Biliary Fascioliasis presented with recurrent Pruritus

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Introduction:

Fascioliasis is a waterborne disease which caused by FH, usually attacks cattle or sheep but human can be their accidental hosts due to contaminated water or raw green vegetables. Fascioliasis mainly involve hepatobiliary and has two phase of manifestation: acute (hepatic) VS chronic (biliary). Hepatic phase typically occurs 6-12 weeks after ingestion, which characterized with right upper quadrant abdominal pain, elevated liver enzyme and eosinophilia. Adult flukes migrate to biliary tracts after acute phase, and can be asymptomatic for many years, or present with biliary obstruction, cholangitis and pancreatitis. In endemic region, FH should be considered for idiopathic biliary obstruction. There are more than 3 million people infected by FH worldwide even in developed country during past decade and Iron has one of the highest infection rate among them. Here, we report a 36 years old male from Iron who presented with recurrent pruritus and was found to has FH which was removed by ERCP.

Case Presentation:

A 36-year-old male from Iran was initially admitted due to recurrent non-rash pruritus accompanied by occasional vague abdominal pain for about two years. PMH was unremarkable and family history of gastric cancer in his father and grandfather. The patient was seen by a dermatologist and prescribed antihistamines which were not effective. He was referred to local hospital to evaluate the nonspecific vague abdominal pain. We evaluated the patient's liver enzymes and other relevant laboratory data indicated below, and in addition requested an Abdominal Ultrasound. Laboratory test showed a Hgb of 14.3mg/dl, WBC of 7500/mm3, eosinophil count of 290/mm3(3%), total bilirubin of 1mg/dl (normal=0.6-1.2), direct bilirubin of 0mg/dl (normal=0.1-0.3), AST of 35Iu/l (normal<40), ALT of 30Iu/l (normal<40), alkaline phosphatase of 300Iu/l (normal= 50-350), CRP of 45g/l (normal= 0-5), ESR of 10 mm/h (normal< 15), BUN of 12 mg/dl (normal= 7-21) and creatinine of 0.86 mg/dl (normal=0.5-1.4).

Due to positive Familial Gastric Cancer history, upper and lower endoscopy were done which the results were completely normal. Due to the vague abdominal pain and pruritus, an Abdominal US was performed, and it revealed dilated CBD without any significant findings. Follow up EUS showed dilated CBD with the diameter of 16mm accompanied by some filling defects and two live parasites (Figure). On the same day, an ERCP was performed on the patient for therapeutic purposes. During the ERCP procedure, CBD cannulation failed, so we postpone it to another session for another day. Due to two failed attempts in two separate occasions, we did the procedure under the guide of EUS during the third time. CBD cannulation was done with Rendezvous method and biliary stent was inserted under fluoroscopic guide. For medical treatment 10mg/kg triclabendazole was administered as a single dose 4 days after the procedure. Another ERCP was performed with the aim of biliary stent removal and parasite extraction, which lead to the live trematode being extracted.

After a month, the patients follow up lab tests showed a Hgb of 14.2 mg/dl, WBC of 7200 mg/dl, eosinophil count of 150/mm3 (2%), total bilirubin of 0.9 mg/dl (normal=0.6-1.2), direct bilirubin of 0.2mg/dl (normal=0.1-0.3), AST of 34Iu/l (normal<40), ALT of 32Iu/l (normal<40), alkaline phosphatase of 254Iu/l (normal= 50-350), CRP of 4g/l (normal= 0-5), ESR of 8 mm/h (normal< 15), BUN of 12 mg/dl (normal= 7-21) and creatinine of 0.86 mg/dl(normal=0.5-1.4) and no clinical relapse were detected. We recommended for follow up EUS after 6 months, but the patient came after 1 year without any complaints, in follow up EUS, CBD was not dilated or containing any stone, sludge or lesion.

Discussion:

Although livestock animals are the definite hosts, human can be accidental hosts for Fasciola Hepatica. The disease has two descriptions of evolution acute or first phase which larva enter the peritoneal cavity, the liver capsule, hepatic tissue and ultimately the bile ducts. Hepatic lesions are produced by migration of fluke produce micro-abscesses and tunnel like area of parenchymal necrosis. Possible symptoms of this phase are urticaria, cough, shortness of the breath, our patient did not have these symptoms. In chronic phase mature fluke migrate to bile duct and produce eggs. Direct irrigating and induction effect of high proline concentration in bile duct promoted the hyperplasia and hypertrophy of ductal epithelium, which created enveloping periductal fibrosis and thickening of the wall. During this phase, patients could be asymptomatic or present following symptoms: cholangitis, biliary obstruction, cholecystitis, gallstones.

Egg presentation in stool samples is recommended in using for diagnosis FH, however in this stool samples are not always as reliable, and they are complex. Therefore, we used CT scan and US are to assist. However, it is important to use serology and/or parasite testing for confirmation of diagnosis of FH. In chronic phase clinical and radiological findings with the support of serology made our diagnosis precise.

For treatment, our first line was $10_{mg/kg}$ triclabendazole, repeated the dose for this major infection as recommended in 12-24h. It is important to mention that in treatment failures using double dose is considered as routine therapeutic intervention. In some countries triclabendazole is not available, we advise the of use bithionol. Symptomatic relief and disappearance of eosinophilia can predict favourable outcome. Medication therapy eliminates parasites and there are high risk of biliary obstruction and related complication so endoscopic clearance is mandatory and extraction of flukes by balloon extraction or basket are very effective and safe intervention .

Figure: EUS Showing the Parasite