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Editorial Note on Nephrogenic Diabetes Insipidus

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Editorial

Central Diabetes Insipidus (CDI) and nephrogenic diabetes insipidus are the two most common types of diabetes insipidus (NDI). The failure of the hypothalamic-pituitary axis to produce or release the proper amounts of vasopressin results in CDI. The collecting duct main cells, which are responsible for water reabsorption through coordinated actions of apical AQP2 and basolateral AQP3 and AQP4, develop NDI as a result of vasopressin hypo responsiveness or insensitivity. The kidneys are unable to concentrate urine in both CDI and NDI, resulting in the generation of excessive urine, or polyuria. NDI can be divided into two types: acquired and congenital. Acquired NDI is much more common than congenital NDI, and it can occur in a variety of pathophysiologic situations. The most prevalent cause of acquired NDI is continuous lithium treatment, which is commonly used to treat bipolar disorder, a common chronic psychiatric condition. NDI is caused by long-term lithium medication, but it can also be caused by obstruction of the urinary system, hypercalcemia, hypokalemia, and protein malnutrition. Congenital NDI is a hereditary condition that we'll go over in depth later.

Congenital NDI is an uncommon disorder caused by mutations in the arginine vasopressin receptor 2 (AVPR2) genes, although it can also be inherited as an autosomal recessive or dominant trait caused by mutations in the aquaporin-2 (AQP2) gene. These genetic variants of NDI are normally present since birth, and if water intake is insufficient, they can cause life-threatening dehydration and neurologic impairment. The AVPR2 gene is found on the X-chromosome (region Xq28), has three coding exons, and encodes a G protein-coupled receptor (GPCR) with seven transmembrane domains that is 371 amino acids long. The basolateral membrane of the main cells of the collecting duct in the kidneys, where water reabsorption occurs, is expressed by AVPR2. Vasopressin binding to AVPR2 in the kidney stimulates adenylyl cyclase, which raises intracellular cyclic adenosine monophosphate (cAMP), which activates cAMP-dependent protein kinase. This activation causes the AQP2 channel to be phosphorylated and trafficked, followed by AQP2 being inserted into the apical cell membrane of the collecting duct, allowing water to enter the cell and lowering renal water excretion.

For bipolar affective disorder, lithium is still the first-line maintenance medication (BPAD). In the treatment of schizoaffective disorder, it is also used to supplement antidepressant medicine and as a mood stabiliser. Lithium has just lately been deemed superior in the prevention of suicide and severe depressive episodes. Despite its therapeutic superiority and adaptability, lithium's usage in clinical practise is limited due to its side effects. Over half of all patients may need to stop taking lithium at some point, and the most common reason is side effects.

Lithium has been linked to a higher incidence of renal function decline. Tubular dysfunction can arise weeks after starting lithium treatment, although glomerular damage takes years to manifest. Tubular dysfunction causes a drop in urine concentration and, as a result, polyuria. Later on, nephrogenic diabetes insipidus (NDI) may develop, in which tubular cells lose their ability to respond to vasopressin [antidiuretic hormone (ADH)]. NDI can affect up to 20%-40% of people who are taking lithium. It's uncertain whether NDI can be reversed once lithium is removed [1-5].

The risk of hypernatraemia may increase in the context of polyuria-related fluid imbalances and NDI. When more free water is lost than is taken in, hypernatraemia develops. If not treated promptly and effectively, hypernatraemia can become a serious or even life-threatening condition. Even modest hypernatraemia is linked to an increased risk of morbidity and mortality. Hypernatraemia can manifest itself in a variety of ways, including lethargy, weakness, and irritability. Muscle twitching, convulsions, and coma are all possible outcomes. As a result, unless a blood test is performed, the issue can easily be overlooked. Clinicians, for example, may misinterpret changes in mental status caused by hypernatraemia as indicators of psychiatric illness. Other side effects, such as dry mouth, hypertension, or oedema, can occur with any psychiatric drug, not just lithium. As a result, it is critical for mental health providers to be aware of hypernatraemia and to recognise risk factors.

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