Hyperkalemia associated to hepatitis in a peritoneal dialysis patient

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Abstract Hyperkalemia is an unusual complication in peritoneal dialysis patients, having a prevalence of around 0.8% among the continuous ambulatory peritoneal dialysis (CAPD) population. The main cause of hyperkalemia in this group is the release of potassium from sources such as gross haematomas and rhabdomyolysis. However, there is no previous report regarding hyperkalemia induced by intracellular potassium shift into the intravascular compartment secondary to drug-induced acute hepatitis in peritoneal dialysis. We present the following case report of a peritoneal dialysis patient, well dialyzed and on a low-potassium diet, who was admitted in our hospital with paralysis secondary to hyperkalemia (serum potassium: 7 mmol/l). Both disorders disappeared using continuous automated peritoneal dialysis (APD) until liver enzymes normalized. We concluded that acute hepatitis can be a cause of hyperkalemia in a properly nourished and well-dialyzed peritoneal dialysis patient.

Keywords Hyperkalemia · Peritoneal dialysis · Hepatitis

Introduction

Hyperkalemia is an unusual complication in peritoneal dialysis patients, having a prevalence of around 0.8% in CAPD. Causes of hyperkalemia in this population include internal potassium-releasing sources such as gross haematomas and rhabdomyolysis [1]. There is no previous report regarding hyperkalemia induced by release of intracellular potassium into the intravascular compartment secondary to acute hepatitis in a peritoneal dialysis patient, and in this paper we present our experience with such a case.

Case report

This 28-year-old Caucasian female suffered from chronic renal failure secondary to focal segmental glomerulosclerosis. She was on peritoneal dialysis treatment for 15 months. Her hypertension was treated with methyl-dopa 1 g/day. Other medications that she was also on were: folic acid, vitamin B, and calcium carbonate (8 g/day). She was seen because of a 24 h evolution of systemic paralysis and was found to have hyperkalemia: 7 mmol/l. She was admitted and started on continuous
automated peritoneal dialysis. As plasma potassium levels were normalized paralysis disappeared.

She had no previous episodes of hyperkalemia, nor antecedent of familiar periodic paralysis, and was adequately dialyzed with a Kt/V of 2.2 and weekly creatinine clearance of 66 l/week (Table 1). She did not suffer from diabetes mellitus and her plasma glucose level was normal.

The patient insisted that she was strictly following instruction for a low-potassium diet and we could not identify any drug that could cause hyperkalemia, such as nonsteroidal anti-inflammatory drugs, angiotensin receptor blockers (ARBs), beta-blockers, nor angiotensin converting enzyme inhibitors (ACEIs).

Since there was no obvious source of potassium intake nor evidence of decreased excretion/removal, a releasing source of intracellular potassium to the intravascular compartment was sought.

A thoracic and abdominal computed tomography scan showed neither a large hematoma nor enlarged lymphatic nodes (excluding the hypothesis of a tumoral lysis syndrome). Normal serum aldolase and creatine kinase ruled out the presence of rhabdomyolysis. Besides there was no evidence of bleeding nor hemolysis. Nevertheless, serum glutamic oxaloacetic (GOT) and glutamic pyruvic transaminases (GPT) had recently been increased: GOT: 60 IU/L (normal range: 5–30 IU/L) and GPT: 116 IU/L (normal range: 5–35 IU/L), indicating an acute hepatic inflammation. Since there was no clinical or laboratory evidence of sepsis and/or viral hepatitis and the only potentially hepatotoxic drug that she was taking was methyl-dopa, hepatitis was attributed to this drug and it was discontinued.

In order to avoid rebound hyperkalemia continuous automated peritoneal dialysis was extended until both serum potassium was normalized and hepatic transaminases had started to decrease (four days). Subsequently the patient continued with her usual peritoneal dialysis schedule (APD 10 l/day) and her serum potassium and serum hepatic transaminases remained normal and slightly high, respectively (Table 2).

Despite improvement in her serum transaminases levels they had not normalized six weeks after discontinuation of the drug. Subsequently liver biopsy showed nonspecific findings of isolated foci of lobular necrosis focus. Hepatic inflammation was attributed to methyl-dopa toxicity. Liver enzymes were normalized after two months.

The case was finally described as a drug-induced hepatitis (methyl-dopa).

**Discussion**

Hyperkalemia is a complication of chronic renal insufficiency, which is controlled by dialysis [1]. Even though potassium removal by peritoneal dialysis is lower than that achieved by hemodialysis, the incidence of hyperkalemia in peritoneal dialysis patients is low [2], while hypokalemia is the most frequent plasma potassium derangement in this population. This phenomenon cannot be explained by the external potassium balance achieved by peritoneal dialysis, and it is attributed to the potassium shift into the intracellular compartment resulting from insulin release following glucose absorption from the peritoneal cavity [3]. This hypothesis was reinforced by a previous study that found that intracellular potassium content is higher in peritoneal dialysis patients respect to hemodialysis ones [4]. The aforementioned mechanism of potassium redistribution in peritoneal dialysis patients can also play a role in diabetics since insulin stimulates intracellular potassium shift even in this group [5].

Main causes of hyperkalemia in peritoneal dialysis patients are high potassium diet, hyper-

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Laboratory values at admission</th>
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<tbody>
<tr>
<td>Crp 13</td>
<td>Up 101</td>
</tr>
<tr>
<td>Normal</td>
<td>&gt;8 mg/dl</td>
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