

Fatal Ascaris Pancreatitis

Suhair Al-Saad, CABS, FRCSI, CST* Hamdi M Al-Shinawi, FRCSI**
Basma Malallah, MBBS***

Gastrointestinal parasites are an uncommon cause of biliary and pancreatic obstruction in the Arabian Gulf; it could have serious complications and even mortality. A Bengali male thirty-two year old patient recently arrived in Bahrain for presented with all the features of acute pancreatitis, no evidence of gallstones or alcohol abuse. He died despite all the therapeutic measures. Endoscopy revealed an *Ascaris lumbricoides* within the ampulla of Vater. Ascariasis is uncommon cause of acute pancreatitis, which should not be ignored.

Bahrain Med Bull 2011; 33(3):

Infestation with *Ascaris* disease in humans is common. The exact figure of *Ascaris* infestation in the world is not known but it is estimated to be 25%. The life-cycle starts with the eggs in the soil ingested with food, or water; the larvae travel from the duodenum to the cecum and to the portal system and the liver; some larvae travel to the heart and lungs through the hepatic veins¹⁻³.

The larvae could reach the lungs through the bowel lymphatics and thoracic duct. From the tracheo-bronchial tree, the larvae are swallowed; the cycle takes nearly 2 months. It takes several months to mature in the small intestine and the eggs are passed in the feces¹⁻³.

Ascariasis is endemic in China, Southeast Asia, Africa and Latin America^{1,2}. Acute ascaris pancreatitis is infrequent. In China, a study reported 42 patients with acute ascaris pancreatitis, mostly in young females⁴. Severe ascaris pancreatitis is rare, only 4.8% were necrotizing pancreatitis. Although it is a disease of tropical countries, it has been seen rarely in Western countries⁵.

Ascaris infections are clinically asymptomatic and mild but pulmonary, intestinal, peritoneal, appendiceal, hepatobiliary, and pancreatic disease could be serious^{1,3}. The worms travel to and exit the biliary tree or pancreatic duct via the ampulla¹⁻³.

Biliary colic, acalculous cholecystitis, acute cholangitis, acute pancreatitis, and hepatic abscess could be the clinical presentations of hepato-biliary and pancreatic Ascariasis (HPA)².

The aim is to report a case of fatal acute pancreatitis in a young patient and to increase awareness for better diagnosis and a prompt protocol for management.

* Consultant Surgeon and Assistant Professor Arabian Gulf University
** Consultant Surgeon and Assistant Professor Arabian Gulf University
*** Senior Surgical Resident
Surgical Department
Salmaniya Medical Complex
Email: alsaadsuhair@gmail.com

The Case

Thirty-two year old Bengali male patient arrived in Bahrain recently to work as a driver. He presented to emergency department with one-day history of severe constant epigastric abdominal pain, which was radiating to the entire abdomen. He had continuous vomiting, one vomitus contained Ascaris worm. There was no loss of appetite or change in bowel habits. Detailed history was not possible due to language barrier.

On examination, the patient was conscious and oriented but was very irritable due to severe pain. Chest and cardiovascular examinations were normal. The abdomen was tender and tense with generalized guarding mostly at the epigastric region. Bowel sounds were positive.

Investigations showed serum amylase 2158 IU and urinary amylase 17073 IU. Serum cholesterol and triglycerides were normal, see table 1.

Table 1: Complete Blood Count (CBC), Liver Function Test (LFT), Urea, Sugar, Creatinine, Arterial Blood Gases (ABG), Electrolytes, Serum Calcium and Coagulation Profile for Three Days

CBC	First day	Second day	Third day
WBCs	22.9 (3.6 - 9.6 x 10 ⁹ /L)	16.1 x 10 ⁹ /L	13.1 x 10 ⁹ /L
Platelets	408 (140 – 440 x 10 ⁹ /L)	292 x 10 ⁹ /L	202 x 10 ⁹ /L
LFT			
Total Bilirubin	14 (< 18)	39 (D:24, ID:15)	39 (D:24, ID:15)
SGPT	52 (30 - 65 u/L)		3151 u/L*
CPK (MB)	6 (0 - 10 u/L)	17 u/L (high)	124 u/L*
LDH	220 (135 - 225 u/L)	579 u/L	4624 u/L*
Urea	4.1 (3 - 7 mmol/L)	18.9 mmol/L	24.3 mmol/L*
Creatinine	84 (62 - 140 umol/L)	330 umol/L	400 umol/L*
Electrolytes			
K+	4 (3.9 - 5 mmol/L)	4.9 mmol/L(high)	6.2mmol/L(high)*
HCO ₃	23 (24 - 30 mmol/L)	12 mmol/L	13 mmol/L *
Arterial Blood Gases			
PH	7.376	7.293	7.328 *
CO ₂	40.1	28.3	27.2 *
O ₂	93.5	106.1	72.8 *
HCO ₃	23.0	13.4	14.0 *
BE	-2.0	-11.6	-10.4*
SO ₂ %	94.4%	95.6%	90.5% *
Serum calcium	(2.13 - 2.63 mmol/L)	1.86 mmol/L	1.62mmol/L
Coagulation profile			
Prothrombin (PT)	10 (9.8 - 12.7 seconds)	16 seconds	21 seconds*
INR	0.8 (0.7 - 1.3)	1.3	1.8*
APTT	22 (26 - 33 seconds)	33 seconds	36 seconds*
D. Diemer	(0.09 - 0.33 mg/L)		18.8 mg/L (high)*

* indicate gradual increase of the level of liver enzymes, cardiac enzymes, urea, sugar, creatinine, Potassium level, metabolic and respiratory acidosis, hypoxia, and disturbance of coagulation profile indicating DCIS.

Erect chest X-ray showed no air under diaphragm. CT scan abdomen and pelvis showed that the pancreas is diffusely swollen and hypodense due to edema but there was no necrosis. There was stranding of the peripancreatic and mesenteric fat. Fluid collection was seen in the pararenal spaces

on both sides, subdiaphragmatic area and paracolic gutters. These findings were consistent with severe pancreatitis (Balthogor type E)⁹. Numerous longitudinal filling defects in the third part of the duodenum and jejunum were seen, most probably ascariasis. The common bile duct was not dilated and the pancreatic duct was not visualized. Diffuse fatty liver infiltration was seen, see figure 1.



Figure 1: CT Scan of Abdomen and Pelvis, the Pancreas Diffusely Swollen and Hypodense Due to Edema. Stranding of Peripancreatic

He was admitted with the diagnosis of acute severe pancreatitis and dehydration most probably secondary to *Ascaris* infestation. The patient was closely observed for vital signs and urine-output hourly. He was kept fasting on nasogastric tube (NGT) free drainage, and on intravenous fluid rehydration. He was also started on intravenous Omeprazole 20 mg twice a day and on Mebendazole syrup 200 mg BD through NGT.

On the second day, the patient had an upper endoscopy. The findings were severe gastritis and in the 2nd part of duodenum, there was a smooth tubular structure protruding through the ampulla of Vater. It was extracted in its entirety with a biopsy forceps and it was measuring 8 cm, it was identified as an *Ascaris* worm. Bile was seen in the duodenum after extraction, see figure 3 (a,b,c).

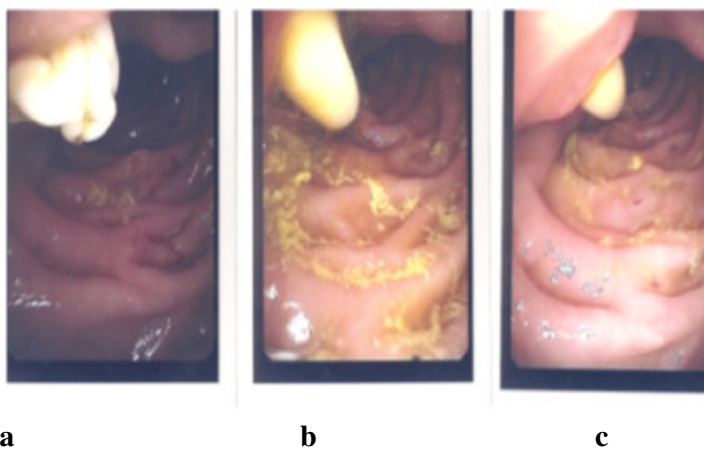


Figure 3 (a, b, c) (from Right to Left): A Smooth Tubular Structure (*Ascaris* Worm) is protruding through the Ampulla of Vater, Extracted with a Biopsy Forceps

In the afternoon, the patient's general condition worsened. He was still fully conscious and oriented but his pulse became 140/minute, BP 130/100, temperature rose to 38⁰ C and he started desaturating. Chest examination showed decrease basal air entry bilaterally with crepitations. He needed O₂ supply through a facemask up to 10 L/minute, to raise the saturation to 97%. Cardiovascular examination was normal. Abdomen was still distended, tense with guarding in epigastric region. Investigations showed elevation of K⁺ to 4.9, urea to 18.9 and creatinine to 300, see table 1.

Intravenous antibiotics were started, the electrolyte imbalance was corrected and he received 6 L/24 hours of intravenous fluids. The urine output was 60 ml/hour.

On the third day, he became very sick, drowsy and tachypneic. His respiratory rate was 54/minute, although he was still on O₂ mask 10 L/minute. Chest examination showed bilateral basal decrease in air entry with crepitations. Abdominal examination was showing more tenderness and distention.

He was not responding to high O₂ intake through the mask, and his PaO₂ was not improving indicating that he went into adult respiratory distress syndrome (ARDS). He was electively intubated and kept on a ventilator support. He was tachycardic (135/minute), and hypotensive (BP was unstable ranging between 57/33 and 102/60) indicating intraperitoneal hemorrhage or massive fluid loss due to severe pancreatitis. He also became anuric. His blood investigations were showing renal failure, gradual elevation of urea, potassium and creatinine, and elevation of liver enzymes. Intravenous blood transfusion and crystalloids were given to raise the blood pressure. Intra-venous infusion of Norepinephrine was started to improve the blood pressure and renal perfusion.

Chest X-ray showed bilateral pleural effusion in the left more than right with signs of ARDS. Later the patient went into refractory shock, he became more tachypneic (Respiratory Rate 54/minute), although he was on the ventilator; the pulse was 135/minute and had unstable low BP. Renal function worsened and the patient became acidotic, finally he went into ventricular fibrillation and asystole. Resuscitation failed and the patient declared dead. The death was most probably due to severe pancreatitis complicated with ARDS, hypovolemic shock and multi-organ failure; unfortunately, in our institute, postmortem examination is not performed routinely in case of hospital death.

DISCUSSION

Ascaris lumbricoides is the most common parasite to cause pancreatitis. Ascariasis is second to cholelithiasis as a cause of pancreatitis in some parts of India¹⁻³. *Ascaris* pancreatitis could be due to invasion of the biliary tree, pancreatic duct or both. Invasion of the pancreatic duct is rare due to its smaller caliber.

Pancreatitis presents in adults more than children do with a mean age of 35-42 years due to small-caliber ducts in children¹⁻³. The presented patient was in the typical age group. It was found in the literature that *ascaris* pancreatitis affects women more than men 3:1. *Ascaris*-related pancreatitis present with abdominal pain, back pain, fever, jaundice, nausea, vomiting, and worm emesis¹⁻³.

Most cases are mild, patients with severe infection and pancreatitis might die^{1-3,6}. The presented patient had typical symptoms but the pancreatitis course was severe enough to end with multi-organ failure and death.

Khuroo et al reported 500 cases in India of HPA due to ascaris, 56% had biliary colic, 24% had acute pancreatitis, 13% had cholecystitis, 6% had acute pancreatitis and 1% had hepatic abscess^{3,7}. Sandouk et al reported 300 cases of pancreatic-biliary ascariasis in Syria and noted that 16% had acute cholangitis, 4% had acute pancreatitis and 1% had obstructive jaundice⁴. Additionally, Sandouk et al reported that 80% of the patients had a history of either cholecystectomy or endoscopic sphincterotomy⁶. In a study from China, 42 patients had biliary and pancreatic ascaris infection, 4.8% had acute pancreatitis and the mortality rate was 0%⁸.

Ascaris usually causes pancreatitis due to obstruction of ampulla of Vater, invasion of common bile duct, or invasion of pancreatic duct. In our presented patient, the cause was most probably due to obstruction of ampulla of Vater by the worm.

High degree of suspicion is important in the diagnosis of Ascaris pancreatitis. The worms move freely in and out of the biliary tree and therefore can be easily missed. Furthermore, stool studies lack sensitivity and specificity. The diagnosis can be attempted with ultrasonography, computed tomography (CT), or ERCP. It is most commonly diagnosed by using ultrasonography and ERCP^{3,7,8}.

Ultrasonography is a simple, non-invasive test, sensitive and specific method for detection of worms in the biliary tree, and it can be repeated frequently to monitor response to therapy and worm movement. It reveals long, linear, echogenic strips that may show acoustic shadowing. It has a sensitivity of 50% to 86% for worms in the biliary tree, but the sensitivity for detecting worms in the pancreatic duct is not known¹. Additionally, ultrasonography cannot diagnose ascariasis in the duodenum. CT scan can also be useful but has a lower sensitivity than ultrasonography⁹.

Our patient had CT scan on admission, which showed the severity score of pancreatitis as well as the ascaris worms in the duodenum and jejunum. The CBD was not dilated and the pancreatic duct was not visualized.

Pancreatic necrosis and peripancreatic inflammatory fluid collection or called phlegmon are two prognostic indicators of severity of acute pancreatitis on CT. These two are associated with early complications and death¹⁰.

Endoscopic retrograde cholangiopancreatography (ERCP) is the gold standard method for identifying and removing the worm from the duodenal, biliary or pancreatic tract. The use of ERCP permits better recognition of worms in the duodenum and in the pancreatic-biliary tree and it provides a safe, therapeutic option of removing the worms^{11,12}. Sandouk et al found that ERCP made a definitive diagnosis by showing a smooth filling defect in the common bile duct, a worm protruding from the ampullary orifice or evidence of damage to the ampulla in 93% of patients⁴. Worm extraction from the ampullary orifice, biliary duct or pancreatic duct was successful in 98% of patients; only 13% required a sphincterotomy^{3,6,7}. Grasping forceps or balloon catheters could extract the worms. Thus, ERCP has now become the investigation and the treatment modality of choice^{11,12}.

In the acute stage, conservative therapy is recommended: bowel rest, intravenous fluids, analgesics, and antibiotics followed by Mebendazole once the acute symptoms subside². Endoscopic intervention is reserved for patients who fail conservative therapy or have worms in the ducts after 3 weeks of observation². Others advocate ERCP to clear pancreatic and biliary ducts from the worms^{2,11,12}. Surgery is required occasionally for definitive therapy². In a review of 42

patients diagnosed as ascari pancreatitis in China, the diagnosis was based on the detection of worms by US, high serum amylase level and the presence of abdominal pain with tenderness⁸. In the same study, the treatment was conservative in 40 patients and only 2 were operated. Laparotomy is recommended if patient fails to respond to conservative therapy, blood in the peritoneal fluid on aspiration, evidence of infection around the pancreas and dead worms remain within the biliary tree⁸.

Anthelmintic therapy with Mebendazole or Albendazole is effective in eradicating ascariasis in 84% to 100% of cases¹. These agents inhibit phosphorylation in the mitochondria, causing a depletion of the worm's glucose. The worms are then expelled by normal gastrointestinal peristalsis. Despite these excellent results, patients can have a reinvasion of the organism from either reinfection or ineffective therapy in 15% to 28%¹.

Our patient most likely was infected while in Bangladesh. This theory is plausible, since it takes approximately 2 months for the development of an adult worm after egg ingestion.

The differential diagnosis of pancreatitis includes gallstone disease, alcohol abuse, drugs, toxins, infections, metabolic abnormalities, vascular abnormalities and ascariasis in patients with recent arrival from endemic areas. The prognosis of Ascaris-induced pancreatitis is excellent if the patient is diagnosed and treated early. The mortality rate is 3% in endemic areas and it could be higher if Ascariasis not considered in the differential diagnosis^{1,13}.

CONCLUSION

Although Ascaris pancreatitis is very rare in the GCC countries, suspected cases should be treated early and promptly, otherwise it could lead to fatal complications. ERCP is the best diagnostic and therapeutic tool. The worms could be extracted from the pancreatic and the biliary ducts. Most patients respond to conservative therapy, surgical intervention is rarely needed.

Author Contribution: All authors share equal effort contribution towards (1) substantial contributions to conception and design, acquisition, analysis and interpretation of data; (2) drafting the article and revising it critically for important intellectual content; and (3) final approval of the manuscript version to be published. Yes

Potential Conflicts of Interest: No

Competing Interest: None **Sponsorship:** None

Submission date: 9 May 2011 **Acceptance date:** 8 July 2011

Ethical approval: Department of Surgery at SMC.

REFERENCES

1. De U, Mukherjee M, Das S, et al. Hepato-pancreatic Ascariasis. Trop Doct 2010; 40(4): 227-9.

2. Kenamond CA, Warshauer DM, Grimm IS. Ascaris Pancreatitis. <http://radiographics.rsna.org/content/26/5/1567>. Accessed on 16.02.2010.
3. Khuroo MS. Ascariasis. *Gastroenterology Clin North Am* 1996; 25(3): 53-77.
4. Sandouk F, Haffar S, Zada MM. Pancreatic-biliary Ascariasis: Experience of 300 Cases. *Am J Gastroenterol* 1997; 92: 2264-67.
5. Galzerano A, Sabatini E, Duri D. Case Report-Ascaris Lumbricoides Infection: An Unexpected Cause of Pancreatitis in a Western Mediterranean Country. *East Mediterr Health J* 2010; 16(3): 350-1.
6. Krige JE, Lewis G, Bornman PC. Recurrent Pancreatitis Caused by a Calcified Ascaris in the Duct of Wirsung. *Am J Gastroenterol* 1987; 2(3): 256-7.
7. Khuroo MS, Zargar SA, Yattoo GN. Worm Extraction and Biliary Drainage in Hepatobiliary and Pancreatic Ascariasis. *Gastrointest Endosc* 1993; 39(5): 680-5.
8. Chen Daojin, Li Xiaorong. Forty-two Patients with Acute Ascaris Pancreatitis in China. *J Gastroenterol* 1994; 29(5): 676-8.
9. Grover SB, Pati NK, Rattan SK. Sonographic Diagnosis of Ascaris-induced Cholecystitis and Pancreatitis in a Child. *J Clin Ultrasound* 2001; 29(4): 254-9.
10. Balthazar EJ, Robinson DL, Megibow AJ, et al. Acute Pancreatitis: Value of CT in Establishing the Prognosis. *Radiology* 1990; 174(2): 331-6.
11. Bhushan B, Watal G, Mahajan R. Endoscopic Retrograde Cholangiopancreatographic Features of Pancreaticobiliary Ascariasis. *Gastrointest Radiol* 1988; 13(4): 327-30.
12. Chen YS, Den BX, Huang BI, et al. Endoscopic Diagnosis and Management of Ascaris – Induced Acute Pancreatitis. *Endoscopy* 1986; 18(4): 127-8.
13. Maddern GJ, Dennison AR, Blumgart LH. Fatal Ascaris Pancreatitis: An Uncommon Problem in the West. *Gut* 1992; 33(3): 402-03.