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Fish gallbladder consumption almost costing life

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ABSTRACT

Young male of 28 years admitted with anuria, breathlessness and jaundice after consumption of raw Rohu Fish (Labeo rohita belongs to Carp Family) gall bladder which subsequently progressed into acute renal failure and hepatic failure requiring Hemodialysis. His renal Parameter improved over period and Liver function normalized with conservative treatment and the patient was discharged subsequently after about 3 weeks.

Keywords: Rohu Fish, Acute Renal Failure, Hepatic Failure, Hemodialysis

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INTRODUCTION

The practice of consuming Fish gallbladder for some medical purpose is not uncommon in Southeast Asia especially in north eastern India, China, Japan.[1,2] People are in belief that fish gallbladder of grass carp helps in digestion, improves vision, cures joint pain, and cures asthma.[3] There are literatures suggesting fish gall bladder consumption leads to acute renal failure, acute liver injury, and multi organ dysfunction syndrome. Mortality rate following poisoning is quite high.[4] Since there has been increase in people consuming fish gall bladder in this part of the world, we report a young patient who developed acute renal failure and Hepatitis following consumption of Fish gall bladder and recovered completely in due course of time.

CASE REPORT

28 year young male presented to the hospital with the history of 15 episodes of vomiting with passage of about 10 episodes of loose stool over a period of 5 hours with severe pain abdomen colicky in nature and he did not pass urine since then. He was treated symptomatically to start with intravenous fluids, antiemetic and proton pump inhibitor and blood investigations were sent. He started to develop yellowish discoloration of sclera. On further questioning, he gave history of consumption of raw Rohu Fish gall bladder previous night for some digestion problem advised by his family members.

On physical examination, there was dehydration, the pulse rate was 106/min, respiratory rate was 28/min and breathing was acidotic and blood pressure was 130/80 mm of Hg, temperature was 97.8°F and he was Icteric. He was neither pale nor cyanotic. No clubbing, lymphadenopathy or pedal edema was noticed. Abdomen was soft and there was no evidence of organomegaly. Cardiovascular, respiratory and cranial nervous system examinations were normal. Blood investigations performed: haemoglobin 12.2g/dl, total leukocyte count was 8200/mm^3 neutrophil predominant and platelet count was 152000/mm^3 .Erythrocyte sedimentation rate was 18mm in 1st hr and peripheral blood picture showed normochromic, normocytic red blood cells. Urine examination showed abundant granular casts, protein 2+, no RBC. Serum bilirubin 2.0 mg/dl,

SGPT(ALT) 1200 IU/l, SGOT(AST) 881IU/l, Serum protein level- 7.2mg/dl, albumin 3.8 g/dl, alkaline phosphatase 165 IU/l, serum creatinine 6.3 mg/dl and blood urea nitrogen 147 mg/dl. Serum Na^+ 142 mmol/l, K^+ 4.5 mmol/l, Serum amylase 45 mg/dl, Serum lipase 40 mg/dl, Serum calcium 8.7 mg/ dl, magnesium 2.7 mg/dl. Arterial blood gas indicated metabolic acidosis. Chest X-ray, X ray KUB and ECG were normal. Ultrasonography of abdomen showed bilateral minimal pleural effusion and minimal ascites which was further confirmed by non contrast CT Abdomen. HBs Ag, Anti-HCV and HIV were negative. IgM Hepatitis A virus and IgM Hepatitis E virus were negative. Serology for malaria and leptospira were negative. Stool Culture, Blood Culture and Urine culture were sterile. With strong suspicion and evidence of deteriorating renal function, he was put on Hemodialysis along with supportive treatment. He was given lactulose, proton Pump Inhibitor, antiemetic and antibiotic.

Next day, his diarrhea and vomiting subsided, pain abdomen reduced but he was anuric and icteric. On day two, his Blood creatinine was 14.4 mg/dl. He received 5 Hemodialysis over the course of one week during which his urine output started and slowly increased day by day reaching 2.2 l/day after 10 days. Blood creatinine reduced subsequently which was 4 mg/dl on day 12 and Liver function tests were normal: Serum bilirubin 0.8 mg/dl, SGOT (AST) 43 IU/l, SGPT (ALT) 34 IU/l. The patient was then discharged on day 21 with creatinine 2.3 mg/dl, normal liver functions and adequate urine output. Patient was discharged and was asked to follow up after 15 days. His serum Creatinine repeated after 15 days was 1.2 mg/dl and liver function tests were normal.

DISCUSSION

Traditionally, Fish gall bladder has been used for various medical purposes in some parts of Southeast Asia. This can lead to renal failure and Toxic hepatitis in patients who consume raw gall bladder which is reported in India, China, Japan.[1,2] Toxicity is attributed to the Fishes belonging to the family Cyprinidae. The family includes grass carp (*C idellus*), common carp, and silver carp.[5] Amongst these, fish of the grass carp variety has been commonly reported for its toxicity. Rohu (Labeto rohita) the Indian fish carp is commonly consumed in northeastern and eastern region of India. Toxin, sodium cyprinol sulfate which is a C27 bile acid[6] is responsible for systemic effects. The toxin is heat stable and insoluble in alcohol as cases are reported even after consumption of cooked bile.[7] In most of the cases reported, fish gall bladder lengths were over 1 cm long. The volume of bile ingested varied from 15 to 30 ml. 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 several hours later, followed by the manifestations of oliguria and renal failure. The hepatic picture usually precedes the impairment of renal function, but may be concomitant with nephrotoxicity[9] leading to acute tubular necrosis. Kidney biopsy reveals proximal tubular cell damage with focal destruction of epithelial cells on light microscopy.[10] Electron microscopy showed that mitochondria crista of epithelial cells in the proximal tubules were decreased and the renal mesangium was extended. Glomerular cells were swollen and podocytes were partially fused.[10] The toxin is believed to damage lysosomes and inhibit cytochrome oxidase thus blocking cellular metabolism, leading to necrosis of the proximal tubular epithelial cells. When it was administrated to rats, their liver showed multiple focal necrosis and their kidney showed congestion and cloudy swelling of tubular epithelium. This in addition to loss of fluid 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 ingestion.[2,4] Bich Huyen Nguyen Xuan et. al, conducted a large study in Vietnam over 5 years and concluded that the consumption of bile from certain freshwater fish is associated with toxin induced acute tubular necrosis. Acute renal failure after fish gallbladder ingestion has an excellent prognosis. However, death from fulminant hepatic failure can occur.[11] In North Eastern India, Dwijen Das et. al, published a case series on fish bile toxicity and concluded that fish bile toxicity can cause renal failure and hepatic dysfunction which can be lethal if not treated in proper time. Proper management can save lives of these patients.[12] Recently, studies have shown that fish gall bladder can also damage the

heart, liver, gastro-intestinal tract and lead to multiple organ dysfunction syndrome (MODS) in addition to acute renal failure.[13] Treatment comprises of hemodialysis and supportive management. Most of the reported cases till date have undergone hemodialysis in view of renal failure[14] and reported patient responded to the same line of management.

CONCLUSION

High degree of suspicion and proper clinical history should be elicited in order to ensure early detection of cases and timely management. Simultaneous renal and hepatic damage may occur following exposure to a variety of toxins, including trichloroethylene, chloroform , carbon tetrachloride, amantita phalloides (mushroom poisoning), copper sulfate and chromium, and drugs including paracetamol overdose and fluorinated anaesthetic agents such as methoxyfluorane and fluoxene. When the patient with acute renal failure and hepatitis denies exposure to such toxins and drugs, the possibility of ingestion of raw carp gall bladder should be raised. Good awareness can aid diagnosis and prevent death from such a dangerous preventable cause of acute kidney injury.

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