

Mass Forming Biliary Ascariasis Masquerading as Gallbladder Cancer: A Case Report and Review of Literature

Lokesh Agarwal¹, Nihar Ranjan Dash^{1*}, Adil Afsan¹ and Kumble Seetharama Madhusudhan²

¹Department of Gastrointestinal Surgery and Liver Transplant, AIIMS, New Delhi, India ²Department of Radiodiagnosis, AIIMS, New Delhi, India

*Corresponding Author: Nihar Ranjan Dash, Department of Gastrointestinal Surgery and Liver Transplant, AIIMS, New Delhi, India.

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Abstract

Biliary ascariasis is a rare clinical entity and its presentation as a mass forming variant mimicking carcinoma gall bladder is unusual. We describe a rare case of biliary ascariasis masquerading as carcinoma gall bladder in a young female. A 24-year-old female with clinico-radiological features of a malignant gall bladder lump turned out to be biliary ascariasis. This report highlights the importance of keeping this infectious inflammatory etiology in the list of differential diagnosis of a gall bladder mass, especially in young patients from ascaris endemic areas.

Keywords: Biliary Ascariasis; Masquerade; Gall Bladder Cancer; Gall Bladder Mass; Ascaris

Abbreviations

GB: Gall Bladder; GBC: Gall Bladder Cancer; MR: Magnetic Resonance; MRCP: Magnetic Resonance Cholangiopancreatogram; FNAC: Fine Needle Aspiration Cytology; USG: Ultrasound; PV: Portal Vein; HA: Hepatic Artery; PVE: Portal Vein Embolization; FLR: Future Liver Remnant; HIDA: Hepatobiliary Iminodiacetic Acid; CHD: Common Hepatic Duct; PTBD: Percutaneous Transhepatic Biliary Drainage

Introduction

Ascariasis remains an important clinical condition in the developing world [1,2]. It is among the most common helminthic infections in humans. The infection spreads via the faeco-oral route and hence it's increased prevalence in tropical and subtropical developing countries, where poverty, poor sanitation and overcrowding are prevalent. The adult ascaris normally resides in the small intestine and has been known to migrate to various organs such as the lungs, urinary bladder, peritoneum, and biliary system [3]. Involvement of gall-bladder is rare, constituting 2.1% of the total cases of biliary ascariasis [4]. This rarity is explained by the narrow and tortuous cystic duct which makes it difficult for the worm to negotiate through. Sometimes in patients with non-specific symptoms, differentiating inflammation from malignant etiology on imaging can be a diagnostic challenge. This is of further concern in countries like India, with a high burden of both infections and malignancies. Gallbladder (GB) cancer is the most common biliary tract malignancy and manifests as either thickening of the GB wall or as a GB mass arising from the fundus, neck or body of the GB. We report a case of a GB lump with jaundice, with imaging characteristics of gall bladder cancer (GBC), which eventually turned out to be biliary ascariasis.

Case Presentation

A 24-year-old female, resident of North-India presented with complaint of intermittent pain abdomen for the last 10 months. The pain was predominantly over the right upper quadrant of the abdomen and was moderate in intensity. She noticed increasingly

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yellowish discoloration of her eyes and urine over the last three months. She also experienced itching over the arms and legs that worsened at nighttime. She had complaint of loss of appetite and an unintentional weight loss of 6 kg over the last 3-months. She did not report any fever episode during this period. She had no medical comorbidities. She was educated up to twelfth standard and belonged to a poor socioeconomic status. On examination the patient had a good performance status. The lady was deeply icteric, and a hard non tender gall bladder lump was palpable on abdominal examination. No abnormality was noted on per-rectal examination.

Investigations

Laboratory workup at presentation revealed conjugated hyperbilirubinemia (Table 1). An ultrasound (USG) of the abdomen showed a distended thick-walled GB with few sub-centimetric calculi within the lumen. A hypoechoic mass of 3.4 x 2.7 cm was noted at the GB neck, infiltrating common hepatic duct with proximal intrahepatic biliary radical dilatation. Based on the clinical and USG findings, a working diagnosis of GBC was made.

Laboratory Parameters	At presentation	3-weeks after PTBD	Reference range
Hemoglobin (gm/dL)	11	11.6	12-15
TLC (cells/mm ³⁾	8900	7400	4000-10,000
Platelet count (cells/mm ³⁾	4,00,000	3,80,000	1,50,000-4,00,000
Urea/Creatinine (mg/dL)	14/0.5	16/0.7	10-40/0.5-1.0
Total/Direct bilirubin (mg/dL)	8.8/7	1.4/0.8	0.2-1.2/0.1-0.3
Serum Alkaline Phosphatase (IU/dL)	1355	768	80-240
AST/ALT (IU/dL)	93/57	45/34	5-40/5-45
Total protein/Albumin (gm/dL)	6.7/3.5	7.2/3.9	6-8/3.5-5.0

Table 1: Showing patient's blood investigations at presentation.

Abbreviations: TLC: Total Leucocyte Count; AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase; PTBD: Percutaneous Transhepatic Biliary Drainage.

A contrast-enhanced MRI was done to further stage the disease (Figure 1). The clinico-radiological diagnosis was a malignant appearing locally advanced GB mass with right PV and HA encasement. USG-guided fine needle aspiration from the GB mass failed yield adequate sample for histopathological analysis.

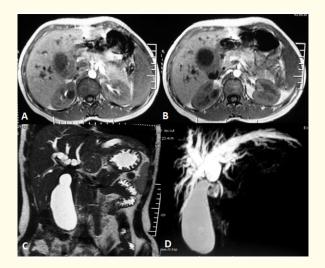


Figure 1: A, B) Axial post gadolinium MRI sequences shows asymmetrically thickened GB wall with intra-hepatic biliary radical dilation (IHBRD). C, D) MRCP coronal and thick slab images show bilateral IHBRD with distended GB. No intraluminal filling defect was noted in the GB.

Treatment

The patient was planned for an extended right hepatectomy with biliary reconstruction. However, the volume of the left lateral segments formed 18% of the total liver volume and was insufficient. A left sided percutaneous transhepatic biliary drainage (PTBD) was done to decrease the serum bilirubin levels pre-operatively. Three weeks after the PTBD a right portal vein embolization (PVE) was done which adequately increased the volume of the future liver remnant (FLR) after four weeks (Figure 2). Intraoperatively, a hard GB mass infiltrating the antrum of the stomach was noted (Figure 3a). There was multiple enlarged peri- choledochal and peri-portal lymph nodes. When the common hepatic duct was divided at the level of the primary confluence, an ascaris worm was seen protruding through its distal cut end (Figure 3b). A right hepatectomy with a left Roux-en-Y hepaticojejunostomy and distal gastrectomy with gastrojejunostomy was performed (Figure 3c). The left PTBD was retained postoperatively. Histopathology report showed eosinophilic infiltrates in the portal tracts and presence of giant cells with intracytoplasmic lamellated structures, suggestive of parasitic etiology. No features to suggest malignancy were noted.

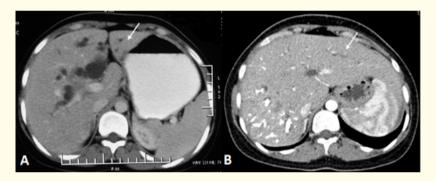


Figure 2: A) Shows pre- PVE liver with bilateral intra-hepatic biliary radical dilation (IHBRD). Arrow showing segment II and III B) Post PVE axial CT section through the liver, shows hypertrophied segments II and III (arrow). Lipiodol-glue noted in the right portal venous branches.

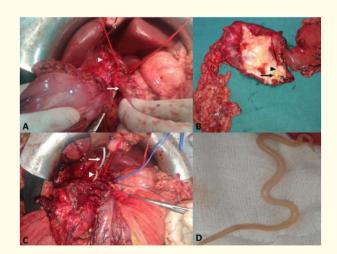


Figure 3: A) Intra-operative image showing large GB mass infiltrating the common bile duct (CBD) and the antrum of stomach; B) Ascaris worm (arrowhead) was seen peeping through the CBD. PTBD catheter noted (arrow); C) Ascaris worm shown over a surgical sponge. D) Resected specimen showing mass in GB neck with GB calculus.

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Outcome and follow-up

Post-operatively patient had bile leak from the cut liver surface, which was managed conservatively. Antihelminth were started (tab. albendazole 400 mg BD x 4 weeks). She was discharged on post-op day 22 with sub-hepatic drain *in situ* (daily output 50 ml bile/day) and PTBD stent kept on external drainage. One month postoperatively the bile leak stopped, and the drain was removed. PTBD gram and HIDA scan 2-months after the surgery, showed normal bilio-enteric drainage and no leak. The PTBD catheter was then removed. Now four years after the surgery the patient is doing well.

Discussion

Ascaris infections are usually asymptomatic, but a large burden of infection may result in abdominal pain, especially if there is intestinal obstruction. Ascaris has been noted to infect other sites including the bile duct, liver, and appendix. Although bile duct ascariasis has been reported frequently, GB remains an uncommon location for the nematode. Clinical presentation of GB ascariasis mimics that of acute cholecystitis [5]. On imaging the GB may be normal or have a thickened wall, with dilated biliary ducts. USG may reveal the nematode as a non-shadowing, echogenic, tubular structure within the GB lumen or the bile duct [6-8]. On MRCP worms are seen as linear low-signal intensity filling defect within the biliary system [9]. These typical imaging findings are however not always present.

There are a few case reports of ascaris infection and associated extra-hepatic cholangiocarcinoma [10,11]. The inflammatory response to infection may lead to an increased risk of cholangiocarcinoma in a similar manner as is observed with liver fluke infection. The inflammatory response of the host against the worm may cause asymmetrical GB wall thickening, mimicking a malignant pathology on imaging [12]. FNAC from the areas suspicious of malignancy, have a low diagnostic yield. A negative cytopathological examination does not change the management in the presence of a strong clinico-radiological suspicion of malignancy.

The established treatments for biliary ascariasis are anti-helminthic drug therapy, endoscopic extraction, and surgical extraction. Bile duct ascariasis responds better to anti-helminthic drug therapy and endoscopic extraction. GB ascariasis however often requires surgical management [4]. Although biliary ascariasis masquerading as choledocholithiasis and cholangiocarcinoma has been described [13], its presentation as a mass forming GBC is extremely rare [12].

Conclusion

The present case adds one more to the masquerades of GBC. A number of inflammatory conditions have been reported to mimic GBC. The value of differentiating such inflammatory pathologies from malignant etiologies cannot be overemphasized. In our case a young female with clinico-radiological features of a malignant GB lump turned out to be biliary ascariasis. In patients from an ascaris endemic area, with a history atypical for malignancy and jaundice especially in young age, possibility of inflammatory etiologies should be kept in mind.

Data Sharing Statement

All relevant data supporting the conclusions of this article are included within the article.

Ethical Approval

The need for institutional ethics approval for this case report was waived.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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Author Contributions

All authors made substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data; took part in drafting the article or revising it critically for important intellectual content; agreed to submit to the current journal; gave final approval of the version to be published; and agreed to be accountable for all aspects of the work.

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Disclosure

The authors of this article declare that they have no conflicts of interest.

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