

# Acute Kidney Injury due to Fish Gallbladder Ingestion: A Case Report from Cambodia

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## Keywords

Hemodialysis · Self-treatment of acne · Fish gallbladder poisoning · Ichthyotoxicity

## Abstract

We report the case of a 22-year-old woman with a 3-day history of watery stool, generalized abdominal pain, repeated vomiting, and decreased urine output following the consumption of fish gallbladder for self-treatment of acne. She was admitted and received empirical antiemetic, proton pump inhibitor, and intravenous saline treatment. Urine output reduced drastically with markedly elevated urea and creatinine, and she underwent urgent hemodialysis (HD). Based on thorough history taking and prompt biochemical investigations that showed extremely high urea and creatinine levels, we made a diagnosis of acute kidney injury (AKI) secondary to fish gallbladder poisoning. Renal function improved over a period of 5 weeks. Fish gallbladder poisoning is quite frequent in several developing countries in Asia. General physicians in these countries should note that various types of food poisoning can be involved in the etiology of AKI. The condition is commonly reversible, and therefore proper history taking is important and prompt biochemical investiga-

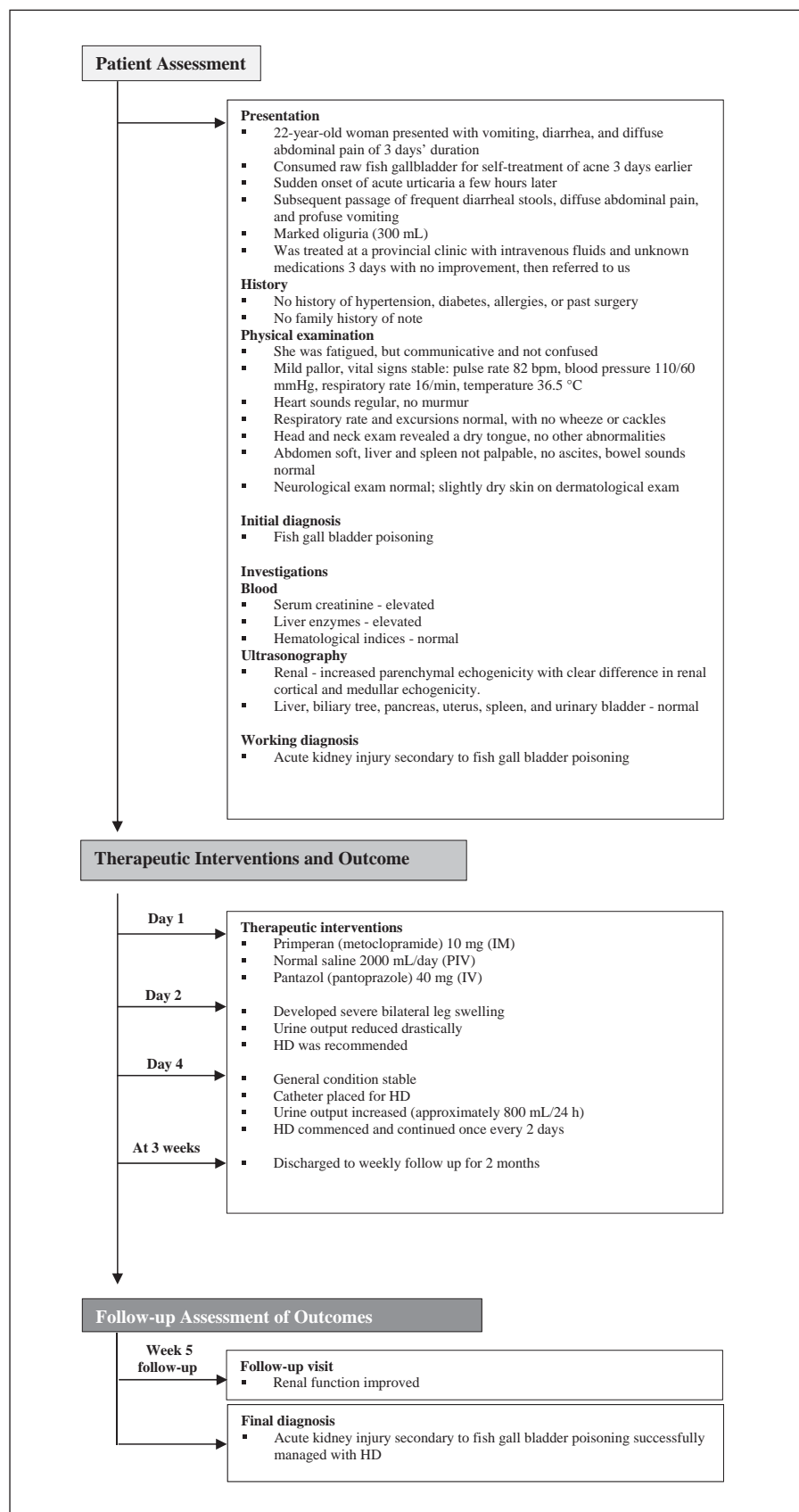
tions including blood urea and creatinine are needed to enable early diagnosis and fast institution of treatment, which may include HD.

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## Introduction

Acute kidney injury (AKI) is a condition that is characterized by a sudden episode of kidney damage that develops within a few hours or days after renal insult. It causes a build-up of waste products in the blood, resulting in compromise of renal function and difficulty in maintaining normal kidney fluid and electrolyte balance in the body [1, 2]. The mainstay of treatment includes correction of fluid overload, acidosis, hyperkalemia, and other abnormalities; however, in some cases hemodialysis (HD) is indicated to replace kidney function temporarily. AKI is commonly reversible, particularly with early detection, which allows appropriate treatment to be

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**Fig. 1.** Timeline showing the clinical course in this patient.

**Table 1.** Laboratory results on hospital days 1 and 2

Hemoglobin, g/L	11.9
Hematocrit, %	35.9
Mean corpuscular volume, fL	78.8
Leucocytes, $\times 10^9/L$	15.3
Peripheral blood neutrophils, %	83
Monocytes, %	9
C-reactive protein, high sensitivity, mg/L	15.6
Alanine-aminotransferase, U/L	1,000
Aspartate-aminotransferase, U/L	130
Urea, g/L	1.43
Creatinine, $\mu\text{mol/L}$	727

given to the patient. This case of fish gallbladder poisoning highlights the importance of history taking and is a reminder of an important cause of AKI for general physicians to note.

### Case Report

The patient was a 22-year-old woman who presented with repeated vomiting, diarrhea, and diffuse abdominal pain of 3 days' duration. She admitted to having consumed raw fish gallbladder for self-treatment of acne 3 days prior to presentation and suddenly developed acute urticaria a few hours later. This was followed by profuse watery, diarrheal stools several times per day associated with diffuse abdominal pain and profuse vomiting. There was generalized fatigue and markedly reduced urine output (300 mL). She had received intravenous fluids and some unknown medications at a nearby provincial clinic for 3 days with no improvement. She was then referred to us. She had no history of hypertension, diabetes, allergies, or surgery and no relevant family history.

On general examination, she appeared fatigued, but was communicative and not confused. There was mild pallor and her vital signs were stable: pulse rate 82 bpm, blood pressure 110/60 mm Hg, respiratory rate 16/min, and temperature 36.5°C. Cardiac exam revealed regular heart sounds and no murmur. Respiratory rate and excursions were normal, with no wheeze or crackles. Head and neck exam revealed a dry tongue, but no other abnormalities. The abdomen was soft, the liver and spleen were not palpable, there was no ascites, and bowel sounds were normal. Neurological exam was normal and on dermatological exam the skin was slightly dry.

Laboratory results on admission (hospital day 1) and on hospital day 2 showed elevated liver enzymes and other results suggestive of liver and kidney pathology (Table 1). Sonography of the liver, biliary tree, pancreas, uterus, spleen, and urinary bladder were all normal. However, increased renal parenchymal echogenicity was observed with a distinct difference in echogenicity between the renal cortex and medulla.

Treatment was commenced empirically on hospital day 1 with Primperan (metoclopramide) 10 mg (IM), normal saline solution 2000 mL/day (PIV), and Pantazol (pantoprazole) 40 mg (IV). Vital

signs and urine output were monitored hourly. On hospital day 2, she developed severe bilateral leg swelling. Urine output reduced drastically and serum urea and creatinine were markedly elevated, warranting discussion with the patient and her family, during which HD was recommended. Her general condition was stable enough on day 4 to withstand catheter placement for HD. Urine output was approximately 800 mL/24 h. HD was continued once every 2 days. She was eventually discharged 3 weeks after admission and was followed up weekly for 2 months. Renal function improved over 5 weeks in total.

Figure 1 shows the timeline of the clinical course in this case.

### Discussion

Consumption of fish gallbladder is a rather common practice in several parts of Asia and is considered to be a traditional medicine for acne (as in this case), urticarial rheumatism, and poor vision [1]. Several reports from India [3], Hong Kong [4, 5], and Japan [6] show that this practice can sometimes result in acute renal failure or acute liver injury. The ichthyotoxicity (i.e., toxicity produced by fish or toxicity to fish) is caused by the toxin sodium cyprinol sulfate, a C27 (fish) bile acid. Sodium cyprinol sulfate is heat stable and insoluble in alcohol and so, even after cooking, the fish can still be toxic. In Cambodia, there are two types of poisonous fish, the Black sharkminnow (*Labeo chrysophekadion*) and the Thicklip bard (*Probarbus labeamajo*), which can cause such ichthyotoxicity.

Physicians, especially general practitioners, should be aware of such cases because the presentation in some patients may be similar to that of general food poisoning with diarrhea, vomiting, and fatigue. For patients with possible toxicity from food, it is important to check blood urea nitrogen (BUN) or serum urea and creatinine particularly in regions where the use of traditional medicine is widely practiced.

In this case, there did not seem to be a prerenal component from other medications. This is because although serum creatinine level is elevated in many patients with AKI, BUN is also increased. In our patient, this was unlikely to have been due to other medications because the provincial clinic would most probably have treated her symptomatically with medications for diarrhea or nausea.

In conclusion, ingestion of fish gallbladder is a common practice not only in Cambodia, but also in other Asian countries such as India, China, and Thailand. This case highlights the significance of history taking, the utility of prompt biochemical investigations including blood

urea and creatinine, and the timely commencement of HD, when necessary, in the management of patients with food poisoning.

### Acknowledgements

The author thanks Florence Orim, MD, PhD, and Caryn Jones of ThinkSCIENCE, Japan, for medical writing support.

### Statement of Ethics

The patient provided written informed consent for her case to be published.

### Disclosure Statement

Writing support, for educational purposes, was funded by Nipro Corporation, Japan. Nipro Corporation had no role in the selection of the case, the collection and analysis of the data, or in the preparation of the manuscript.

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