

Case Report

A Case Report of Acute Kidney Injury Following the Use of Herbal Vaginal Pessary

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ABSTRACT

The demand and use of complementary and alternative medicine (CAM) is on the increase in many countries. Majority of the population using these herbal medicines are ignorant of their potential toxicities. We present an uncommon case of a 22 year old female who developed oliguric AKI following the use of herbal vaginal pessary. She was managed with haemodialysis and had complete renal recovery. This case highlights the potential nephrotoxicity of some herbal medication even when used locally and the urgent need to regulate CAM practice.

KEYWORDS: acute kidney injury, herbal, vaginal pessary

INTRODUCTION

The demand and use of complementary and alternative medicine (CAM) is on the increase in many countries.^[1,2] According to the World Health Organization Media Centre, about 80% of the population in some Asian and African countries depend on traditional medicine for their primary health care.^[1]

Majority of the population using these herbal medicines are ignorant of their potential toxicities because of the erroneous beliefs that medicines made directly from plants are natural and harmless.^[2,3] Some of the major challenges facing CAM practice, especially in developing countries, include poor regulation and standardization by appropriate regulatory institutions;^[4] hence, the side effects profile of these medicines are largely unknown.

Herbal medication accounts for 11–37.5% of acute kidney injury (AKI) managed in the hospital.^[5-9] It is responsible for up to 50% of the cases of acute toxic nephropathy.^[8] There is also the possibility of an underestimation of the contribution of herbal medicine to etiology of AKI in the tropics due to failure to elicit the history owing to ignorance of the physician or denial by the patient when the history is being elicited because of fear of social stigmatization.

We present a case of a 22-year-old female student who developed AKI following the use of herbal vaginal pessary, thereby highlighting the potential nephrotoxicity of herbal medicine, even when locally administered and the need for strict regulation of these medicines by regulatory authorities.

CASE REPORT

We present a 22-year-old female tertiary college student who was referred to our hospital on account of 8-day history of progressive decrease in urinary output and 2-day history of persistent hiccups.

Her symptoms started following insertion of a locally prepared herb (semi-solid) into her genital tract which she procured from a traditional doctor in an attempt to terminate an unwanted pregnancy at about 5 weeks gestation.

A day after the use of the herbal medicine, she developed generalized abdominal pain. There was associated vomiting, although the quantity was less than 500 ml. There was no history of fever, passage of loose stool, jaundice, or foul smelling vaginal discharge. Six days after the use of herbal medication, she noticed progressive decrease in her urinary output and at the time of presentation she was anuric. There was no associated swelling of the legs or face. There was no history of seizures, altered consciousness, or difficulty in breathing. There was history of persistent hiccup that started 2 days before referral to our hospital.

At presentation, she was conscious, not in distress, well hydrated, afebrile, not pale, anicteric, and without pedal swelling. Her pulse was 96 beat per minutes, regular, with

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Access this article online

Quick Response Code:



Website:
www.ajmhs.org

DOI:
10.4103/2384-5589.209485

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How to cite this article: Adejumo OA, Akinbodewa AA, Ogunleye A, Agoi OF, Abolarin OS. A case report of acute kidney injury following the use of herbal vaginal pessary. *Afr J Med Health Sci* 2017;16:65-7.

normal volume; the blood pressure was 110/70 mmHg, and the heart sounds were normal. Respiratory examination was essentially normal. She had tenderness in the epigastric and right iliac fossa regions, but there was no palpably enlarged organ.

Urinalysis showed pH of 6.0, the specific gravity of 1.020, a protein, glucose, and blood were negative while microscopy showed granular cast. A diagnosis of acute kidney injury secondary to herbal vaginal pessary was made.

Investigation results at presentation

The following results were observed at presentation: Urea: 33.5 mmol/L, creatinine: 1035 µmol/L, sodium: 117 mmol/L, potassium: 4.2 mmol/L, chloride: 95.1 mmol/L, and bicarbonate: 10.8 mmol/L. The white cell count was 11,000 cells/cmm, hematocrit was 33%, and the platelet count was 172,000 cells/cmm. The liver function test was essentially normal. Abdomino-pelvic scan showed normal liver and bulky uterus with product of conception. The kidneys were normal-sized but there was increased echogenicity.

She had manual vacuum aspiration of retained product of conception and was placed on levofloxacin and metronidazole. Repeat pelvic scan showed empty uterus. She had three sessions of hemodialysis and was discharged on request due to financial constraint. She was subsequently followed up in the out-patient clinic. At the time of her last clinic visit which was 90 days post-presentation, she was in stable clinical state, and her renal function showed complete renal recovery with an estimated glomerular filtration rate of 79 ml/min/1.72 m² [Table 1].

DISCUSSION

This case is peculiar because majority of the cases of AKI from herbal medication followed oral ingestion unlike this index case where AKI resulted from insertion of herbal vaginal pessary in an attempt to terminate an unwanted pregnancy. This showed that some of these herbal medications could be highly nephrotoxic. The constituent of the herbal medication

used by this patient could not be identified despite several attempts that were made which are similar to previous reports.^[9-11] However, Kadiri *et al.*^[12] identified some plants that have been used in traditional herbal preparation such as leaves and bark of mango (*Magnifera indica*), shoots of cashew leaves (*Anacardium occidentale*), paw paw leaves (*Carica papaya*), lime leaves (*Citrus aurantifolia*), *Solanium erianthum*, *Morinda lucida* leaves, and bark of *Azadirachta indica* leaves as causing acute renal failure in Southwest Nigeria.

Kidneys are particularly vulnerable to toxic injury because of their high blood flow rate, large endothelial surface area, high metabolic activity, and active uptake by tubular cells. The nephrotoxic effects of herbal medicines may occur through one or more common pathogenic mechanisms such as alteration of intra-glomerular hemodynamics, tubular cell toxicity, inflammation, crystal nephropathy, rhabdomyolysis, and thrombotic microangiopathy.^[12,13]

Factors contributory to nephrotoxicity of herbal medications are adulteration of indigenous medicines, incorrect identification by inexperienced personnel which may lead to substitution of a medicinal plant with a toxic one, and concomitant administration of other nephrotoxic medicines that may also potentiate herbal nephrotoxicity.^[13]

Most patients with acute toxic nephropathy from herbal medication present with oliguric type of AKI as seen in this patient.^[9-11] Spectrum of renal syndromes from herbal medications include electrolyte abnormalities, hypertension, acute and chronic interstitial nephritis, papillary necrosis, urogenital tract cancer, urolithiasis, Fanconi's syndrome, and acute tubular necrosis.^[14] The presence of granular cast, the absence of red blood cells on microscopy and the clinical course of complete renal recovery in this patient are suggestive of acute tubular necrosis. However, this was not confirmed in this patient because renal biopsy was not done on account of financial constraint. Kidney injury may either be the sole manifestation or part of a multisystem involvement that includes acid-base disturbances, liver failure,

Table 1: Biochemical profile of the patient

Number of days post-presentation	1	6	12	19	26	40	51	90
Urea (mmol/L)	33.5	17.3	22.6	16.3	16.3	2.8	2.8	5.0
Creatinine (µmol/L)	1035	754	736	451	346	173	162	98
Sodium (mmol/L)	117	126	131.6	135.2	136.4	111.7	138	137.8
Potassium (mmol/L)	4.2	3.0	4.3	4.0	4.2	4.4	4.1	3.8
Chloride (mmol/L)	95	104	121	110	119	112	110	111
Bicarbonate (mmol/L)	10.8	17.2	24.8	18.3	18.2	18.4	17.8	16.9

Reference values: Urea (2.5–5.8 mmol/L), creatinine (50–132 µmol/L), sodium (120–140 mmol/L), potassium (3–5 mmol/L), bicarbonate (20–30 mmol/L), chloride (96–110 mmol/L).

neurologic abnormalities, disseminated intravascular coagulation, or respiratory failure.^[13] In our patient, AKI was the sole manifestation of the toxic effect of the herbal medicine.

Management of AKI from herbal medication may require renal replacement therapy and it improves the overall outcome when it is available and accessible as seen in this patient. Other factors that possibly contributed to a favorable renal and overall outcome in this patient were young age and absence of other co-morbidities such as hepatotoxicity.

CONCLUSION

This case highlights the potential nephrotoxicity of some herbal medication, even when used locally. There is therefore urgent need for government and regulatory authorities to regulate and standardize CAM practice. The public should also be educated on the potential health risks of herbal medications.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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