Death by Coconut

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Coconut water has become an increasingly popular sports drink because of its natural ingredients, electrolytes, and mineral content. Marketing has focused on the product’s low levels of fat, calories, and carbohydrates while promoting health benefits that are as-of-yet unproven. Coconut water, when consumed in excess, has been noted in case reports to cause severe hyperkalemia.1 We present a case of a 42-year-old otherwise healthy man who presented to our emergency department following an episode of exertional syncope after consuming multiple servings of coconut water.

Case Presentation

A 42-year-old black man without any medical history presented to our hospital after a syncopal episode. He was playing tennis outdoors all day in temperatures in excess of 90° Fahrenheit. He reported drinking a total of eight 11-ounce bottles of coconut water throughout the day. He experienced the sudden onset of lightheadedness and was witnessed to have lost consciousness. He did not experience any significant head trauma and regained consciousness almost immediately. Afterward he complained of generalized weakness and lightheadedness.

He was brought to the emergency department by ambulance. There he was noted to have a blood pressure of 67/45, a regular pulse in the 50s, and a temperature of 36.3°C. On examination, he was disoriented and his skin was warm. There was no jugular venous distention. Lungs were clear to auscultation. Cardiovascular examination disclosed a regular bradycardic rhythm without any murmur or gallop. Point of maximal impulse was not displaced. Abdominal examination was benign. There was no edema on examination of the lower extremities, and his distal pulses were present but thready.

ECG demonstrated sinus arrest with a junctional escape rhythm at a rate of 51 beats per minute, a high take-off of coved ST-segment elevations resembling a Brugada pattern and peaked T waves (Figure [A]).2 He then had a prolonged pause associated with altered mental status. He was paced externally with consistent capture, and atropine 0.5 mg was administered intravenously. Subsequently his intrinsic sinus rhythm returned with normal AV conduction. A temporary transvenous pacemaker was placed.

Subsequent laboratory evaluation disclosed a serum potassium of 7.8 mmol/L, blood urea nitrogen of 22 mg/dL, and creatinine of 2.1 mg/dL. Initial creatinine kinase was 1615 U/L with a normal MB fraction and troponin. He was admitted to the cardiac care unit for rhabdomyolysis, acute kidney injury, and hyperkalemia. He was aggressively rehydrated with intravenous fluids. The hyperkalemia was treated with calcium gluconate, albuterol sulfate by nebulizer, sodium polystyrene sulfonate, and intravenous dextrose and insulin.

During his hospital stay, he remained in normal sinus rhythm without any further significant arrhythmic or hemodynamic events. His potassium down-trended to normal levels and his renal function improved (Table I in the Data Supplement). Repeat ECG showed sinus bradycardia and memory T-wave inversions secondary to recent pacing (Figure [B]). He was discharged in stable condition on day 3 with close follow-up planned with his primary physician and cardiologist. He was also instructed to avoid coconut water, remain well hydrated, and avoid excessive exercise in the extreme heat.

Discussion

Coconut water has become a popular oral rehydration solution in the United States and Europe, in part, because of marketing and celebrity endorsement. The makers of coconut water purport health benefits that have not yet been verified by clinical trials. Marketing materials claim that the drink prevents kidney stones, lowers cancer risk, and strengthens the immune system.3 Coconut water is a hypotonic solution that is more acidic than plasma. It contains high concentrations of sugars and potassium, with lower amounts of sodium, chloride, and phosphate. There have been documented cases of the use of coconut water as an intravenous solution in developing countries where saline solution was not readily available.3

When consumed in excess, coconut water has been shown in case reports to cause severe hyperkalemia.3 Eight ounces of coconut water contain ≈600 mg of potassium. One popular brand has as much as 690 mg per serving (Table II in the Data Supplement). The suggested adequate daily intake of potassium for an adult without chronic kidney disease is ≈4.7 g per day.7 Our patient, who drank 8 servings of coconut water within a short period of time would have ingested ≈5.5 g of potassium. In combination with acute kidney injury and rhabdomyolysis, his potassium level was life-threatening.
Our patient did not have any significant predisposing factors for rhabdomyolysis on presentation. His body mass index is 23 kg/m², which is in the normal range. He had no history of alcohol or drug abuse (including cocaine and amphetamines). His phosphorus level was 2.2 mmol/L at presentation, likely not low enough to precipitate rhabdomyolysis on its own. He had no evidence of infection during the admission, such as fever or leukocytosis.

Our case demonstrates the potential dangers associated with excessive consumption of potassium-rich coconut water. To our knowledge, this is the first reported case of a life-threatening bradyarrhythmia associated with the use of coconut water. Patients who consume such beverages, especially those with renal impairment, should be advised of the potential dangers associated with drinking unrestricted volumes.

Disclosures
None.

References

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Figure. 

**A**, ECG showing junctional rhythm with tall, deformed T-waves. **B**, ECG after treatment showing sinus bradycardia and memory T-wave inversions secondary to recent pacing.
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