Sociedad Española de Nefrología

nefrología
Revista de la Sociedad Española de Nefrología
www.revistanefrologia.com

Case report

Hyponatremia in refractory congestive heart failure patients treated with icodextrin-based peritoneal dialysis: A case series

*Margarita Kunin**, *Liat Ganon*, *Eli J. Holtzman*, *Dganit Dinour*

Nephrology and Hypertension Institute, Sheba Medical Center and Sackler Faculty of Medicine, Tel-Hashomer, Israel

ARTICLE INFO

Article history:
Received 21 November 2016
Accepted 2 May 2017

Keywords:
Hyponatremia
Congestive heart failure
Peritoneal dialysis

ABSTRACT

Severe congestive heart failure (CHF) patients are prone to hyponatremia. Peritoneal dialysis (PD) is increasingly used for long-term management of refractory CHF patients. The glucose polymer icodextrin was proposed to be a good option for fluid removal in such patients. A small, although statistically significant reduction in serum sodium (~2 mmol/l) consistently observed in multiple trials, is considered as not clinically relevant. Here we reported five refractory CHF patients who demonstrated sodium drop by median of 8 meq/l (range 5.4–8.3 meq/l) after icodextrin was added to their program. It seems that icodextrin may contribute to clinically relevant hyponatremia if the hyponatremia is compounded by other factors. Patients with extremely severe congestive heart failure are susceptible to this complication.

Hiponatremia en pacientes con insuficiencia cardíaca congestiva resistentes al tratamiento tratados con diálisis peritoneal con icodextrina: una serie de casos

Palabras clave:
Hiponatremia
Insuficiencia cardíaca congestiva
Diálisis peritoneal

RESUMEN

Los pacientes con insuficiencia cardíaca congestiva grave son propensos a sufrir hiponatremia. La diálisis peritoneal se utiliza cada vez más para el tratamiento a largo plazo de los pacientes con insuficiencia cardíaca congestiva resistentes al tratamiento. El polímero de glucosa icodextrina se propuso como una buena opción para la ultrafiltración. Una reducción pequeña, aunque estadísticamente significativa, del sodio sérico (~2 mmol/l) observada sistemáticamente en numerosos ensayos no se considera de relevancia clínica. En este documento informamos de 5 casos de pacientes con insuficiencia cardíaca congestiva resistentes al tratamiento que presentaron una caída de las concentraciones de sodio en una mediana

* *Corresponding author.*
E-mail address: Margarita.Kunin@sheba.health.gov.il (M. Kunin).
http://dx.doi.org/10.1016/j.nefroe.2017.05.017

de 8 mEq/l (intervalo 5,4-8,3 mEq/l) después de la adición de icodextrina a su programa. Parece ser que la icodextrina puede contribuir a una hiponatremia clínicamente relevante si se combina con otros factores. Los pacientes con insuficiencia cardíaca congestiva muy grave son propensos a esta complicación.

Introduction

Removal of extensive fluid overload is one of the most difficult challenges in the management of severe congestive heart failure (CHF), particularly in patients who do not respond to diuretic therapy. Peritoneal ultrafiltration (UF) is a simple choice for daily fluid removal. Today, peritoneal dialysis (PD) is increasingly used to treat hypervolemic CHF patients who are resistant to conventional therapies, in particular when complicated by renal insufficiency (reviewed in Refs. 1, 2). Icodextrin was proposed to be a good option for UF in refractory CHF patients.[3–6] Icodextrin solution is a long-acting osmotic agent that allows the patient's UF volume to gradually increase for up to 12 h.[1,3] In a recently published systematic review[7] it was found that mortality in CHF patients treated with peritoneal UF was associated with less use of icodextrin.

Icodextrin is generally well tolerated. A small, although statistically significant reduction in serum sodium (~2 mmol/l) consistently observed in multiple trials, is likely not clinically relevant.[8] Two female diabetic patients treated by icodextrin-based PD developed severe hyponatraemia with plasma Na < 121 mmol/l and neurological complications.[9] The mechanism by which icodextrin causes hyponatremia is unclear. It was proposed that icodextrin solution leads to accumulation of maltose in the extracellular volume and this gradient induces osmotic flow by a mechanism similar to isosmolar colloid osmosis. The resultant shift in water from the intracellular to the extracellular space can result in dilutional hyponatremia.[8] It was demonstrated that icodextrin increased peritoneal Na removal.[10]

Five refractory CHF patients treated with peritoneal ultrafiltration in our unit, developed significant symptomatic hyponatremia with Na < 130 mmol/l after icodextrin was added to their program (Table 1).

Cases description

Patient 1

A 63-year-old woman with history of diabetes mellitus (DM), restrictive cardiomyopathy and severe pulmonary hypertension was referred to PD due to significant volume overload. Her eGFR was 27 ml/min/1.73 m^2. She had no proteinuria and renal sonography showed small kidneys. Her kidney disease was attributed to nephrosclerosis with cardiorenal component. She was treated with one dianeal glucose solution 4.25% exchange per day for 6 weeks, then icodextrin was added to her program. She continued the treatment with furosemide and was on sodium restriction diet. Her urinary sodium at icodextrin start was 78 meq/l. Following icodextrin start a serum sodium dropped from 136.3 to 128.3 meq/l (average of 3 consecutive values) with the lowest value measured of 126 meq/l. The patient complained of extreme weakness and fatigue. Icodextrin was stopped and dianeal glucose solution 2.5% was started. After icodextrin stop serum sodium raised to 134.7 meq/l (average of 3 consecutive values). Due to difficulty to maintain adequate ultrafiltration and worsening diabetes mellitus control, icodextrin was restarted. Serum sodium dropped to 128.7 meq/l (average of 3 consecutive values), lowest value measured was 128 meq/l. The patient complained of extreme weakness and fatigue, therefore icodextrin was stopped again. A month later average serum sodium was 133 meq/l. The patient died a month later from heart failure exacerbation.

Patient 2

A 64-year-old woman with hypertrophic cardiomyopathy was referred to PD due to significant volume overload. She had no proteinuria and demonstrated normal kidneys in renal sonography. Her kidney disease was attributed to cardiorenal syndrome. At PD start the patient's eGFR was 23 ml/min/1.73 m^2. From the very beginning she turned to be hypotensive and oliguric. The patient was 8 months on PD with 3 glucose 2.5% exchanges per day. Furosemide was stopped due to oliguria and hypotension. Urinary sodium was 49 meq/l and the patient continued to be edematous. After icodextrin addition serum sodium dropped from 133.7 to 126.3 meq/l (average of 3 consecutive values) with the lowest value measured of 125 meq/l. The patient continued with icodextrin despite low serum sodium. Non-adherence to free water restriction was suspected. Despite the attempts to restrict free water consumption by the patient serum sodium remained low. The patient died 5 months later from septic shock due to infected cardiac pacemaker electrode.

Patient 3

An 82-year-old man with ischemic cardiomyopathy and low LVEF started PD due to resistant volume overload. At the admission his eGFR was 26 ml/min/1.73 m^2. He had non-nephrotic proteinuria and reduced parenchyma size in kidney sonography. His renal disease was attributed to nephrosclerosis with cardiorenal component. He was treated with PD for one month with 2 exchanges per day: glucose 2.5% and glucose 4.25%. He continued treatment with furosemide and was on sodium restriction diet. His urinary sodium was 53 meq/l. After icodextrin addition serum sodium dropped from 134.7