Lesions are usually described as oval, ill-defined nodules, either unique or more frequently multiple. Imaging reveals multiple clustered complex cystic lesions, best appreciated on portal venous phase, with little or no peripheral enhancement or surrounding edema. These are usually small, less than 2 cm in size and likely to be along the portal vein branches or liver periphery with occasional traversing of the portal vein through the larger lesions. However, we encountered two cases with lesions showing diffusion restriction and distinct peripheral enhancement on portal venous phase and can thus mimic amoebic or pyogenic abscesses. Furthermore, imaging revealed portal vein thrombosis and abscess rupture in two of our patients. To the best of our knowledge, these complications have never been reported in literature in cases of hepatic larva migrans. Treatment with albendazole alone or in combination with ivermectin is reported to be effective,[6] although long term therapy is needed for complete eradication.

References

1. Chang S, Lim JH, Choi D, Park CK, Kwon NH, Cho SY, Choi DC. Hepatic visceral larva migrans of Toxocaracanis: CT and sonographic findings. AJR Am J Roentgenol. 2006 Dec;187(6):W622-9.
2. Lim JH. Toxocariasis of the liver: visceral larva migrans. Abdom Imaging. 2008 Mar-Apr;33(2):151-6.
3. Glickman LT, Magnaval JF, Domanski LM, Shofer FS, Lauria SS, Gottstein B, Brochier B. Visceral larva migrans in French adults: a new disease syndrome? Am J Epidemiol. 1987 Jun;125(6):1019-34.
4. Hayashi K, Tahara H, Yamashita K, Kuroki K, Matsushita R, Yamamoto S, Hori T, Hirono S, Nawa Y, Tsubouchi H. Hepatic imaging studies on patients with visceral larva migrans due to probable Ascaris suum infection. Abdom Imaging. 1999 Sep-Oct;24(5):465-9.
5. Mukund A, Arora A, Patidar Y, Mangla V, Bihari C, Rastogi A, Sarin SK. Eosinophilic abscesses: a new facet of hepatic visceral larva migrans. Abdom Imaging. 2013 Aug;38(4):774-7.
6. Raffray L, Le Bail B, Malvy D. Hepatic visceral larva migrans presenting as a pseudotumor. Clin Gastroenterol Hepatol. 2013 Jun;11(6):e42.

Syndrome of Acute Hepatitis and Acute Renal Failure after Raw Fish Bile Consumption: A Case Series

Prasanna K S

Apollo Hospital, Guwahati.

Corresponding Author: *Dr Prasanna K S*
Email: prasannaks19@gmail.com

Rohu (*Labeorohita*) fish is commonly found and consumed in South East Asia (India, Pakistan, Bangladesh, Nepal and Myanmar) as it is abundantly available in this area. It is a common practice among certain groups of people to consume raw gall bladder of fish to prevent malaria, for vision improvement and for good health.[1]

We report one of the rare causes of toxic hepatitis along with acute renal failure which is caused by consumption of fresh fish bile. There are few case reports from Asian countries and one from USA which have reported renal failure along with acute hepatitis after fish bile consumption.[1-3]

Four people from North Assam consumed bile of a Rohu fish. Two of them consumed small quantity of bile (5-10 ml) and had abdominal discomfort and vomiting and the other two had renal and liver injury for which they were referred to our hospital.

Case 1

A young 25 year old male from Lakhimpur, north Assam had consumed about 25 ml fish bile (Rohu fish), 2 -3 hours after consuming bile patient noted upper abdominal pain along with nausea and vomiting. When he was evaluated next day at his place his liver and kidney function tests were abnormal and patient even noted decrease in urine output. With above details he was referred to our hospital. On admission he was hemodynamically stable,

he had Icterus, abdomen was soft, and there was no hepatomegaly). His urine output was low but was not acidotic. His liver and renal function tests were deranged. Ultrasound abdomen revealed acute renal parenchymal disease changes.

He was started on intravenous fluids and was monitored carefully. His urine output gradually improved over next 3-4 days and following that his creatinine and liver function tests improved significantly. He was discharged in stable condition from hospital. After a week he came for follow up, he was asymptomatic and his creatinine was 1.8 mg% and his liver function was normal.

Case 2

Another young male of 30 yrs age also consumed same bile (about 50 ml) following which he noticed pain abdomen and vomiting. He was also found to have renal and liver injury on evaluation at their place and was referred to out hospital.

By next day patient had significant decrease in urine output and by the time he came to our center he was anuric and was found to have metabolic acidosis secondary to acute renal injury. He also had hepatitis. In view of anuria and acidosis he underwent hemodialysis along with other supportive therapy. Total of 6 sessions of dialysis were done over 2 weeks period and following that his urine out improved. He was discharged from hospital in a stable condition.

Case 3

Young male of 30 years age who had consumed small quantity (10 ml) fish bile along with above two cases presented to us after 10 days of consumption. He had developed nausea and vomiting 2-4 hours following bile consumption and was treated at his place and apparently improved over next 24 hours. By the time he presented to us his liver and kidney function tests were normal.

Discussion

Those who consume fish bile in significant quantity may develop various problems starting with mild gastrointestinal discomfort to multi organ dysfunction. Most of the patients develop liver and kidney injury.[3]

The volume of bile consumed is the main determinant of severity of toxicity caused by bile consumption.[2] As in our case series, one who consumes large quantity (approximately >25 ml) will develop severe liver and kidney injury. In our case series, patients also reported history of similar fish bile consumption without any adverse effects many times in the past but in small quantity which again points towards dose related toxicity of fish bile.

Patients may present either with oliguric or non-oliguric renal failure as in our case series, both have good prognosis. Few case reports have reported even multi-organ damage including pancreas and heart in addition to kidney and liver

It is very important to note that usually the prognosis is very good with supportive therapy but there is a reported case of death due to fulminant liver failure following fish gall bladder consumption from Vietnam[3] which needs to be always kept in mind.

Gall bladder size and bile yield from fish is very minimal quantity. According to one study bile yield is 1% of the total body weight4, in certain geographical areas like in Assam where the fresh water fish quantity and size is remarkably higher and in turn the amount of bile from those fish, this can explain the incidence of such cases restricted to specific geographic areas. As this is limited to specific geographical location we can try and educate people about these effects so that these can be prevented in future.

The causative agent in bile which causes this toxicity is not yet clear, toxin in fish gall bladder is believed to damage or break lysosomes, meanwhile inhibiting cytochrome oxidase and blocking cellular energy metabolism, so as to cause necrosis of the proximal tubular epithelial cells. The toxin believed to be behind all this nephrotoxicity and hepatotoxicity is cyprinol sulphate or cyprinol, a C27 bile acid.[5]

In conclusion, fish gall bladder or bile consumption can cause severe liver and renal injury, the severity depends on the amount of bile consumed and

usually patients recover with supportive therapy. This can be prevented by creating awareness in those areas where this practice is prevalent.

References

1. P. C. Bhattacharyya, M. Nayak, and A. Barkataky; Acute renal failure following consumption of fish gall bladder. Indian J Nephrol. 2009 Oct; 19(4):161-2
2. Dwijen Das, Kallol Bhattacharjee, Amit Kr. Kalwar, Bhaskar Debnath. "A Case Series on Fish Bile Toxicity". Journal of Evidence based Medicine and Healthcare; Volume 2, Issue 33, August 17, 2015
3. Xuan BH, Thi TX, Nguyen ST, Goldfarb DS, Stokes MB, Rabenou RA, Ichthyotoxic ARF after fish gallbladder ingestion: a large case series from Vietnam, Am J Kidney Dis. 2003 Jan;41(1):220-4.
4. P T Mathew, K G Ramachandran Nair, P Madhavan, P V Prabhu, Isolation of bile from fish gall bladder and identification by thin layer chromatography, Journal of fishery technology, volume 23, 1986
5. Hwang DF, Yeh YH, Lai YS, Deng JF: Identification of cyprinol and cyprinol sulfate from grass carp bile and their toxic effects in rats.Toxicon. 2001;39:411-4

A Salvage for Upper Gastro Intestinal Bleeding: Better Use of Existing Resources

Sanjeev K Thakur[1], Sachindra K Astik[2], Vijay Prakash[3]

[1]Department of Gastroenterology, Bihar Institute of Gastroenterology, Patna. [2]Department of Medicine, Nalanda Medical college, Patna. [3]Department of Gastroenterology, Patna Medical College, Patna.

Corresponding Author: *Dr Sanjeev K Thakur*
Email: sanjeevtk02@yahoo.co.in

Upper Gastrointestinal (UGI) Bleeding is a common medical emergency. Variceal bleeding accounts for nearly 15% of all UGI Bleeds.[1] It is sometimes torrential and associated with high morbidity and mortality with reported rates varying from 10-20%.[2] Combination therapy with vasoactive drugs (within 30 min of hospitalization) and endoscopic variceal ligation (door to scope time within 12 hrs of admission) is the current standard of care.[3] Resource poor places have limited availability to both. Many cases in such situations have adverse outcome because of delayed availability of proper medical care and failure of initial hemostasis. We herewith present a case where a novel use of widely available medical equipment averted a crisis.

Case Report

Forty two year male, chronic alcohol consumer, a known case of cirrhosis with portal hypertension, had Child A status and small esophageal varices. He was lost to follow up after initial work up. He presented to us one morning with history of recurrent hematemesis, malena and giddiness. He was started on Intravenous Somatostatin, antibiotics and IV fluids and Ryle's Tube (RT) aspirations. He was planned for Endoscopy after initial stabilization. He continued to bleed, his sensorium worsened and his blood pressure was dropping despite doubling the dose of Somatostatin. We had no Intensive care back up and Sangestaken Blakemore (SB) Tube was not available. His condition and rapid progression of events were not allowing for a transfer to a better equipped place.

We modified the RT by tightly securing a Latex condom at the end it (above the proximal feeding holes). A leak test was performed by filling the condom with 300 ml of water outside. After insertion up to 60 cms, we instilled 100 ml of water through the RT, 50 ml of air was then pushed and gush was heard in left hypochondriac region, to ensure position in to the stomach. It was filled further with 200 ml of water and a gentle pull back was done till resistance was felt. Tube position was marked at this place with a tape and was secured at the nostril with the help of Needle cap and tapes.

Bleeding stopped immediately, hemodynamic parameters started improving. He was transfused one unit of packed blood. We deflated the system after 12 hrs and took him up for do upper Gastro Intestinal Endoscopy. He had four columns of small varices with red colour signs