

CT demonstration of acute gastrointestinal bleeding caused by gallbladder carcinoma eroding the pyloric canal. A case report.

A Hakeem, N Wani, T Gojwari, H Shafi, S Rasool, M Wani

Citation

A Hakeem, N Wani, T Gojwari, H Shafi, S Rasool, M Wani. *CT demonstration of acute gastrointestinal bleeding caused by gallbladder carcinoma eroding the pyloric canal. A case report.* The Internet Journal of Surgery. 2009 Volume 22 Number 2.

Abstract

Gall bladder cancer is a rare cause of gastrointestinal bleeding, and it is extremely rare for hematemesis to be the initial manifestation of invasive gall bladder cancer. We report a case of a 45-year-old woman who had undergone open cholecystectomy one year back for gall stone disease and now presented with hematemesis. She was found to have a bleeding ulceration of the pylorus on upper gastrointestinal endoscopy. CT showed a heterogeneous gall bladder fossa mass infiltrating into the adjacent liver with loss of the fat plane between the mass and the thick-walled pylorus, with air within the mass compatible with invasion of pylorus. Image-guided fine-needle aspiration biopsy confirmed the lesion histopathology as gall bladder adenocarcinoma.

INTRODUCTION

Primary carcinoma of the gallbladder is an uncommon, aggressive malignancy that affects women more frequently than men. The symptoms at presentation are vague and are most often related to adjacent organ invasion. Therefore, despite advances in cross-sectional imaging, early-stage tumors are not often encountered. Imaging studies may reveal a mass replacing the normal gallbladder, diffuse or focal thickening of the gallbladder wall, or a polypoid mass within the gallbladder lumen. Adjacent organ invasion, most commonly involving the liver, is typically present at diagnosis, as is biliary obstruction. Periportal and peripancreatic lymphadenopathy, hematogenous metastases, and peritoneal metastases may also be seen. The vast majority of gallbladder carcinomas are adenocarcinomas. Because most patients present with advanced disease, the prognosis is poor, with a reported 5-year survival rate of less than 5% in most large series. The radiologic differential diagnosis includes the more frequently encountered inflammatory conditions of the gallbladder, xanthogranulomatous cholecystitis, adenomyomatosis, other hepatobiliary malignancies, and metastatic disease.

CASE HISTORY

A 45-year-old female presented to the emergency department with recurrent episodes of hematemesis. She did

not give any history of drug intake and there was no such history in the past. She was hemodynamically stable with a pulse rate of 96bpm and a blood pressure of 110/60mmHg. Her hemoglobin was 8g/dL, TLC and platelet count were within normal limits. Her kidney function and liver function tests were normal; there was no evidence of generalized coagulation disorder. The patient underwent emergency upper gastrointestinal endoscopy which showed a bleeding ulcer in the wall of the pylorus from which a biopsy was taken. EGD demonstrated a pyloric canal ulcer which was presumed to be the cause of upper gastrointestinal bleeding based on the endoscopic findings. Subsequently, abdominal ultrasonography showed a large heterogeneous mass in the liver in the region of the gall bladder fossa. MDCT of the abdomen was performed next to characterize the mass. CECT study showed a heterogeneous attenuation mass in the liver in the gall bladder fossa with infiltrating margins with the adjacent liver [Fig. 1, 2 & 3].

Fig. 1. Axial CT image through the liver showing a heterogeneous attenuation mass in the gall bladder fossa region involving segments IV and V with areas of enhancement, necrosis and air in it. The mass is infiltrating the adjacent liver, and there is no fat plane of separation between the mass and the pylorus which shows thickening of the wall.

Fig. 2. Coronal reformatted CT image which shows a heterogeneously enhancing mass in the gall bladder fossa with areas of necrosis and air inside the mass, the mass is infiltrating into the pylorus which shows thickening of the wall. Enhancing nodular lesions are seen abutting the hepatic flexure of the colon in the mesocolon.

Fig.3. Sagittal reformatted CT image showing a mass in the gall bladder fossa with heterogeneous attenuation and loss of fat plane between the mass and the adjacent pylorus. The wall of the pylorus is thickened, more so on the side abutting the mass.

There were foci of air attenuation in the mass [Fig. 1, 2 & 3] with loss of the fat plane between the mass and the pylorus which showed thickening of the wall, more pronounced on the side towards the mass [Fig. 1, 2 & 3]. There were no definite foci of calcification in the mass and no definite gall bladder was visualised. These findings were compatible with invasion of the pylorus by the gall bladder fossa mass. Rounded, nodular lesions were seen in the transverse mesocolon [Fig. 3] and in the hepatorenal pouch suggesting mesenteric and peritoneal deposits from the mass. There were no significant enlarged nodes in the vicinity of the mass and there was no ascites. The bile ducts were not dilated and portal vein branches were normal. The large gut was unremarkable. Image-guided fine-needle biopsy of the mass was done which matched with the endoscopic biopsy report of adenocarcinoma. Non-visualisation of gall bladder, disproportionate involvement of liver and asymmetric involvement of pyloric canal contiguous with the mass on MDCT suggested external invasion of the pylorus as the cause of ulceration and bleeding as against the preliminary impression of primary gastric ulcer producing hematemesis in our case. Diagnosis of gall bladder carcinoma invading the pylorus was made on the basis of imaging and histopathology findings.

DISCUSSION

The majority of patients with gallbladder carcinoma present with advanced disease. Symptoms are typically indolent. Chronic abdominal pain, anorexia, or weight loss are common initial complaints [8]. Other reported manifestations of carcinoma of the gall bladder include gastric outlet obstruction, distant metastasis and stroke [1,2,6]. Physical examination may demonstrate a palpable mass, hepatomegaly, and jaundice. The cross-sectional imaging patterns of gallbladder carcinoma have been described as a mass replacing the gallbladder in 40%-65% of

cases, focal or diffuse gallbladder wall thickening in 20%-30%, and an intraluminal polypoid mass in 15%-25% [3,7]. In such cases, contrast material-enhanced CT may demonstrate a hypoattenuating or isoattenuating mass in the gallbladder fossa and soft-tissue invasion of the liver, with protrusion of the anterior surface of the medial segment of the left lobe [3,7]. CT is more useful than US for detecting lymph node involvement, adjacent organ invasion, and distant metastasis [5,7,10]. The most common mode by which gallbladder carcinoma spreads to adjacent organs is direct extension, followed by lymphatic and vascular extension. Intraperitoneal, intraductal, and neural spread of tumor also occur [7,10]. The liver is the organ most frequently involved by direct contiguous spread (65% of cases), followed by the colon (15%), duodenum (15%) and pancreas (6%) [7,10].

Contiguous spread of tumor is facilitated by the thin gallbladder wall, which lacks a substantial lamina propria and has only a single muscular layer. In addition, the perimuscular connective tissue of the gallbladder is continuous with the interlobular connective tissue of the liver [3,7]. Loss of fat planes between the tumor and the adjacent organs on CT has been used as a criterion for diagnosing infiltration by the tumor [7,9]. The loss of fat plane between the gall bladder fossa mass and pylorus, thickening of the wall of the pylorus (more on the side towards the liver than inferiorly) and presence of air inside the mass were the unequivocal evidences of invasion of the pylorus in our case. Gastric spread from gall bladder carcinoma may manifest as gastric outlet obstruction [2]. Invasion of pylorus and duodenum with adjacent carcinoma such as pancreatic carcinoma may present as hematemesis [11]. MDCT facilitates the evaluation of spread by providing multiplanar reformats and depicting adjacent vascular and nodal involvement [9]. The presence of vascular invasion, infiltration of contiguous organs, and hepatic and peritoneal metastases are the primary criteria for considering gallbladder carcinoma as unresectable [3,7,10].

Gallbladder carcinoma is highly lethal, as anatomic factors promote early local spread. The ease by which this tumor invades the liver and surrounding structures including the biliary tree contributes to its high mortality [7,8]. The median survival is 6 months, indicating that the majority of patients present with advanced disease. In general, the therapeutic options for gallbladder carcinoma are limited because of the late stage of disease at presentation in most

cases [8]. In addition, there is no standardized therapy for gallbladder carcinoma. Systemic or regional chemotherapy has had little success. Some reports have demonstrated an increase in survival time for patients who underwent palliative or adjuvant radiation therapy [4].

In our case, the endoscopically diagnosed ulceration of the pyloric canal, proved on biopsy as adenocarcinoma, was actually caused by adenocarcinoma of gall bladder eroding the pyloric canal. Timely cross-sectional imaging and histopathology have a substantial role in classifying the origin of such lesions.

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Author Information

Aijaz Hakeem, MD

Departments of Gastroenterology, Radiodiagnosis, & General Surgery, SK Institute of Medical Sciences

Nisar Wani, MD

Departments of Gastroenterology, Radiodiagnosis, & General Surgery, SK Institute of Medical Sciences

Tariq Gojwari, MD

Departments of Gastroenterology, Radiodiagnosis, & General Surgery, SK Institute of Medical Sciences

Hakim Shafi, DM

Departments of Gastroenterology, Radiodiagnosis, & General Surgery, SK Institute of Medical Sciences

Shubana Rasool, MD

Departments of Gastroenterology, Radiodiagnosis, & General Surgery, SK Institute of Medical Sciences

Muneer Wani, MS

Departments of Gastroenterology, Radiodiagnosis, & General Surgery, SK Institute of Medical Sciences