

A Case Report on Fish Bile Toxicity- A Rare Cause of Multiple Organ Dysfunction Syndrome

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

Editor(s):

(1) Dr. P. Kiranmayi, GITAM University, India.

Reviewers:

(1) Frieder Keller, University hospital in Ulm, Germany.

(2) Juliana Christyaningsih, Indonesia.

Complete Peer review History, details of the editor(s), Reviewers and additional Reviewers are available here:

<https://www.sdiarticle5.com/review-history/78047>

Case Study

Received 13 October 2021

Accepted 27 December 2021

Published 29 December 2021

ABSTRACT

A case study on fish bile poisoning is reported. After ingestion of gall bladder of *Labeo rohita* fish for allergy treatment purpose initially presented with gastrointestinal symptoms such as cramping pain in, nausea and vomiting within 12 hours after ingestion. Subsequently renal, hepatic dysfunctions and cardiac dysfunction were found in that case. The patient recovered fully with conservative treatment and supportive hemodialysis.

Keywords: *Labeo rohita*; fish bile; hemodialysis.

1. INTRODUCTION

In India, especially in Assam and Chinese people believe that fish gall bladder can improve vision,

treat rheumatism, improves allergy and cure asthma [1]. Due to frequent consumption of fish gall bladder, fish bile poisoning cases are reported more commonly in China, India, Japan,

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and other Asian countries [2,3,4]. There were many reports about fish gall bladder poisoning leading to acute renal failure (ARF), acute liver injury, and therefore increasing mortality [5]. The incidence of ARF in fish bile poisoning is 55%–100%, while the mortality rate accounts 91.7% . Fish gallbladder contains a heat stable toxin which can severely affect gastrointestinal system, renal, liver, central nervous system, cardiovascular system and leading to multiple organ failure (MODS) [1] This is a case of Rohu fish gall bladder poisoning leading to multi organ involvement in a Bangladeshi child. This report is a whole new perspective on the pathogenesis of acute renal failure and other organ involvement in a case of poisoning by fish gall bladder. This has a positive role in guiding treatment of fish bile poisoning, with obvious effect to improve its prognosis. This case not only focuses over presentations but also guides the management as there was no mortality in our case in spite of severity in presentation. The condition is commonly reversible, and therefore proper history taking is important and prompt biochemical investigations including blood urea and creatinine are needed to enable early diagnosis and fast institution of treatment, which may include hemodialysis.

2. CASE FEATURES

A 10-year-old boy who was with history of repeated vomiting and diffuse abdominal pain for 3 days and oliguria for 2 days. His father admitted that he convinced his boy to consume raw fish gallbladder for the purpose of treatment

of allergy 1 day prior to the presentation. This was followed by diffuse abdominal pain and profuse vomiting and he got admitted into a hospital and treated with IV fluid, antiemetic and antiulcerant medications. He developed oliguria along with puffy face and jaundice within 48 hours of presentation. After investigations over that hospital they diagnosed him as a case of Acute Kidney Injury (AKI) and Intermittent peritoneal dialysis (IPD) was started. When there was no improvement, they referred him to our hospital for better management. There was no history of fever, previous history of renal disease or family history of such type of illness.

On general examination, he appeared ill looking, puffy, afebrile but was communicative and oriented. The boy was mildly pale and icteric and on vitals examination pulse rate 82 bpm, blood pressure 130/90 mmHg (above 99th centile), respiratory rate 20/min, and temperature 37.5 ° C. Cardiac exam revealed regular heart sounds and no murmur. His breath sound was vesicular with no wheeze or crackles. Abdomen was soft, mildly tender with IPD catheter in situ. There was no organomegaly but ascites was evident by positive fluid thrill, bowel sound was present. Nervous system with all other systems examination revealed no abnormalities.

After getting three sessions of hemodialysis and conservative management patient was on improving pattern and got discharged. After two weeks of follow up his all-biochemical parameters become normal.

Table 1.His initial Investigations showed in

Investigations	Findings	Normal Values
Hb%	9.8 gm/dl	11 to 14 gm/dl
WBC	12 000/cumm,	4 000 to 11 000/cumm
Neutrophil	78%	40 to 70%
Lymphocytes	15%	20 to 40%
Platelet	245 000/cumm	150 000 to 40 000/cumm
Serum Creatinine	4.7 mg/dl	0.3 to 0.8 mg/dl
Blood urea	18 m mol/L	1.3 to 5.8 m mol/L
Serum Sodium	143 m mol/L	135 to 145 m mol/L
Serum Potassium	5.2m mol/L	3.5 to 5.4 m mol/L
Serum Chloride	105m mol/L	96 to 108 m mol/L
Serum Albumin	25 gm/L	35 to 60 gm/L
Serum Calcium	2.1 m mol/L	2.02 to 2.1 m mol/L
C-Reactive protein	7 mg/L	< 5 mg/L
SGOT	2196 U/L	Up to 37 U/L
SGPT	7283 U/L	Up to 40 U/L
Serum Bilirubin	2.5 mg/dl	0.2 to 1 mg/dl

Investigations	Findings	Normal Values
PT INR	12 seconds, INR 1	12 seconds
APTT	29 seconds	35 seconds
Serum Ferritin	288 ng/ml	15 to 300 ng/ml
Serum Procalcitonin	6.45 ng/ml	< 0.50 ng/ml
D-dimer	1.44 mg/L	< 0.5mg/L
Cardiac Troponin I	0.00 ng/ml	0 to 0.034 ng/ml
NT ProBNP	27262 pg/ml	<125 pg/ml
RT PCR COVID 19	Negative	
Antibody (IgG) of Covid 19	Negative	
Urine RME	Albumin 2+, RBC 10-15/HPF	Nil Nil
Chest X ray	Normal	
USG of Whole Abdomen	<ul style="list-style-type: none"> Cortical Echogenicity of both kidneys raised with poorly differentiated Cortex and medulla Mild ascites Normal Kidney sizes Right Kidney 8.0 cm Left Kidney 8.1 cm 	
Echocardiography	<ul style="list-style-type: none"> Dilated coronaries with loss of distal tapering Mild LV Systolic Dysfunction 	
Blood Culture	No growth	
Urine Culture	No growth	
Complement C3	0.76 g/L	0.75 to 1.65 gm/L
Complement C4	0.21 g/L	0.15 to 0.45 gm/L
ANA	Negative	
Anti Ds DNA	Negative	
Serum PTH	78 pg/ml	
ABG	PH 7.35 HCO3 13 PO2 138 BE – 8.2	7.35 to 7.45 18 to 23 80 to 100 mmHg -2 to +2
Renal Biopsy	Features of Tubular Injury	

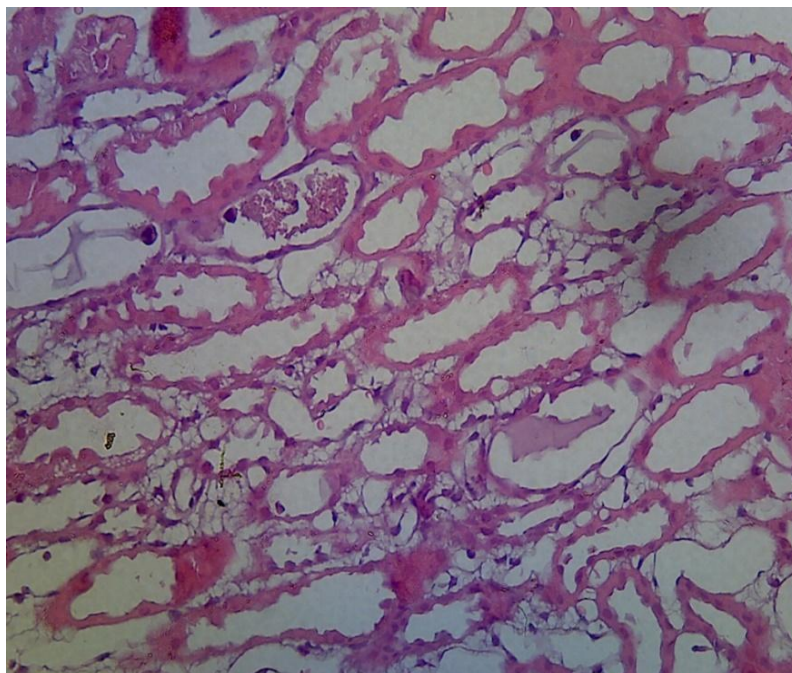


Fig .1. Renal biopsy showing tubular necrosis

3. DISCUSSION

Fish bile induced renal failure and toxic hepatitis cases have been reported in various parts of Asia & Southeast Asia [5]. Toxicity is attributed to the fishes belonging to the family Cyprinidae. The family includes grass carp, common carp, and silver carp. Amongst these, fish of the grass carp variety has been commonly reported for its toxicity. Rohu (*Labeo rohita*) the Indian fish carp is commonly consumed in north eastern and eastern region of India. Its bile contains a toxin, sodium cyprinol sulfate, which occurs in three forms: toxins in visceral organs (ichthyosarcotoxic), reproductive organs (ichthyootoxic), or blood (ichthyohemotoxic). It is heat stable and alcohol insoluble, so cases are reported even after consumption of cooked bile [6,7]. Toxicity is directly proportional to the size and quantity of gall bladder or bile consumed [8]. After ingestion, initial manifestations include abdominal pain, nausea, vomiting and watery diarrhea, followed by oliguria and renal failure. The hepatic impairment and cardiac dysfunction usually precedes renal dysfunction, but may be concomitant with kidney injury. Kidney biopsy reveals proximal tubular cell damage on light microscopy. Electron microscopy shows decreased mitochondria cristae in the proximal tubular epithelial cells, swollen glomerular cells and partially fused podocytes. The toxin is believed to damage lysosomes and inhibit cytochrome oxidase enzyme, thus blocking cellular metabolism and causing necrosis of the proximal tubular epithelial cells. In addition, loss of fluid due to vomiting, diarrhea can lead to decreased effective circulating blood volume and eventually leads to oliguric or the non-oliguric form of acute renal failure, usually within 48-72 hours after toxin ingestion [9, 10]. BichHuyen Nguyen Xuan *et al.* from Vietnam have conducted a large study and showed the effects of certain freshwater fish bile associated acute tubular necrosis. In India, Dwijen Das *et al.* have published a case series on fish bile toxicity causing lethal renal failure and hepatic dysfunction. Fish bile can also damage other organs causing multiple organ dysfunction syndrome (MODS) [11,12]. However, acute renal failure after fish gallbladder ingestion has an excellent prognosis, though death from fulminant hepatic failure can occur. Proper management comprises of hemodialysis and supportive management, is essential to save lives of these patients [13]. Eliciting a proper clinical history in such cases is of paramount importance as many different substances can produce simultaneous

renal, hepatic and cardiac damage. This includes variety of toxins like carbon tetrachloride, trichloroethylene, chloroform, copper sulfate and chromium, mushroom poisoning and drugs including paracetamol overdose and fluorinated anesthetic agents such as methoxyfluorane and fluoxetine. Hence these more common etiological agents should be first ruled out by detailed history taking before considering uncommon fish bile as the injurious agent [14].

4. CONCLUSION

This case report should help the general physicians in developing countries to be aware of the fact that various types of food poisoning can cause AKI and fish bile can be a possible but rare cause of reversible acute renal & hepatic failure. Proper history taking is important in these circumstances and prompt biochemical investigations are needed for an early diagnosis and institution of proper treatment for the purpose of fruitful outcome.

Footnotes: Authors' Contribution: Study concept and design: FY; Acquisition of data: FY; Analysis and interpretation of data: FY; Drafting of the manuscript: FY; Critical revision of the manuscript for important intellectual content: SA,TF; Administrative, technical, and material support: FY,SA,UT,SB. Conflict of Interests: There are no conflicts of interest to declare. Funding/Support: No funding was needed for this study.

INFORMED CONSENT

We obtained informed written consent from the father.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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Peer-review history:

The peer review history for this paper can be accessed here:
<https://www.sdiarticle5.com/review-history/78047>