ACUTE RENAL FAILURE FOLLOWING JERING INGESTION

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ABSTRACT

We report two cases of acute renal failure that followed the ingestion of jering. Features of jering poisoning included clinical presentation of bilateral loin pain, fever, nausea, vomiting, oligo-anuria, haematuria and passage of sandy particles in the urine. Blood urea (40.8 mmol/l; 21.9 mmol/l) and serum creatinine (1249 μmol/l; 693 μmol/l) were markedly elevated. With conservative therapy which included rehydration with normal saline and alkalinisation of the urine with sodium bicarbonate, the acute renal failure resolved.

Keywords : Acute renal failure, jering.

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INTRODUCTION

Acute renal failure in the tropics has diverse aetiologies, including pre-renal ureamia secondary to fluid and electrolyte loss eg. infective diarrhoea, food poisoning, vomiting, acute dysentery, typhoid fever and cholera[1-3], intravascular haemolysis in G6PD deficient patients[4], snake bites[5,6], insect bites[6,7], leptospirosis[3,6,8] and malaria[6,7,9,10]. Acute renal failure following jering ingestion has been reported only in Indonesia. We report two cases of acute renal failure following excessive jering ingestion in Malaysia.

CASE REPORT

Case 1

A 21 year old Indonesian man was admitted with symptoms of anuria for 3 days, one episode of macroscopic haematuria, bilateral loin pain which radiated to the tip of the penis, fever and nausea. Prior to the onset of anuria, the patient passed sandy particles in the urine. There was no past history of trauma or renal disease. He had however consumed 20 cooked jering (Pithecellobium Jiringa) two days prior to his complaints.

Physical examination was unremarkable except for an elevated blood pressure (160/100 mm Hg). The renal punch was positive bilaterally.

On admission, the blood urea was 40.8 mmol/l, sodium was 131 mmol/l, potassium was 4.2 mmol/l, creatinine was 1249 μmol/l, haemoglobin was 11.8 g/dl, total white cell count was 8200/μl with a differential count of 73% polymorphs, 25% lymphocytes and 2% eosinophils, platelet count was 290,000/μl, blood glucose was 3.6 mmol/l, the liver profile was normal, anti-nuclear factor and rheumatoid factor were negative and the erythrocyte sedimentation rate was 53 mm/hr. Urinalysis showed numerous red blood cells and urine culture revealed no growth of organisms. Abdominal X-ray did not reveal radioopaque calculi. Ultrasound of the kidneys showed increased echogenicity with prominent pyramids. There was no hydronephrosis. The bipolar length of the right kidney was 10.5 cm and the left kidney was 10.0 cm.

The acute renal failure resolved spontaneously with conservative management which included rehydration with normal saline and alkalinisation of the urine with intravenous sodium bicarbonate. At the time of discharge (five days later) the blood urea was 9.7 mmol/l, sodium was 138 mmol/l, potassium was 3.4 mmol/l and creatinine was 114 μmol/l and the patient was passing adequate amounts of urine (2-3 litres/day).

Case 2

A 25 year old Malay man was admitted for oliguria of three days duration, haematuria, passing sandy particles in the urine, fever, chills, nausea, vomiting and bilateral loin pain. He had ingested 10 jering fruits three days prior to the onset of oliguria.

On examination, he was found to be hypertensive (160/110 mm Hg). The renal punch was positive bilaterally. The rest of the examination was unremarkable.

The haemoglobin was 15 g/dl, total white cell count was 12800/μl, blood urea was 21.9 mmol/l, sodium was 132 mmol/l, potassium was 3.4 mmol/l, creatinine was 693 μmol/l, the anti-nuclear and rheumatoid factor were negative. Urinalysis showed numerous red blood cells and urine culture revealed no growth of organisms. Abdominal X-ray did not reveal radioopaque calculi. Ultrasound of the kidneys did not show any calculi or features of obstruction. The bipolar length of the right kidney was 9.9 cm and the left kidney was 10.8 cm.

He recovered with conservative management which included rehydration with normal saline and alkalinisation of the urine with intravenous sodium bicarbonate. At time of discharge (five days later) the blood urea was 10.4 mmol/l, sodium was 138 mmol/l, potassium was 3.8 mmol/l and creatinine was 120 μmol/l.

DISCUSSION

Jering (Pithecellobium Jiringa) is a pod that is consumed by Malays as a delicacy. It is known by them that Jering has antidiabetic properties[11] and that excessive ingestion can cause oliguria, anuria and haematuria. Acute renal failure due to jering poisoning has mainly been reported in Indonesian journals[12,13]. It occurs predominantly in males (male to female ratio being 9:1) and has a seasonal incidence, peak incidence

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being between September and January. This peak incidence corresponds with the rainy season and the blossom time of jering[12]. The development of renal failure was not dependent upon the method of preparation and the number and age of the fruits consumed[12]. This is contrary to an earlier report which stated that the renal failure could be prevented by boiling, frying, roasting or cutting the fruits into chips[13]. Symptoms included dysuria (48%), haematuria (42%), vomiting (20%), loin pain (22%), anuria (16%) and turbid urine (20%)[12]. Time of onset of symptoms following ingestion of fruits ranged from 2 to 36 hours[12]. The prognosis is good and most patients recover from the renal failure. The pathogenesis of acute renal failure following jering ingestion is unclear. It has been postulated that djengkolic acid crystals are deposited in the urinary tract[12]. Djengkolic acid is a sulphated amino acid. The amino acid has been shown to be the thioacetal form of cysteine.

In both our cases, the temporal relationship of jering ingestion and the onset of acute renal failure suggest that jering ingestion was the probable cause of acute renal failure. History and physical examination excluded prerenal causes for the acute renal failure and abdominal X-ray and ultrasonography excluded obstructive causes. Urine cultures excluded urinary tract infection.

We are presently undertaking further studies to gather data on Jering consumption amongst Malaysian population and to determine the pathogenesis of acute renal failure following ingestion of jering.

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