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# A Life-Threatening Complication of a Sodium Glucose Cotransport-2 Inhibitor in an Elderly Patient with Dementia

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**Abstract**

The Sodium Glucose Cotransport- 2 (SGLT-2) inhibitors are a newer class of oral antidiabetic drugs which act by preventing reabsorption of glucose in the Proximal Convoluted Tubule (PCT) and facilitate its excretion in urine. As glucose is excreted, its plasma levels fall leading to an improvement in all glycemic parameters [1]. Even though they are a preferred choice of oral antidiabetic drugs due to their many benefits, caution must be exercised while initiating them in certain classes of patients; especially the elderly with dementia and concurrent comorbidities which might limit their capacity to drink water. Here we illustrate a case of life-threatening hypernatremia in an elderly female patient with dementia after she was started on SGLT-2 inhibitor dapagliflozin.

**Keywords:** Sodium glucose cotransport-2(SGLT-2) inhibitors, Dapagliflozin, Elderly, Dementia, Hypernatremia, Diabetic nephropathy, Hyperlipidemia, Urinary incontinence, Normal glucose tolerance, Empagliflozin

## Introduction

Dapagliflozin is a highly selective and potent inhibitor of SGLT2. Single dose administration of dapagliflozin produces a dose-dependent glucosuria in Normal Glucose Tolerance (NGT) individuals that lasts for >24 hours [2]. The cumulative amount of glucose excreted per 24 hours in NGT individuals was 58 grams and 49 grams at 1 and 14 days, respectively, following initiation of dapagliflozin treatment with 20 mg daily dose [3]. It has a unique mechanism of action independent of b cell function and insulin sensitivity which makes it a viable option in combination with other oral antidiabetic agents in all the stages of the disease.

Apart from blood sugar control, it has other benefits including weight loss, reduction in blood pressure, improved cardiovascular outcomes and delaying the progression of diabetic nephropathy [2]. The adverse effects that are described include water and sodium deficit in the initial phase of therapy and increased risk of urogenital infections and ketoacidosis. Electrolyte abnormalities are not frequently described even though hyponatremia may be expected due to sodium deficit. Here we describe a case of hypernatremia in an elderly demented patient, possibly due to osmotic diuresis and inability to compensate the water deficit due to underlying comorbidities.

## Case presentation

A seventy-nine-year-old female patient with past medical history of vascular dementia, cerebrovascular accident with resultant right hemiparesis, urinary incontinence, hypertension, hyperlipidemia, diabetes mellitus and chronic kidney disease presented to the geriatric clinic with generalized weakness and decreased functional status for 4 days. Of note, the patient was started on dapagliflozin by her primary care provider 3 days ago. While at the clinic a neuro stoke was called for her symptoms and patient was taken to emergency room. Evaluation in emergency room was significant for hypernatremia with sodium level of 175 mmol/L, glucose 395 mg/dl and creatinine of 2.7 mg/dl (baseline creatinine 1.1 mg/dl). Urine studies showed high specific gravity 1.030, glucose >1000 mg/dl; urine sodium 68 mmol/L, potassium 28 mmol/L, Chloride 61mmol/L and urine osmolality of 597. Blood ketones were negative. Calculated Fractional Excretion of Sodium (FeNa) was 0.8. There was no evidence of infection. CT brain was negative for acute findings. The high urine osmolarity and FeNa less than one suggested volume depletion with calculated free water deficit of around 8 liters. The patient was subsequently shifted to the intensive care unit and started on half normal saline

infusion with frequent monitoring of her electrolytes. By day 5 her sodium was corrected to 141 mmol/l and creatinine to 1.2 mg/dl. Her functional status improved to her baseline. The dapagliflozin was stopped before patient was discharged home.

## Discussion

SGLT-2 inhibitors may cause osmotic diuresis and water deficit. This is however a rare occurrence as the increased water excretion is usually around 350-400 ml which is equal to one extra void and is not sufficient to cause symptomatic volume depletion [4]. In symptomatic individuals this is often combined with other exacerbating factors like limited water intake due to reduced functional capacity as illustrated above. Even though osmotic diuresis and natriuresis occurs with SGLT-2 inhibitors, there is usually no notable change in serum sodium levels [5]. To the best of our ability, review of literature does not show any case reports of hypernatremia caused by SGLT-2 inhibitors, though there has been reported cases of hyponatremia [6]. But the SGLT-2 inhibitor empagliflozin has been studied as a treatment option for syndrome of inappropriate anti-diuretic hormone secretion, which in theory increases the sodium levels by osmotic diuresis [7] the same mechanism may cause pathological hypernatremia in a different setting as above.

Older adults are more likely than their younger counterparts to have impaired thirst in the setting of hypertonicity and/or to have a condition that reduces their access to water (physical immobility alone and/or a diminished level of consciousness). In addition, they have change of body composition including decrease in water content. Comorbidities such as dementia and neurologic disorders increase risk of hypernatremia too. Before starting SGLT-2 inhibitors counselling must be given to patients and caregivers to maintain adequate water intake and they must be made aware of the symptoms of dyselectrolytemia [4]. One must also be careful while prescribing loop diuretics with SGLT-2 inhibitors [8] as this increases the chances of electrolyte imbalance.

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