Primary Polydipsia

Primary polydipsia is characterized by increased free water intake, which is commonly caused by psychiatric diseases and results in polyuria and hyponatremia. It can be diagnosed by normal or decreased plasma osmolality, decreased urine osmolality, and significant elevation of urine osmolality during the water deprivation test. The main management strategy is water restriction.



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Characteristics

Increased Free Water Intake
Up-arrow Water
Primary polydipsia is characterized by excessive voluntary water intake, often leading to complaints of increased urination (polyuria). The resulting urine output may be large in volume and is typically dilute.

CAUSES

Hypothalamic Lesions
Hippo-Thor and Leaches
Damage to the hypothalamus, especially areas involved in thirst regulation, can impair osmoreceptor function, leading to inappropriate thirst and excessive water intake. This can result in primary polydipsia, even in the absence of psychiatric illness. Common causes include trauma, tumors, or neurosurgery affecting the hypothalamus.

Psychiatric Diseases
Psychic
Primary polydipsia is commonly associated with chronic psychiatric conditions, especially schizophrenia. It may also be seen in patients with bipolar disorder and other psychotic disorders.

Diagnosis

Hyponatremia
Hippo-salt-shaker
Hyponatremia in primary polydipsia results from excessive free water intake that exceeds the kidneys' ability to excrete dilute urine, leading to dilutional hyponatremia (serum sodium <135 mEq/L). This occurs despite appropriately suppressed antidiuretic hormone (ADH) levels and is often accompanied by low serum osmolality. The kidneys are normally capable of clearing up to approximately 15–25?L of maximally dilute urine per day (around 50–100?mOsm/kg). However, when water intake surpasses this limit—especially in the setting of low dietary solute—excess water is retained, leading to dilutional hyponatremia.

Normal or Decreased Plasma Osmolality
Normal-sign Down-arrow Plasma-TV Ozzy-mole
Primary polydipsia presents with normal or decreased plasma osmolality (?280 mOsm/kg). This differentiates it from diabetes insipidus, which presents with an increased plasma osmolality (?300 mOsm/Kg).

Decreased Urine Osmolality
Down-arrow Urinal Ozzy-mole
Primary polydipsia patients present with dilute urine. Labs will reveal a decreased urine osmolality (less than 100 mOsm/kg). Urine osmolality will be less than serum osmolality. Unlike SIADH, urine sodium tends to be low or normal because the kidneys are attempting to excrete water, not retain sodium. Due

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to free water retention, patients often present with hyponatremia and a serum osmolality ??280?mOsm/kg.

Water Deprivation Test

Water-bottle Restricted-belt Test-tubes

During a water deprivation test, restricting water intake significantly increases urine osmolality (>700 mOsm/kg) in individuals with normal physiology and those with primary polydipsia. This finding helps distinguish primary polydipsia from diabetes insipidus, where urine osmolality remains low or shows only a minimal increase despite dehydration. In contrast to central or nephrogenic diabetes insipidus, patients with primary polydipsia retain the ability to concentrate urine when appropriately stimulated.

Management

Water Restriction

Water-bottle Restricted-belt

Water restriction is the ideal treatment for patients with primary polydipsia. Avoid rapid correction of hyponatremia as it can lead to osmotic demyelination syndrome.

Treatment of the underlying psychiatric condition, if the patient has any, and cognitive-behavioral therapy (CBT) if the psychiatric condition is mild

COMPLICATIONS

Cerebral Edema

Cerebral Edamame

Severe hyponatremia resulting from excessive water intake can overwhelm the kidneys' ability to excrete free water, leading to water intoxication. This dilutional hyponatremia lowers plasma osmolality, causing water to shift into brain cells and resulting in cerebral edema.

Clinically, this may manifest as headache, nausea, confusion, lethargy, and in acute or severe cases, seizures, coma, or even death. Rapid-onset hyponatremia poses a greater risk of neurological complications due to the brain's limited time to adapt to osmotic changes.

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