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CASE REPORT

A CASE SERIES ON FISH BILE TOXICITY

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ABSTRACT: A case series of 3 cases of fish bile poisoning are reported. After ingestion of gall bladder of Labeo rohita for alleged vision improvement, generally presented with gastrointestinal symptoms such as cramping pain abdomen, nausea and vomiting within 12 hours after ingestion. Subsequently renal and hepatic dysfunctions were found in all the three cases. The patient recovered fully with conservative treatment and supportive haemodialysis.
KEYWORDS: Labeo Rohita, Fish bile, haemodialysis

INTRODUCTION: In northeast region especially in Assam, fish is very commonly consumed food by the people and particularly Rohu (Labeo rohita) which is a species of fish of the carp family but along with that some people also tend to eat raw fish gall bladder due to the fact that they believe it is good for health, improves vision and cures rheumatism[1,2] and some people especially the young ones in Barak Valley ingest it as a part of fun and challenge as it is very bitter in taste. Acute renal failure and hepatitis have been reported from China,[3,4,5] Japan,[6] USA[7] and India[2,8,9] following ingestion of fish gall bladder. We are reporting 3 cases from Barak Valley, Assam, India with acute renal failure and liver injury following consumption of raw fish gallbladder. Our study not only focuses over their presentations but also guides the management as there was no mortality in our cases in spite of their severity in presentation.

CASE REPORT 1: A 24 year male presented at the outpatient department at Silchar Medical College and Hospital with decreased urine output, dysguisea, nausea and malaise for 2 days. Prior 2 days he suffered from severe abdominal cramps, nausea and vomiting. He was then admitted to nearby health centre where he received IV fluids, PPI, anti-emetics and antibiotics but still there was no improvement in his condition so they planned for investigation on 2nd day and found that his serum creatinine was 14.18mg/dl, SGOT 86 IU/L, SGPT 385 IU/L. He was then immediately referred to Silchar Medical College and Hospital.

On physical examination, the pulse rate was 80/min, respiratory rate was 22/min and acidotic breathing pattern and blood pressure was 130/80 mm of Hg. Pallor, cyanosis, oedema, and clubbing were absent. Icterus was present. Apart from mild abdominal distension without ascites, rest of the systemic examination was normal without any organomegaly clinically. Investigations revealed a Hb of 11.4 g/dl with a total leucocyte count of 9,020/cumm with 65% neutrophils, 20% lymphocytes, 1% monocytes and 14% eosinophils. Erythrocyte sedimentation rate was 20 mm in 1st hr and peripheral blood picture showed normochromic, normocytic red blood cells with adequate platelets and total serum albumin was 3.2mg/dl. Kidney function tests revealed a serum creatinine 13.62mg/d, urea 120mg/dl, Sodium 129mEq/L and potassium was 4.34mEq/L. His Bilirubin was 5.4mg/dl, SGPT 567 IU/L, SGOT 912IU/L. His Ultrasound abdomen

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shows hepatomegaly and bilateral renal swelling with maintained cortico medullary differentiation. Routine urine examination reveals blood 3+, protein 3+, RBC 4-5/HPF and Epithelial cells 7-10/HPF. His virology profile (HBs Ag, Anti-HCV, HIV I & II) comes out to be normal. Serology for malaria and leptospira were negative.

It was provisionally thought to be a case of Acute Viral Hepatitis with Hepatorenal syndrome (Non-B Non-C) or a case of Septicemia with multi organ failure. But patient was not having any history of viral prodrome before his presentation and the level of renal impairment relatively more severe. On the other hand his manifestations and investigations do not support completely to be a case of septicemia.

The vomiting was not very severe and we were not getting any clue why the patient landed with renal failure. After repeated enquiry we could elicit history of ingestion of 2no.s of fish gall bladder of big size Rohu fish out of fun with his friends. On search of literature we found some cases reported from Manipur, Odissa and even from Assam and China who presented in the similar way as our patient. We also started thinking in the same line and ultimately he was diagnosed to have fish bile induced renal failure and toxic hepatitis.

The patient was then put on IV fluids at the rate of 60drops/min, pantoprazole, ondensatron. Urine output and input were closely monitored and on next day of admission he was put on Haemodialysis. Next day his repeat creatinine was 11mg/dl and features of azotemia subsided. After receiving 3 settings of haemodialysis his creatinine falls to 2.3mg/dl and urine output becomes 4l/d and this fluid loss is maintained via IV fluids and after 5 days his urine output comes to normal and creatinine was 1.2mg/dl, SGOT and SGPT returns to normal and there was no increase in bilirubin level. He was discharged and on follow up after 2 weeks his biochemical and hematological parameters were normal.

Consequently within few days of the presentation of the first case we got another similar case as follow.

CASE REPORT 2: A young male of 27yrs age presented with pain abdomen, nausea and vomiting within 12hrs of ingestion of raw gall bladder of rohu fish as he thought it might improve his vision for which he seeks to local physicians but after 48hrs there was decrease in urine output and yellowish discolouration of urine for which he came to Silchar Medical College where his serum creatinine level found to be 3.6mg/dl and urine output was 150ml/d with dehydration on examination. As such IV fluids started at the rate of 100ml/hr and monitoring of input and output with regular chest auscultation for crackles was started. His SGOT was 1518 IU/L, SGPT 2049 IU/L and serum bilirubin was 5.38 mg/dl. Ultrasound abdomen shows mild hepatomegaly with normal renal echo with normal viral markers, negative serology for malaria and leptospira, so he was maintained on oral glucose, lactulose and pantoprazole. Next day his ceatinine level increased to 5.18mg/dl so he was prepared for hemodialysis but his SGOT falls to 170 IU/L, SGPT to 775 IU/L and bilirubin to 4.89 after 2 days. After receiving 2 settings of haemodialysis his creatinine level was 2mg/dl and urine output increased to 3500L/d. On 10^{th} day of admission his serum creatinine, SGOT, SGPT comes to normal level and on follow up after 2 weeks his all blood parameters were normal.

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CASE REPORT 3: A 35yr male with a history of ingestion of fish gall bladder 4 days back presented with pain abdomen, nausea, vomiting within 12hrs of intake and yellowish discoloration of urine with anorexia from past 1 day but his urine output was normal. On examination icterus was present, rest were normal and investigation revealed Urea- 66mg/dl, creatinine- 1.49mg/dl, SGOT–1641IU/L, SGPT- 2463IU/L and bilirubin was 4.79mg/dl, urine examination shows Albumin 1+,nonreactive viral markers, serology for malaria, rest including ultrasound of abdomen were within normal limit. He was then put on IV fluids, oral glucose, oral lactulose and proper bed rest. After 2 days his repeat investigations show creatinine- 1.08, SGOT- 212 IU/L, SGPT- 874 IU/L and bilirubin was still 4.36mg/dl. He was then discharged on conservative management on follow up after 2 weeks his SGOT, SGPT and bilirubin and creatinine level comes to normal.

DISCUSSION: Rohu (Labeto rohita) the Indian fish carp is commonly consumed in northeastern and eastern region of India but cases of fish bile poisoning are not very common. Apart from our case there are cases reported from Manipur,[9] Guwahati[2] and Orissa.[8] All 3 patients of the present series presented initially with gastrointestinal upset including abdominal pain, nausea, vomiting, within 12 hours of ingestion. In all patients hepatotoxicity and nephrotoxicity occurred. The hepatitis picture, observed within hours of ingestion, was attributed to hepatotoxins rather than to an infective agent in the raw bile.[4]

Spontaneous resolution of hepatic dysfunction within a few days is usual. More serious is the nephrotoxicity, which culminates in either the oliguric or the non-oliguric form of acute renal failure, usually within 48-72 hours after ingestion while hepatic dysfunction improved with conservative management as we got in other studies.[8,2,10,9] Although the bile component(s) responsible for this syndrome have not been characterized fully,[3] cyprinol, a C27 alcohol found in the bile of cyprinid fish, may have a direct toxic affect on the kidneys.[4] It was reported that the toxic effect of 5α-cyprinol sulfate on the kidney functions was more harmful than that of 5α-cyprinol.[11] Five species of fish belonging to the order Cypriniformes have been associated with bile-induced hepatitis and renal failure.[5] Ingestion of either one, or two, or more large gallbladders, may be responsible for poisoning. Several case series, describing a cause of fish poisoning from gallbladder ingestion with resultant gastrointestinal, renal, hepatic, cadiac, and neurological toxicities, are reported. Most of the poisoning cases developed after swallowing gallbladders from grass carp or silver carp, and their lengths were over 1 cm long. The volume of bile ingested varied from 15 to 30ml. Therefore, the larger the size of the ingested Cyprinid fish is the higher is the risk of intoxication.[11] In our cases with proper fluid management and dialysis renal failure can be reverted and for hepatitis conservative management and extensive monitoring is sufficient.

CONCLUSION: Though uncommon but fish bile toxicity can lead to renal failure and hepatic dysfunction. No definite marker for its diagnosis is available but its diagnosis needs high index of suspicion and thorough clinical history.

Our idea of presenting these case reports is only to sensitize that even fish bile toxicity can cause renal failure and hepatic dysfunction which can be lethal if not treated in proper time but proper management of these patients can save lives of these patients.

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