Test Your Knowledge: Evaluation of Polyuria

A recent article by Bhasin et al published in the March issue of *AJKD* describes a case of mixed polyuria in the outpatient setting associated with high solute and water intake. The following questions will test your knowledge on this condition.

1. What is the normal daily solute intake?

A. 100 to 350 mOsm
B. 300 to 550 mOsm
C. 500 to 750 mOsm
D. 700 to 1050 mOsm

2. Which of the following is not a complication of polyuria?

A. Hypokalemia
B. Hyponatremia
C. Hypovolemia
D. Hydronephrosis

3. Which of the following scenarios best describe a polyuria due to solute diuresis?

A. Daily urine output = 3 L/day, urine osmolality = 100 mOsm/kg
B. Daily urine output = 4 L/day, urine osmolality = 200 mOsm/kg
C. Daily urine output = 3 L/day, urine osmolality = 300 mOsm/kg
D. Daily urine output = 4 L/day, urine osmolality = 400 mOsm/kg

4. Which one of the following causes of polyuria constitutes pure water diuresis?

A. Head trauma
B. Recovery phase of acute tubular necrosis
C. Hyperglycemia
D. High protein diet

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Solutions to AJKD Blog's Test Your Knowledge: Evaluation of Polyuria

1. **C. 500 to 750 mOsm**

   According to Bhasin et al, the daily solute excretion is roughly 10 mOsm/kg body weight, or 500 to 750 mOsm. Under steady state conditions, the amount of solutes excreted in the urine is equal to the amount of solutes ingested and generated. The volume of urine (and water) excreted is directly proportional to the amount of solute that needs to be excreted in the urine. When the daily solute intake exceeds 900 mOsm, there will be an increased urinary water loss from osmotic diuresis. On the other hand, low daily solute intake can lead to hyponatremia (e.g., beer potomania, tea and toast diet) by limiting urinary water excretion.

2. **B. Hyponatremia**

   Polyuria can be incapacitating by causing disruptions in patients' lives, such as sleep cycle disorders (Bhasin et al). Other metabolic abnormalities caused by polyuria are hypokalemia, which is thought to be due to activation of flow-mediated potassium channels (i.e., BK potassium channels) in the distal nephron by increased urine flow. Hypernatremia is also a common complication of polyuria due to significant urinary electrolyte-free water loss. Sodium loss with polyuria can cause hypovolemia, although excessive use of intravenous fluids during the recovery phase of acute tubular necrosis can cause solute diuresis and perpetuate the polyuria. Finally, excessive urine production can cause distension of urinary tract resulting in (non-obstructing) hydronephrosis.

3. **D. Daily urine output = 4 L/day, urine osmolality = 400 mOsm/kg**

   Polyuria can be classified as solute diuresis, water diuresis, or mixed polyuria. Solute diuresis is characterized by an abnormally high daily excretion of urinary osmoles (greater than 900 mOsm) associated with a urine osmolality greater than 300 mOsm/kg. The daily excretion of urinary osmoles is calculated by multiplying the daily urine output by the urine osmolality (4 L/day x 400 mOsm/L = 1600 mOsm/day in choice D). Under these circumstances, a solute such as glucose (e.g., hyperglycemia), urea (e.g., high protein diet), mannitol, or electrolytes (e.g., intravenous normal saline) obligates water excretion.

4. **A. Head trauma**

Water diuresis is characterized by a normal daily excretion of urinary osmoles between 500 and 750 mOsm and a urine osmolality of less than 100 mOsm/kg. Water diuresis is caused by a decrease in vasopressin release (e.g., central diabetes insipidus caused by head trauma) or a decrease renal response to vasopressin (e.g., nephrogenic diabetes insipidus caused by lithium). Mixed polyuria is a combination solute and water diuresis usually associated with a urine osmolality between 100 and 300 mOsm/kg. Typical examples of mixed polyuria are post-obstructive diuresis and post-acute tubular necrosis (ATN) diuresis where the kidneys are excreting excess solutes accumulated during acute kidney injury couples with a component of water diuresis caused by transient nephrogenic diabetes insipidus during the recovery phase. Recovery phase of ATN has been associated with decreased expression of aquaporin 2 protein in collecting duct cells.