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MULTI ORGAN DYSFUNCTION FOLLOWING INGESTION OF FISH GALL BLADDER

Authors

***Dr. Suprabhat Giri[1], Dr. Sijoy Kurian[2], Dr. Namita Mohanty[3], Dr. Suvendu S Acharya[4], Dr. Nihar R Sahu[5]**

Junior Resident[1,2], Associate Professor[3],Assistant Professor[4],Senior Resident[5]

Department Of General Medicine, MKCG Medical College And Hospital Berhampur, Odisha-India 760004

Corresponding Author

Dr. Suprabhat Giri[1]

Department Of General Medicine, MKCG Medical College And Hospital Berhampur, Odisha-India 760004

ABSTRACT

Fish gall bladder has been used as a delicacy as well as traditional medical remedy for variety of ailments in India. A case of multi organ dysfunction including cardiac dysfunction after consumption of fish gall bladder for traditional medical remedy is reported. The patient fully recovered. Hepatic failure was managed conservatively. The risk of multiple organ dysfunction syndrome following ingestion of fish gall bladder is highlighted.

KEYWORDS: fish gall bladder, poisoning, MODS, cardiac dysfunction

INTRODUCTION:

Consumption of fish's gall bladder as a food item is common amongst the non-vegetarian people of various parts of India. Many people believe that fish's raw gall bladder of grass carp variety (*Ctenopharyngodon idellus)* as food is good for health, improves vision and cures rheumatism. Chinese people believe that fish's gall bladder cures fever and asthma[1]. But many case reports have shown that fish gall bladder can damage the kidney, liver, gastrointestinal tract and lead to multiple organ dysfunction syndrome (MODS). The cause of acute renal function failure can be attributed to necrosis of the proximal tubules[2]. The mortality and morbidity rate following ingestion of gall bladder of fish is significant. Most of the cases reported have a variable combination of renal and hepatic dysfunction but the incidence of cardiac dysfunction is very rare. Because of its clinical significance, we report a case of MODS following consumption of grass carp gall bladder in middle aged male.

CASE REPORT:

A 40 year old male presented to our hospital with 5 days history of yellow discoloration of sclera and decreased urination and 3 days history of head reeling. Patient gave a history of generalized pruritus followed by diarrhea, vomiting and spasmodic pain abdomen 7 days ago after ingestion of a single piece of raw carp gall bladder. 3 days prior blood investigations were done which revealed Sr. urea=164mg/dl, Sr Creatinine=5.4mg/dl, SGOT =240U/L, SGPT=1800U/L, Sr Bilirubin(T) =3.9mg/dl, Urine routine examination revealed Albumin ++, pus cell= 1-2 cell/HPF, Urine bile pigment=trace. Patient presented to our hospital 7 days after ingestion of the fish gall bladder. At presentation he was conscious, oriented, afebrile having HR=36/min, BP=110/70 mm Hg, RR=18/min, Icterus +, no pedal edema, no cyanosis and all other physical

examination normal. Urine output was 100 ml over previous 24 hours. ECG revealed sinus bradycardia. Lab investigations revealed Hb=12 gm%, $N_{78}L_{19}E_2M_1B_0$, TLC=13600/cu mm, Platelet count=3 lakh, Urine albumin + , Sr. Urea= 79 mg/dl, Sr. creatinine=8.6mg/dl , Sr. Na^+=122meq/L , Sr. K^+=5.9meq/L, Sr. SGOT=54U/L, SGPT=385U/L, ALP=181U/L, Bilirubin(T)=1.42mg/dl, Bilirubin(D) =0.9mg/dl and Sr Trop. I =0.2 ng/ml. 2D Echocardiography was normal Patient was started on iv frusemide, hepatoprotectives and atropine. Next day output was 200 ml over 24 hours. On day 3 of admission patients had Sr. urea =95 mg/dl and creatinine =12.5 mg/dl. Patient was put on hemodialysis and after 3 sessions of dialysis on consecutive days his renal function test came down to Sr. urea= 44 mg/dl and creatinine =4.1 mg/dl with urine output 3L over 24 hours. LFT became normal by day 4 of admission. Heart rate became normal without atropine by day 7 of admission . Patient was discharged on day 8 of admission with Pulse= 68/min, BP= 120/80 mm Hg, Sr. urea=40 mg/dl, Sr. creatinine= 2.1mg/dl, Sr Bilirubin(T) =1.1mg/dl and Sr Bilirubin (D)=0.5mg/dl.

DISCUSSION:

Among fish poisoning, ciguatera and scombroid poisoning are the most commonly recognized. However, the raw gall bladder of the grass carp (*Ctenopharyngodon idellus*) with both nephrotoxic and hepatotoxic properties is less known. After ingestion, most patients present initially with gastrointestinal upset including abdominal pain, nausea, vomiting and watery diarrhoea several hours later, followed by the manifestations of the central nervous system and oliguria. The hepatic picture usually precedes the impairment of renal function, but may be concomitant with nephrotoxicity[3]. Spontaneous resolution of hepatic dysfunction within a few days is usual. More serious is the nephrotoxicity, which culminates in oliguric or non-oliguric

acute renal failure, usually within 48–72 h after ingestion while hepatic dysfunction is resolving. Deaths following ingestion of raw carp gall bladder have been described mostly due to AKI[4]. Acute renal failure occurs in 50-100% of all fish gall bladder poisoning and fish gall bladder poisoning accounts for 91.7% of the mortalities due to ARF[1]. Various causes have been implicated in the pathogenesis of acute renal failure after the ingestion of raw carp gall bladder. These include direct nephrotoxicity of the bile, bradycardia and systemic hypotension secondary to the accumulation of the bile, and volume depletion either due to initial natriuresis and diuresis, or protracted diarrhoea. Histological studies of kidney and live tissue specimens from patients usually demonstrate acute tubular necrosis and focal hepatitis. Deng et al. found that light microscopy showed damages to epithelial cells in the proximal tubule and focal destruction of epithelial cells. Electron microscopy showed that mitochondria crista of epithelial cells in the proximal tubules had decreased or disappeared and the renal mesangium was extended. Glomerular cells were swollen and podocytes were partially fused; lysosomes were broken. Partial potocytic processes are fused. It is believed that the toxin in fish gall bladder damages or breaks lysosomes, meanwhile inhibiting cytochrome oxidase and blocking cellular energy metabolism, so as to cause necrosis of the proximal tubular epithelial cells[5]. Although the bile components responsible for these pictures have not been characterized fully, cyprinol, a C27 bile alcohol found to be present in bile of the family represented by the common carp may have a direct effect on the kidneys and liver[6]. No specific antidote or treatment is available for fish gall bladder poisoning. If ingestion is recent gastric content should be removed with emetics and purges. Hydration should be maintained to prevent development of acute renal failure. Hemodialysis plays a

key role in the management of acute renal failure. Spontaneous resolution of liver dysfunction is usual and can be managed conservatively. Atropine improves the sinus bradycardia and gastrointestinal symptoms[7]. With this multi modal management patients with MODS usually recover.

CONCLUSION

Simultaneous renal and hepatic damage may occur following exposure to a variety of toxins, including carbon tetrachloride, trichloroethylene, chloroform, 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. In the light of these toxic effects, clinicians should not only be alert to these toxic complications after the ingestion of raw carp gall bladder, but also aware of eating habits and food exposure that may pose a risk for their patients[8].

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