

A Typical Presentation of Hepatocellular Carcinoma

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Abstract

Introduction: Hepatocellular carcinoma is commonly seen in chronic liver disease patients due to hepatitis B or C infection. Melena and haematemesis are the common gastrointestinal symptoms due to intraluminal haemorrhage. Surgical resection is the treatment of choice.

Case presentation: We report a case of hepatocellular carcinoma presenting as gastric outlet obstruction in a 64 years male with chronic hepatitis B virus infection. Hepatocellular carcinoma was detected during evaluation for gastric outlet obstruction. Intra-operatively there was no invasion to the duodenum; rather there was extrinsic compression and adhesions around the duodenum. Patient underwent successful right posterior sectionectomy.

Conclusion: Hepatocellular carcinoma presenting as gastric outlet obstruction is rare and curative surgery is possible in selected patients.

Keywords: Hepatitis; Hepatocellular carcinoma; Gastric outlet obstruction; Hepatectomy B virus

Introduction

Hepatocellular carcinoma (HCC) is usually seen in patients with chronic hepatitis B or C infection or underlying chronic liver disease. It is usually seen in the 6th and 7th decades of life [1]. HCC rarely infiltrates the gastrointestinal (GI) tract with reported incidence of 0.5% to 2% [2]. They usually present with haematemesis and melena. Gastric outlet obstruction is less common presentation in GI tract invasion by HCC. Here we present a case of HCC presenting as gastric outlet obstruction (GOO) due to extrinsic compression on the duodenum.

Case Report

A 64 years male patient from northeast India presented with persistent vomiting and weight loss of 6 kgs in 3 months. The vomiting was intermittent once in 2-3 days in the beginning, but progressively increased to 2-3 times daily in the last one month. He was a known case of hepatitis B virus carrier for the last 3 years. On examination patient was dehydrated, there was fullness in the upper abdomen with visible peristalsis and non-tender hepatomegaly 3 cms, below the right coastal margin. Routine hematological investigations showed anemia (Hemoglobin-10.2 gm%), platelet count 168 10³/microliter, hypokalemic hypochloremic alkalosis. Liver function tests showed mild elevation of liver enzymes (AST 58 U/L and ALT 64 U/L), albumin 3.6 g/L, and rest of the liver function tests were normal. Child-Pugh score 5. Endoscopy showed normal oesophagus, no varices, hugely dilated stomach with edematous mucosa, and old food material, endoscope could not be passed beyond 1st part duodenum, suggestive of gastric outlet obstruction.

Computerised tomography (CT) scan of abdomen showed a large tumor in the right lobe (segment VI and VII) of liver, of size 10.5 × 8.4 × 7.2 cms, with area of central necrosis and early washout in arterial phase. The lesion was compressing the duodenum, leading to huge dilatation of the stomach, likely malignant morphology causing gastric outlet obstruction (Figure 1). No other lesions were seen in other segments of liver or in the abdomen. Spleen was normal in size and there were no features of portal hypertension or ascites. Serum Alfa-fetoprotein (AFP) level significantly elevated (900 ng/ml). Carbohydrate antigen 19.9 and carcinoembryonic antigen were within normal limit. Our probable diagnosis was HCC in a hepatitis B carrier. The tumor was a resectable tumor, as per the Barcelona clinic liver cancer staging system [3].

The patient was taken up for surgery. On exploration, the liver tumor was originating from segment VI and VII. The tumor was adherent to the 1st and 2nd parts of duodenum, causing outlet obstruction and huge dilatation of the stomach. All the adhesions were separated from the duodenum and no gross serosal invasion was seen. Right posterior sectionectomy (segment VI and VII) of liver was carried out (Figure 2). Post-operative recovery was uneventful. Postoperatively, patient was relieved of vomiting. Histopathology report showed HCC-clear cell type; well differentiated with surgical resection margin free from tumor, Lymphovascular invasion was present. Liver had features of cirrhosis with regenerative atypia. Gallbladder showed features of chronic cholecystitis (Figure 3). Postoperative recovery was uneventful and patient had gained 4 kgs weight in 10 weeks follow up. Follow up AFP levels were 105 ng/ml and follow up CT scan showed well regenerated liver (Figure 4) with normal liver function.



Figure 1: Preoperative CT image showing grossly dilated stomach with tumor mass seen compressing the duodenum.

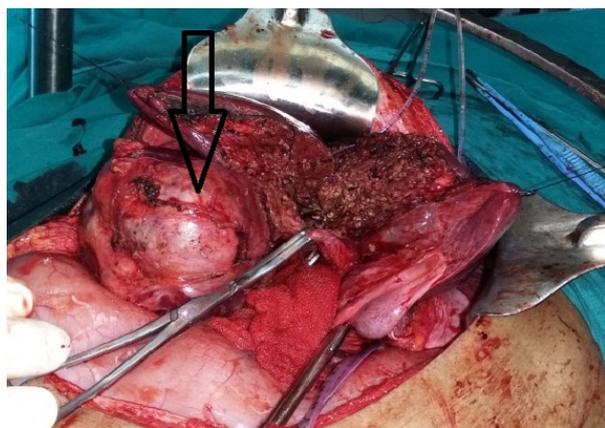


Figure 2: Intra-operative image showing liver parenchymal transection.

Discussion

HCC is one of the most common cancers worldwide; in general it arises from a precursor condition including chronic hepatitis B or C or liver cirrhosis [4]. In most of the reported cases the most common initial GI symptom was melena and few cases had associated hematemesis [2,5]. Endoscopy is essential to determine the source of bleeding [5]. In our patient presentation was unusual. He presented with GOO and weight loss, mimicking an obstructive lesion in the distal stomach and duodenum. There were only few reported cases of HCC presenting as GOO. The cause of GOO in our case was due to extrinsic compression by the tumor over the 1st and 2nd parts of duodenum. Earlier reported cases described the direct tumor invasion of the GI tract. But in our case, there was only extrinsic compression by the tumor without direct invasion of the duodenum. Preoperative

diagnosis of HCC was difficult, without imaging because of the rarity of the presentation in HCC as GOO.

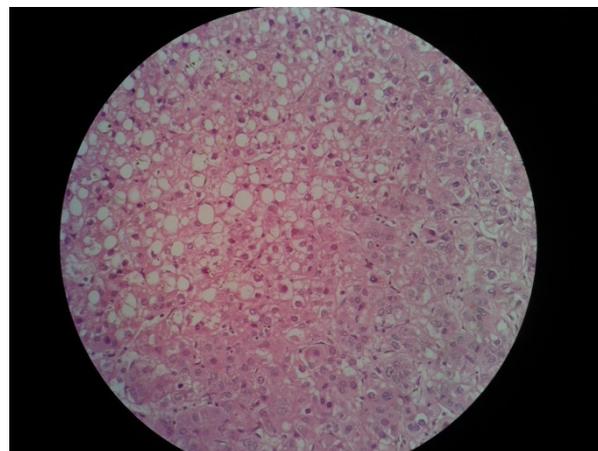


Figure 3: 40X H&E Stain. Photomicrograph displaying tumor cells arranged in lobules, Individual tumor cells are having abundant vacuolated cytoplasm.



Figure 4: Follow up CT image at 6 weeks, showing well regenerated liver with normal stomach.

Direct invasion or extrinsic compression of the GI tract by HCC is uncommon [2,6]. The most common site of invasion is duodenum, followed by stomach, jejunum, colon and pancreas [7]. The presumed mode of direct involvement of the GI tract is initiated by the adhesion of the serosal side of the adjacent organ with a bulky, exophytic tumor. But in our case there was no invasion to serosa of duodenum. Chen et al., who found that GI tract invasion by HCC in 8 of 396 patients (2%), were the first to report GI tract involvement by HCC during the course of the disease. The mode of metastasis was presumed to be haematogenous spread in 2 patients and direct invasion in 5 patients (invasion to stomach in one and of the duodenum in four), but was undetermined in the remaining one patient, in whom the stomach was

involved [2]. Dimitris et al. reported a case of direct invasion of both the stomach and pancreas from causing HCC.

Upper GI tract obstruction Tanaka et al. [8] reported that in 19 of the 29 patients, in whom HCC involved directly the upper GI tract; initial endoscopic assessment revealed an ulcerative hemorrhagic tumor protruding into the lumen of the stomach or duodenum [9]. But in most of these cases were very advanced HCCs unlike our case where R0 resection was achieved due to extrinsic compression on duodenum without serosal invasion.

In most of these cases of HCC invasion of the GI tract, the patients had received some form of regional therapy like transarterial chemoembolization (TACE) and intra-arterial chemotherapy (IA), either alone or in combination, because of unresectability and/or had experienced abdominal surgery [10]. The various possible mechanisms direct involvement of HCC are due to its natural course of invading behavior, post-operative adhesions and scarring and TACE or IA chemotherapy-induced tumor necrosis, causing promotion of subcapsular tumor adhesion to the GI serosa Fuji et al. [10]. Reported that the patients who undergo curative surgery, survive significantly longer compared to those receiving nonsurgical or supportive treatment and the median survival of these patients were 9.7, 3.0, and 1.2 months, respectively [10]. Complete en bloc surgical resection of the tumor along with the involved segment of GI tract with negative margins may be the treatment of choice in order to control symptoms such as obstruction or haemorrhage and to obtain oncologic cure in selected patients with an appropriate hepatic functional reserve [10].

Conclusion

HCC presenting with GOO is a less common presentation. Curative surgery is possible in selected patients with good hepatic functional reserve.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report and its accompanying images. A copy of

the written consent is available for review on demand from the by the Editor-in-Chief of this journal.

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