CASE REPORTS

# Recurrent cholangitis secondary to Ascaris lumbricoides – a rare occurrence

Nawin Kumar[1], Rajesh Nair*[1], Anuja Sinha[2], Amit Kumar[1]

[1] *Department of General Surgery, Kasturba Medical College, Manipal University, Manipal, India*
[2] *Department of Anatomy, Kasturba Medical College, Manipal University, Manipal, India*

**Received:** June 17, 2015 **Accepted:** July 19, 2015 **Online Published:** August 8, 2015
**DOI:** 10.5430/css.v1n1p23 **URL:** http://dx.doi.org/10.5430/css.v1n1p23

## ABSTRACT

Partial obstruction of sphincter of Oddi, secondary to Ascaris, is a clinically descried entity. Herewith we describe our institutional experience in a 46-year old gentleman who presented with obstructive jaundice and recurrent cholangitis. On surgical exploration of the common bile duct (CBD), a round worm was found obstructing the proximal lumen and on extraction of the worm, the patient was rendered symptom free postoperatively. He remained asymptomatic after three weeks and on further follow up. Here we describe our case.

**Key Words:** *Ascaris lumbricoides*, T-tube cholangiogram, Recurrent cholangitis

## 1. INTRODUCTION

*Ascaris lumbricoides* is the largest intestinal nematode, often found in humans, which is widely distributed in the tropical and subtropical region. Transmission is usually accidental, following consumption of food infected with eggs of the parasite or by droplet transmission. Biliary ascariasis is usually secondary to intestinal infestation and the worm usually moves through the ampulla of vater and part of worm may remain within the duodenum, causing surgical jaundice. They usually cause partial obstruction, due to spasm of the sphincter of Oddi, and it eventually causes cholangitis secondary to chemical irritation with or without bacterial infection. Complications following partial obstruction include empyema, perforation of the gall bladder and occasionally jaundice secondary to acalculous cholecystitis.

## 2. CASE REPORT

We present the case of a 46-year-old male, who presented to the hospital with high-grade fever, vomiting, jaundice, abdominal pain and oliguria since one week. One year earlier, he had had a similar bout with the exception of jaundice. He was a farmer by occupation and there was no significant travel history, to any tropical or subtropical region. Laboratory examination revealed elevated liver enzyme levels with biochemical evidence of biliary obstruction (Alkaline phosphatase [ALP] 257 U/L; AST 142 U/L; ALT 148 U/L; and Bilirubin of 5.7 mg/dl), along with anaemia (Hb 6.9%) and leukocytosis (TLC 17,500) with left shift, suggestive of toxic change. His renal parameters were also deranged (urea 176 mg/dl, creatinine 6.1 mg/dl). Blood culture grew Escherichia Coli and ultrasonography of the abdomen depicted a dilated common bile duct (CBD) with the lumen filled with an echogenic linear shadow. Endoscopic retrograde cholangiopancreatography was attempted (4th day post admission) but abandoned due to the presence of a deformed first part of duodenum. Open cholecystectomy with choledochotomy and open exploration of the CBD was planned. Intraoperative exploration of the CBD revealed a coiled round worm ob-

*Correspondence: Rajesh Nair; Email: rajeshnair39@yahoo.com; Address: Department of General Surgery, Kasturba Medical College, Manipal University, Manipal, India.

structing the lumen entirely, which was then carefully milked out and extracted in toto and delivered out (see Figure 1). On-table cholangiogram (OTC) was performed and T tube was placed into the cleared CBD. Postoperatively, patient was dewormed with a single dose of 400 mg Albendazole and antispasmodics. A T-tube check-cholangiogram was done on post-operative day fourteen, which showed free flow of bile into the proximal and distal CBD with no filling defects, it was co-related with an abdominal ultrasound, which showed persistence of CBD dilatation with T-tube in situ and absence of echogenic material. T-tube was pulled out following the study. The patient was discharged three weeks later and his abdominal symptoms had resolved and the deranged liver-enzymes and renal parameters had returned to normal values. The patient was doing well without signs of CBD obstruction and cholangitis and is on close follow up.

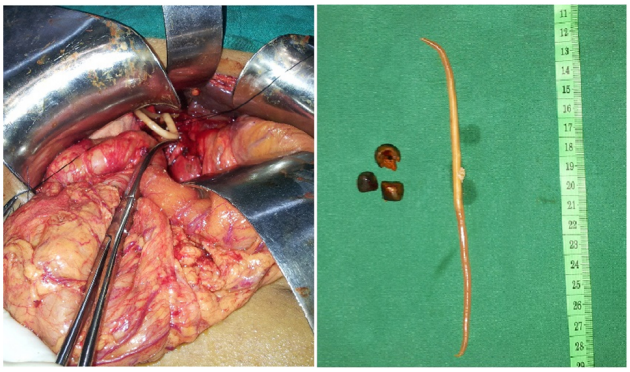

**Figure 1.** Intraoperative image showing the coiled up Ascaris within the lumen of the CBD after choledochotomy and the round worm after complete extraction from within the CBD

## 3. Discussion

Biliary ascariasis is often reported in high prevalence from the Indian subcontinent, parts of Middle East, Africa and Latin America. Being a usual parasite within the adult small intestine, it migrates into the unusual sites only once the worms load increases over a 1000.[1] Patients usually present to the hospital with biliary colic, which is secondary to migration of the worms across the papillae. Chronic symptoms include cholangitis, strictures, calculi, pancreatitis and cholecystitis, which are usually due to persistemce of the worm or parts of the worms within the bile duct and gall bladder.[2]

Multiple hypotheses, from previous literature, have postulated the strong association of recurrent cholangitis with biliary infestation with Ascaris lumbricoides and this strong association was restrengthened by followup studies.[3] The study also showed that age, sex and cholesterol gall stones did not show such strong association with recurrent pyogenic cholangitis (RPC).[3] This also cast away the doubts that recurrent pyogenic cholangitis and biliary parasites are common in regions with low socio-economic status and that their co-existence was a mere coincidence because of the high prevalence without any cause and effect relationship.[3]

Hepatobiliary ascariasis, after cholelithiasis, is the second most common cause for obstructive biliary symptoms worldwide.[3] Endoscopic retrograde cholangiopancreatography (ERCP) can play a diagnostic as well as therapeutic role as it allows direct visualization and extraction of the worm.[4–7] Sphincterotomy is best avoided because it can facilitate recurrent infestation of the worms in the biliary tract. Dilatation of Oddi's sphincter is a better and amiable alternative in these situations.[8] Surgical intervention is required in some cases, if endoscopic extraction fails. Laparoscopic visualization and extraction of the worm is yet another preferred route but such procedures are usually challenging and require a skillful surgeon.[9] Standard of care dictates treatment with antihelminthics following intervention to address the intestinal infestation and to repeat stool examination for ova or cyst after two weeks.

The various mechanisms by which Ascaris causes RPC have been enumerated, which include:

- It carries enteric organisms with it and causes partial obstruction of the biliary drainage, further causing bile stasis and a nidus for infection to set in.
- It also causes papillitis, which induces motor abnormalities resulting in delayed biliary drainage and recurrent cholanigits.
- Most often the worm moves out of the biliary system after inducing infection, however in the rare event they might get trapped and die within the bile ducts and their degradation products contain beta glucoronidase, which facilitate deconjugation of bile pigments.
- Dead worms, ova and worm fragments act as potential nidus for stone formation.[3]

Since biliary ascariasis return to the duodenum within 24-48 hours after inducing chemical cholangitis, antihelminthic should be restricted till this time else the nematodes die and are retained within the biliary tract leading to complications.

## 4. Conclusion

Though more than 95% of the patients with uncomplicated biliary ascariasis respond well with conservative management, surgical intervention is mandated in failed ERCPs with the worm within the bile duct causing chronic complications such as recurrent biliary cholangitis. In endemic areas, this should be considered as a differential diagnosis and the patient should be offered surgical intervention in the event of failure of conservative management.


ISSN 2377-7311 E-ISSN 2377-732X

## REFERENCES


[1] Mukhopadhyay M. Ascariasis in the Indian Subcontinent: A Study of 42 cases. Saudi J Gastroenterol. 2009; 15(2): 121-124. PMid: 19568577. http://dx.doi.org/10.4103/1319-3767.48970

[2] Louw JH. Biliaray ascariasis in childhood. S Afr J Surg. 1974; 12: 19-25.

[3] Khuroo MS, Mahajan R, Zargar SA, *et al*. Prevalence of biliary tract diseases in India. A sonographic study in adult population in Kashmir. Gut. 1989; 30: 201-205. PMid: 2649414. http://dx.doi.org/10.1136/gut.30.2.201

[4] Sandouk F, Haffar S, Zada MM, *et al*. Pancreatic-biliary ascariasis: experience of 300 cases. Am J Gastroenterol. 1997; 92: 264-7.

[5] Jessen K, Mofleh Al, Moferrah Al. Endoscopic treatment of ascariasis causing acute obstructive cholangitis. Hepatogastroenterology. 1986; 33: 275-7. PMid: 3542773.

[6] Jamsheer NS, Malik N, Al-Qamish J. Biliary ascariasis: Sonographic diagnosis. Saudi J Gastroenterol. 2001; 7: 69-70. PMid: 19861772.

[7] Al-Karawi MA, Salem I, Mohammed AS. Endoscopic diagnosis and extraction of biliary ascariasis. Ann Saudi Med. 1989; 9: 80-1.

[8] Zargar SA, Lehan BA, Javid G, *et al*. Endoscopic management of early postoperative biliary ascariasis in patients with biliary tract surgery. World J Surg. 2004 Jul; 28(7): 712-5. PMid: 15175897. http://dx.doi.org/10.1007/s00268-004-7183-0

[9] Jethwani U, Singh GJ, Sarangi P, *et al*. Laproscopic Management of Wandering Biliary Ascariasis case rep surg. 2012; 561-563.