

Djenkolism: An uncommon cause for Acute Kidney Injury.

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ABSTRACT

Djenkolism is a condition that results in acute kidney injury and occurs after the consumption of djenkol beans, which are a local delicacy and traditional medicine in some Southeast Asian countries. The pathogenesis of acute kidney injury secondary to djenkolism is not well understood but the current hypothesis is acute tubular necrosis secondary to renal obstruction due to djenkolic acid crystals (principal toxin of djenkol beans). This report describes a previously healthy 28-year-old man experiencing acute kidney injury after the consumption of djenkol beans. He presented with features of acute djenkolism which included suprapubic pain, dysuria and hematuria. With conservative treatment based on the principles of rehydration with normal saline and alkalinisation of urine with sodium bicarbonate, the acute kidney injury resolved. Healthcare practitioners in the Southeast Asian region need to consider this uncommon cause as one of the differentials of acute kidney injury.

Keywords: Acute Kidney Injury, Asia, Southeastern, Haematuria, Hydronephrosis, Hydroureter, Kidney Tubular Necrosis

INTRODUCTION

Djenkol beans are a local delicacy and a form of traditional medicine in some Southeast Asian countries. Djenkolism is a condition that affects a small proportion of people who consumes djenkol beans, resulting in acute kidney injury (AKI). The clinical presentation of djenkolism is varied but generally presents as a spasmodic loin to groin pain and AKI, with evidence of urinary obstruction. The onset of AKI appears to be independent of the method of preparation or the number and age

of the fruits consumed.^{1,2} The clinical presentation may range from mild to severe symptoms. If toxicity occurs after the first meal, this does not indicate subsequent meals would produce an adverse reaction. Toxicity may also present after many symptom-free meals.³ Symptoms may occur immediately or as late as 36 hours after consumption.¹ A history of acute djenkolism doesn't produce immunity or hypersensitivity to subsequent consumption of djenkol beans.⁴ In this case report, a previously healthy adult male developed AKI after the consumption of djenkol beans which resolved with rehydration with normal saline and alkalinisation of urine with sodium bicarbonate.

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CASE REPORT

A 28-year-old healthy man was referred from a private general practitioner to our tertiary hospital with difficulty in passing urine, vomiting, bilateral loin and suprapubic pain after the consumption of 20 pieces of "jering" (Figure 1: Djenkol beans, scientific name: *Archidendron Jiringa*) a few hours ago. He also experienced post-voiding dribbling, dysuria and hematuria.

Physical examination was unremarkable. On admission, a blood renal panel showed a creatinine of 274 $\mu\text{mol/L}$, urea of 6.5 mmol/L, with sodium of 137 mmol/L and potassium of 4.1 mmol/L. A full blood count showed a haemoglobin of 14.5 g/dl, total white cell count was 16,300/ μl and platelet was 204,000/ μl . Liver function test was normal. Urinalysis confirmed urine protein level of 30.0g/L, red blood cells of 250 cells/ μL , leucocyte of 10 WBC/ μL and nitrate negative. An ultrasound revealed bilateral mild hydronephrosis and proximal hydroureter; and tiny calculi in both kidneys. A subsequent CT urography revealed mild left hydronephrosis and proximal hydroureter. Urine culture was negative for growth after 48 hours.

He was managed conservatively with rehydration using normal saline and alkalisation of urine with sodium bicarbonate over 24 hours. A urinary catheter was inserted for continuous bladder drainage. He was also given oral sachet and cefuroxime antibiotic intra-

venously (IV) 1.5g stat followed by a regular dose of 750mg three times a day for 7 days. IV tramadol 50mg was given for analgesia and he was encouraged to take oral fluids.

After 7 days, the patient's symptoms including hematuria resolved. He was discharged in view of a reducing trend of blood Creatinine levels. Upon discharge, the blood urea was 3.4 mmol/L, sodium was 138 mmol/L, potassium was 3.8 mmol/L and the creatinine was 77 $\mu\text{mol/L}$.

DISCUSSION

Epidemiologic studies revealed that acute tubular necrosis (ATN) due to community-acquired infections are the commonest cause of AKI in the tropical region while cardiogenic shock, industrial accidents, drugs, trauma and renal transplantation rejection are the common causes in the developed world.⁴ The burden of AKI is further worsened in the tropical regions by some of the herbal medicines used by traditional healers.⁵ Djenkol beans are commonly known as krakos (Cambodia), jering (Malaysia), niang-yai (Thailand), jenkol, genkol, yiniking, yi-ring, ma-niang, cha-niang, niang, kra-niang.⁶ Djenkolism is a condition characterised by acute kidney injury following ingestion of djenkol beans. It is not commonly encountered but is an important etiology of AKI amongst natives of Southeast Asia. Djenkol beans are eaten raw at meal times to purify the blood.⁷ It is also a local snack in Southeast Asia, sold all year round in the markets and are consumed raw, roasted or fried.⁶ Despite being available during most of the year, some reports states that djenkolism has a seasonal incidence with the peak incidence being between September and January, which corresponds with the rainy season and the blossom time of djenkol beans.¹ Typically, a pungent odour is detected in the breath and urine after consuming djenkol beans.⁸



Figure 1: Djenkol beans, scientific name: *Archidendron Jiringa*. (Click to enlarge)

Most people are able to consume

djenkol beans without ill effects, however AKI occurs in a small proportion of the population. The pathogenesis of djenkolism is still not well understood. Experiments in rats and mice had been inconclusive, but the pathological findings are suggestive of ATN. The principal toxin has been identified as djenkolic acid, a sulphur-containing non-protein amino acid. The current hypothesis is ATN secondary to obstruction in the renal tubules due to djenkolic acid crystals.⁹ However, this has been difficult to prove due to: 1) acid crystals were not found in all animal models (histologic preparation may dissolve the crystals) and 2) renal biopsies are rarely performed on patients with acute djenkolism (one case report of human renal biopsy demonstrated findings of ATN).^{8,10}

Bunawan et al reported male predominance (70%) and common presenting signs and symptoms being colicky abdominal pain (70%), dysuria (66%), oliguria (59%) and hematuria (55%).¹¹ Needle-shaped crystals were inconsistently reported in the urine analysis. Creatinine elevation ranged between 1.7-14.1 mg/dL. Treatment was mainly rehydration and alkalinisation of urine. In few instances, dialysis, surgery, urethral and bladder irrigation or urethral stenting was required.

Similarly, Vachvanichsanong and Lebel reported a history of consuming djenkol beans causes an increased risk of hematuria in children by four times although the amount consumed was not a factor. However, there is no relationship between consumption of djenkol beans and other urine abnormalities, including pyuria and crystaluria.¹²

In our case, the most probable cause of AKI would be associated with the recent history of ingestion of djenkol beans. History, physical examination and investigations had ruled out other pre-renal causes for the AKI. The symptoms are also supportive of djen-

kolism. Imaging studies supported an obstructive pathology and urine culture ruled out an infection.

CONCLUSION

There are few reports of djenkolism in the medical literature but as treating doctors, we have to be aware of this condition as it is uncommon and remains an important cause of AKI in Southeast Asia. Knowledge of its clinical presentation, suggested pathophysiology and principles of therapy are relevant for healthcare professionals in the Southeast Asian region.

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REFERENCES

- 1: 1. Suharjono A, Sadatun OE. "Djengkol" intoxication in children. *Paediatr Indones.* 1968 Jan-Feb; 8(1): 20-9.
- 2: Moenadjt WR, Sadatun OE, Oey KN. Soal kerat-junan djengkol. *Madj Kedokt Indon.* 1963; 2: 51-5.
- 3: Reimann HA, Sukaton RU. Djenkol bean poisoning (djenkolism): A cause of hematuria and anuria. *Am J Med Sci.* 1956 Aug; 232(2): 172-4.
- 4: Chugh KS, Sitprija V, Jha V. Acute renal failure in tropical countries. In: Davidson AM, Cameron JS, Grunfeld JP, et al. editors. *Oxford textbook of clinical nephrology.* Oxford: Oxford University Press; 2005: 639-58.
- 5: Burdmann EA. Acute kidney injury in the tropics: Introduction. *Semin Nephrol.* 2008 Jul; 28 (4): 319.
- 6: Barceloux DG. Djenkol Bean [Archidendron Jiringa (Jack) I. C. Nielsen]. *Dis Mon.* 2009 Jun; 55(6): 361-4.

- 7: Ong HC, Norzalina J. Malay Herbal Medicine In Gemencheh, Negri Sembilan, Malaysia. *Fitoterapia*. 1999;70(1):10-4.
 - 8: Segasothy M, Swaminathan M, Kong NC, Bennett WM. Djenkol bean poisoning (djenkolism): an unusual cause of acute renal failure. *Am J Kidney Dis*. 1995;25(1):63-6.
 - 9: Areekul S, Kirdudom P, Chaovanapricha K. Studies on djenkol bean poisoning (djenkolism) in experimental animals. *Southeast Asian J Trop Med Pub Health*. 1976;7(4):551-8.
 - 10: Areekul S, Muangman V, Bohkerd C, Saenghirun C. Djenkol bean as a cause of urolithiasis. *Southeast Asian J Trop Med Public Health*. 1978 Sep; 9(3):427-32.
 - 11: Bunawan NC, Rastegar A, White KP, Wang NE. Djenkolism: case report and literature review. *Int Med Case Rep J*. 2014; 7: 79-84.
 - 12: Vachvanichsanong P, Lebel L. Djenkol beans as a cause Of hematuria in children. *Nephron*. 1997; 76(1): 39-42.
 - 13: H'ng PK, Nayar SK, Lau WM, Segasothy M. Acute renal failure following jering ingestion. *Singapore Med J*. 1991 Apr; 32(2): 148-9.
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