An unusual cause of cholecystitis: a worm in the bag
N Elaldi, M Turan, M Arslan, I Dokmetas, M Bakir

CASE REPORT
A 58 year old woman attended the emergency department with a one month history of right upper abdominal pain, vomiting, and fever. Her physical examination revealed tenderness in the right upper quadrant.

Laboratory test results at presentation revealed mildly increased liver enzymes aspartate transaminase:353 U/L, alanine transaminase:218 U/L, γ-glutamyltransferase:110 U/L, alkaline phosphatase:192 U/L; serum total bilirubin was normal. Blood haemoglobin was 12 mg/dl, packed cell volume:35.8%, and leucocyte count was 18 × 10⁹/l with mild eosinophilia. Serum amylase level was within normal limits at admission. Her stool examination was positive for Ascaris lumbricoides ova. A sagittal ultrasonographic image of the gall bladder showed a ribbon-like, non-shadowing structure with a highly echogenic wall and a less echogenic centre. Gall bladder wall was 5 mm in thickness. The structure was 4 mm in diameter, and 5 cm in length (fig 1).

Cefotaxime was started intravenously with the diagnosis of acute cholecystitis. After her clinical status subsided, she began receiving mebendazole orally 100 mg twice daily and continued for three days. On the eighth day of her admission, subcostal pain of the patient recurred. Liver enzymes mildly increased and serum amylase level increased to 184 U/L. After her clinical status subsided, on the 13th day of admittance, she underwent surgery. In operation, cholecystectomy and choledochus exploration were performed. Any worm type of parasite was not detected in the gall bladder and biliary tract. But ascaris eggs were seen in the microscopical examination of the bile samples taken from the gall bladder. She was uneventful in the postoperative period and discharged nine days after surgery.

DISCUSSION
Although intestinal infestation of ascariasis is often asymptomatic, migration of worms into the biliary tree may cause serious complications such as biliary colic, cholecystitis, cholangitis, intrahepatic abscesses, or pancreatitis. But, these complications are rare even in endemic areas.1,2 Worms in the duodenum and invading the ampullary orifice induce biliary colic or acute pancreatitis and in the common bile duct they usually block the cystic duct and cause distension of gall bladder, which lead to episodes of acute cholecystitis.

The diagnosis of biliary ascariasis can be established by means of microscopic examination of ascaris eggs in the bile samples, as represented in our case. Hepatobiliary ultrasonography is also a safe and non-invasive procedure that permits the identification of the roundworm, which moves and changes position while alive.2,3 A ribbon-like, non-shadowing structure with a highly echogenic wall and a less echogenic centre image at the sagittal ultrasonography of the gall bladder is strongly demonstrated as a live ascaris in the gall bladder (fig 1).

Various drugs used for the treatment of intestinal ascariasis are not effective for biliary ascariasis, except for piperazine citrate when instilled through a nasobiliary drain.1 Mebendazole is not effective because of little absorption from the intestinal tract. Some authors reported that biliary ascariasis generally has a good response to conservative treatment.4,5 This may not hold true for gallbladder ascariasis. It usually requires laparotomy and cholecystectomy. But in some cases, worms may migrate spontaneously to the common bile duct and be excreted to intestine through the Oddi sphincter, as in this case.6 Endoscopic procedures are useful in the management of ascariasis in biliary and pancreatic ducts and piperazine citrate instillation through a nasobiliary drain is a good choice in ascariasis of biliary ducts. But they are not so useful for gallbladder ascariasis. Therefore this patient was operated on and cholecystectomy and choledochus exploration were performed.

Although ascariasis is predominantly a disease of developing countries, clinicians need to be aware of gallbladder ascariasis in view of world travel and population migration.

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Figure 1 Sagittal ultrasonographic image of the gall bladder shows a ribbon-like, non-shadowing echogenic region (arrows) within the gall bladder (GB).
A 53 year old man attended the department after hitting a lamp post while driving at about 40 mph. As a result of the impact the driver airbag inflated causing injury to the right side of the face. Examination revealed extensive periorbital abrasions, subconjunctival haemorrhage, a small hyphaema, and a sluggishly reacting pupil. Fluorescein staining revealed an extensive corneal abrasion (fig 1). The visual acuity was markedly impaired with the patient able to detect hand movements only. Intraocular pressure was 10 compared with 12 in the uninjured eye. At a one week review the acuity had increased to 6/36 with a small amount of blood remaining in the anterior chamber, no obvious retinal damage was seen at this stage. The patient made a satisfactory recovery thereafter.

DISCUSSION

Although there is ample evidence in the literature to suggest that airbags save lives by protecting the driver against head and chest trauma, little has been written concerning the potential injury as a result of airbag inflation. A routine search using Medline has shown that airbag deployment has been shown to cause injuries to the soft tissues and facial skeleton but few cases have been reported about direct injury to the globe, occasional cases such as corneo-scleral laceration and lens dislocation have been reported.

Clearly airbag inflation has not only the potential to cause injury to the facial skeleton but also to the eye itself, the authors would therefore recommend a careful eye examination in all such injuries.

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EMERGENCY CASEBOOK

Occular trauma related to airbag deployment

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