Gallbladder Stone Migrating in the Liver and Mimicking Gallbladder Cancer (GBCA)

Ahmed Elaffandi, Hany Nada, Samah Mohamed, Ahmed Farahat, Ghada Mohamed, Hussein Soliman* and Amr Attia

Surgical Unit, National Cancer Institute Hospital, Cairo, Egypt

Abstract

We report a case of gallbladder stone that eroded the posterior wall of the gallbladder and migrated into the liver parenchyma giving a similar picture to Gallbladder Cancer (GBCA) on imaging. Such a clinical scenario is uncommon in the presence of raised serum tumor markers (CA 19.9) and significant weight loss. It is to the authors' knowledge that a similar case has hardly been reported in the literature.

Keywords: Hepatic focal lesions; Gall bladder mass

Introduction

Gallstones and biliary lithiasis are very common in the western world. Up to 4% of the asymptomatic population may start developing symptoms annually. The most common presentations are biliary colics (56%) and acute cholecystitis (36%) [1]. Other presentations and complications can also occur with less common incidence as obstructive jaundice, cholangitis, pancreatitis, empyema, gallstone ileus and the least common of all is erosion into adjacent viscera causing fistula or gastric outlet obstruction (Bouveret syndrome) [2]. A gallstone eroding into the liver parenchyma mimicking GBCA both clinically and radiologically has barely been reported in the literature.

Case Report

A 48 years old mediterranean female presented to the Outpatient Clinic (OPC) at the National Cancer Institute (NCI) with a history of weight loss, right hypochondrial pain and gastric discomfort for 4 months. She was laparoscopically explored outside the NCI where a gall bladder mass suspicious of being cancer was found and therefore the laparoscopic attempt to remove the gall bladder was terminated and no tissue biopsy was taken. Preoperative Liver Function Tests (LFTs) and inflammatory markers were all within normal range. Abdominal ultrasound before the laparoscopic attempt showed thick walled calculus gallbladder in the absence of extra & intrahepatic biliary duct dilatation, and hence, the diagnosis of benign calcular gallbladder disease was made.

Following her visit to the OPC at the NCI a complete history analysis and physical examination was done. The patient had the LFTs, inflammatory and tumor markers (CA 19-9, alpha-Fetoprotein and CEA), repeated. All blood tests came back within normal range, apart from the CA 19.9 (277). The patient was rescanned by whole body Computerized Tomography (CT) and Triphasic CT of the abdomen, which in turn showed asymmetrical gallbladder wall thickening and associated adjacent gall bladder bed hypodensity, with obliterated line of cleavage (Figure 1). The presence of regional lymphadenopathy was clearly evident and hence, the presence of a malignant disease involving the gallbladder was hard to exclude.

A second exploration performed at the NCI showed normal liver with no evidence of any peritoneal disease. The gall bladder was hard and shrunken. While doing the cholecystectomy, a 2.5 cm gallstone was found that has eroded through the posterior wall and migrated within the liver parenchyma. Sampling of regional lymph nodes was done and analysed intraoperative using frozen section. Both the results of the gallbladder specimen and lymph node were proven histologically to be negative for malignancy following an immediate analysis, which was confirmed later by paraffin study (Figures 2-4). The patient had an uneventful postoperative course and was discharged on the 8th postoperative day.

Discussion

Gallstone disease is one of the most common of all digestive diseases. Up to 54.5% of the population in upper Egypt is having gallbladder disorders (gallstones/gallbladder wall thickening) [3]. Many factors...
have been strongly linked with gallstone formation such as obesity, diabetes mellitus, oestrogen & pregnancy, haemolytic diseases, and cirrhosis. There were no factors in the presented case that suggested any association with such a benign pathology. Instead, the presence of weight loss over a short period of time and biochemically high CA 19.9 shifted our attention to the presence of a malignant pathology within.

Ultrasonography remains the diagnostic test of choice, being 90–95% sensitive. The traditional description of a gallstone is that of an echogenic focus with posterior acoustic shadowing [2,4]. However, uncommon inflammatory pathology of gallbladder such as xanthogranulomatous cholecystitis may sometimes be difficult to differentiate from gallbladder cancer even with the use of CT [5]. Similarly, gallbladder cancer could hardly be differentiated from a migrating gallstone in our presented case.

Gallstones cause various problems besides simple biliary colic and cholecystitis. With chronicity of inflammation caused by gallstone obstruction of the cystic duct, the gallbladder may fuse to the extrahepatic biliary tree, causing Mirizzi’s syndrome, or fistulise into the intestinal tract, causing so-called gallstone ileus [2]. Other uncommon path of gallbladder stones is erosion into nearby structures such as distal stomach or proximal duodenum causing Bouveret syndrome, small intestine, aorta or cystic artery causing intraperitoneal haemorrhage or the skin causing cutaneous biliary fistula [2].

Patient found to have GBCA usually present with nonspecific symptoms of jaundice and indigestion that can be identical to symptoms of benign gallbladder disease [6]. The main risk factors for GBCA include cholelithiasis (especially untreated chronic symptomatic gallstones), obesity, and chronic gallbladder infections [7]. Significant overlap between GBCA and benign diseases of the gallbladder often exist on imaging [8]. We have experienced such an overlie which along with raised serum tumor markers and significant loss of weight made us think more of an underlying malignant disease.

Figure 2: A (H & E stained slide (Magnification 200X): Gall bladder wall, mucosa is Focally ulcerated in this view with related mild to moderate infiltration by chronic inflammatory cell infiltrate; composed predominantly of lymphocytes, with fewer numbers of plasma cells and histiocytes). B (H & E stained slide (Magnification 100X): This view shows Rokitansky-Aschoff sinuses (a characteristic feature of chronic cholecystitis) seen within gall bladder wall. It contains bilious material. The mucosa lining the Rokitansky-Aschoff sinus is thin and extensively ulcerated. The surrounded tissue is fibrotic and shows granulomatous reaction).

Figure 3: A (H & E stained slide (Magnification 200X): Gall bladder wall shows granulomatous reaction incited by escaping of biliary material from the Rokitansky-Aschoff sinuses). B (H & E stained slide (Magnification 100X): Chronic cholecystitis with fibrosis of the wall, and extension into subserosal connective tissue. The surface epithelium has been denuded, and mucosal folds are obliterated).

Figure 4: (H & E stained slides; Mag. 200X): Shows an adherent hepatic tissue with blood accumulation in the sinusoids (mild congestion).
In our reported case, patient presented with symptoms of chronic Calculus cholecystitis with laboratory tests and tumor markers of equivocal significance.

Similarly, the CT findings, along with pre-existing clinical findings made us think more of a malignant disease affecting the gall bladder area. Benign disease such as gallstones can look very similar to GBCA and hence should always be on the list of differentials for the operating hepatobiliary surgeon. Similarly, on mere suspicion we should always exclude the presence of underlying malignant disease.

References