Case Report

Gastric Outlet Obstruction Due to Extraluminal Compression: An Unusual Etiology

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Abstract

Ascariasis caused by the nematode Ascaris lumbricoides is a very common disorder amongst the Asian population. Adult roundworms are generally found in the intestine. But their agility often takes them through different orifices into ducts and cavities. Hepatobiliary and pancreatic ascariasis (HPA) refers to a group of diseases caused by migration of the Ascaris from the intestine into the biliary tree and pancreatic duct through the ampulla of Vater. Depending on the position of the nematode, HPA has widespread manifestations like biliary colic, acalculous cholecystitis, obstructive jaundice, cholangitis, pancreatitis and liver abscess. Surgical interventions are rarely required as approximately 95% of the patients with uncomplicated biliary ascariasis respond to conservative management. Ultrasonography of the abdomen, CECT & ERCP all can demonstrate ascaris as linear filling defect . Here we present a case that presented with features of acute gastric outlet obstruction and obstructive jaundice as a result of extraluminal antral compression by a worm ball.

Keywords: Ascaris lumbricoides, Hepatobiliary and pancreatic ascariasis, obstructive jaundice, gastric outlet obstruction

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Introduction

Ascariasis caused by the nematode Ascaris lumbricoides is a very common disorder amongst the Asian population. Adult roundworms are generally found in the intestine (mainly jejunum). But their agility often takes them through different orifices (1). Hepatobiliary and pancreatic ascariasis (HPA) refers to a group of diseases caused by migration of the Ascaris from the intestine into the biliary tree and pancreatic duct through the ampulla of Vater. In most cases, the nematode moves back into the intestine within 72 hours. Depending on the position of the nematode, HPA has widespread manifestations. We present a case that presented with acute gastric outlet obstruction and obstructive jaundice caused by a ball of ascaris.

Case Report

A fifty year old male patient presented with intermittent colicky pain in right hypochondrium for 1 month, for which he was taking medication as prescribed by his physician. Patient also developed jaundice with passage of high coloured urine, clay coloured, stool, pruritus and anorexia. He gave a history of projectile vomiting of food taken more than 24 hours ago for last 5 days. The patient was brought to the emergency in hypovolemic shock. Inspection of abdomen showed visible peristaltic waves from left to right. Right hypochondrium was highly tender with localized rigidity. Auscultopercussion revealed a dilated stomach. Investigations revealed mild leucocytosis, raised serum urea and creatinine, increase in total (7mg/dl) and direct bilirubin with moderately raised liver enzymes. Patient was resuscitated and haemodynamically stabilized. Further investigations were done to find the cause of gastric outlet obstruction and obstructive jaundice.

USG revealed dilated intrahepatic biliary radicles (IHBR), an ill defined 7.7 x 7 x 5cm collection with interval echoes and multiple echogenic tiny foci with posterior acoustic shadow in gall bladder (GB) fossa, 13mm dilated common bile duct (CBD) containing multiple calculi and a moving round worm in CBD (Figure 1A).

Upper GI endoscopy (UGIE) revealed a bulge in antrum of stomach due to extrinsic compression. Duodenum was deformed, ampulla could not be identified due to distorted anatomy (Figure 2). Contrast enhanced CT scan showed dilated IHBRs, irregular GB with increased attenuation in lumen, CBD dilated up to lower end with calculus impaction, and a 6x6cm collection around GB fossa containing linear slender structures, (suspected to be worms), compressing against stomach (Figure 1B). CT guided aspiration from the collection yielded bilious fluid.

The patient vomited quite a few living round worms two days after admission. He was given piperazine citrate syrup by Ryle's Tube for five consecutive days followed by ivermectin and albendazole tablets orally with a purgative to flush out dead worms.

By fifth day of administering piperazine, the patient improved significantly. There was cessation of vomiting and abdomen became soft. Bilirubin level came down.

Repeat UGIE showed disappearance of the extraluminal bulge from gastric antrum and clear visibility of ampulla. USG also revealed a gall bladder lumen free of ascaris. Endoscopic papillotomy followed by extraction of multiple stones and dead worms from CBD was done. Patient was subsequently discharged and kept on follow up. He has not developed any further attacks since discharge.

Discussion

Ascaris lumbricoides is the commonest pathogenic nematode found in the small intestine of human beings. Although most of the cases remain asymptomatic, there may be varied presentations of this condition, the commonest being colic and passage of worms with stools. However, some patients may



Figure 1: A) USG showing mass at gall bladder fossa B) CECT scan showing worm ball at gall bladder fossa causing extrinsic compression of antrum of stomach.



Figure 2: Upper GI endoscopy showing huge antral bulge.

present with malabsorption, malnutrition and abdominal distension. In extreme cases intestinal obstruction by worm bezoars, perforation, strictures, suppuration or abscess at various levels of the gastrointestinal tract may occur. Worms that migrate by retrograde flow may present as HPA. The worms may occupy the common bile duct, hepatic ducts; enter the gallbladder through the cystic duct or enter the pancreatic duct. But in view of the narrow and tortuous nature of the cystic and pancreatic duct, the incidence of worms in the gall bladder and pancreatic duct is rare. Depending on the position of the nematode, HPA has widespread manifestations like biliary colic, acalculous cholecystitis, obstructive jaundice, cholangitis, pancreatitis and liver abscess (2,3,4, 5). Dead or calcified worms may act as a nucleus for stone formation in biliary tree. Previous surgeries in biliary tree increases the chance of recurrent biliary ascariasis (4) due to increased level of cholecystokinin, which leads to relaxation of the sphincter of oddi.

Ultrasonography is recommended as a safe, noninvasive diagnostic modality with high sensitivity for biliary ascariasis, which are demonstrated as long linear echogenic shadows in the biliary tree (6). Real time sonography can also effectively monitor the movement of the worms through the ampulla of vater and back. ERCP is the gold standard as it not only visualizes the worms, but may also be used for their extraction from the biliary tree. However, it must be performed during active symptoms, as the worms may otherwise move back into the duodenum.

Worms in stomach are generally vomited out or form worm bezoars. Cases of intrinsic gastric outlet obstruction by worm bezoars are frequently reported. But review of literature did not reveal any reporting of gastric outlet obstruction due to extraluminal compression by a worm ball in GB fossa.

Surgical interventions are rarely required as almost 95% of the patients with uncomplicated biliary ascariasis responds to conservative management (7). As far as patients with gall bladder ascariasis goes, if they expel the worms with conservative management, they have to be followed up regularly, as they may have similar attacks again. Those who improve symptomatically with conservative management, but fail to expel the worms, need to undergo cholecystectomy (8). ERCP has proved to be extremely useful in extracting dead worms from the biliary tree, particularly if they are also associated with calculi in the biliary tree.

Conclusion

HPA is a known entity commonly involving the intra and extrahepatic biliary tree. Involvement of the GB is less common and may lead to cholecystitis. But extrinsic compression of the antrum of the stomach by GB ascariasis leading to gastric outlet obstruction has not been reported in literature so far. Though most patients respond to conservative management, there is a role of cholecystectomy for patients who do not expel the worms from the GB with conservative management. A high index of suspicion should be there when dealing with patients of acute onset gastric outlet obstruction and biliary-pancreatic disorders, particularly if the patient comes from an endemic area.

References

- 1. Langewar DN, Maheshwari MB, Wegholikar UL. Hepatic perforation due to ascariasis. Indian J Pediatr. 1993; 60(3): 457–9.
- Sandouk F, Haffar S, Zada MM, Graham DY, Anand BS. Pancreatic-biliary ascariasis: Experience of 300 cases. Am J Gastroenterol. 1997; 92(12): 2264 –7.
- Louw JH. Biliary ascariasis in childhood. S Afr J Surg. 1974; 12(4):19–25.
- Khuroo MS, Zargar SA, Mahajan R. Hepatobiliary and Pancreatic ascariasis in India. Lancet 1990; 335(8704):1503-6.
- 5. Lloyd DA. Massive hepatobiliary ascariasis in childhood. Br J Surg. 1981; 68(7):468–73.
- 6. Khurro MS, Zargar SA, Yatoo GN, et al. Sonographic findings in gall bladder ascariasis.J Clin Ultrasound 1992(9);20:587-589.
- Gomez NA, Ortiz O, León CJ, Iñíguez S. Ascariasis of the gallbladder. Report of 2 cases and review of the literature. Acta Gastroenterol Latinoam. 1992; 22(2):129-31.
- 8. Javid G, Wani N, Gulzar GM, Javid O, Khan B, Shah A. Gallbladder ascariasis: presentation and management. Br J Surg. 1999; 86(12):1526-7.