

# Self induced Water Intoxication Associated with Routine Employment Drug Screen Leading to Symptomatic Hyponatremia: A Case Report

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## Abstract

Acute symptomatic hyponatremia is a rare complication of water intoxication. Excessive water intake alone is unlikely to cause symptomatic hyponatremia unless there is impairment of water excretion. We report a case of hyponatremia associated with non-psychogenic polydipsia and an overdose of an herbal diuretic, uva ursi in preparation for a routine workplace drug screen.

## INTRODUCTION

Acute symptomatic hyponatremia is a serious medical condition manifested by seizures, headache, nausea, fatigue, encephalopathy, confusion, coma and occasionally death.<sup>(1)</sup> Diuretic are the most common cause. Less commonly, psychogenic polydipsia and iatrogenic etiologies are implicated. <sup>(2)</sup> Excessive water intake alone is unlikely to cause severe hyponatremia unless there is abnormal renal function or secretion of antidiuretic hormone (SIADH).<sup>(3)</sup> We report a case of non-psychogenic self induced water intoxication associated with an overdose of a herbal diuretic, uva ursi leading to hyponatremia as a complication of a routine workplace urine drug screen.

## RESULTS

Case report: A 43-year-old white man with no prior medical history was transferred to our hospital for symptomatic hyponatremia. He had recently smoked marijuana and fearing a pre-employment drug screen he took 20 capsules of the herbal, uva ursi (500 mg of hydroquinone) and drank 5 gallons of water over the period of a few hours. Several hours later he became combative, confused and had one episode described as lip smacking and "foaming at the mouth" lasting 3-5 minutes. No tonic, clonic or myoclonic movements were noted.

On admission, his physical examination was remarkable for lethargy and orientation to self. The rest of the neurological and general examinations were normal. His serum chemistries revealed sodium 114 mmol/L and osmolality

236 mOsm/kg. Urine drug screen was negative. Urinalysis had a specific gravity of 1.004, pH of 7.0, moderate blood and no RBCs or WBCs. CT scan of the head was normal. EEG was not obtained. Lumbar puncture revealed a protein 31 mg/dl, 2 WBC, 1 RBC. He was treated with 3% saline at 40 cc/hr for 12 hours with gradual improvement in serum sodium and mental status over 48 hours. Additionally he had massive diuresis (> 9 liters) over the same period. Patient was transferred out of the ICU. Forty-eight hours after admission alanine aminotransferase (ALT) and aspartate aminotransferase (AST) and began to rise with a maximum of 466 u/L and 1586 u/L on day 4, respectively. The creatine phosphokinase (CPK) was 71,115 u/L and gamma-glutamyl transpeptidase (GGT) was 12 u/L on day 5. Ultrasound of the abdomen showed normal hepatobiliary system. On day 6, the ALT decreased to 390 u/L, AST decreased to 806 u/L, and CPK decreased to 20773 u/L. The patient was improved and was discharged home.

## DISCUSSION

Hyponatremia due to acute water intoxication in young patients is rare because normal kidneys are efficient at excreting free water, handling up to 20 liters per day unless other mechanisms exist such as diuretic usage or chronic renal insufficiency.<sup>(3)</sup> In psychogenic polydipsia, it is believed a combination of SIADH and chronic water consumption lead to hyponatremia. <sup>(4,5,6)</sup> In addition to psychogenic polydipsia, acute water intoxication has been described in a variety of situations: patient-physician miscommunication in preparation for tests,<sup>(7,8,9)</sup> preparation

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for drug screen,<sup>(10)</sup> and polydipsia in the setting of prolonged strenuous exercise<sup>(11,12,13)</sup>. The latter is associated with stress induced SIADH.<sup>(13)</sup>

Our patient had normal renal function and no prior psychiatric history. He likely exhibited transient SIADH in relation to emotional stress from using marijuana and taking the pre-employment drug screen. Transient SIADH was supported by the development of hyponatremia and hyposmolarity in the setting of extracellular volume expansion. Additionally, uva ursi toxicity may have played a role. Uva ursi is thought to have diuretic properties that are derived from its active ingredients ursolic acid, isoquercetin and arbutin.<sup>(14,15)</sup> Although the mechanisms of diuresis are not known; one component, arbutin, has been shown to increase sodium excretion.<sup>(14)</sup>

Our patient's rhabdomyolysis was more consistent with hyponatremia-induced rhabdomyolysis and not related to the seizure. Rhabdomyolysis after a seizure peaks at 48 hours with serum CPK ranging between 300-1,200 u/L.<sup>(16)</sup> In contrast, hyponatremia induced rhabdomyolysis is associated with serum CPKs between 18,00-98,000 u/L and is delayed 48-96 hours which is more consistent with our patients course.<sup>(17)</sup>

The role of hyponatremia in rhabdomyolysis is not completely known but a plausible explanation is the acute hyponatremia leads to cellular swelling due to extracellular hypo-osmolality. Intracellular potassium is extruded 4 to 24 hours later leading to a reduction in cell size. This potassium depletion eventually induces the rhabdomyolysis.<sup>(18)</sup> Additionally, the delay in potassium movements may explain the delay between presentation with symptoms attributed to hyponatremia and development of rhabdomyolysis in our patient.

The morbidity and mortality associated with acute hyponatremia is almost entirely related to cerebral edema. Most deaths are preventable by rapid accurate diagnosis and treatment.<sup>(12)</sup> Treatment is focused on correcting the hyponatremia with either fluid restriction or hypertonic saline the latter is usually reserved for severe symptomatic hyponatremia with levels between 115-120 mmol/L.<sup>(9)</sup> The rate of correction may be as high as 1-2 mmol/hour with a maximum correction of 25 mmol in 48 hours.<sup>(19)</sup>

### **CONCLUSION**

Herbal medications are not benign products and physicians

should educate their patients about the possibility of side effects and potential drug interactions. We present the first case of hyponatremia associated with self induced water intoxication complicated by uva ursi overdose and the second case of hyponatremia resulting while attempting to pass a workplace urine drug screen. Additionally our patient illustrates the importance of well-designed studies to look at the potential benefit and side effects of herbal medications.

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