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**Acute kidney injury associated with consumption of starfruit juice: A case report**

Zuhary TM *et al*. Starfruit induced acute kidney injury

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**Abstract**

BACKGROUND

This study aims to highlight the potential serious complications of acute kidney injury (AKI) resulting from the consumption of excessive amounts of starfruit, a common traditional remedy.

CASE SUMMARY

A 78-year-old male with a past medical history of hypertension, diabetes mellitus and hyperlipidemia without prior nephropathy presented to the emergency department (ED) with hiccups, nausea, vomiting and generalized weakness. In the preceding 1 wk, he had consumed 3 bottles of concentrated juice self-prepared from 1 kg of small sour starfruits. His serum creatinine was noted to be 1101 μmol/L from baseline normal prior to his ED visit. He was diagnosed with AKI secondary to excessive starfruit consumption.

CONCLUSION

Consumption of starfruit can cause acute renal failure, with a good outcome when promptly identified and treated.

**Key Words:** Acute kidney injury; Acute renal failure; Starfruit; Hemodialysis; Case report

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**Core Tip:** Physicians should have a high index of suspicion on possible interactions and toxicities that may occur with the use of traditional medications in combination with prescription drugs in susceptible patients. This report highlights the toxicity of starfruit when consumed as a traditional remedy for diabetes mellitus resulting in acute kidney injury.

**INTRODUCTION**

The starfruit (Averrhoa carambola) is a popular fruit in tropical countries due to its nutritional and medicinal benefits[1], and is used to treat various ailments such as diabetes mellitus, rheumatism, and cough. The starfruit is used as a traditional remedy in Asian countries such as Malaysia and Indonesia to treat diabetes mellitus due to its hypoglycemic properties[2]. Despite its frequent consumption, many people are unaware of the dangers of overindulging in starfruit. When consumed in large quantities, the fruit contains high levels of oxalic acid, which can be nephrotoxic. Starfruit-induced neurotoxicity and nephrotoxicity, which manifests as acute kidney injury (AKI) in individuals with underlying renal dysfunction, is well documented[3,4]. AKI in individuals with normal renal function is rare. We present a case report of AKI following the consumption of starfruit.

**CASE PRESENTATION**

***Chief complaints***

A 78-year-old male presented to the emergency department (ED) with hiccups, nausea, vomiting and generalized weakness.

***History of present illness***

In the preceding week, he had consumed 3 bottles of concentrated juice which were self-prepared from 1 kg of starfruits. Following ingestion of the third bottle of the fruit juice, he developed bouts of severe nausea and vomiting without abdominal pain or diarrhea.

***History of past illness***

He had a past medical history of hypertension, diabetes mellitus and hyperlipidemia.

***Personal and family history***

No significant family history.

***Physical examination***

On arrival at the ED, his vital signs were stable (temperature was 36.8°C, pulse rate 60 bpm, respiratory rate 18 breaths/min, and blood pressure 161/78 mmHg) and there was no pitting edema. Examinations of his cardiovascular, respiratory, abdominal and neurological systems were normal.

***Laboratory examinations***

Laboratory examination results are shown in Figure 1 and Table 1.

***Imaging examinations***

No imaging was undertaken.

**MULTIDISCIPLINARY EXPERT CONSULTATION**

The patient was initially seen in the ED and admitted under renal medicine for specialized care.

**FINAL DIAGNOSIS**

Acute kidney injury.

**TREATMENT**

The patient was treated with 4 sessions of hemodialysis and supportive care such as intravenous fluid. After each session of hemodialysis, blood tests to determine renal function were repeated. Progressive improvement in renal function was noted with each session of hemodialysis.

**OUTCOME AND FOLLOW-UP**

The patient's renal function returned to normal.

**DISCUSSION**

Starfruit has several toxins including caromboxin, an excitatory central nervous system stimulant and oxalate a nephrotoxic agent[5-7]. The sour type of starfruit has higher levels of oxalate than the sweet type. Homemade and medicinal supplements often have high levels of oxalate. When consumed in large amounts, especially when fasting or dehydrated, deposits of calcium oxalate crystals in the renal tubules lead to kidney damage[6]. Chronic kidney disease has been identified as a major risk factor for starfruit-induced kidney toxicity. Starfruit juice volume of approximately 25 mL is known to cause nephrotoxicity in patients with chronic kidney disease. Other known risk factors include dehydration, the amount of starfruit ingested, and consumption on an empty stomach. Patients with starfruit toxicity show gastrointestinal symptoms such as nausea, vomiting, and abdominal discomfort immediately after ingestion. These symptoms are believed to be due to the direct corrosive effects of dietary oxalates rather than systemic effects[8].This may be followed by a decrease in urinary output, which can lead to renal dysfunction and acute renal failure. Typical histological findings are the intraluminal and intraepithelial deposition of colorless oxalate crystals. There is no specific treatment for acute kidney damage from starfruit. In patients requiring renal replacement therapy, hemodialysis and hemoperfusion are preferred[9].

Our patient had no evidence of pre-existing renal failure or other contributory factors predisposing to AKI such as sepsis, dehydration, nephrotoxic drugs or obstructive urological causes based on clinical evaluation and tests done. In addition, over the course of four sessions of hemodialysis, he had gradual restoration of his renal function. The temporal relationship between the ingestion of large amount of fruit juice and the onset of symptoms in this case strongly suggests starfruit intoxication as the transient and reversible etiology likely due to resolving oxalate nephropathy.

**CONCLUSION**

In Asian countries where starfruit is commonly consumed as a traditional remedy, it is imperative for emergency physicians to be aware of starfruit toxicity in patients with unexplained AKI. This will help identify and treat these patients promptly to prevent starfruit-induced nephrotoxicity. Patient history is the key to reaching an early diagnosis. It is essential to prevent starfruit nephrotoxicity by educating the public and especially diabetics on the risks of consuming excess starfruit. Consumption of starfruit as a traditional remedy to control blood sugar levels in diabetics should be discouraged by educating the public.

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**Footnotes**

**Informed consent statement:** Informed written consent was obtained from the patient.

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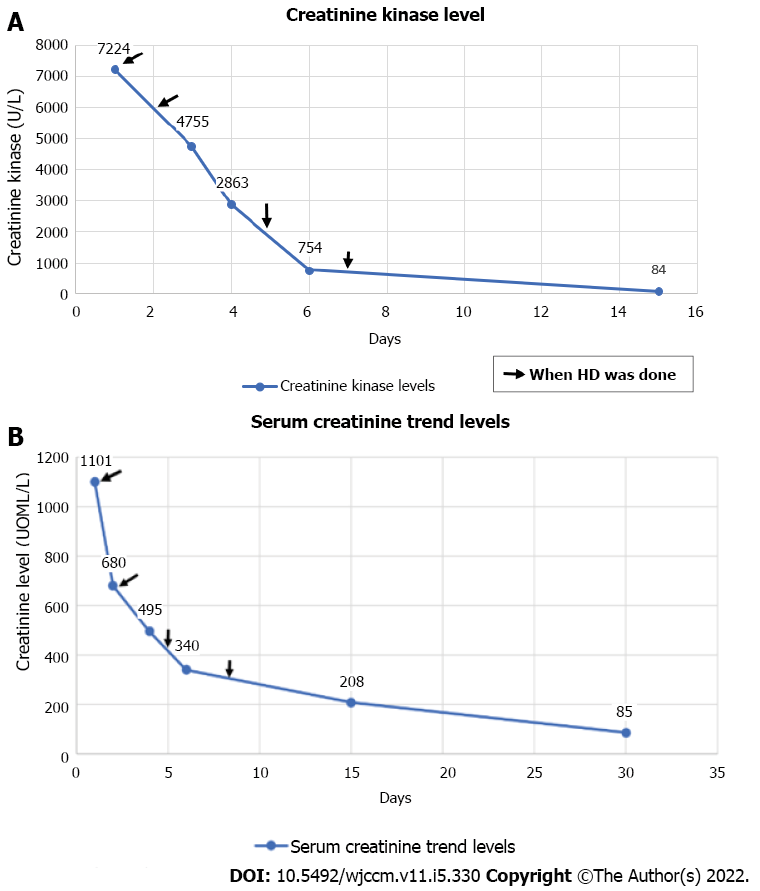
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**Figure Legends**



**Figure 1 Laboratory examination results.** A: Trend in creatinine kinase following hemodialysis; B: Trend in serum creatinine.

**Table 1 Trend in patient’s blood investigations**

|  |  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | **Day 1** | **Day 2** | **Day 3** | **Day 4** | **Day 5** | **Day 7** | **Day 13** | **Day 17** | **Day 24** | **Day 60** | **Day 135** |
| **Renal function** |  |  |  |  |  |  |  |  |  |  |  |
| Serum creatinine (μmol/L) | 1101 |  | 680 | 659 | 495 | 340 | 328 | 208 | 177 | 127 | 99 |
| Serum urea (mmol/L) | 38.1 |  | 23.1 | 27.1 | 22.0 | 14.5 | 25.2 | 17.4 | 10.6 | 12.4 | 6.2 |
| **Electrolytes** |  |  |  |  |  |  |  |  |  |  |  |
| Sodium (mmol/L) | 134 |  | 142 | 146 | 147 | 137 | 135 | 136 | 138 | 140 | 144 |
| Potassium (mmol/L) | 4.4 |  | 3.5 | 3.5 | 3.1 | 4.0 | 4.3 | 4.0 | 4.1 | 3.8 | 3.9 |
| Chloride (mmol/L) | 101 |  | 105 | 102 | 100 | 98 | 101 | 102 | 105 | 108 | 110 |
| Bicarbonate (mmol/L) | 15.9 |  | 22.8 | 26.8 | 31.1 | 24.6 | 28.3 | 23.7 | 24.6 | 23.5 | 24.9 |
| Magnesium (mmol/L) | 0.91 |  |  |  |  |  |  |  |  |  |  |
| **Liver function** |  |  |  |  |  |  |  |  |  |  |  |
| Total protein (g/L) | 60 |  |  |  |  |  |  |  |  |  | 76 |
| Serum albumin (g/L) | 32 |  |  |  |  |  |  |  |  |  | 41 |
| Total bilirubin (mmol/L) | 07 |  |  |  |  |  |  |  |  |  | 09 |
| Alkaline phosphatase (U/L) | 58 |  |  |  |  |  |  |  |  |  | 65 |
| Alkaline transaminase (U/L) | 57 |  |  |  |  |  |  |  |  |  | 17 |
| **Routine tests** |  |  |  |  |  |  |  |  |  |  |  |
| White blood cells (× 109/L) | 9.33 |  |  |  |  | 10.25 |  |  |  |  | 9.89 |
| Neutrophil (%) | 78.8 |  |  |  |  | 74.6 |  |  |  |  | 74.1 |
| Lymphocytes (%) | 11.1 |  |  |  |  | 11.6 |  |  |  |  | 15.9 |
| Hemoglobin (g/dL) | 12.3 |  |  |  |  | 13.8 |  |  |  |  | 14.1 |
| Platelet count (× 109/L) | 208 |  |  |  |  | 307 |  |  |  |  | 281 |
| **Coagulation** |  |  |  |  |  |  |  |  |  |  |  |
| APTT (secs) | 27.0 |  |  |  |  | 28.5 |  |  |  |  |  |
| Prothrombin time (secs) | 11.2 |  |  |  |  | 11.4 |  |  |  |  |  |
| **Other indicators** |  |  |  |  |  |  |  |  |  |  |  |
| Creatine kinase (U/L) | 7224 |  |  | 4755 | 2863 | 754 |  | 84 |  |  | 84 |
| PTH (pg/mL) | 11.0 |  |  |  |  |  |  |  |  |  |  |
| Urine creatinine (μmol/L) |  |  | 5233 |  |  |  |  | 3862 | 7747 |  | 8035 |

APTT: Activated partial thromboplastin time; PTH: Parathyroid hormone.



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