Coconut water drink and the risk of hyperkalaemia in diabetes

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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.
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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.[1] 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.[2]

The micronutrient constituents of a fresh CW depend on the age of the nut. Its osmolality is around 300–452mOsm/L; pH is 4.7–6.0, sodium <5mmol/L, potassium 4–10mmol/L, calcium 3–11mmol/L, phosphate 4.8mmol/L, magnesium 2–10mmol/L, vitamin B group with B_1 (nicotinic acid) and pantothenic acid at approximately 0.5ug/ml.[2–4]

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.[5]

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,[6] 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.[7]

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.[8] 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[9] 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 0.7mmol (Table 1), and post-cessation decreased to within 0.3mmol 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 HbA_{1c} 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

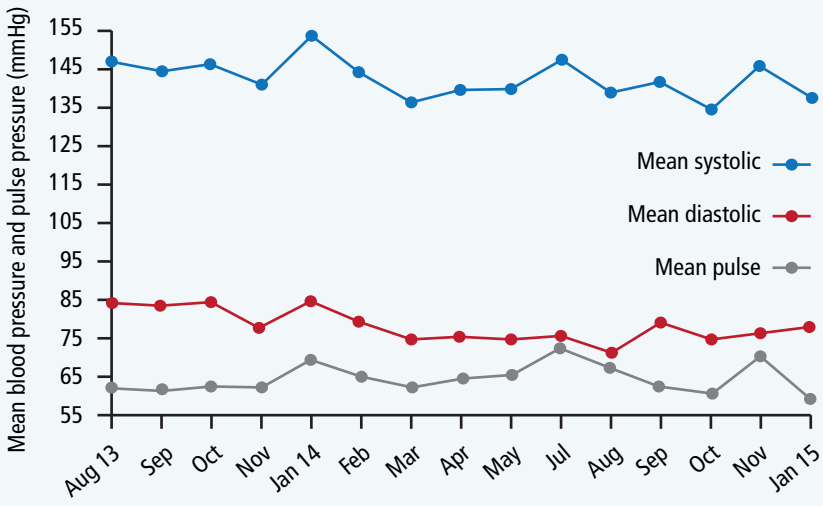

Figure 1. The patient's mean systolic, diastolic and pulse pressure during the period August 2013 to January 2015. Coconut water consumption occurred between February and June 2014

Analyte	Reference interval/ limit	Results: mean/SD		
		Before CW	During CW	After CW
Alkaline phosphatase, U/L	30–120	52/9	36/11	26/3
Alanine transferase, U/L	<51	24/12	15/2	13/1
Creatine kinase, U/L	<151	105/26	68	159/27
Creatinine, µmol/L	60–120	86/11	115/13	114/11
Estimated glomerular filtration rate, ml/min/1.73m^2	>59	96/5	57/6	58/4
Fasting glucose, mmol/L	4.0–7.0	10.2/4.6	6.8/2.0	7.3/0.4
HbA_{1c}, % total haemoglobin	20–42	54/6.4	45/0.7	45/0.7
Potassium, mmol/L	3.5–5.0	4.6/0.29	5.3/0.21	4.9/0.07
Sodium, mmol/L	135–145	139/1.5	138/0.0	138/0.0
Total bilirubin, µmol/L	<22	14/2	4/1	7/1
Total bicarbonate, mmol/L	21–28	29/2	28/1	24/1
Urea, mmol/L	2.7–7.5	3.9/0.6	5.7/0.6	6.9/0.2
Urine albumin, mg/L	0–20	15/6	11/3	13/4
Albumin–creatinine ratio, mg/mmol	<2.5	1.7/0.6	1.3/0.2	1.3/0.5

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.

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

thyroid stimulating hormone were within the reference range, and did not show any change from the pre- to the post-CW period. Creatine kinase decreased when on CW, and increased on its cessation. Alkaline phosphatase was low when on CW, and it stayed below the RI in CW's absence. Serum copper was 14.3µmol/L (RI 10–22) and zinc 10.4µmol/L (RI 11–18). There is no record of results for these trace metals pre-CW consumption. Whether the low zinc status is a result of micronutrients in CW or is due to dietary change remains unanswered. Reduction in eGFR is noticeable and raises concerns about the daily quantity of CW that can be safely consumed by people with diabetes or when on potassium-retaining medication. Daily use of LoSalt can provide around 35mmol of potassium; its use in diabetes is clinically detrimental.[8] In comparison, potassium load from CW (<50mmol daily in the present case) is likely to be equally harmful in patients with reduced renal excretion and in those with prescription drugs affecting the renin-aldosterone system.

The subject's mean blood pressure before, during and after cessation of CW drinks was 146/82mmHg, 140/74mmHg and 140/77mmHg, respectively (Figure 1). During the CW consumption period there was a 10% decrease in the diastolic blood pressure (t=5.82, p<0.0003) and a statistically non-significant reduction (4%) in the pulse pressure (t=0.53, p>0.61).

In medical emergency, as a temporising alternative to standard intravenous fluids, fresh CW use resulted in potassium increase of around 1.5–2.8mmol/L.[10] In contrast to fresh CW, 100ml of Coco Vita contains zero fat and protein, carbohydrates 5g, salt 0.03g, potassium 195mg (5mmol), vitamin C 40mg, energy value 72kJ (18kcal), and 99% CW with 1% fruit sugars.[9] The recommendation for dietary potassium in adults is around 90mmol/day.[11] At this level of potassium intake there is a reduced risk of cardiovascular disease, stroke and coronary heart disease. Research work on rats suggesting CW has a protective effect on the kidneys in diabetes needs independent verification;[12] there is no similar research reported on human subjects.

In health, CW consumption in moderation may provide and restore some lost micronutrients. However, increased intake of potassium by patients with diabetes, including those with reduced renal excretion[13–19] or on potassium-retaining medication,[5,20] has the potential to increase the risk from hyperkalaemia. These groups of people should be advised on the potential harm from consumption of excessive amounts of CW drinks.

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

Declaration of interests

There are no conflicts of interest declared.

References

1. Zelman KM. The truth about coconut water. www.webmd.com/food-recipes/truth-about-coconut-water [last accessed 24 July 2015].
2. Prades A, *et al.* Coconut water uses, composition and properties: a review. *Fruits* 2012;67:87–107.
3. Vigliar R, *et al.* Biochemical profile of coconut water from coconut palms planted in an inland region. *J Pediatr (Rio J)* 2006;82:308–12.
4. Yong JWH, *et al.* The chemical composition and biological properties of coconut (*Cocos nucifera L.*) water. *Molecules* 2009;14:5144–64.
5. The electronic Medicines Compendium (eMC). Lisinopril. Summary of Product Characteristics. https://www.medicines.org.uk/emc/medicine/30946 [last accessed 30 November 2015].
6. Omron M6, Omron Healthcare Co Ltd, Kyoto, Japan. www.omron-healthcare.com/en/products/bloodpressuremonitoring.
7. Devgun MS. Delay in centrifugation and measurement of serum constituents in normal subjects. *Clin Physiol Biochem* 1989;7:189–97.
8. Ayach T, *et al.* Postoperative hyperkalemia. *Eur J Int Med* 2015;26:106–11.
9. Vita Coco: vitacoco.com [last accessed 24 July 2015].
10. Campbell-Falck D, *et al.* The intravenous use of coconut water. *Am J Emerg Med* 2000;18:108–11.
11. WHO. Guideline: Potassium intake for adults and children. Geneva: World Health Organisation, 2012.
12. Nwangwa EK. The reno-protective effects of coconut water on the kidneys of diabetic Wistar rats. *J Health Sci* 2012;2:1–4.
13. Doorenbos CJ, Vermeij CG. Danger of salt substitutes that contain potassium in patients with renal failure. *BMJ* 2003;26:35–6.
14. Cheng C-J, *et al.* Acute hyperkalemic paralysis in a uremic patient. *J Nephrol* 2005;18:630–3.
15. Hakimian J, *et al.* Death by coconut. *Circ Arrhythm Electrophysiol* 2014;7:180–1.
16. Rees R, *et al.* Coconut water-induced hyperkalaemia. *Br J Hosp Med (Lond)* 2012;73:534.
17. Medeiros VFLP, Medeiros AC. Therapeutic use of coconut water. *J Surg Clin Res* 2012;3:75–83.
18. Adams W, Bratt DE. Young coconut water for home rehydration in children with mild gastroenteritis. *Trop Geograph Med* 1992;44:149–53. http://europepmc.org/abstract/med/1496708.
19. Eiseman B, *et al.* Clinical experience in intravenous administration of coconut water. *AMA Arch Surg* 1954;69:87–93. doi:10.1001/archsurg.1954.01270010089013.
20. European Food Safety Agency. Tolerable upper intake of levels for vitamins and minerals. 2006, 409–22. www.efsa.eu.int [last accessed 24 July 2015].

