ACUTE PANCREATITIS CAUSED BY ASCARIES LUMBRICODES

ARMEEN Z. OONWALA, FARZANA MEMON, SAEED QURAISHY
Department of Surgery, Dow University of Health Sciences & Civil Hospital, Karachi

ABSTRACT
Ascaris lumbricoides is a rare cause of acute pancreatitis. Significant morbidity and mortality is associated with the concomitant complication of hepatobiliary and pancreatic ascariasis, and early diagnosis and management is of utmost importance. This is a report of a case of a 25 year old woman who presented with epigastric pain and vomiting. She was diagnosed as having acute pancreatitis. ERCP revealed ascaris lumbricoides as the cause. The worm was successfully removed during endoscopy.

KEYWORDS: *Pancreatitis, Ascaris Lumbricoides, ERCP*

INTRODUCTION

Ascariasis lumbricoides is the most common parasitic disease of the human gastrointestinal tract.[1] Migration of the worm through orifices and into ducts leads to complications such as cholangitis and pancreatitis. Treatment of hepatobiliary and pancreatic ascariasis (HPA) used to be surgical but now endoscopic removal of the worm has been successfully performed.[2] This is a report of a case in whom an Ascaris in the duodenal ampulla led to acute pancreatitis; the worm was successfully removed endoscopically after performing sphincterotomy.

CASE REPORT

A 25 year old married woman from northern Pakistan presented with epigastric pain of five days duration. Pain was sudden in onset, severe and radiated to the back. It was associated with four to six episodes of vomiting. Patient also had fever 101° F. One episode of vomiting contained a round worm pink in color and around 10 inches in length. Past medical and surgical history was insignificant. On examination patient had mild jaundice and pallor. She had slight tenderness in the epigastrium. Rest of the examination was unremarkable.

Correspondence:
Dr. Armeen Z. Oonwala
C-100, Block C, North Nazimabad, Karachi-74700.
Phones: 021-6636892.
E-mail: armeen_z@hotmail.com

Complete blood picture showed a hemoglobin of 9.9 g/dl, leukocyte count of 4300/mm^3 with a platelet count of 61,000/mm.^{3} Serum electrolytes, urea and creatinine were normal. Liver function tests showed a total bilirubin of 7.0mg/dl, direct bilirubin 3.2 mg/dl, a serum aspartate aminotransferase level of 80 U/l and alkaline phosphate of 240U/l. Serum amylase was 1677 U/l at the time of admission. Serum calcium was 7.4 mg/dl. Serum LDH was 295 U/l. PT, INR were 11seconds and 1.0 respectively. Abdominal sonography showed an enlarged pancreas and slightly enlarged liver. Patient was diagnosed as acute pancreatitis and was advised restriction of oral intake with intravenous fluids and antibiotics.

Endoscopic retrograde cholangiography (ERCP) was done on the third day of admission which showed a large worm coming out of ampulla of vater. The worm was removed with the help of a grasper followed by a cholangiogram which was normal (Fig.1). The serum amylase levels fell to a level of 318 U/l after endoscopy. Patient was prescribed oral mebendazole 100 mg twice daily for three days and made an uneventful recovery.

DISCUSSION

Hepatobiliary and pancreatic ascariasis (HPA) can present as acute cholecystitis, acute cholangitis, biliary colic, obstructive jaundice, acute pancreatitis and hepatic abscess.[3] Acute pancreatitis may be caused by actual worm invasion of the pancreatic duct.[4] This blockage coupled with the simultaneous obstruction to the bile duct, causes bile and organisms to regurgitate into the pancreatic duct activating pancreatic enzymes with the subsequent development of acute pancreatitis.[5] Pregnant women and patients with a history of biliary tree surgery who are infected with Ascaris are at an especially

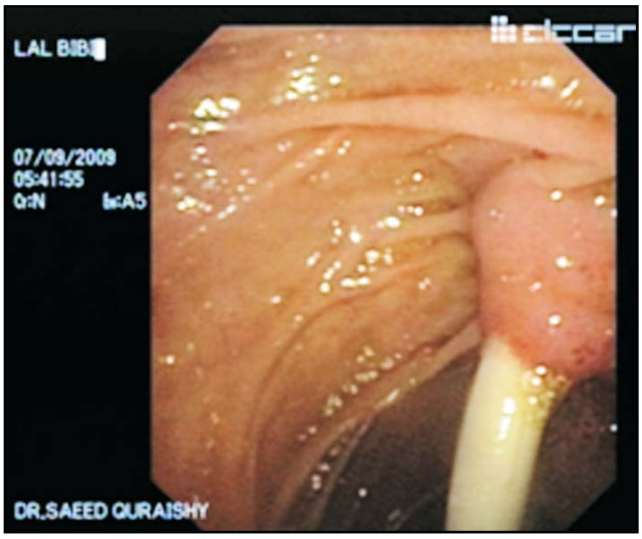

Fig. 1a. Worm coming out of duodenal ampulla

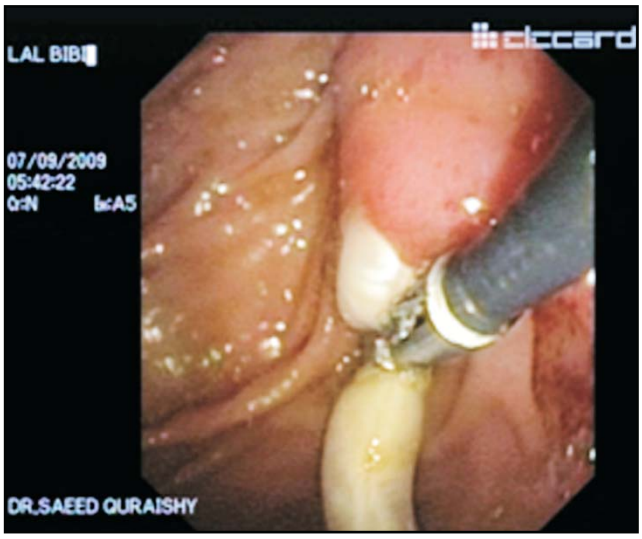

Fig. 1b. Worm held with the help of a grasper

heightened risk for contracting HPA.[5] A diagnosis of pancreatic ascariasis is often made on the basis of a high index of suspicion. Patients present with midepigastric pain that is referred to the back; vomiting of worms 25% to 48% of cases[6,7]; and elevated levels of pancreatic markers, such as serum amylase and lipase.

In the above case the serum amylase was markedly increased consistent with pancreatitis. Serum LDH and serum calcium were also deranged. The liver function tests were abnormal with a high bilirubin which was due to obstruction in the extrahepatic biliary ducts. Ultrasonography is a particularly specific and sensitive mode for detecting biliary and pancreatic ascariasis. In cases of hepatobiliary ascariasis, the longitudinal plane of the ultrasonogram will reveal a hypoechoic strip containing a central anechoic tube within the common bile duct. The transverse plane may display the worm inside the tubular duct.[8] In patients with acute pancreatitis, ultrasonography will show an enlarged "echo-poor" pancreas.[5] In the above case the pancreas was enlarged but no worms were identified. The test may be negative if worms transiently migrate into intestine. The sensitivity of this test in identifying pancreatobiliary ascariasis ranges from 25% to 91%; being lower in those with pancreatic disease.[3]

Initial management of HPA is restriction of oral intake; intravenous fluids; antibiotics; and analgesics. Antihelminthic therapy is started once acute symptoms have remitted.[5] Conservative treatment is continued usually for 3 days. During this period migration of the worm should be monitored by serial ultrasonography.[3] The gold standard of diagnosis and treatment of HPA is endoscopic retrograde cholangiopancreatography (ERCP).[3,5,7,9] This procedure should be performed if initial conservative management (discussed above) fails to provide relief of symptoms. With ERCP, worms can be detected virtually everywhere within the duodenum and hepatobiliary and pancreatic trees and can be extracted using forceps.[2] Used alone, ERCP can diagnose 53-58% of cases but the sensitivity approaches almost 100% when used in conjunction with real-time sonography.[3]

Most authors recommend early intervention with endoscopy since this has brought about a major reduction in the morbidity and mortality of this disease.[3,10] But it must be balanced against potential complications and cost of procedure. Sphincterotomy during ERCP for worm extraction predisposes to recurrent worm infestation. Worms within the pancreatic duct are not amenable to easy endoscopic extraction and therefore, these patients carry a poor prognosis.[3] The need for surgical intervention has been gradually superseded by the introduction of ERCP in the management of HPA.

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