Iatrogenic water intoxication during pelvic ultrasonography in a patient with diabetes insipidus

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Pelvic ultrasonography (US) is a simple and non-invasive radiologic test to evaluate the pelvic organs. It requires a full bladder for better visualization. Our case is a 14-year-old female with diabetes insipidus (DI) who admitted to the pediatric emergency service with the complaints of seizure and agitation after drinking 4 liters of water in one hour for a pelvic US examination due to work-up for delayed puberty. Her biochemical and clinical evaluation revealed water intoxication (WI). To our knowledge, this is the first WI case developed in a patient with DI. Here, we discuss the underlying factors leading to this complication and recommended an approach to obtain a better sonographic image without necessitating oral water intake to fill the urinary bladder.

Key words: child, diabetes insipidus, hyponatremia, water intoxication.

Pelvic ultrasonography (US) is a simple and non-invasive radiologic test to evaluate the pelvic organs. To obtain a better visualization, patients are usually required to consume fluid orally for distention of the urinary bladder prior to the procedure. Parenterally and/or orally consuming an excess of water for radiologic tests such as pelvic US[1], uroflowmetry[2], fluorescein angiography[3], hysteroscopy[4], and colonoscopy[5] may result in iatrogenic water intoxication (WI).

We report a diabetes insipidus (DI) patient who suffered from a serious complication of iatrogenic WI due to excessive oral water intake before the US, which led to hyponatremia, related seizure and confusion. In addition, we discuss the underlying factors leading to this complication and recommend an approach to obtain a better sonographic image without necessitating oral water intake to fill the urinary bladder.

Case Report

A 14-year-old female was brought to the hospital with the complaints of sudden onset of generalized seizure after pelvic US and delayed recovery of consciousness after the seizure. After initial stabilization, it was discovered from the patient's history that she was instructed to take plenty of oral fluid, nearly four liters, before the US. Systemic examination and vital signs were normal except a Glasgow Coma Scale score of 11. The neurologic examination was normal. It was learned that she had been receiving sublingual desmopressin (Minirin®) with a diagnosis of DI for 10 years. Her DI medication included sublingual Minirin® 120 microgram twice a day. She had received the medication before the US.

Her blood glucose was 100 mg/dl, sodium 126 mEq/L, potassium 2.9 mEq/L, blood urea nitrogen (BUN) 9 mg/dl, and creatinine 0.43 mg/dl. Serum osmolarity and urine sodium level were normal. Brain tomography revealed mild edema. The seizure was considered to be secondary to hyponatremia; thus, the electrolyte disturbance was properly corrected with hypertonic saline solution aiming for an elevation of sodium level by 0.5 mEq/L/hour. Her sodium level was 138 mEq/L on the second day. Minirin® was discontinued during treatment. She was discharged on the fifth day without any neurological deficit.

Discussion

Pelvic US is a simple and non-invasive radiologic test. Before sonography, adequate fullness of the bladder improves the quality of imaging. However, there is not enough information in the literature regarding the amount of liquid that has to be received before US. Considering the routine slogan ″more water, better image!″ stated by staff to patients without any investigation of preexisting diagnoses can lead to uncontrolled water intake. Consumption of a large volume of parenteral/oral water within a short time may exceed the renal excretion capacity of kidneys and therefore may cause iatrogenic WI, leading to dilutional hyponatremia[6].

Clinical findings may vary depending on the sodium level and the rapidity of the onset. Neurological symptoms may emerge when an acute decrease in serum sodium level occurs below 120 mEq/L[7]. Furthermore, severe hyponatremia (<110 mEq/L) can cause cytotoxic brain edema that may result in transient neurological impairment such as confusion and seizures[6].

Consideration of a treatment strategy in WI depends on the cause and degree of hyponatremia and the rapidity of its onset. When hyponatremia occurs secondary to fluid overload, then fluid restriction is the mainstay of treatment. The correction rate of the hyponatremia depends on the occurrence of neurological symptoms. Patients with mild hyponatremia usually require only water restriction and a close follow-up. Hyponatremia can be corrected by administration of 1 to 2 ml/kg/h of hypertonic saline (3% NaCl)[7]. Too rapid a correction of serum sodium concentration should be avoided to prevent the occurrence of neurological sequelae, referred to as 'central pontine myelinolysis'. The co-administration of a loop- diuretic can be beneficial by enhancing free water excretion[1].

Among healthy subjects, WI was reported in psychogenic polydipsia, diarrhea, marathon runners, abused children, young soldiers with heat-stroke, and with drug use such as desmopressin[8,9] . Desmopressin is the first-line agent for treatment of central DI, and its most common complication is WI and hyponatremia. The risk of hyponatremia can be reduced by careful dose titration when starting therapy and by close monitoring of serum osmolarity and sodium concentration during the follow-up[9].

To our knowledge, a total of 9 WI cases due to US procedure have been described so far[1]. The WI case we report herein differs from the others by its occurrence in a patient with DI.

In conclusion, to prevent WI, excessive water intake before US should be avoided by careful instruction, particularly in DI patients. In addition, considering the points below may be helpful:

1. The initiation of water consumption right after Minirin® intake facilitated our patient's WI. Therefore, additional factors such as drug ingestion time during the day (i.e., evening, morning), route of administration (i.e., oral, sublingual, intranasal, etc.), and accompanying systemic disease (i.e., renal disease), which affect the pharmacodynamics of desmopressin, should be born in mind, particularly in DI patients.
2. On maintaining normal water balance, the DI patient does not differ from a healthy individual if he/she receives desmopressin regularly at an instructed suitable dose. However, in case of excess water consumption, especially in a short time period, the DI patient even under treatment with a suitable dose of desmopressin might be more susceptible to WI than a healthy subject owing to the long half-life and potency of desmopressin in comparison to anti-diuretic hormone (ADH). Therefore, we suggest that it would be sufficient to consume nearly 1 liter of water before the procedure to distend the bladder.
3. Waiting for the bladder to fill naturally without rapid intake of excessive water or filling the bladder retrogradely by means of a urinary catheter instead may be safer approaches.
4. Before instructing the patients to consume excess fluid, physicians should inquire about preexisting diagnoses.

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