

Case Report

A case of renal failure developing in association with African mango consumption

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Abstract: Chronic kidney disease continues to represent a significant health problem in all societies. One of the main factors accelerating renal progression is nephrotoxins. The African mango is a plant added to many foods and commonly consumed in West Africa. No toxic effect has to date been shown. Our aim was to discuss the 42-year-old patient who became dialysis-dependent through developing rapid renal progression following 2.5-month African mango use. To the best of our knowledge, our patient is the first case of chronic renal insufficiency developing in association with African mango consumption.

Keywords: African mango, herbal medicine, renal failure

Introduction

Chronic kidney disease (CKD) continues to represent an important health problem. Thirteen percent of the adult population of the USA is known to have a glomerular filtration rate (GFR) below 60 ml/min [1]. As that figure shows, understanding the nephron loss mechanisms and the factors that affect progression in CKD, a major health problem, will permit significant advances to be made in treatment. One important factor that accelerates renal progression is nephrotoxins. These include many antibiotics, analgesics, contrast materials and particularly herbal drugs sold without prescription [2].

Irvingia gabonensis, also known as the African Bush Mango, is a traditional fruit widely used in West African cuisine and produced commercially. The pulp and seed are used in the preparation of many foods. Studies have shown that African mango seed extract has health benefits, is an effective antioxidant and has weight-reducing effects [3, 4]. A toxicity study in healthy rats showed that >2500 mg/kg per day of *Irvingia gabonensis* extract has no toxic effect on any organ. However, no information is available regarding the results of its use in rats or humans with kidney function disorder [5].

The purpose of this study was to discuss acute on chronic kidney failure developing in an obese patient under observation for CKD following 2.5-month use of African mango for weight loss purposes and subsequent dialysis dependence.

Case history

A 42-year-old patient under observation due to hypertension for seven years and hypertensive nephrosclerosis for one year had a creatine level of approximately 1.4-1.6. The patient was using amlodipine 10 mg and doxazosin 2×4 mg for hypertension, and blood pressure had been regulated. Three months previously she started using 2×500 mg African mango for slimming purposes without consulting a physician. After using the drug for 2.5 months, she became aware of swelling in the body and a decreased urinary level. The patient had begun experiencing nausea, vomiting and lethargy in the preceding few days, and applied to our clinic for that reason. At physical examination, her height was 1.50 m, weight 85 kg, body mass index 37.8 kg/m² and blood pressure 140/90 mmHg. 2+ edema was determined in the lower extremity, while other system examinations were normal. At biochemical tests his blood urea nitro-

African mango and renal failure

Table 1. Biochemical and hematological parameters of the patient

	One year ago	When started African Mango	After 2, 5 months (At the time of hospitalization)	When started hemodialysis	When discharged	After 6 months
Glucose (mg/dL)	90	95	89	92	85	95
BUN (mg/dL)	25	27	57.5	62.7	23.7	68
Cr (mg/dL)	1.6	1.4	5.88	6.92	5.12	6.13
Na (mmol/L)	138	135	138	137	133	139
K (mmol/L)	4.8	4.5	5.46	6.01	3.49	5
Ca (mg/dL)	9.8	9.5	8.7	8.9	7.4	8.5
P (mg/dL)	2.8	3.5	4.5	5.0	3.78	4.8
TP (g/dL)	7.8	8.0	7.8	7.9	7.6	7.7
Alb (g/dL)	3.9	3.7	3.8	3.65	3.84	3.9
Hb (g/dL)	13.2	12.5	10.3	10.8	10.6	11.4
HCT (%)	39.2	38.6	30.9	31.5	30.8	34.5
WBC (U/L)	5700	6300	6800	7500	7800	8800
Platelet ($\times 10^3/\mu\text{L}$)	155	210	225	250	245	225
hs CRP (mg/dL)			0.35	0.45		0.35
Urinalysis density		1015	1012		1015	
Protein (mg/dL)		-	-		-	
Leukocyte		-	-		-	

Abbreviations: BUN, Blood urea nitrogen; Cr, Creatinine; TP, Total protein; Alb, Albumin; Ca, Calcium; P, Phosphorus; Na, Sodium; K, Potassium; WBC, White blood cells; Hb, Hemoglobin; HCT, Hematocrit; PLT, Platelets; hs CRP, high sensitive C-reactive protein.

gen (BUN) level was 57.5 mg/dl, creatinine 5.88 mg/dl and albumin 3.8 gr/dl. Density of 1012 was determined at complete urine analysis, while protein was negative. Other laboratory tests are shown in the **Table 1**. Pulmonary imaging was normal. No pathological finding apart from left ventricular hypertrophy was determined at echocardiography. Grade 2 hypertensive retinopathy was determined at fundus examination. At renal ultrasonography kidney dimensions were right kidney 91 mm, parenchymal thickness 10 mm, left kidney 85 mm and parenchymal thickness 9 mm. A grade 2 increase was determined in parenchymal echogenicity, but no finding of urinary obstruction was detected. Renal Doppler ultrasonography was compatible with renal parenchymal disease. The patient's history revealed that she had been under observation for CKD for one year and that his creatine levels were approximately 1.4-1.6 mg/dl. Acute on chronic renal failure was suspected and the patient was admitted for observation and treatment. Blood and urine cultures were taken in order to detect any probable infection. The patient was given 3 l i.v. hydration daily. Urine output monitored over the subsequent days were approximately 2 l, although there was a rise in creatinine levels. The patient's nausea persisted, but there was no growth in the cultures. When the nausea, vomiting and creatinine levels increased, we decided to start hemodialysis. A hemodialysis catheter was attached to the jugular vein and dialysis began. After 15 days, the patient required 3 HD a week. Fever of 38.5°C and redness in the hemodialysis catheter exit site suggested catheter infection. The catheter was removed and vancomycin therapy at 1 g/h i.v. was started. No growth was detected in culture from the catheter, and treatment was concluded at the end of the first week. The patient was monitored for 28 days. There was no improvement in renal functions and the need for 3 HD weekly persisted. Over the next two months she continued on the 3 HD weekly program through a tunneled HD catheter. With no improvement in renal functions at the third month, permanent damage to renal functions associated with African mango drug taken for slimming purposes was suspected. The patient was diagnosed with chronic kidney failure and an arteriovenous fistula was opened. She was placed on a 3 chronic HD a week program. Observation and treatment continued for six

months, but there was no improvement in renal functions.

Discussion

CKD represents a significant health problem involving both high mortality and morbidity and high costs. Studies have determined that 13% of patients in the USA have GFR below 60 ml/min [1]. The most important cause of CKD is DM, the second being hypertension [2]. Our patient's seven-year history of hypertension and a one-year history of renal function disorder, left ventricular hypertrophy and Grade 2 hypertensive retinopathy at fundus examination suggested HT-related CKD.

Several studies have been performed on both the pathogenesis and treatment of CKD in recent years, and these have shown that treatment of various factors can slow the progression of the disease. These, known as reversible factors, include activation of primary disease, uncontrolled hypertension, proteinuria above 1 g, urinary system obstruction, urinary infection, use of painkillers and analgesics, exposure to other nephrotoxins, congenital or subsequent nephron loss, diseases causing a rise in intraglomerular pressure, DM, pregnancy, dyslipidemia, cigarette use, vitamin D deficiency, hyperuricemia and metabolic acidosis [6-9]. None of these reversible factors was present in our patient, and we think that the factor leading to rapid progression in CKD was African mango he used for slimming purposes.

African mango (*Irvingia gabonensis*) is a regional leafed plant grown in humid tropical forests in West Africa. The root, leaves and bark of the mango are used in traditional medicine in Africa. The leaf is used to treat diarrhea, colic and dysentery, as well as skin diseases [10]. Extract obtained from the seed has been shown to reduce blood glucose levels in diabetic patients [11]. In addition, clinical studies in recent years have shown it has a weight-reducing effect in obese patients [3]. Kothari et al. showed that the administration to rats of African mango seed extract at 2500 mg/kg over 90 days caused no subchronic organ toxicity, including in the kidney [5]. However, the fact that all the rats used in that study were healthy animals makes it impossible to predict whether its use will give rise to any toxicity in individuals with CKD. Our patient's creatinine levels had

been approximately 1.4-1.6 mg/dl for the previous year. When he began using African mango at 2×500 mg/day his creatinine level rose from 1.4 mg/dl to 5.88 mg/dl. Since no other reversible factor was present in the patient, who continued to require dialysis in the subsequent period, we thought that rapid renal progression had developed in association with African mango use. This makes our case report the first of its kind.

Non-prescription herbal medicine is frequently consumed in modern society. The World Health Organization reports that 70%-80% of the population use herbal medicines in the treatment of various diseases [12, 13]. The reasons why herbal medicine exhibit no toxic effects in vivo and in vitro but give rise to renal toxicity during clinical use are thought to be incorrect descriptions of herbal plants or inaccurate description of the dose that will exhibit toxic effects, herbal plants being contaminated with various drugs, heavy metals or hormones or interaction with conventional drugs [12]. Various studies have shown that the incidence of CKD is higher in countries where herbal medicines are used on an intensive basis. While the mechanism of the nephrotoxic effect is not completely clear, there are reports of acute tubular necrosis, acute interstitial nephritis, Fanconi syndrome, papillary necrosis, urinary tract neoplasia and chronic interstitial renal fibrosis [12, 13]. The reason why African mango use led to rapid progression in CKD in our patient may be that the dose at which the drug will not be toxic in rats or human beings with CKD is unknown and that at 2×500 mg/day recommended in healthy individuals he may have taken an excessive dose and a nephrotoxic effect developed as a result. In addition, as with other herbal medicines, African mango may be contaminated with heavy metals, hormones or drugs. Had renal biopsy been performed in our case, we could have established the kind of damage African mango had established in the kidney. However, the fact that our patient's kidney dimensions were small, she was obese and had been under observation for CKD for one year led us not to perform biopsy. Biopsy would certainly have provided more illuminating information had it been performed.

In conclusion, consumption of herbal medicines for the purpose of both losing weight and treating primary disease by natural means is

excessive. African mango has begun being frequently used for slimming purposes. Consumption of these drugs without prescription or medical advice over the internet or through television advertising may lead to organ toxicity. In using herbal medicines such as African mango or any non-prescription drug, patients with CKD should seek the advice of a nephrologist. As in our case, patients may become dialysis-dependent when they could have been monitored for years without the need for dialysis.

Disclosure of conflict of interest

None.

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