

## Acute kidney injury in patient with djenkolism: a case report



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### ABSTRACT

**Background:** Djenkolic poisoning or djenkolism is one of the causes of acute kidney failure common in Southeast Asia. Djenkol bean or jering (*Archidendron pauciflorum*) is one of the foods commonly found in Southeast Asia. These seed plants are usually processed by boiling or frying, but often they are also consumed raw. Djenkolism sometimes occurs, albeit infrequently after ingesting djenkol beans. The clinical presentation of djenkolism is varied but generally presents as a spasmodic loin to groin pain and acute kidney injury (AKI), with evidence of urinary obstruction.

**Case description:** We report a previously healthy 40-year-old male developed AKI after consuming a large amount of uncooked djenkol beans, which was resolved with rehydration with normal saline and conservative therapy.

**Conclusion:** We highlight the importance of healthcare practitioners, especially in the Southeast Asian region to consider this rare cause of acute kidney injury to provide early diagnosis and prompt treatments.

**Keywords:** djenkolism, acute kidney injury.

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### INTRODUCTION

Djenkolic poisoning or djenkolism is one of the causes of acute kidney failure in Southeast Asia, including Indonesia. Djenkol bean or jering (*Archidendron pauciflorum*) is one of the foods commonly found in Southeast Asia, including Indonesia, Thailand, Malaysia, and Myanmar. Djenkol bean is a food that is extremely popular with Indonesians, especially in Java and Sumatra. These seed plants are usually processed by boiling or frying, but often they are also consumed raw. Djenkolism sometimes occurs, albeit infrequently after ingesting djenkol beans.<sup>1</sup>

The clinical presentation of djenkolism is varied but generally presents as a spasmodic loin to groin pain and acute kidney injury (AKI), with evidence of urinary obstruction. The onset of AKI appears to be independent of the method of preparation or the number and age.<sup>2</sup> The purpose here is to report this rare case of djenkolism; a man previously healthy developed AKI after consuming djenkol beans, resolved with rehydration with normal saline and conservative therapy. This case report aims to enhance clinical

awareness to recognize djenkolism.

### CASE REPORT

A 40-year-old Sumatran presented to the emergency ward with 4 hours of severe bilateral flank pain. The patient experienced acute, bilateral, severe, and spasmodic flank pain, which radiated to the groin. He had trouble with urination for 9 hours prior. He also has a sulfurous odor on his breath. A family member later revealed that the patient had consumed about 1000 grams of djenkol beans three days before his pain started.

The physical examination revealed tenderness in the suprapubic and bilateral costovertebral regions. The vital signs were blood pressure 130/80mmHg, heart rate 110 bpm, and afebrile. A laboratory investigation revealed an elevated white cell count of  $14.7 \times 10^9$  /L, a hemoglobin count of 16.2 g/dL, a platelet count of  $186 \times 10^9$  /L, Blood Urea Nitrogen (BUN) level of 12.5 mg/dL (8- 23 mg/dL), and elevated creatinine level of 2.49 mg/dL (0.5-0.9 mg/dL). An abdominal radiograph (KUB) did not show any urological stones or free air. He was admitted to the urology service

for suspected renal stones and consulted to nephrology. Urgent abdominal ultrasonography (USG) was taken of the patient's abdomen; it revealed mild ascites and bilateral hydronephrosis. He was managed conservatively with rehydration using normal saline 1 ml/kg body weight over 24 hours. A urinary catheter was inserted for continuous bladder drainage. Paracetamol 1000 mg three times a day was given for analgesia, and he was also encouraged to take oral fluids. After five days, the patient's symptoms resolved. He was discharged because of increasing urine output and a reducing trend of blood creatinine levels. Upon discharge, the blood BUN was 13.3 mg/dL, and the creatinine level was 2.16 mg/dL.

### DISCUSSION

Djenkolism is a condition characterized by acute kidney injury following ingestion of djenkol beans. It is not commonly encountered but is an essential etiology of AKI amongst natives of Southeast Asia. Djenkol beans are eaten raw at mealtimes to purify the blood. It is also a local snack in Southeast Asia, sold all year round

in the markets and are consumed raw, roasted, or fried. Despite being available during most of the year, some reports state 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. Typically, a pungent odor is detected in the breath and urine after consuming djenkol beans. Most people are able to consume djenkol beans without ill effects; however, AKI occurs in a small proportion of the population.<sup>3,4</sup>

Djenkolism has a substantial male-to-female predominance (7:1) and needs large ingestion. One study in Thailand reported a higher incidence of hematuria in children with long-term djenkol bean consumption patterns.<sup>2</sup> Preventive measures for djenkolism are lacking, mainly because the incidence is low and sporadic. However, one study proposed boiling djenkol beans in dilute alkali to remove the djenkolic acid before consumption.<sup>1,5</sup> History djenkol consumption is not necessarily a predictor with following same clinical features further, as was right in the case. Our patient has a history of eating djenkol bean in large amounts, primarily consumed in an uncooked state, and has a sulfurous odor of the breath.<sup>1,4</sup> The djenkol bean contains a large amount of the djenkolic acid in the range of 0.3–1.3 g/100 g wet weight; 93% of the acid exists in a free state. The beans are eaten raw, fried, boiled, or roasted; sometimes, they are served as a dessert. Regardless of the preparation method, even one bean's ingestion causes the breath and urine to have a sulfurous odor.<sup>1</sup>

The pathogenesis of AKI from djenkol beans is not well understood. However, it is thought to occur due to either hypersensitivity or a direct toxic effect of djenkol bean metabolites resulting in AKI and/or urinary tract obstruction by djenkolic acid crystals, sludge, and/or possible ureteral spasms.<sup>2,6</sup> In most severe cases, this could cause post-obstructive renal failure.<sup>5</sup> Meanwhile, experiments in rats and mice had been inconclusive, but the pathological findings are suggestive of Acute Tubular Necrosis (ATN). The principal toxin has been identified as djenkolic acid, a sulfur-containing non-protein amino acid. The current hypothesis

is ATN secondary to obstruction in the renal tubules due to djenkolic acid crystals. 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).<sup>4,7</sup> 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 djenkolism. Imaging studies in our patient supported an obstructive pathology.

The two primary clinical syndromes of djenkolism are characterized as follows: a) mild presentation of suprapubic pain and hematuria resulting from transient ureteral obstruction due to djenkolic acid crystal; and b) severe presentation in which pain and hematuria are accompanied by hypertension, oliguria, and azotemia.<sup>7</sup> The pain stems from ureteric colic from crystal precipitation and stones. However, as in our case, the pain severity can exceed that seen with ureteric colic; the pain may be a more severe form of ureteric colic. The syndrome is likely determined by the amount of djenkol beans consumed. Regardless, early recognition is of the utmost importance. Therefore, awareness of the potential consequences of djenkol beans and inquiry into patients' djenkol bean consumption patterns are critical in general practice.

The mainstay of djenkolism treatment is aggressive hydration and alkalization of the urine to clear the crystal and relieve pain. In severe cases, renal replacement therapy may be required. This case presented classical findings associated with djenkol bean toxicity. Diagnosis of acute kidney injury in this patient was based on persistent anuria (24 hours) following the bean's ingestion. The diagnosis is made based on clinical presentation and laboratory data. It is reported that most cases resolve within three days with supportive care. Mild djenkolism requires no specific treatment except pain control and hydration.<sup>1</sup> Our patient recovered within five days of hydration

and conservative therapy. However, there are also reports of surgical interventions being used to relieve obstruction caused by the crystal, sludge, or calculi.<sup>2,5</sup>

## CONCLUSION

There are few reports of djenkolism in the medical literature, but as healthcare practitioners, we must be aware of this condition as it remains a significant cause of AKI in Southeast Asia. Knowledge of its clinical presentation, pathophysiology, and therapy principles is relevant, especially for healthcare professionals in the Southeast Asian region, to make swift and early recognition and prevent any misdiagnosis and mistreatment.

## ETHICAL CONSIDERATION

The patient had received information and given consent regarding data publication before any data collection.

## DISCLOSURE

The author reports no conflicts of interest in this work.

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## AUTHOR CONTRIBUTION

All authors equally contributed in preparing the manuscript.

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