptoms, such as urinary urgency, nocturia, incontinence, urinary tract infection, and prostatic hyperplasia. To identify polyuria, a 24-hour urine sample is obtained. If completing this test at home, educate the patient about the importance of saving all urinary output. When completing this testing in a healthcare setting, ensure that all staff members, as well as the patient, understand the importance of saving all urine.

Polyuria is confirmed with the following urinary output:

- urine volume greater than 150 mL/kg/24 hours at birth
- urine volume greater than 100 to 110 mL/kg/24 hours up to age 2 years
- urine volume greater than 50 mL/kg/24 hours in older children and adults.

For example, a patient who weighs 150 lb (68.2 kg) would need a urinary output of 3,410 mL or higher to confirm polyuria. When caring for adults, it's considered reassuring when the 24-hour urine volume is less than 2.5 L. Throughout the diagnostic process, the patient's urine is monitored for output and how dilute it is.

With DI in mind, we measure osmolality in the urine and blood. To review, osmolality refers to the number of particles in a fluid. You'll typically see increased serum osmolality and decreased urine osmolality (hypotonic urine). Basically, the patient's blood becomes more concentrated and his or her urine becomes less concentrated. Hypotonic urine has an osmolality of less than 300 mOsm/kg. A plasma osmolality of 285 mOsm/kg will typically make a person feel thirsty. As the plasma osmolality increases, so does thirst.

In addition to osmolality, which only measures the number of particles, urine specific gravity may be checked to also measure the concentration of particles. In general, when urine osmolality is in the normal range, urine specific gravity will be between 1.003 and 1.030. When monitoring urine specific gravity in the patient with DI, it will often be less than 1.005. Both urine specific gravity and urine osmolality are relatively easy to measure and noninvasive in most cases unless a catheter must be used to collect the urine sample. The urine should also be monitored for glucose because DM, which can lead to increased urine output, will result in glucose in the urine whereas DI won't.

Serum sodium levels may also be checked in the patient with potential DI. If the serum sodium levels are elevated, it can help identify central or nephrogenic

DI; if the serum sodium levels are normal or low, dipsogenic DI is suspected.

Fluid deprivation tests involve the restriction of fluid intake while measuring changes in body weight and urine concentration. The two types of fluid deprivation tests that may be carried out are the short test and the formal fluid deprivation test. Before either of these tests, electrolyte abnormalities must be identified and corrected. Additionally, medications that can affect urinary output should be discontinued for 24 hours before the test.

For the short test, the patient stops drinking liquids at a specified time, a urine sample is collected at a different specified time, and the concentration of the sample is tested.

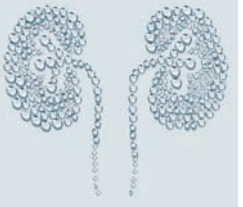
The formal fluid deprivation test is performed in the hospital setting. The patient needs to be monitored closely for adverse reactions such as dehydration. The patient is weighed and his or her urine is tested at specified intervals, in some cases as often as every hour. The test will be stopped if one or more of the following occurs: hypotension or tachycardia, weight loss of 5% or more, and/or urine concentration increase in two to three consecutive tests. At the completion of the test, serum sodium, vasopressin levels, and urine concentration are checked. In patients who don't have DI (excepting dipsogenic DI), the increase in plasma osmolality that occurs due to decreased intake stimulates the release of vasopressin, effectively causing the urine to become more concentrated, with increased urine osmolality. In patients who have DI, this process doesn't happen, and the urine will remain dilute.

At the completion of the fluid deprivation test, desmopressin may be used for patients who have continued dilute urine to help differentiate between nephrogenic DI and central DI. Patients who have central DI should be able to concentrate urine after the administration of desmopressin, whereas those with nephrogenic DI won't be able to.

An additional test that may be carried out is the hypertonic saline infusion test. Depending on the facility, nurses may find that some healthcare providers prefer the hypertonic saline test over the fluid deprivation test. In fact, some research studies have shown that the hypertonic saline infusion test is a more effective way of testing for DI. This test is conducted in a healthcare setting, typically lasting 3 hours or less. As with the fluid deprivation test, medications affecting output need to be stopped 24 hours before the test. Baseline blood testing will be completed for copeptin (an arginine vasopressin surrogate), sodium, glucose, urea, and plasma osmolality.

At the start of this test, hypertonic saline is given as a 250 mL bolus over 10 to 15 minutes and then slowed and administered as a continuous infusion. Serum sodium and osmolality are measured every 30 minutes for the duration of the test. Once the serum sodium levels are greater than or equal to 150 mOsm/L, the test is terminated. At the completion of the test, the patient is asked to drink water at 30 mL/kg over 30 minutes followed by an infusion of dextrose 5% (D5) at 500 mL/h for 1 hour. After the infusion of D5, the

Accurate diagnosis is essential because the treatment plan will differ depending on the type of DI identified. Diagnostic steps include confirmation of polyuria and determination of the underlying etiology.

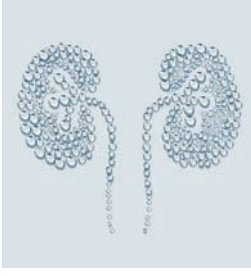


patient's serum sodium levels are checked to ensure that they've returned to normal.

Throughout the course of the hypertonic saline infusion test, the patient must be closely monitored, particularly frequent vital signs assessment. A benefit to this test is that it doesn't require urine to be collected and tested. When reviewing the lab results, a plasma copeptin level of less than 4.9 pmol/L is indicative of central DI and a level greater than or equal to 4.9 pmol/L indicates primary polydipsia. A baseline copeptin value of greater than 21.4 pmol/L is indicative of nephrogenic DI.

may be performed. Baseline and post-op neurologic assessments should be completed. Patient education includes the avoidance of activities that can put pressure on the site, such as coughing, sneezing, blowing the nose, straining for a bowel movement, and bending at the waist. Although this surgery can be used to treat DI, it can also cause DI. The post-op patient should be monitored for signs and symptoms of DI and appropriate treatments initiated if it develops.

With nephrogenic DI, it's important to identify and treat the cause because treating



Astute assessment skills will help you identify signs and symptoms of DI and complications if they develop. Knowing what to look for can save your patient's life.

Treatment

Patients with diagnosed or suspected DI may be referred to a nephrologist or endocrinologist. The type of treatment varies, depending on the type of DI.

With central DI, the hormone desmopressin will be prescribed to replace vasopressin in the body. This medication can be administered via injection, nasal spray, or pill. Desmopressin has been shown to be a safe medication, but it can cause the following adverse reactions: headache, upset stomach, congestion, runny nose, and/or nosebleeds. Taking too much of this medication or drinking too much fluid while taking it can lead to fluid overload, which results in headaches, dizziness, bloating, and hyponatremia. Signs and symptoms of hyponatremia include headache, confusion, and nausea/vomiting. Although desmopressin can help control the patient's symptoms, this treatment isn't curative.

If a pituitary tumor is present, a hypophysectomy (pituitary gland removal) the cause may cure the condition. If nephrogenic DI is caused by a medication that's damaged the kidneys, the medication may need to be discontinued and kidney function monitored. In some cases, stopping the medication will be enough to address the problem. When electrolyte abnormalities are identified as a potential cause, those abnormalities should be treated and reversed. In some instances, such as when ADH is lacking, a thiazide diuretic may be prescribed because it paradoxically reduces urine production. When used for central and nephrogenic DI, thiazide diuretics allow for water and sodium reabsorption at the proximal tubules, which causes decreased urine output. Adverse reactions associated with thiazide diuretics include orthostatic hypotension, indigestion, sensitive skin, and/or erectile dysfunction. Additionally, aspirin and ibuprofen may reduce urine production when used in conjunction with thiazide diuretics.

Treatment for dipsogenic DI can be challenging; in many cases, there's no

Understanding iso-, hypo-, and hypertonic solutions

Isotonic: The concentration of dissolved solutes in two environments is equal. 0.9% sodium chloride is an isotonic solution. When administered to patients, 0.9% sodium chloride solution won't cause any fluid shifts.

Hypotonic: The concentration of dissolved solutes in two environments is unequal, with the hypotonic solution having less dissolved solutes than other solutions. 0.45% sodium chloride is a hypotonic solution. When administered to patients, 0.45% sodium chloride solution will cause fluid to shift from the bloodstream and move into the cells.

Hypertonic: The concentration of dissolved solutes in two environments is unequal, with the hypertonic solution having more dissolved solutes than other solutions. 3% sodium chloride is a hypertonic solution. When administered to patients, 3% sodium chloride solution will cause fluid to shift from the cells and move into the bloodstream.

effective treatment. If a mental health disorder is a cause, treating the psychiatric illness may be beneficial. If medications are identified as causing dry mouth for the patient, it may be possible to explore different medications. If it isn't possible to alter medications causing dry mouth, interventions such as dry mouth tablets, mouthwashes, and sprays may be used to help with symptom control. Treatment may also include fluid restriction and psychosocial care.

When a patient has gestational DI, desmopressin, which has been shown to be safe during pregnancy, may be prescribed. Note that the placenta doesn't destroy desmopressin like it destroys vasopressin. In some cases, no treatment is necessary. The patient will be encouraged to drink extra fluid and monitored closely. If treatment is started, it will typically be stopped after delivery because symptoms usually resolve within a few weeks.

In general, the patient with DI must drink enough liquid to prevent dehydration and the associated complications. In mild cases of DI, the individual may only need to drink more water. Additionally, hypotonic I.V. fluids, such as 0.45% sodium chloride solution, may be given to replace fluids without increasing sodium levels. (For more information on hypotonic fluids, see *Understanding iso-, hypo-, and hypertonic solutions*.) Regardless of the treatment, the patient with DI should be closely monitored and educated about the disease and how to manage it.

Complications

The two main complications associated with DI are dehydration and electrolyte imbalances. Signs and symptoms of dehydration include:

- thirst
- dry skin, mouth, and lips
- sunken features
- fatigue
- dizziness
- lightheadedness
- confusion
- headache
- muscle cramps
- decreased urine output
- concentrated urine
- syncope
- nausea
- tachycardia
- tachypnea.

Mild dehydration can lead to changes in BP, heart rate, and temperature. Severe dehydration can present with weakness and confusion, progressing to brain damage, kidney damage, and death if left untreated.

Dehydration must be monitored when it occurs. Be mindful that dehydration, vomiting, fever, sweating, and hot weather can increase the risk of or

key points

Priority nursing assessments

When caring for a patient with DI, priority nursing assessments include the following:

- Obtain a health history, including medications taken, history of pregnancy, family history of DI, kidney disease, and mental health disorders.
- Monitor for polyuria, polydipsia, nocturia, and signs and symptoms of dehydration.
- Assess skin turgor, vital signs trends, daily weights, and intake and output.
- Closely observe urinary output, including amount, color, and clarity of urine.
- Monitor all requested labs, paying close attention to serum sodium levels and kidney function tests.

worsen dehydration. If any of these risk factors develop, be ready to communicate your findings and treat the identified cause. For mild cases of dehydration, I.V. fluids or increased fluid intake may be prescribed. Electrolytes may need to be replaced to prevent further complications.

The most common electrolyte imbalance that develops in patients with DI is hypernatremia, or an elevated serum sodium level. Serum sodium concentration is controlled by water homeostasis. Thirst, vasopressin/ADH, and the renin-angiotensin-aldosterone system all play a role in water homeostasis. When a patient develops hypernatremia, the first symptom that typically develops is increased thirst. The patient may also present with mental status changes, such as agitation, confusion, and personality changes. He or she may experience muscle twitches, unusual muscle contractions, skeletal muscle weakness, nausea, vomiting, lethargy, irritability, spasticity, seizures, and in severe cases, coma.

Normal serum sodium levels are 135 to 145 mEq/L. With hypernatremia, serum sodium levels are above 145 mEq/L. Although some facilities recognize 135 to 145 mEq/L as the normal range for serum sodium, each facility uses its own ranges. Defer to your facility's ranges when caring for patients with serum sodium abnormalities. Patients with hypernatremia must be monitored and receive treatment to decrease their serum sodium levels. In instances in which a fluid imbalance accompanies hypernatremia, the fluid imbalance should be corrected, followed by a serum sodium level check to determine if the corrected fluid balance also corrected the hypernatremia. If the patient continues to have elevated serum sodium levels, I.V. fluid replacement without sodium may be enough to correct the problem. Lastly, the patient may be placed on a sodium-restricted diet.

Nursing interventions

Nurses play an essential role when caring for patients with DI, including assessing daily weights, intake and output, skin turgor, mucous membranes, vital signs, changes in LOC, and labs. When completing daily weights, it's necessary to obtain the patient's weight at the same time each day on the same scale. All staff members caring for the patient, in addition to the patient and his or her family/caregivers, should be educated about the importance of accurate intake and output measurement. Assess skin turgor and mucous membranes for dryness because these two assessments provide important information about the patient's hydration status. Vital signs should be monitored, being mindful of a decrease in BP and increased pulse. Also monitor for changes in LOC in addition to agitation, anxiety, and restlessness. Be aware of all lab tests ordered and ensure that the results are reviewed and reported to the healthcare provider as needed.

When polyuria is occurring, encourage increased fluid intake. If the patient is taking medications such as desmopressin, encourage decreased fluid intake. Be mindful of patients who have a diminished thirst mechanism or those who can't

report thirst, such as infants or patients who are confused. These patients are at a much higher risk for complications associated with polyuria if they're unable to receive adequate amounts of fluid. It may be beneficial to educate the patient about wearing a medical-alert bracelet stating that he or she has DI.

At the ready

Astute assessment skills will help you identify signs and symptoms of DI and complications if they develop. Knowing what to look for can save your patient's life. ■

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