Case study

A Case Report on Fish Bile Toxicity- a rare cause of Multiple Organ Dysfunction Syndrome

ABSTRACT: A case of fish bile poisoning is reported. After ingestion of gall bladder of Labeo rohita for allergy generally presented with gastrointestinal symptoms such as cramping pain abdomen, 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

INTRODUCTION:

In India, especially in Assam and Chinese people believe that fish gall bladder can improve vision, treat rheumatism, improves eyesight and cure asthma [1]. Due to frequent consumption of fish gall bladder, fish bile poisoning cases are reported more commonly in China, India, Japan, 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 HD.

CASE FEATURES

Sani Abdullah, a 10-year-old boy who presented with history of repeated vomiting and diffuse abdominal pain for 3 days and oliguria for 2 days. His father admitted that he forced Sani 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 and other medications. He developed oliguria along with puffy face and yellowish discoloration of skin and sclera within 48 hours of presentation. After investigations over that hospital the diagnosed him as a case of AKI and Intermittent peritoneal dialysis was started. When there's no improvement they refer him to our hospital to better management. There 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. There was mild pallor, mild 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. Respiratory rate were normal with no wheeze or cackles. The abdomen was soft, the liver and spleen were not palpable, there was IPD catheter in situ with ascites, and bowel sounds were normal. Neurological exam was normal.

His initial Investigations showed in Table 1.

Investigations	Findings
Hb% WBC Neutrophil Lymphocytes Platelet	9.8 gm/dl 12000/cumm, 78% 15% 245000/cumm
Serum Creatinine Blood urea Serum Sodium Serum Potassium Serum Chloride	4.7 mg/dl 18 mmol/L 143 mmol/L 5.2mmol/L 105mmol/L
Serum Albumin Serum Calcium C-Reactive protein	25 gm/L 2.1 mmol/L 7 mg/L
SGOT SGPT Serum Bilirubin PT INR APTT	2196 U/L 7283 U/L 2.5 12 seconds, INR 1 29 seconds
Serum Ferritin Serum Procalcitonin D-dimer	288 ng/ml 6.45 ng/ml 1.44 mg/L
Troponin I NT ProBNP	0.00 ng/ml 27262 pg/ml
RT PCR COVID 19 Antibody (IgG) of Covid 19	Negative Negative
Urine RME	Protein 2+, RBC 10-15/HPF
Chest X ray	Normal
USG of Whole Abdomen	• Cortical Echogenicity of both kidneys raised with poorly differentiated Cortex and medulla

	• Mild ascites
Echocardiography	• Dilated coronaries with loss of distal tapering • Mild LV Systolic Dysfunction
Blood Culture Urine Culture	No growth No growth
Complement C3 Complement C4 ANA Anti Ds DNA Serum PTH	0.76 g/L 0.21 g/L Negative Negative 78 pg/ml
ABG	PH 7.35 HCO3 13 PO2 138 BE – 8.2
Renal Biopsy	Features of Tubular Injury

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.

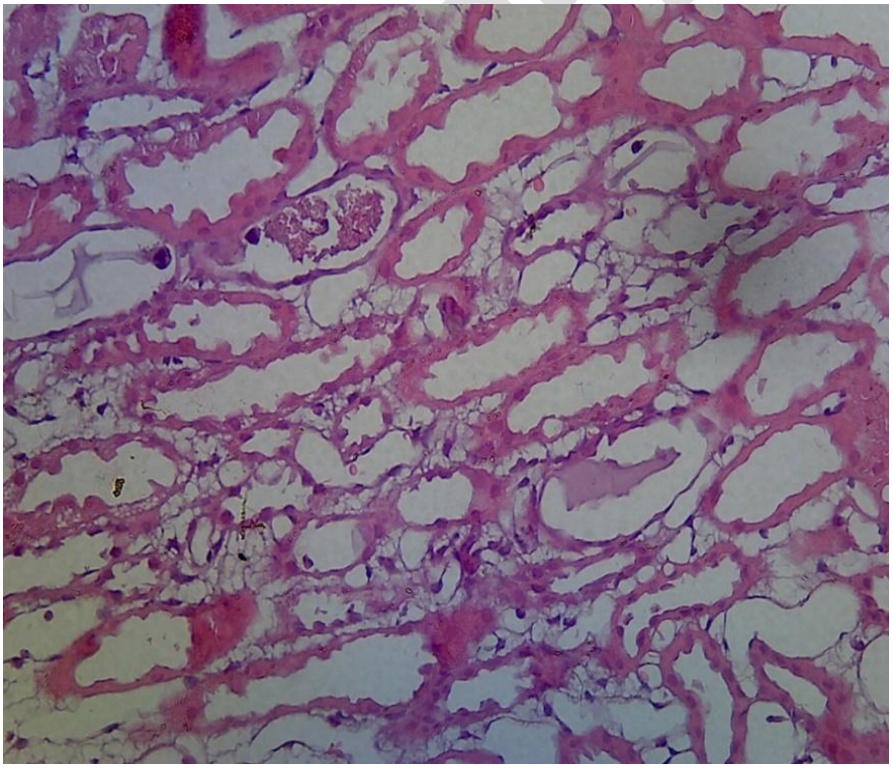

Fig 1: Renal biopsy showing tubular necrosis

DEPARTMENT OF PATHOLOGY
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RENAL BIOPSY REPORT

Patient Name : XXXXXXXXXX
Lab No : 2155-00458
Ref Doctor : BSMMU
Request No : 0001496854
Gender : MALE
Age : 10 Yrs 0 Mons 1 Day
Nationality : BANGLADESHI

Clinical Presentation : 1. Swelling of whole body, oliguria.
2. H/O fish bite poisoning and excess intake of fruits and fruit juice.
3. Urine R/E: Protein (2+), RBC 10-15/HPF, S. Albumin: 27.1 gm/L, Blood urea: 26.9 mmol/L, S. Creatinine: 3.6 mg/dL, Serum: C3 : Deceased, C4: Normal; ANA: Negative, Anti DNA: Negative.

Gross Examination :

Received in 10% formalin.
Number of tissue : One
Shape : Linear
Measurement : 1.0 cm.

Received in normal saline
Number of tissue : One
Shape : Linear
Measurement : 0.7 cm.

Light Microscopic Examination :
Glomerulus :
Number : 16
Size : Mildly enlarged
Cellularity : Segmental increase
Matrix : Increased
GBM : Not thickened.

Tubule :
T atrophy :
Epithelium : Features of protein reabsorption.
TBM :
Cast : Hyaline, granular.

Necrosis :
Tubulitis :
Crystals : In the lumen.
Others : Features of tubular injury.

Interstitium :
Inflammation : Mild, chronic.
Unremarkable :

Fibrosis :
Others : Oedema

Blood Vessels :
Unremarkable.

Special Stain :
PAS, silver & Masson's trichrome.

DIF finding :

Deposited antibody	IgG	IgM	IgA	C3	C1q	Fibrin	Kappa	Lamda
Remarks :	No deposition of any antibody is seen.							

Dx : Tubular injury.

Note: Please explore the cause of proteinuria.

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Fig 2: Renal biopsy report showing features of tubular injury

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 (icthyohemotoxic).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 crista 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*. 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 fluoxene. 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].

CONCLUSION

Our report should help in 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 scenarios and prompt biochemical investigations are needed for an early diagnosis and institution of proper treatment.

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