Gastric metastasis of hepatic sarcomatoid carcinoma: a case report and literature review

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Case Report

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Abstract

Background

Hepatic sarcomatoid carcinoma (HSC), an extremely rare malignant tumor in clinical practice, mostly occurs in the right lobe and rarely in the left lobe of the liver. This paper reports a case of sarcomatoid carcinoma that occurred in the left outer lobe of the liver, accompanied by gastric stromal tumors (GSTs) of the greater curvature of the stomach, and the patient developed gastric metastasis of liver sarcomatoid carcinoma in the later stage.

Case presentation:

A 63-year-old male patient was diagnosed with both HSC and GST. With fever as the main clinical manifestation, the liver malignant tumor was detected after he underwent abdominal computer tomography (CT) and ultrasound-guided percutaneous liver biopsy at the local hospital. The patient came to our hospital for further treatment. During the next electronic gastroscopy, we found multiple submucosal tumors (SMT) in the stomach. Without multiple metastases in other parts, we performed left hepatic lobe resection with gastric partial resection, and the postoperative pathology determined the presence of HSC and GST. One month after surgery, the patient felt well, and the imaging examination found no obvious space-occupying lesions in other areas. Unfortunately, four months later, the patient showed pain in the upper left abdomen and found metastases in the stomach. We performed the operation on him again, and the patient recovered well after the operation. The patient was still alive at the time of writing this paper.

Conclusion

Hepatic sarcomatoid carcinoma lacks characteristic clinical manifestations and is highly malignant. Surgery is the first choice for the treatment of patients with HSC. For sarcomatoid cancer that occurs in the left lobe of the liver, we need to be highly vigilant against the tumor's invasion of the stomach tissue, especially when the tumor breaks through the capsule of the liver. In addition, during the operation, doctors need to perform delicate operations to avoid dissemination and metastasis of the gastrointestinal tract.

Background

HSC, as a rare malignant tumor with unknown pathogenesis, accounts for 2% of surgically removed hepatocellular carcinoma (HCC) and 3.9–9.4% of autopsy cases [1, 2]. The age of onset of HSC patients is about 60 years, and the disease is more common in men, with its incidence 3–4 times that of women [3, 4]. HSC may be a tumor of monoclonal origin, but its histological origin is still controversial [5]. Compared with HCC and intrahepatic cholangiocarcinoma (ICC), HSC has a higher histological grade,

recurrence rate and metastasis rate, therefore, its prognosis is extremely poor. At present, surgical treatment is the first choice of treatment, and adjuvant treatments include radiotherapy, chemotherapy, interventional therapy and other comprehensive approaches.

Herein, we report the case of a 63-year-old male patient with fever as the main clinical manifestation, and the postoperative pathological diagnosis made as both HSC and GST. We performed left hepatic lobectomy combined with partial gastrectomy on the patient, and the patient recovered well after the operation. However, four months later, the patient was diagnosed with gastric metastases, and we operated on him again. In this paper, we share the clinical characteristics, as well as the diagnosis and treatment process for this patient with the aim to assist colleagues in their associated work.

Case Presentation

Patient information

At the time of hospital admission, the patient was a 63-year-old male with fever as the main clinical manifestation. His body temperature fluctuated between 37.5-38°C. The patient was evaluated outside and referred to our hospital, and he was eventually diagnosed with HSC in the left lobe of the liver and GST in the greater curvature of the stomach. The patient presented no jaundice, and denied a history of chronic diseases such as hypertension and diabetes, a history of infectious diseases such as hepatitis B and tuberculosis, as well as a family history of tumors. During the course of the disease, the patient was in good spirits, and his general state was normal. He had normal eating and sleeping patterns, and healthy urine and bowel movements. In addition, he reported no significant increase or decrease in his body weight for the period leading up to his admission.

Diagnosis and treatment process

The patient, mainly presented with fever, had a physical examination at the local hospital. The ultrasound examination of the liver, gallbladder, pancreas and spleen found space-occupying lesions in the left lobe of the liver. The CT examination of the upper abdomen indicated liver abscess, multiple lymphadenopathies in the abdominal cavity, and a cyst in the left kidney. However, a subsequent (2 days later) ultrasound-guided percutaneous liver biopsy revealed a malignant tumor in the left lobe of the liver. The patient came to our hospital for further treatment and was admitted to the Department of Hepatobiliary Surgery for liver tumors. The patient had no jaundice, and there was no swelling of superficial lymph nodes throughout the body. The abdomen was flat and soft, and showed no tenderness and rebound pain. The liver and spleen were not palpable under the ribs, and no mobile dullness was heard. The blood test showed increased levels of white blood cells ($11.14 \times 10^9/L$), neutrophils ($8.45 \times 10^9/L$), eosinophils ($0.72 \times 10^9/L$), basophils ($0.08 \times 10^9/L$), and content of monocytes ($0.86 \times 10^9/L$). The blood biochemical examination indicated that alanine aminotransferase (37U/L) and aspartate transferase (18U/L) were within the normal range. The total protein content (62.8 g/L) and the white ball ratio (1.0) was decreased, while the C-reactive protein (215.70 mg/L) was abnormally increased. The immune screening detected no hepatitis B virus (HBV) or hepatitis C virus (HCV) infection.

The tumor marker screening found that carcinoembryonic antigen (CEA) (1.24 ng/mL), alpha-fetoprotein (AFP) (4.47 ng/mL), prostate-specific antigen (PSA) (2.98 ng/mL), carbohydrate antigen 19-9 (CA19-9) (6.32 IU/mL) and carbohydrate antigen 15-3 (CA15-3) (18.10 IU/mL) were all within the normal range. As mentioned above, the patient had no HBV infection, the liver function was not significantly abnormal, and AFP was within the normal range, which caught our attention. To further observe the characteristics of the tumor, we performed imaging examinations. The Color Doppler ultrasound (hepatobiliary, pancreatic, spleen and portal vein) showed that the left outer lobe of the liver had a mixed echo of 103 cm*112 cm*110 cm. The tumor was mainly hypoechoic with irregular shape and clear boundaries. The internal echo was uneven, the color showed that there were dots of blood flow signals in and around it, and there was no abnormality in the portal vein. The magnetic resonance imaging (MRI) of the tumor (plain scan + enhanced + functional imaging) detected irregular masses of long T1 (Figure 1. A) and long T2 (Figure 1. B) abnormal signals in the space between liver and stomach. As seen in the images, the size is about 9.5 cm*8.3 cm, the circular short T1 long T2 signal can be seen in it, and the boundary is clear. The lesion and the left lobe of the liver are unclearly decomposed. The diffusion-weighted imaging (DWI) (Figure 1. C) shows a strong signal, and the enhancement (Figure 1. D-F) indicates progressive heterogeneous enhancement. There was irregular thickening of the stomach wall on the side of the corresponding lesser curvature of the fundus. The gallbladder was not enlarged, and its wall was not thickened. There was no dilation of the intrahepatic and extrahepatic bile ducts and no obvious filling defect in the main portal vein and its branches. The morphologies of the pancreas and spleen were not abnormal, and no obvious signal indicating irregularity was observed. In the imaging range, a round-like long T2 signal could be seen in the left kidney, which was strong without enhancement, and multiple small lymph nodes could be seen in the retroperitoneum. We performed a chest X-ray, CT and electronic gastroscopy for the overall assessment of patient status. The chest X-ray and CT showed no abnormalities in either lung, the heart and the diaphragm, whereas the CT showed mass low-density shadows in the left lobe of the liver. The electronic gastroscope indicated a submucosal bulge with a diameter of about 0.8 cm in the fornix of the stomach and the greater curvature of the stomach. The surface of the protuberance was smooth, tough to touch with the biopsy forceps, and was inactive, suggesting multiple SMT. In the lesser curvature of the stomach near the corner, there was a sheet-like shallow erosion with smooth mucosa flushing in color, which showed no ulcers or bleeding. The biopsy was submitted for examination, and the pathology revealed chronic superficial gastritis of the mucosa, which was severe, active and locally accompanied by ulcers and HP infection. The patient's stool occult blood test was negative. Considering the above examination results and anatomical relationship, we preliminarily judged the presence of liver tumor with gastric metastasis, but we could not rule out the possibility that both were primary. It is understood that liver tumors may also be caused by tumor metastasis. The patient did not have multiple metastases in other organs. Ten days after the definite diagnosis of liver tumor, we performed left hepatic lobectomy combined with partial gastrectomy with the patient's and his family's consent. During the operation, we found a mass of about 10 cm in diameter in the left lobe of the liver, with off-white color and hard texture. The tumor broke through the liver capsule, the visceral surface was densely adhered to the lesser curvature of the stomach, and enlarged lymph nodes were palpable in the abdominal cavity. There were two superficial tumors on the greater curvature of the stomach wall. The right liver was normal in size

and soft in texture. The size of the gallbladder was about 7 cm*4 cm*3 cm. No tumor metastatic nodules were detected in the omentum, diaphragm, jejunum or pelvis. During the surgical exploration, it was found that the liver tumor had invaded the left gastric artery, which was removed from the root. The operation was relatively successful, lasted for about 6 hours, and the bleeding volume was about 200 mL. The postoperative specimens were subjected to pathological examination and immunohistochemistry (IHC). The liver tumor tissue specimen was diagnosed as a high-grade pleomorphic malignant tumor of the left lateral lobe of the liver with necrosis. The immunological markers were as follows: CK (+) (Figure 2. B), Vim (-), CD117 (-), Dog-1 (-), CD34 (-), SMA (-), Ki-67 (+, about 60%), GPC-3 (-), Hepar-1 (-). The tissue specimen was also sent to another hospital for consultation, and the immunohistochemical results showed AE1/AE3(+) (Figure 2. C), P16(-), CDK4(+), MDM2(-), Desmin(-), Myogenin(-), MyoD1(-). Molecular detection: MDM2 gene status (-), no amplification. Based on IHC, a sarcomatoid carcinoma with a length of 10 cm, and a negative cut-off edge of the liver section was considered. The gastric tissue specimen was diagnosed as representing spindle cell proliferative disease, combined with immunological markers in line with low-risk GST, featuring 2 pieces with a length of 1.0-1.5 cm. The relevant immunohistochemical markers were as follows: Vim (+), CD117 (+) (Figure 2. E), CD34 (+), Dog-1 (+) (Figure 2. F), Ki-67 (+, approximately 5%), SMA (-), S-100 (-). The patient recovered well and was discharged from our hospital on the eighth day after the operation. The patient did not receive adjuvant therapy. The patient was followed up in the outpatient clinic one month after discharge, when he presented no obvious symptoms or discomfort. There were no abnormalities in the blood routine test and biochemical profile, and AFP (10.5 ng/ml) had no significant increase compared with the first measurement (4.47 ng/mL). The hepatobiliary, pancreatic and splenic ultrasound showed no obvious space-occupying lesions. However, four months later, the patient presented with persistent pain in the upper left abdomen, which worsened paroxysmally. The imaging examination revealed that there was an irregular and substantial mass in the area between the greater curvature of the stomach and the omental sac (Figure 3. A), and the enhanced CT scan showed uneven enhancement (Figure 3. B - Figure 3. D). We performed gastric tumor resection with partial resection of the transverse colon on him. The size of the tumor is 53mm*78mm*100mm, the color is gray and there is necrosis inside (Figure 3. E and Figure 3. F). Results of postoperative pathology and immunohistochemistry confirmed that the tumor in the stomach was metastasized from hepatic sarcomatoid carcinoma. The patient recovered well after the operation. Four months later, the patient died from multiple metastases to other sites.

Discussion And Conclusion

Hepatic sarcomatoid carcinoma

Sarcomatoid carcinoma (SC) is an extremely rarely encountered disease in clinical practice. It can occur in multiple organs throughout the body even at the same time, while the lung and the bladder are the most common sites [6,7], and cases affecting the liver are rare. HSC is a rare malignant tumor that mostly occurs in the right lobe but rarely in the left lobe of the liver. It accounts for 1.8% of all surgically resected HCCs and 3.9-9.4% of autopsy cases [1,8]. It is also believed that the sarcoma-like components in liver cancer are derived from the dedifferentiation of liver cancer [1]. The etiology and pathogenesis of HSC are

still unclear, but there are two main opinions in the medical field. One is that HSC is the sarcomatoid transformation of HCC, and the other is that it is the combination of liver cancer and sarcoma. The former is accepted by more scholars [9]. Previous studies have reported that sarcoma transformation may be related to certain preoperative anti-liver cancer treatments, such as transcatheter hepatic artery chemoembolization (TACE), radiofrequency ablation (RFA), and percutaneous ethanol injection therapy [10-12]. However, there are also reports that some patients who have not received such anti-cancer treatments developed sarcoma changes [13-15]. HSC is a mixed tumor composed of cancer and sarcomatoid components. According to the different cancer components, HSC is mainly divided into sarcomatoid hepatocellular carcinoma (SHC), intrahepatic sarcomatoid cholangiocarcinoma (ISCC), and undifferentiated cancer. Under normal circumstances, the volume of HSC is larger than that of HCC, which helps to distinguish between the two. In a study of 28 HSCs, the average HSC size was 6.1 ± 2.8 cm, and 18 cases (64.3%) had tumors larger than 5 cm [16]. Another study involving 17 cases of HSC pointed out that its long diameter was 6.12 ± 3.18 cm, while the HCC's long diameter was 4.21 ± 2.38 cm [17]. In our case, the HSC had a long diameter of about 10 cm, which is consistent with the previous reports. For larger HSCs, the probability of false capsules and bleeding may increase. In the study including 28 patients, 9 (32.1%) had pseudocapsule formation, most of which were incomplete [16]. The other study showed that the probability of HSC (47.1%) of pseudocapsule formation was less than that of HCC (84.0%) but more than that of ICC (12.0%) [17]. In the patient of our case, T1WI showed a high signal in the tumor, suggesting the possibility of intratumoral hemorrhage. HSC is highly aggressive and is associated with a particularly poor prognosis. In the above study, it was found that up to 47.1% of HSC patients had intrahepatic metastasis, which was far greater than the 12% of HCC patients, but there was no significant difference from ICC [17]. A total of 93% of HSC patients were reported to have extrahepatic metastases during treatment, with the most common metastatic organs being the lung, peritoneum, pleura, pancreas, adrenal gland, intestine and spleen. In the study including 28 patients, it was found that the number of patients with local invasion, vascular invasion, bile duct invasion, and lymph node metastasis was 6 (21.4%), 8 (28.6%), 3 (10.7%), and 7 (25 %), respectively [16]. Similar characteristics were found in other studies [16, 18]. In this case, no other organ metastases were found at the first visit, but gastric metastases appeared four months later. HSC has a high pathological grade, poor tumor differentiation, proneness to vascular infiltration and local infiltration, and poor prognosis. In the study involving univariate analysis [16], it was pointed out that TNM stage, pseudocapsule formation, local invasion, vascular invasion, Child-Pugh classification, and radical resection are the prognostic factors of OS in HSC patients. In a different study involving 79 cases of HSC, the 1-year and 3-year overall survival rates were 63.3% and 35.4%, respectively [19]. In addition to these poor survival rates, HSC is prone to recurrence. It was pointed out that during the follow-up period, 25 of 28 (89.3%) patients who underwent radical surgical resection had tumor recurrence, and the cumulative recurrence rate at 6, 12, and 24 months was 53.6%, 78.6%, and 85.7%, respectively [16]. The patient of our study was re-examined in the clinic one month after the operation, and he complained of no symptoms involving significant discomfort, and there were no abnormalities in the blood routine test and biochemical profile, or AFP (10.5 ng/mL). In addition, the hepatobiliary, pancreatic and splenic ultrasound showed no obvious metastases. However, four months later, the patient presented with persistent pain in the upper left

abdomen, which worsened paroxysmally. The imaging examination revealed that there was an irregular and substantial mass in the area between the greater curvature of the stomach and the omental sac, and the enhanced CT scan showed uneven enhancement. We performed gastric tumor resection with partial resection of the transverse colon on him. Postoperative pathology confirmed that the tumor in the stomach was metastasized from hepatic sarcomatoid carcinoma. The patient recovered well after the operation.

Diagnosis of hepatic sarcomatoid carcinoma

The diagnosis of HSC depends on the clinical features, imaging data, pathology, and IHC. The characteristics of HSC and GST are compared in Table 1.

Clinical features

The clinical parameters recorded for HSC patients include age, gender, clinical manifestations, Hepatitis B surface antigen (HBsAg) and Hepatitis C virus antibody (anti-HCV), liver cirrhosis background, liver function test, serum tumor markers AFP, CA19-9, CEA, etc. The age of onset of these patients is about 60 years. The condition is more common in men, and its incidence is 3-4 times of that in women [3,4]. Symptomatic HSC cases account for 68%-75% of cases. Abdominal pain is the most common symptom [4,9,16], followed by fatigue, fever, and jaundice. The typical systemic symptoms caused by liver decompensation are jaundice, fever, abdominal distension and weight loss. HSC patients can also show gastrointestinal symptoms, such as persistent pain in the upper right quadrant, nausea, vomiting, weight loss, and palpable mass in the xiphoid [20]. In addition, a small percentage of patients are asymptomatic(17.6%), which, to a certain degree, is the opposite of liver cancer that tends to exhibit no symptoms for most patients(70.0%)[17]. In one study, it was pointed out that about half of HSC patients had HBV infection [17], which helps to differentiate HSC from HCC, especially for HBV-negative cases, because 92.0% of HCC patients have HBV infection. In another study [21], it was found that 14 of 19 HSCs (73.7%) had a history of liver cirrhosis. A number of studies have also pointed out the potential relationship between chronic liver disease and HSC [22-24]. The incidence of HSC chronic liver disease(70.6%) was found to be lower than that of HCC(96.0%), and the prevalence of liver cirrhosis was shown to be higher than that of ICC [17]. The diagnostic value of serum AFP, CEA, CA19-9 and CA-125 and other tumor markers is very limited, since they may be normal, slightly elevated, decreased or even negative. although some studies have pointed out that these indicators present different rates of abnormal increase in HSC [3,9,21]. Nonetheless, this helps with the differential diagnosis of HCC, in which CEA, CA19-9 and CA-125 are often negative, and AFP is abnormally high in nearly half of affected patients [17]. The patient of our case report was a 63-year-old elderly male, with fever as the main clinical manifestation. The patient's physical examination showed no other positive signs of disease, the lack of HBV or HCV infection, and no manifestations of liver cirrhosis. The tumor indicators CEA, AFP, PSA, CA19-9, and CA15-3 were all within the normal range.

Imaging data

Although the diagnosis of HSC relies on a large number of pathological signs and IHC evaluation, imaging data play an indispensable role in making a definitive diagnosis. In particular, enhanced MRI is crucial for the differential diagnosis of HSC. Sarcoma components are composed of poorly differentiated cells that grow rapidly with the formation of new blood vessels, however, these blood vessels cannot adequately supply the fast-growing malignant cells, leading to necrosis. On an enhanced MRI image, HSC is typically manifested as peripheral enhancement, central necrosis, and different enhancements with or without a tumor envelope in the solid part [25]. The varied tissue composition of HSC determines its enhancement mode, which also means that the imaging results of HSC are complex and diverse. In one example, more than half of HSC cases showed progressive enhancement in the Gd-DTPA-enhanced MRI scan, and about one-third of cases showed continuous high enhancement [25]. Another study involving 10 cases of HSC reported that the enhancement mode of MRI was mainly "progressive" [26]. In a different study on HSC, the mass mainly showed ring enhancement [21]. The continuous enhancement mode of HSC is one of the key points in the identification of HCC, because the latter is mostly manifested as the "fast in and out" of the contrast agent. Unfortunately, this enhancement mode is similar to intrahepatic cholangiocarcinoma (ICCA). In addition, intrahepatic metastasis [22,23], lymphadenopathy [17] and adjacent biliary tract dilation [24,27] also help to distinguish HSC from HCC, because these characteristics are mostly found in HSC. In ICCA, typical "target signs" can appear on the MRI image, but the probability of false cysts and bleeding is significantly lower than that in HSC [17]. The GST of the liver can also be manifested as hemorrhage, necrosis or cystic degeneration, while the imaging shows uneven hypervascular disease [28,29], and HSC is mostly a mass lacking blood vessels. In our case, the T1WI showed a strong signal (indicating possible bleeding), the enhancement showed progressive unevenness and the lack of "target sign", and found retroperitoneal lymph node swelling. When the patient comes to see the doctor four months later, the imaging examination revealed that there was an irregular and substantial mass in the area between the greater curvature of the stomach and the omental sac, and the enhanced CT scan showed uneven enhancement.

Pathology and immunohistochemistry

As discussed above, the accurate diagnosis of HSC relies on a large number of pathological signs and IHC observations. HSC is composed of spindle cells and epithelial components that are identifiable in ultrastructure, IHC and morphology [30]. The tumor components of HSC are mostly sarcoma-like tissues, which usually make up over 50%. The epithelial component has the common characteristics of multinucleus, meganucleus and mitosis. Under the electron microscope, some sarcoma cells have epithelial desmosome connections and ultrastructural features, such as intracytoplasmic tension microfilaments, clearly showing the morphological characteristics of these two components [20]. The immunohistochemical staining results of HSC were positive for the sarcoma-like component marker Vimentin (Vim), the epithelial component markers keratin (CK) and epithelial membrane antigen (EMA) [31,32] as well as the anion exchanger (AE) 1/AE3, 34 β E12, CAM 5.2, c-Kit, S-100 protein, HHF-35, kinesin-like protein-1, CD34 and HAM-56, which is helpful for the diagnosis of HSC [33]. The case discussed in this study featured necrosis in the liver tumor specimen, which had a length of 10 cm, a negative cut edge of liver section, and the immunohistochemical markers were: AE1/AE3(+), Vim(+) , CD117 (-), Dog-1 (-),

CD34 (-), SMA (-), Ki-67 (+, about 60%), GPC-3 (-), Hepar-1 (-), therefore, it was considered HSC. The molecular detection showed the MDM2 gene status (-) with no amplification. In addition, the microscopic examination indicated that the gastric tumor specimens were characterized by spindle cell proliferative lesions with low risk, featuring 2 pieces with a length of 1.0-1.5 cm, and the immunohistochemical markers were as follows: tumor cells Vim(+), CD117(+), CD34(+), Dog-1 (+), Ki-67 (+, about 5%), SMA (-), S-100 (-), in line with GST. The Ki-67 proliferation index of >35% of SC patients was associated with a poor prognosis [34]. In our case, the Ki-67 index was about 60%. Although GSTs have the longest metastasis to the liver, the pathological and immunohistochemical results ruled out this possibility.

Treatment of hepatic sarcomatoid carcinoma

Surgical resection has been shown as an effective method for the treatment of HSC. Radical surgery can significantly prolong the overall survival of patients with HSC, and the associated survival period is significantly higher than that of palliative resection [16,18]. Whether systemic lymph node dissection can improve the prognosis of patients with HSC lymph node metastasis remains to be further confirmed. In one study, 5 out of 7 patients with lymph node metastasis underwent systemic lymph node dissection, and 4 had early recurrence (within 6 months) [16]. If the HSC edge is detected as aggressive, adjuvant radiotherapy and chemotherapy are needed. HSC is poorly differentiated, with half of all cases classified into grade III and grade IV [3]. More than half of HSC cases cannot be resected due to peripheral invasion and extensive extrahepatic metastasis. For unresectable tumors, systemic chemotherapy and local radiotherapy can be selected [35]. For multiple metastases, combined treatments, such as radiotherapy, chemotherapy and hyperthermia, can alleviate symptoms, but they cannot change the overall survival (OS) [20]. Due to the poorly differentiated carcinogenesis and aggressiveness of HSC, it will be easy to recur even in the early stages of the disease. Vascular invasion and local invasion are independent risk factors for short disease-free survival (DFS) [16]. Although previous studies found that HSC is mainly manifested as vascular clumps, adjuvant TACE is considered a risk factor related to poor prognosis [16]. These results are consistent with a previous study [18], where the average survival time of TACE + surgery and surgery alone was 10.6 months and 18.3 months, respectively [18]. Among patients with intrahepatic recurrence after hepatectomy, the median survival time of those who received TACE treatment was 14.6 months, which was significantly longer than the 8.1-month survival time of patients that received supportive treatment alone [16]. These findings provide evidence that TACE may be beneficial for localized intrahepatic recurrence and may improve the survival rate of these patients. In the presented case, we performed liver lobectomy and performed follow-up one month after the operation. The patient had no obvious discomfort, the tumor marker AFP was not abnormally increased, and no obvious metastases were found in the liver, gallbladder, pancreas, or spleen. However, four months later, the patient presented with persistent pain in the upper left abdomen, which worsened paroxysmally. The imaging examination revealed that there was an irregular and substantial mass in the area between the greater curvature of the stomach and the omental sac, and the enhanced CT scan showed uneven enhancement. We performed gastric tumor resection with partial resection of the transverse colon on him. Postoperative pathology confirmed that the tumor in the stomach was metastasized from hepatic

sarcomatoid carcinoma. The characteristics of the three separately reported cases of HSC are shown in Table 2.

Hepatic sarcomatoid carcinoma is highly malignant, therefore, timely detection, accurate diagnosis and personalized treatment have a great impact on the prognosis of patients. This is an interesting case. When the patient first visited the doctor, we considered that the tumor in the stomach was a metastasis of liver tumor, but the postoperative pathology broke our guess. Then, four months later, a real metastasis of liver tumor appeared in the stomach. Through the evaluation of the case reported herein and that of previous studies, the following points could be made: (1) HSC is relatively rare, and has no characteristic clinical or imaging features. This type of cancer is easy to be misdiagnosed as another type of disease, such as liver abscess, HCC, and ICC. In this case, our colleagues confused it with a liver abscess based on imaging at the first time, which has also been reported in previous studies. (2) In fact, for liver tumors with no abnormal increase in AFP and swollen retroperitoneal lymph nodes but with increased heterogeneity or progressive enhancement in imaging, the possibility of liver sarcomatoid carcinoma needs to be considered. (3) Radical surgery is the first choice for liver sarcomatoid carcinoma, but the integrity of the tumor should be paid attention to during the operation, and the spread and metastasis of the tumor should be highly vigilant. We shared the patient's clinical characteristics and the corresponding diagnosis and treatment process as a significant reference for medical workers in this field.

Abbreviations

Hepatic sarcomatoid carcinoma, HSC; gastrointestinal stromal tumor, GST; computer tomography, CT; submucosal tumor, SMT; hepatocellular carcinoma, HCC; intrahepatic cholangiocarcinoma, ICC; hepatitis B virus, HBV; hepatitis C virus, HCV; carcinoembryonic antigen, CEA; alpha-fetoprotein, AFP; prostate-specific antigen, PSA; carbohydrate antigen 19-9, CA19-9; carbohydrate antigen 15-3, CA15-3; magnetic resonance imaging, MRI; diffusion-weighted imaging, DWI; immunohistochemistry, IHC; sarcomatoid carcinoma, SC; transcatheter hepatic artery chemoembolization, TACE; radiofrequency ablation, RFA; sarcomatoid hepatocellular carcinoma, SHC; intrahepatic sarcomatoid cholangiocarcinoma, ISCC; hepatitis B surface antigen, HBsAg; hepatitis C virus antibody, anti-HCV; Vimentin, Vim; epithelial membrane antigen, EMA; anion exchanger, AE; overall survival, OS; disease-free survival, DFS.

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Committee of Bengbu Medical College. The patient was aware of this research and provided corresponding support.

Consent for publication

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

Availability of data and materials

The datasets used are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

MSS and ZDY collect relevant medical history materials and write drafts. ZGR and DS help gather data and review drafts. ZXL helped organize and review imaging materials. WQ helped organize and review pathology materials. LZ proposed this idea and made the final revision. All authors read and support the final version.

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Tables

Table 1 and 2 available in the Supplementary Files section.

Figures

Figure 1

MRI manifestations of hepatic sarcomatoid carcinoma.

The magnetic resonance imaging (plain scan + enhanced + functional imaging) shows abnormal signals of irregular mass in the liver, with a size of about 9.5*8.3 cm. The T1WI (A) shows a weak signal, the T2WI (B) shows a strong signal and the DWI (C) also shows a strong signal. The arterial phase (D)

indicates slightly uneven enhancement at the edge of the lesion, with continuous uneven enhancement in the portal phase (E) and delayed phase (F).

Figure 2

Pathology and immunohistochemistry of hepatic sarcomatoid carcinoma and gastrointestinal stromal tumors.

HE staining of hepatic sarcomatoid carcinoma (A); the results of immunohistochemical staining of hepatic sarcomatoid carcinoma shows CK positive X200 (B) and VIM positive X200 (C); HE staining of gastrointestinal stromal tumor (D); the results of immunohistochemical staining of the gastrointestinal stromal tumor shows CD117 positive X200 (E) and DOG-1 positive X200 (F).

Figure 3

Related imaging data and clinical specimens of gastric metastasis of liver sarcomatoid carcinoma.

A plain CT scan of the abdomen showed that there was an irregularly-shaped and uneven-density occupancy in the area between the greater curvature of the stomach and the omental sac(A), the size was about 53mm*78mm*100mm; the enhanced scan showed progressive and uneven enhancement (B, C, D); The specimen is off-white(E) with necrosis inside(F).

Supplementary Files

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