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Acute Kidney & Hepatic Injury Following Fish Bile Ingestion- Two Case Reports

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Case Report

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Abstract: Fish gallbladder is consumed in several developing countries in Asia as part of traditional medicine. It is considered to improve vision, digestion, cure joint pain, skin ailments & asthma. It contains a heat stable toxin which can severely affect gastrointestinal system, renal, liver, central nervous system leading to multiple organ failure.We report two cases with a history of diarrhoea, generalized abdominal pain, repeated vomiting and decreased urine output following the consumption of fish gallbladder. Both underwent urgent hemodialysis (HD) and supportive treatment. Based on thorough history taking and prompt biochemical investigations that showed markedly raised urea and creatinine levels and liver enzymes, a diagnosis of acute kidney injury (AKI) & hepatic injury secondary to fish gallbladder poisoning was made. Kidney biopsies revealed acute tubular injury. Renal & hepatic function improved gradually with continued treatment.General physicians in these countries should be aware of the fact that various types of food poisoning can cause AKI. Proper history taking is important and prompt biochemical investigations are needed for an early diagnosis and treatment.

Keywords:Acute kidney injury, hepatic injury, fish bile.

INTRODUCTION

In rural areas of south & Southeast Asia many types of alternative medicines including animal & plant products are consumed for treatment of various ailments. One such unusual example is fish bile which is considered to improve vision, digestion, cure rheumatism, acne & asthma. But it is also known to cause life threatening side effects & can lead to multiple organ dysfunction syndrome (MOD)[1,2].

Such incidents of organ failure has been reported from China, Japan, South East Asia (Vietnam, combodia), North east india (Manipur, assam) & USA. The incidence of acute renal failure (ARF) in fish bile poisoning is 55%–100%, while the mortality rate accounts upto 91.7% .[3,4] We report a case of a 56 year female presenting with acute renal & hepatic injury following ingestion of fish bile.

CASE 1

56 year female admitted in our hospital with the history of sudden decrease in urine output for three days with swelling all over body. She had episodes of vomiting and passage of about 4 to 6 episodes of loose stool per day with severe colicky pain abdomen. On further questioning, she gave history of consumption of raw Rohu gall bladder intake 4 days back with no history of consumption of any other food or drug.

On physical examination, she had moderate dehydration, pulse rate was 96/min, respiratory rate was 28/min ,breathing was acidotic , blood pressure was 140/80 mm of Hg and she was mild Icteric with bilateral pedal edema. She was neither pale nor cyanotic. No clubbing, lymphadenopathy or organomegaly noticed. Abdomen was soft and tender. Cardiovascular, respiratory and central nervous system examinations were unremarkable. Further investigations revealed HB: 8.8 g/dl, TLC: 11, 600/cmm, DLC: N 78% and TPC: 1,38,000/cmm. Erythrocyte sedimentation rate was 22 mm in 1st hr and peripheral blood picture showed normochromic, normocytic blood picture. Urine routine & microscopy examination showed abundant granular casts, protein 3+. Biochemical examination showed abnormal liver function tests. Serum bilirubin was 1.4 mg/dl, SGPT (ALT): 340 IU/l, SGOT (AST) 1141 IU/l, sr protein: 6.8 g/dl,sr albumin:3.0 g/dl and sr alkaline phosphatase: 146 IU/l. Serum creatinine was raised at 6.1 mg/dl and

sr urea: 84 mg/dl. Arterial blood gas indicated metabolic acidosis. Chest X-ray, X ray KUB and ECG were normal. Ultrasonography of abdomen showed normal renal images and dense liver with minimal ascites. HBs Ag, Anti-HCV and HIV were negative. Stool Culture, Blood Culture and Urine culture were sterile. With strong evidence of deteriorating renal function, she received 3 cycles of hemodialysis along with supportive treatment. Percutaneous renal biopsy was performed and sent for light microscopy & immunofluorescence study. Electron microscopic study & liver biopsy was not performed.

Histopathology of kidney biopsy revealed glomeruli and blood vessels of normal morphology with pathologic changes confined to tubulointerstitial compartment. Simplification & sloughing of tubular epithelium, focal loss of tubular nuclei, loss of brush border and necrosis noted. Proteinaceous deposits present in tubular lumen (Fig 1). Interstitium showed mild edema & focal collection of mixed inflammatory cells comprising of lymphocytes, polymorphs, and few histiocytes (Fig 2). Immunofluorescence against all the immmunoglobulin antisera & complements were negative implying there was no immunological process involved in it. Hence the final histopathological impression was Acute tubular injury, fish bile induced. Though liver biopsy was not done, raised SGPT & SGOT level indicated the degree of toxic hepatitis. Patient recovered after four more cycles of hemodialysis and continued supportive treatment . Blood urea & serum creatinine level as well as liver enzymes came down to normal gradually.

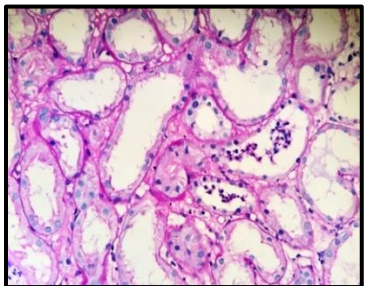

Fig-1: PAS stain: 400x, showing acute tubular injury

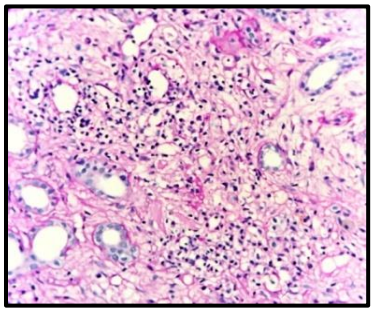

Fig-2: H&E stain 400x, showing interstitial inflammation

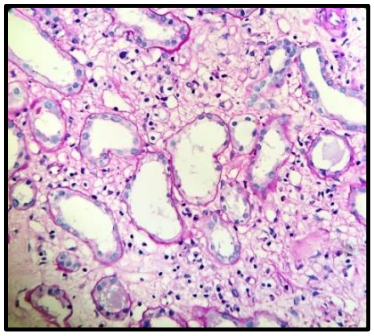

Fig-3: H&E stain 400x, showing acute tubular injury with interstitial edema

CASE 2

A 44-years-old male was admitted to a community hospital after consumption of large grass carp fish gall bladder. Initial symptoms were nausea, vomiting, spasmodic abdominal pain, and watery diarrhea. He was treated in community level hospital with intravenous fluid infusion for three days without any improvement of his condition. After 5 days, he was admitted to our nephrology department, KIMS& PBMH Hospital, for better management. On examination vital signs were stable. However, oliguria or anuria was observed in 24 hours and showed bilateral pitting pedal

edema. Investigations showed the following: HB: 12.3 g/dL, WBC: 6.9 × 10 ^{9}/L,N: 92%, TPC: 50,000/cmm, CPS: Normocytic normochromic blood picture with thrombocytopenia, reticulocyte count: 1%; Urine analysis reported: protein 3+,RBC: plenty/HPF, WBC: 4-5/HPF ; biochemistry reported the following: LDH: 479.25 U/L, Sr Cr: 7.9 mg/dl, sr urea: 108 mg/dl, sr total protein: 6.3 gm /dl, sr albumin:3.5 gm/dl, A/G ratio: 1.25, sr total bilirubin: 2.02 mg/dl, sr direct bilirubin:1.84 mg/dl, sr indirect bilirubin: 0.18 mg/dl, ALT: 776 U/L, AST: 177 U/L, GGT:75.21U/L, sr alkaline phosphatase: 107 U/L, HBsAg /HCV/HIV: Negative . Kidney biopsy revealed acute tubular injury with edema in interstitium on histopathology. (Fig 3) Immunofluorescence was negative for all the immunoglobulins& complements. After 4 cycles of hemodialysis & continued supportive treatment patient recovered.

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 (*C idellus*), common carp, and silver carp. Amongst these, fish of the grass carp variety has been commonly reported for its toxicity. Rohu (Labetorohita) the Indian fish carp is commonly consumed in north eastern and eastern region of India.Its bile contains a toxin,sodiumcyprinol 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 diarrhoea, followed by oliguria and renal failure. The hepatic impairement 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, diarrhoea 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 and hepatic damage. This includes variety of toxins like carbon tetrachloride, trichloroethylene, chloroform, copper sulfate and chromium, mushroom poisoning and drugs including paracetamol overdose and fluorinated anaesthetic agents such as methoxyfluorane and fluoxene. Hence these more common aetiological 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 alerting 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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