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Djenkolism: Acute Kidney Injury Unresponsive to Conservative Therapy - A Case Report

Muhammad Ridho Bilhaq[1], Etriyel Myh[2]

[1]General Practitioner of Universitas Andalas Hospital, [2]Department of Urology, Universitas Andalas Hospital Padang, West Sumatera, Indonesia

Corresponding Author: Muhammad Ridho Bilhaq

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ABSTRACT

Djenkol beans (Archidendron jiringa), a popular food in Southeast Asia, can cause djenkolism - a condition leading to acute kidney injury due to the presence of djenkol acid. This case report details a 26-year-old male who presented with three days of anuria following the consumption of half a kilogram of djenkol beans. Upon admission, he exhibited severe abdominal pain, costovertebral tenderness, and elevated renal markers, leading to a diagnosis of obstructive uropathy caused by djenkolic acid crystals. Initial conservative treatment was ineffective, prompting the need for emergency ureteroscopy and placement of bilateral JJ stents to relieve the obstruction. Post-procedure, the patient experienced significant improvement, with a marked increase in urine output; post-obstructive diuresis in the first 24 hours, and resolution of abdominal pain, alongside improved renal function. This case underscores the importance of recognizing djenkolism as a potential cause of acute kidney injury, and advocating for timely diagnosis and intervention to mitigate kidney damage. Public awareness regarding the risks associated with djenkol consumption is also essential.

Keywords: Djenkolism, Obstructive Uropathy, Post-Obstructive Diuresis, Acute Kidney Injury

INTRODUCTION

Djenkol beans (Archidendron jiringa) are a popular food item in Southeast Asia, especially in Indonesia, Malaysia, and Thailand. These beans are characterized by their large, reddish appearance and dark purple pods. While many people enjoy them without problems, djenkol beans contain djenkol acid, which can cause djenkolism—a condition marked by acute kidney failure, blood in urine, and abdominal discomfort.[1]

CASE REPORT

A 26-year-old male patient was admitted with anuric for three days. Four days before admission, he had consumed half a kilogram of djenkol. He presented with abdominal pain radiating to the umbilicus, costovertebral tenderness, and persistent anuria. He was treated with crystalloid rehydration and bicarbonate for urine alkalinization, which produced 500 cc of reddish urine, but he remained anuric after catheterization. On the third day, he still had no urine output, with elevated urea levels at 53 mg/dl and creatinine levels at 5.0 mg/dl. He was diagnosed with anuria and obstructive uropathy resulting in acute kidney injury. Then, the patient is referred for further management. His blood pressure at 155/96 mmHg and a pulse rate of 78 beats per minute. An ultrasound showed bilateral hydronephrosis and an empty bladder, with serum urea at 87.1 mg/dl and serum creatinine at 9.3 mg/dl.

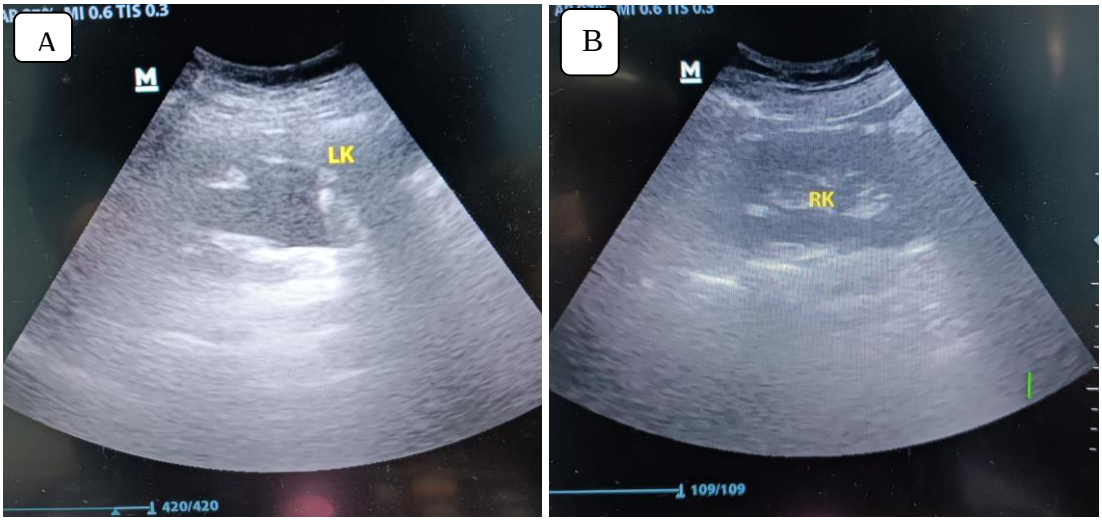

Figure 1. (A) hydronephrosis of left kidney (B) hydronephrosis of Right kidney

After fasting, he underwent an emergency ureterorenoscopy, which revealed djenkolic acid crystals obstructing both ureters. Bilateral JJ stents were placed to relieve the obstruction. Three hours after the procedure, his abdominal and flank pain had decreased, with blood pressure at 140/85 mmHg, pulse rate at 90 beats per minute, and urine output of 1000 cc accompanied by macroscopic hematuria. Six hours later, urine output increased to 2000 cc; after 24 hours, it reached 4500 cc. His abdominal and flank pain resolved, with blood pressure at 130/75 mmHg and pulse rate at 80 beats per minute. Kidney function improved, with serum urea dropping to 85 mg/dl and serum creatinine to 5.3 mg/dl. Three days following the procedure, his urine was clear, blood pressure was 125/70 mmHg, heart rate was 80 beats per minute, and kidney function was reassessed with serum urea at 20.3 mg/dl and serum creatinine levels at 1.2 mg/dl, the patient was discharged home.

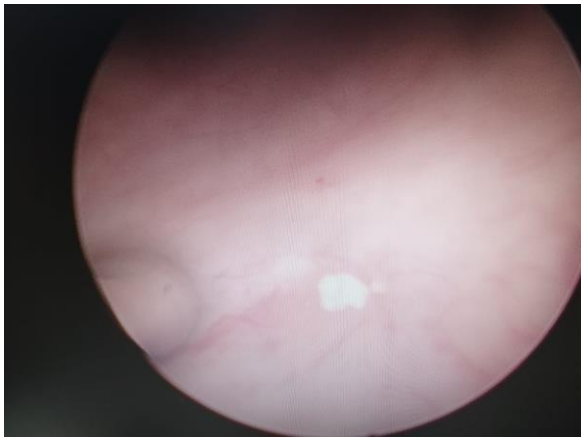

Figure 2. Djenkolic Crystals Shown in Ureteric orifice

DISCUSSION

This case report highlights the success of the endosurgery approach in a 26-year-old young male with djenkolism and acute kidney injury who is unresponsive to conservative treatment. The patient presented with complaints of anuria for the past 3 days. Djenkolism appears to present in two ways: 1) a mild case characterized by pain and hematuria, usually associated with temporary ureteral obstruction caused by djenkolic acid crystals; and 2) a severe cases where symptoms can include hypertension, oliguria, and azotemia. Cases of anuric and death are rare.[2]

Djenkol beans (Archidendron jiringa) are a popular food in Southeast Asia, particularly in Indonesia, Malaysia, and Thailand. These beans are known for their large, reddish appearance and dark purple pods. While many people consume them without issues, djenkol beans contain djenkol acid, which can lead to djenkolism - characterized by acute kidney failure, hematuria, and abdominal pain. Symptoms can appear within hours after consumption, and the risk of toxicity increases when eating raw beans or not staying adequately hydrated. Treatment primarily focuses on supportive care, including monitoring kidney function and ensuring proper hydration. While long-term consumption may lead to urinary problems, most individuals recover without lasting effects.[1]

This patient exhibited symptoms of anuria along with bilateral hydronephrosis. Bilateral obstructive uropathy can lead to anuria and a decrease in the glomerular filtration rate (GFR), which is proportional to the severity of the blockage. In the acute phase of bilateral obstruction, the significant reduction in GFR is directly linked to the obstruction itself.[3] Secondary acute kidney injury due to obstructive uropathy accounted for 10% of all acute kidney injury cases, often associated with a history of urinary tract infections, hematuria, renal stones, and previous urinary procedures. Imaging studies, including CT scans and ultrasounds, revealed the presence of hydronephrosis.[4] Hydronephrosis can occur unilaterally or bilaterally. Secondary hypertension may arise as a consequence of hydronephrosis, with some cases documented in a limited number of animal studies and clinical reports. Hydronephrosis can lead to reversible hyper-reninemic hypertension. While salt and water retention may contribute to this through increased TGF activity, the rapid normalization of blood pressure after relieving the obstruction suggests that systemic vasoconstriction is likely a key factor. Furthermore, renal parenchymal compression from cysts or subcapsular or extrinsic lesions should be assessed in patients presenting with hypertension.[5] In this patient presented hypertension, with no hypertension background before.

The pathophysiology of acute kidney injury resulting from djenkol ingestion was not completely understood. It was believed to stem from a mix of individual factors and the unique properties of the beans consumed. Possible mechanisms for kidney damage and hypersensitivity or a direct toxic effect from a metabolite of the djenkol bean, which can lead to acute kidney injury and urinary tract blockage due to djenkolic acid crystals, sludge, or potential ureteral spasms. In severe instances, this may lead to post-obstructive kidney injury.[6]

Intravenous fluid therapy and oral bicarbonate have been administered to the patient as conservative treatment by the previous healthcare facility; however, the patient did not show any clinical improvement and remained anuric. Aggressive hydration and urine alkalinization using natrium bicarbonate to enhance the solubility of djenkolic acid. In some severe cases of djenkolism with anuria, conservative treatment may be ineffective, necessitating surgical intervention. There are reports of procedures involving urethral irrigation, the placement of urinary catheters, and using stents to bypass or address urinary obstructions caused by djenkolic acid, sludge, and stones.[6]

The patient developed increased urine output following the insertion of bilateral JJ stents to manage the obstruction. Monitoring included checking consciousness, and vital signs, and tracking fluid intake and output balance. The patient remained conscious without any decline, maintained normal blood pressure, and did not exhibit signs of hypovolemic shock. After treating the blockage, urine output may increase, particularly in cases involving both kidneys or a single kidney obstruction. This increased urine output is caused by a notable decrease in receptor transport and reduced natrium reabsorption in the

descending limb of the loop of Henle. Typically, this condition resolves once fluid and electrolyte balance is restored. It is crucial to monitor consciousness levels and vital signs, provide electrolyte supplements, and gradually reduce intravenous fluid intake.[7]

This patient showed polyuria after bilateral JJ stents were performed, this condition was post-obstructive diuresis. Post-obstructive diuresis (POD) is a condition marked by increased urine output following the relief of ureteral obstruction, aimed at eliminating excess solute and fluid. Its pathophysiology involves several mechanisms. Initially, acute obstruction causes a temporary increase in renal blood flow, followed by vasoconstriction and tubular damage. The obstruction activates the renin-angiotensin system, further affecting renal function. Prolonged bilateral ureteral obstruction (BUO) leads to significant structural damage, resulting in apoptosis of renal cells. After relieving the obstruction, there is a decrease in the medullary concentration gradient due to vascular washout and down-regulation of natrium transporters, which can impair the kidneys' ability to concentrate urine, often resulting in vasopressin-resistant partial nephrogenic diabetes insipidus. Altered feedback mechanisms, including reduced tubuloglomerular feedback and potential damage to sympathetic nerve terminals from high intrarenal pressure, also contribute to POD. Additionally, changes in the regulation of local atrial natriuretic peptide further influence the functional changes seen after BUO relief.[8]

The kidney function in this patient has improved, indicated by enhanced levels of urea and creatinine following the procedure, which confirms successful diuresis. Research by Navarro et al. demonstrates that recovery of renal function after acute kidney injury from obstructive nephropathy can vary among individuals. While some patients may fully recover, others may experience a deterioration in kidney function. In a cohort study involving 34 ICU patients with AKI, it was found that 21% of those without chronic kidney disease still exhibited poor renal function three months post-treatment.[4]

CONCLUSION

Djenkol beans are commonly consumed in Indonesia. Healthcare providers should recognize djenkolism as a potential cause of acute renal injury; accurate diagnosis and prompt management can reduce kidney damage and potential complications. Conservative therapy and surgical intervention should be considered in the management of djenkolism. The public needs to be informed about the possible health risks of djenkolism.

Declaration by Authors

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