Concurrent acute kidney injury and acute hepatitis resulting from raw fish gallbladder ingestion: a preventable cause of community acquired organ damage

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\textbf{ABSTRACT}

In south-east Asia, there has been a common belief, mainly in rural areas, that ingesting raw fish gall bladder can help cure certain health problems. Unfortunately, raw fish bile has both nephrotoxic and hepatotoxic effects. In this report, we present a case of 42-year-old man, who presented with acute kidney injury and jaundice. Meticulous history revealed that his ailment resulted from raw Rohu fish gallbladder ingestion.

Key words: acute kidney injury, fish gall bladder, hemodialysis, jaundice, Rohu fish.

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\section*{INTRODUCTION}

Due to frequent consumption of fish gall bladder for the purpose of curing rheumatism, asthma and improving vision, fish bile poisoning cases are commonly reported in China, India, Japan and other Asian countries.\textsuperscript{1} Fish bile poisoning have significant morbidity and mortality.\textsuperscript{2} Recently, studies have shown that fish gallbladder can damage to the heart, liver and gastrointestinal tract and lead to multiple organ dysfunction syndrome (MODS).\textsuperscript{3} Most patients present with nausea, vomiting, abdominal pain, watery diarrhea from a few minutes to several hours after eating, followed by manifestation of the central nervous system (CNS), oliguria due to acute kidney injury (AKI) and toxic hepatitis.\textsuperscript{4} As this is an overlooked cause of simultaneous AKI and hepatitis, we here in present a case of fish gallbladder poisoning, to create awareness among physicians in recognizing and managing such cases.

\section*{CASE REPORT}

A 42-year-old man, known case of ischaemic heart disease and hypertension, presented with 3-day history of vomiting, watery stool and diffuse abdominal pain. Two days later, he developed reduced urine output and yellowish discoloration of skin and sclera. Patient did not take non-steroidal anti-inflammatory drugs (NSAIDs) or other nephrotoxic and hepatotoxic drugs. No history of exposure to chemicals or toxins was found. No other family members were affected. Patient was initially treated in a local hospital, where his serum creatinine (6.8 mg/dl) and serum alaline aminotranferase (ALT) (2408 U/L) levels were found elevated. Meticulous history was taken for searching possible cause of concurrent renal and hepatic impairment and patient admitted taking raw Rohu fish (a member of carp family) gallbladder to cure peptic ulcer disease (PUD) 1 day before symptom onset.
At our center, clinical evaluation revealed tachycardia (heart rate 108 beats/min), icterus and dehydration. Abdominal examination revealed diffuse tenderness without any organomegaly. Bed side urine examination showed trace of protein. Other systemic examination findings were normal.

Patient had leukocytosis and raised erythrocyte sedimentation rate (ESR) (30 mm in 1st hour). Serum creatinine and serum urea levels were 7.6 mg/dl and 126 mg/dl respectively. His serum electrolytes revealed hyponatremia (serum Na 127 mmol/L), hyperkalemia (serum K 6.8 mmol/l) with metabolic acidosis (serum HCO₃ 19 mmol/L). His serum ALT and aspartate aminotransferase (AST) level were 373 U/L and 943 U/L respectively. Serum bilirubin level was 4.1 mg/dl and serum alkaline phosphatase level was 70 U/L. Urine routine examination showed presence of red blood corpuscles (RBC) (20-30/high power field) with trace protein.

As patient’s urine output was still low and features of uremia developed despite proper conservative management, hemodialysis was started. After 6 sessions of hemodialysis, patient’s urine output started increasing and other biochemical parameters also gradually came back to normal level. Patient was discharged with the advice of follow-up after 1 month. On follow-up visit, patient had normal clinical and biochemical parameters.

**DISCUSSION**

Most of fish poison contains ciguatoxin⁵ and mackerel poison mainly found in marine coral fish. Although carp fish is non-poisonous, its bile contains highly virulent bile toxins like water soluble sodium cyprinol sulphate which can lead to MODS.⁶ Toxicity is directly proportional to the size and quality of the gallbladder and bile content.⁷ Renal failure is the most commonly reported effect of fish bile poisoning.⁸ Cyprinol and related compounds in fish gall bladder are thought to be the cause of acute tubular necrosis in such cases.⁹ Furthermore, frequent vomiting, diarrhea and insufficient fluid intake can worsen renal failure due to significantly decreased intravascular fluid volume.²

AKI after fish gall bladder ingestion has a variable prognosis. However, death may result from fulminant hepatic failure.⁷ Treatment comprises of hemodialysis and supportive management⁸ and most of the reported cases till date have undergone hemodialysis in view of renal failure. The patient in the reported case also responded to the same line of management.

Simultaneous renal and liver injury may result from exposure to a variety of toxins, infections including malaria and leptosprirosis but in absence of any history suggesting toxin exposure or fever, the possibility of ingestion of raw fish gallbladder and bile should be considered. As proper management can save lives in such cases, high degree of suspicion and proper clinical history should be elicited in order to ensure early detection of cases and timely management.

**Authors’ contribution:** MRH managed the case, did literature search and drafted the manuscript. SMA did literature search and helped in drafting the manuscript. TAC supervised managing the case. MAR supervised managing the case, revised and edited the manuscript. MAA diagnosed the case and supervised managing the case. SI was the overall supervisor. All authors read and approved the final manuscript.

**Conflict of interest:** Nothing to declare.

**Consent:** Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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