

Gastric hepatoid carcinoma: Report of a case

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ABSTRACT

Gastric hepatoid carcinoma (GHC) is a rare type of gastric cancer with a tendency to have poor prognosis and metastasize to the liver. GHCs generally show histopathologically hepatocellular differentiation and secrete alpha fetoprotein (AFP). AFP production can occur in cancers originating from the embryologically similar liver, gastrointestinal tract, and yolk sac and often metastasizes to the liver. Although GHC is aggressive, it may not always cause liver metastasis and may invade into the other abdominal organs by direct contact. In this article, we present a case of locally advanced GHC with high AFP levels.

Keywords: Alpha fetoprotein protein; gastric cancer; gastric diseases; human.

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Gastric hepatoid carcinoma (GHC) is a rare type of gastric cancer with a tendency to have poor prognosis and metastasize to the liver [1]. GHCs generally show histopathologically hepatocellular differentiation and secrete alpha fetoprotein (AFP) [2]. In this article, we present a case of locally advanced GHC with high AFP levels.

CASE REPORT

A 60-year-old male patient with no chronic disease history was admitted to our outpatient clinic with complaints of weight loss, abdominal pain, weakness, and palpitation for 3 months. There was no feature in family history. The patient had no history of smoking and had frequent alcohol use. Upper gastrointestinal endoscopy revealed a polypoid tumoral mass which localized from greater curvature to the fundus. Histopathological examination of this lesion was reported as poorly differentiated adenocarcinoma. At laboratory tests of patient, hemo-

globin level was low 9.9 g/dL, AFP level was high with 65,146.95 ng/mL (reference value <9 ng/dL), Ca 19.9, Ca 15.3, and CEA ve Ca 72.4 was within normal limits. Positron emission tomography computed tomography (PET CT) examination revealed a tumoral mass (SUDmax: 18.7) invading the spleen and pancreatic tail in the stomach (Fig. 1). The patient was evaluated as locally advanced cancer and neoadjuvant chemotherapy treatment was started. After 4 cycles of neoadjuvant chemotherapy, control PET CT showed regression compared with the previous examination, but there was still a tumoral lesion with invaded to surrounding tissue (SUDmax: 12.6) (Fig. 2). AFP level was regressed to 22,516.75 ng/mL. After diagnostic laparoscopy, R0 resection was performed with total gastrectomy, D2 lymph node dissection, distal pancreatectomy, and splenectomy. Post-operative follow-up, the patient was discharged with surgical recovery. Histopathological examination of the operation specimen revealed a gastric tumor with a 10 cm ×



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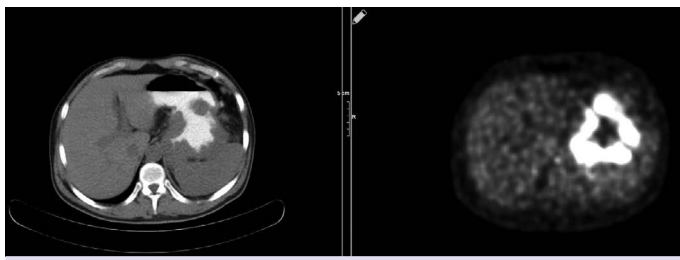


FIGURE 1. Image of positron emission tomography computed tomography, locally advanced gastric hepatoid carcinoma (SUDmax: 18.6).

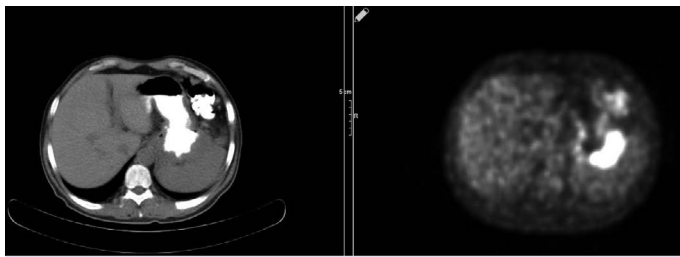


FIGURE 2. Image of positron emission tomography computed tomography, regression after neoadjuvant therapy locally advanced gastric hepatoid carcinoma (SUDmax: 12.7).

6.2 cm×2.3 cm diameter, ulcerated appearance, invading the pancreas and spleen, with pT4bN2M0R0, lymphatic, vascular, and perineural invasion. Immunohistochemical examination revealed MOC31, Glipkan-3, HepPar, SALL4, and pCEA was positive, EMA was focally positive, chromogranin was 10% positive; synaptophysin, PLAP, oct $\frac{3}{4}$, arginase-1, CK20, CDX2, CK7, S100 was negative, and AFP level was too high. Based on these findings, the case was diagnosed as GHC histopathologically. Post-operation follow-ups we send him to medical oncology for adjuvant therapy. Medical oncology planed and started him to four cures adjuvant systemic chemotherapy. After complete two cures, in his post-operation 4th month, patient presented to our hospital's emergency department with the complaint of oral intake disorder. After physical and laboratory examinations, MODS was detected. At the other hand, CT was performed to patient. CT examination revealed multiple metastatic foci in the liver. Because of this, he was hospitalized to intensive care unit (ICU). After complete follow-ups at ICU, and recovered his general condition, he was transferred our clinic. During our follow-ups, the patient was diagnosed as ischemic hepatitis due to elevated liver function tests and deterioration of general condition. Then, he was transferred to ICU again and he died.

DISCUSSION

GHC is a highly malignant histological subtype of GC and may result in spontaneous gastric perforation [3].

GHC is usually seen in the elderly and its pathogenesis is not clear [4]. It seems 2–3 times in men more than women [5]. Patients generally present with abdominal pain and anemia symptoms [6]. Our case was 60 years old and he was anemic.

Immunohistochemical tests such as albumin, alpha-1 antitrypsin, and transferrin are performed for morphological confirmation of this rare histopathological subgroup [2]. In this case, histopathologic diagnosis was made with high level of AFP and immunohistochemical tests. The treatment of patients diagnosed with GHC is surgery and usually requires adjuvant chemotherapy after surgery [7]. Compared with non-hepatoid gastric cancers, patients with a diagnosis of GHC have a worse prognosis with a 5-year survival rate of 9% [8]. This poor prognosis of the GHC may be attributed to these involvements as well as to the production of AFP and presence of AAT/ACT, which have immunosuppressive and protease inhibitory properties, at the other hand, it may be attributed with the extensive venous involvements by tumor cells. It should be kept in mind that when first diagnosed, it may have caused liver metastasis with high AFP and it should be considered in the differential diagnosis of primary liver nodules without any additional disease history such as hepatitis and cirrhosis [6]. GHC can be confused with primary liver cancers at the time of diagnosis due to high AFP values and the frequency of metastasis to the liver and it can be cause to misdiagnosis [9].

Hepatoid adenocarcinoma has been described histopathologically in oral cancers, esophageal, rectal, and prostate cancers. They have poor prognosis like GHCs and there are frequently lung and liver metastasis on first diagnosis [10–13].

AFP production can occur in cancers originating from the embryologically similar liver, gastrointestinal tract, and yolk sac and often metastasizes to the liver [14]. In our case, it was a locally advanced tumor at the time of diagnosis but there was no metastasis to liver.

Although GHC is aggressive, it may not always cause liver metastasis and may invade into the other abdominal organs by direct contact. If hepatic resection can be performed as R0 in GHC patients with liver metastasis, it should be applied in addition to gastric surgery in the

same session. Arterial embolization and subsequent resection can be performed in patients with GHC who are clinically diagnosed with complications of liver metastasis such as bleeding, but survival is very short in these patients [15].

At present, the place of adjuvant systemic chemotherapy in GHC treatment is not clear. Chemotherapy regimens containing cisplatin are used for adjuvant therapy in both metastatic and locally advanced cases [16].

Conclusion

GHC should be considered in patients with high AFP values and no liver mass, and this aggressive tumor should be treated with R0 resection.

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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