Nephrogenic Diabetes Insipidus Characteristics and Presentation

Nephrogenic diabetes insipidus occurs due to a defect in the ADH receptor and is characterized by normal or increased ADH levels and decreased urine osmolality. Causes may include kidney disease, drugs such as lithium, and electrolyte disturbances. Patients may experience polyuria, polydipsia, nocturia, and hypovolemia.

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Characteristics

Defect in ADH Receptor

Ant-tie-die-rocket-harmonica with Broken Receptor

Nephrogenic diabetes insipidus occurs due the resistance of ADH (antidiuretic hormone)/vasopressin at the level of the nephron. It is a result of a defect in ADH receptors or aquaporin-2 protein. This may be hereditary or acquired. This differs from central diabetes insipidus, which presents with decreased release of ADH.

Normal or Increased ADH Levels

Normal-sign Up-arrow Ant-tie-die-rocket-harmonica

ADH has no effect in this disorder due to receptor defects in the nephron. Because of this, the body will try to compensate by increasing ADH levels. ADH level can be normal or increased in nephrogenic diabetes insipidus. This differs from central diabetes insipidus, which presents with decreased ADH levels.

Decreased Urine Osmolality

Down-arrow Urinal Ozzy-mole

Decreased urine osmolality is seen in this disorder. It is also seen in central diabetes insipidus. Patients typically present with a urine osmolality < 300 mOsm/kg in complete DI and 300–500 mOsm/kg in partial DI. It occurs due to the inability of the nephron to concentrate urine.

Etiologies

Kidney Disease

Kidney Diseased

A defect of ADH receptors in the kidney can occur due to an inherited disorder or a chronic kidney disorder.

Drugs

Med-bottle

Drugs that may cause diabetes insipidus include lithium, demeclocycline, methoxyflurane, ifosfamide, ofloxacin, amphotericin B, aminoglycosides, cisplatin, foscarnet, cidofovir, and didanosine.

Lithium

Lithium-Battery

Lithium is the most common cause of acquired nephrogenic diabetes insipidus. Kidney function tests should be obtained every 3 months for patients on lithium therapy. Other drugs that can induce nephrogenic diabetes insipidus include amphotericin B and demeclocycline.

Electrolyte Disturbances

Electric-lights Disturbed

Nephrogenic diabetes insipidus can occur due to electrolyte imbalances, including hypokalemia and hypercalcemia. The decreased ability of the kidney to concentrate urine contributes to this process.

Clinical Features

Polyuria and Polydipsia

Polly-urinating and Polly-drinking

Resistance to antidiuretic hormone in nephrogenic diabetes insipidus causes increased urine excretion, resulting in polyuria. This will trigger thirst, causing the patient to drink more water which will in turn result in polydipsia. Polyuria is characterized by urine output greater than 3 L/day in adults or 2 L/m² in children.

Nocturia

Nocturnal-moon-urine

Excessive urine excretion also results in nocturia. Patients can report that they frequently wake up in the middle of the night to urinate. This can lead to daytime sleepiness and sleep deprivation.

Hypovolemia

Hippo-volume-cup

The increased urine output will cause a decrease in blood volume resulting in hypovolemia. Patients can experience dehydration symptoms such as dry mucous membranes, poor skin turgor, and confusion.