Coconut water drink and the risk of hyperkalaemia in diabetes

Abstract
Coconut water is available as a substitute for table water and as a sports rehydrating drink. Because of the nature of its micronutrients, it may lead to biochemical changes that may not be beneficial for all groups of people.

A patient with type 2 diabetes mellitus consumed daily around one litre of coconut water drink. As a result, there was a gradual increase in serum potassium. On cessation of beverage consumption, serum potassium decreased to within the reference interval. However, an increase in urea and creatinine concentration did not revert to the level seen prior to coconut water consumption. There was a decrease in serum alkaline phosphatase and zinc when consuming the beverage. Reduction occurred in diastolic blood pressure, estimated glomerular filtration rate, serum enzymes and zinc, while serum potassium concentration increased in this patient with type 2 diabetes. The observed changes resulted from consuming excessive quantities of coconut water drinks.

In patients with diabetes and renal impairment and on potassium-retaining medication, there is a high risk of developing hyperkalaemia. Copyright © 2016 John Wiley & Sons.

Key words
diabetes; hyperkalaemia; coconut water; sports drinks

Introduction
Water from the endosperm of coconut is consumed as a hydrating and recovery drink by some top class athletes. Coconut water (CW) has very little fat and protein macronutrients and only a small amount of carbohydrates; hence, its promotion as a sports and hydration drink.

The micronutrient constituents of a fresh CW depend on the age of the nut. Its osmolality is around 300–452 mosm/L; pH is 4.7–6.0, sodium <3mmol/L, potassium 4–10mmol/L, calcium 3–11mmol/L, phosphate 4.8mmol/L, magnesium 2–10mmol/L, vitamin B group with B1 (nicotinic acid) and pantothenic acid at approximately 0.5mg/ml.

As a replacement for table water and carbonated soft drinks, a patient with diabetes started consuming CW on a daily basis. The resulting effects and the clinical outcome are reported in this case study.

Case history
During an annual review in a primary care surgery, it was noticed that a 62-year-old patient with a six-year history of type 2 diabetes had developed mild hyperkalaemia. Past results revealed a slow progressive rise in serum potassium during his last three outpatient clinic visits. He had no history of nephropathy, neuropathy, or peripheral vascular disease. Over the previous year, he had developed moderate non-proliferative diabetic retinopathy with central macular oedema. The patient’s metabolic profile and glycaemia until then was under good control with metformin medication. He also takes fenofibrate to help maintain normal blood lipids and to slow the progression of retinal vascular disease. For hypertension, diagnosed at the same time as his diabetes, he has taken lisinopril since diagnosis. Perusal of his case notes revealed he has been well compliant in taking the blood pressure lowering medication lisinopril, an angiotensin-converting enzyme inhibitor, and that there has been no recent adjustment in drug dose to link it to the development of hyperkalaemia.

Hyperkalaemia in diabetes can arise from type 4 renal tubular acidosis, chronic kidney disease, medication, and non-compliance with insulin. Possible reasons for the change in serum potassium status, along with the investigation results and blood pressure measurements, are summarised. All previous and current laboratory tests used fresh blood specimens taken by an experienced phlebotomist. Spurious
and extraneous factors causing raised potassium are excluded. Detailed examination of case records revealed no particular cause for the progressive rise in potassium concentration. The possibility of intake of LoSalt salt substitutes was excluded. However, the patient admitted to regularly drinking CW since February and that in the past two months he had increased the daily consumption to around a litre of Vita Coco CW as a substitute for table water and carbonated soft drinks. Advice was given to stop further consumption of CW. The patient’s results from the latest clinic visit along with previous data are summarised in Figure 1 and Table 1.

**Discussion**

During the CW drinks period, the patient’s serum potassium rose by 0.7 mmol (Table 1), and post-cessation decreased to within 0.3 mmol of basal concentration. By taking an early decision to stop CW consumption, the development of frank and severe hyperkalaemia was averted. Its effect on serum enzymes, fasting glucose, and HbA1c was a decrease in concentration (Table 1). However, urea increased from basal level but remained within the reference interval (RI). Allowing for the biological variations, there was no progressive trend in creatinine concentration pre-CW – that is, there is no indication of developing microvascular renal complications. Urinary albumin concentration and albumin–creatinine ratio remained stable at all times.

During CW, serum creatinine concentration increase resulted in a lowering of the estimated glomerular filtration rate (eGFR) value. Interestingly, the renal markers did not return to basal level after CW consumption ceased. How much of this potentially adverse change is due to other dietary and lifestyle factors is uncertain. As the patient has retinopathy, it is possible that the increase in creatinine may have resulted from a similar as yet undiagnosed renal microvascular deterioration, and this may have put him at an increased risk from hyperkalaemia. Serum sodium, bicarbonate, triglycerides, total- and HDL-cholesterol, free thyroxine, and

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**Table 1. The patient’s laboratory results before, during and after his consumption of coconut water (CW)**

<table>
<thead>
<tr>
<th>Analyte</th>
<th>Reference interval/limit</th>
<th>Results: mean/SD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Before CW</td>
</tr>
<tr>
<td>Alkaline phosphatase, U/L</td>
<td>30–120</td>
<td>52/9</td>
</tr>
<tr>
<td>Alanine transferase, U/L</td>
<td>&lt;51</td>
<td>24/12</td>
</tr>
<tr>
<td>Creatine kinase, U/L</td>
<td>&lt;151</td>
<td>105/26</td>
</tr>
<tr>
<td>Creatinine, µmol/L</td>
<td>60–120</td>
<td>86/11</td>
</tr>
<tr>
<td>Estimated glomerular filtration rate, ml/min/1.73m²</td>
<td>&gt;59</td>
<td>96/5</td>
</tr>
<tr>
<td>Fasting glucose, mmol/L</td>
<td>4.0–7.0</td>
<td>10.2/4.6</td>
</tr>
<tr>
<td>HbA1c, % total haemoglobin</td>
<td>20–42</td>
<td>54/6.4</td>
</tr>
<tr>
<td>Potassium, mmol/L</td>
<td>3.5–5.0</td>
<td>4.6/0.29</td>
</tr>
<tr>
<td>Sodium, mmol/L</td>
<td>135–145</td>
<td>139/1.5</td>
</tr>
<tr>
<td>Total bilirubin, µmol/L</td>
<td>&lt;22</td>
<td>14/2</td>
</tr>
<tr>
<td>Total bicarbonate, mmol/L</td>
<td>21–28</td>
<td>29/2</td>
</tr>
<tr>
<td>Urea, mmol/L</td>
<td>2.7–7.5</td>
<td>3.9/0.6</td>
</tr>
<tr>
<td>Urine albumin, mg/L</td>
<td>0–20</td>
<td>15/6</td>
</tr>
<tr>
<td>Albumin–creatinine ratio, mg/mmol</td>
<td>&lt;2.5</td>
<td>1.7/0.6</td>
</tr>
</tbody>
</table>

Mean/standard deviation (SD) of results: Before = Dec 2008 to Mar 2012; During = Feb 2014 to Jun 2014; and After = Sep 2014 to Jan 2015. No laboratory test was analysed in 2013.
Coconut water contains a high amount of potassium. In people with diabetes and renal disease, and on potassium-retaining medication, there is an increased risk of developing hyperkalaemia. Coconut water drink micronutrients may affect diastolic blood pressure, serum urea, creatinine, zinc and enzymes.

Key points

- Coconut water drink contains a high amount of potassium
- In people with diabetes and renal disease, and on potassium-retaining medication, there is an increased risk of developing hyperkalaemia
- Coconut water drink micronutrients may affect diastolic blood pressure, serum urea, creatinine, zinc and enzymes

Drug notes

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Alishiken Amlodipine Bisoprolol Bromocriptine Bumetanide Carbamaezpine Cilostazol Clodigorel Coleselam Dagibatran Darbepoetin alfa Dicideo Digeoxin Dipiridamole Domperidone Doxazosin Dronedaron Duloxetine Epleronere Enthyromyc

Ezetimibe Gabapentin Iindapamide Iprabradin Labelatalo Lidocaine Lorcsarinet Losartan Metyldopa Metclopramide Nicorandil Nicedipine Omacor Orlistat Prasugrel Prolonged-release nicotinic acid Quinine sulphate Ramipril Ranolazine Rimonabant Rinaroxaban Rosuvastatin Sibutramine Spironolactone Tadalahil Testosterone Torcetrapib

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Declaration of interests

There are no conflicts of interest declared.

References