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Java Barb Fish Gallbladder–Induced Acute Kidney Injury and Ischemic Acute Hepatic Failure

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INTRODUCTION

In some Asian countries, the fish gallbladder is used as a part of traditional medicine. It improves fatigue, arthritis, and erectile dysfunction.[1] In Tasikmalaya, West Java, Indonesia, people believe if they consume fresh fish gallbladder of Java Barb (*Barbonymus gonionotus,* Cyprinidae family) or so-called *Tawes* will improve their health (Figure 1). Fish in the Cyprinidae family can be toxic.[2] In some reports, eating Java Barb may cause systemic toxicities, such as acute kidney injury and acute hepatic failure. The renal pathology usually demonstrates acute tubular necrosis. Some cases may require conservation treatment and renal dialysis.[1,3]

Acute kidney injury following consumption of fish gallbladder has been reported in several Asian countries, but it has never been reported before in Indonesia. Recently it also has been reported in the United States.[4]

CASE PRESENTATION

We report the case of a hypertensive 44-year-old man after consumption of 5 pieces of Java Barb gallbladders 3 days earlier. He came to the hospital with the chief complaint of shortness of breath beginning 3 days before admission. Symptoms worsened day by day and included profuse vomiting and decreased urine output. He developed edema in limbs and the scrotum within 3 days.

On physical examination, he looked fatigued but was communicative and not confused. He appeared mildly icteric. Vital signs were blood pressure 170/80 mm Hg, pulse rate 96 beats per minute, respiratory rate 28 breaths per minute, and temperature 36.8 °C. He displayed neck distension, with a pallor appearance. Respiratory rate showed an elevated rate with crackles and no wheeze. The cardiac examination showed heart enlargement. His abdomen was soft, with normal bowel sounds, the liver and spleen were not palpable, and there was no ascites. The neurological examination was normal.

Both his serum creatinine and serum urea were elevated to 17.7 mg/dl and 193 mg/dl, respectively. Meanwhile, he also developed ischemic acute hepatitis failure, with an alanine aminotransferase of 56 U/l, and aspartate aminotransferase of 536 U/l. He remained hypertensive (170/80 mm Hg). He was diagnosed as prerenal acute kidney injury and acute hepatic failure. Chest radiograph showed fluid overload (lung edema) and renal ultrasound detected no evidence of abnormalities (Figure 2).

During admission, the patient had been treated conservatively with restricted fluid management, bicarbonate tablet 3 times a day, amlodipine 10 mg once daily, and pantoprazole injection 40 mg once a day. The urine output was more than 2000 ml/24 hours, and no diuretics had been used. The patient did not require dialysis. After 10 days, he was discharged from the hospital with a serum creatinine concentration of

Figure 1. Java Barb fish (*Barbonymus gonionotus*, Cyprinidae family) or so-called *Tawes*.

4.46 mg/dl, urea 90 mg/dl, alanine aminotransferase 17 U/l and aspartate aminotransferase 42 U/l. A week after being discharged, his serum creatinine concentration reached 1.83 mg/dl and urea was 38 mg/dl.

DISCUSSION

There has been a tradition in Asia to ingest raw bile of the grass carp with the belief that it improves visual acuity, rheumatic disease, and health.[1,3] Although some symptoms of acute kidney injury and hepatitis following the consumption of raw fish gallbladders have been reported previously among persons living in Asia,[5] we found 2 cases of this syndrome described in the United States.[4] Because more and more people have emigrated from Asia to Western countries and physicians are often less aware of its toxic effects on liver and kidney, we present a case to illustrate the typical clinical course.

The initial symptoms of the fish gallbladder toxicities are nausea and vomiting, diarrhea, and abdominal tenderness, which usually occur 5 to 12 hours after ingestion. Elevated liver enzymes and icterus occur in 75% to 87% of patients. Acute kidney injury occurs in 72% to 87% of patients and manifests after day 3 to 6.[6]

Patnaik *et al.*[7] reported a patient after fish gallbladder consumption. The patient developed a decrease in urination followed by swelling of the legs and face. These symptoms were similar to our patient, who had decreased urine output and developed edema of limbs and the scrotum within 3 days.

Renal failure is the most commonly reported effect of fish gallbladder poisoning. It is believed that the fish gallbladder results in serious damage to renal tubules. The exact pathomechanism of toxicity is not well-established. The gallbladder of the Cyprinidae family contains 5 alpha-cyprinol sulfate, which is hepatotoxic and nephrotoxic. Mild poisoning may cause some

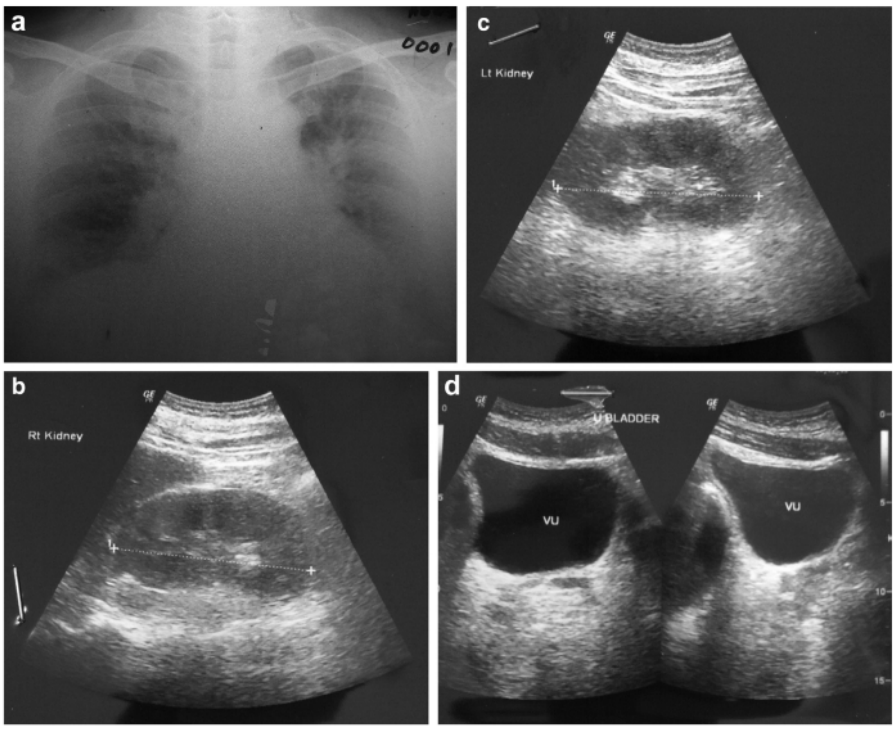

Figure 2. A chest radiograph showed fluid overload (lung edema) and renal ultrasound detected no evidence of abnormalities. (a) A chest x-ray showed lung edema. (b) The right kidney is normal. (c) The left kidney is normal. (d) The other organs, such as the bladder, are normal.

Table 1. Teaching points related to the case.

1. The fish gallbladder of Java Barb (*Barbonymus gonionotus*, Cyprinidae family) contains 5 alpha-cyprinol sulfate compound and may cause renal and liver toxicity.
2. Renal and liver damage can manifest as nausea and vomiting, diarrhea, low urinary output, and swelling of extremities.
3. It is important to make the appropriate diagnosis and management to prevent renal and liver worsening. Some cases need further intervention.
4. After conservative treatment, the patient's symptoms had improved.

gastrointestinal problems. Moderate poisoning affects liver and kidneys, and severe poisoning induces multiorgan failure.[6,8]

Histological findings of kidney and liver tissue specimens from patients usually show acute tubular necrosis and focal hepatitis. These pathological features may be attributed to the toxin with both nephrotoxic and hepatotoxic properties rather than to an infective agent in the raw bile.[9] Electron microscopy showed that mitochondria crista of epithelial cells in the proximal tubules had decreased or disappeared and the renal mesangium was extended. Glomerular cells were swollen and podocytes were partially fused; lysosomes were broken. Partial podocytic processes were fused. It is believed that the toxin in fish gallbladder damages or breaks lysosomes, meanwhile inhibiting cytochrome oxidase and blocking cellular energy metabolism to cause necrosis of the proximal tubular epithelial cells.[8]

Treatment is supportive and often includes hemodialysis.[1,3] This case required supportive treatment only without dialysis. Liver function of the patient returned to normal after 10 days, whereas his daily urine became polyuric, so the patient needed restriction fluid management, and renal function recovered gradually and became almost normal a week after being discharged from hospital (Table 1).

It seems acute kidney injury and acute ischemic hepatic failure after fish gallbladder consumption has an excellent prognosis. We suggested that the case was transient acute kidney injury induced by prerenal causes and toxicity of the fish gallbladder. A renogram and kidney biopsy should be performed and also a toxicological study of the gallbladder should be done to obtain a better diagnosis and treatment.

DISCLOSURE

All the authors declared no competing interests.

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We thank the patient for allowing us the opportunity to publish this case report. Written informed consent was obtained from the patient for publication of this case report and any accompanying images. The written consent was sent to the Editor-in-Chief of this journal.

AUTHOR CONTRIBUTIONS

MR described the patient and drafted the manuscript. RSG and RB provided the critical analysis. LL, RS, and A wrote the discussion. RMAR and DR read and approved the final manuscript.

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