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CASE REPORT

Primary hepatic leiomyosarcoma masquerading as liver abscess: A case report

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Abstract

BACKGROUND

Primary hepatic leiomyosarcoma (PHL) is a rare malignant tumor and has non-specific clinical manifestations and imaging characteristics, making preoperative diagnosis challenging. Here, we report a case of PHL presenting primarily with fever, with computed tomography imaging showing a thick-walled hepatic lesion with low-density areas, resembling liver abscess.

CASE SUMMARY

The patient was a 34-year-old woman who presented with right upper abdominal pain and fever over 4 days before admission. Based on the patient's medical history, laboratory examinations, and imaging examinations, liver abscess was suspected. Mesenchymal tumor was diagnosed by percutaneous liverbiopsy and partial hepatectomy was performed. Postoperative pathology revealed PHL. The patient is currently undergoing intravenous chemotherapy with the AD regimen and shows no signs of recurrence.

CONCLUSION

When there is a thick wall and rich blood supply in the hepatic lesion with a large proportion of uneven low-density areas, PHL should be considered.

Key Words: Primary hepatic leiomyosarcoma; Liver abscess; Magnetic resonance imaging; Computed tomography scan; Immunohistochemistry; Case report

Core Tip: Primary hepatic leiomyosarcoma (PHL) is a rare malignant tumor and has non-specific clinical symptoms and imaging manifestations, and patients have a poor prognosis. Our patient was characterized by fever and a thick-walled cystic lesion, leading to our primary diagnosis of liver abscess. When a computed tomography scan shows the presence of a thick wall and rich blood supply in the hepatic lesion with a large proportion of uneven low-density areas, the possibility of PHL should be considered. Magnetic resonance imaging and biopsy can provide the assessment and diagnosis. If conditions allow, early radical surgery is the most effective treatment. In-depth studies are needed to find effective treatments for unresectable PHL.

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INTRODUCTION

Primary hepatic leiomyosarcoma (PHL) is a rare malignant tumor that may originate from the hepatic blood vessels, bile ducts, or ligaments[1]. It is a subtype of primary hepatosarcoma, with unclear pathogenesis possibly linked to factors such as compromised immunity and so on. Preoperative diagnosis of PHL is difficult due to the lack of specific clinical symptoms and the diversity of imaging features. The definitive diagnosis relies on histopathology and immunohistochemistry[2]. Currently, there have been few reports on PHL, and its standardized treatment strategy has not yet been formulated. Patients with liver abscess typically have abdominal pain, fever, and intrahepatic cystic lesions. In rare instances, the combination of PHL and fever may lead to misdiagnosis of liver abscess. We report a case of PHL who presented with fever and a thick-walled cystic lesion, in the hope of providing a reference for future diagnosis and treatment of such cases.

CASE PRESENTATION

Chief complaints

The patient was a 34-year-old woman who presented with right upper abdominal pain and fever over 4 days before admission.

History of present illness

Four days ago, the patient experienced right upper abdominal pain and a fever, her specific temperature is unknown. She took amoxicillin, but the fever persisted and her symptoms were not relieved.

History of past illness

The patient underwent surgery for a right clavicle fracture due to trauma two years ago and recovered well.

Personal and family history

The patient denied any history of smoking, drinking, or exposure to toxic substances. He also denied any family history of genetic disease.

Physical examination

The patient's temperature was 37.3 °C, heart rate was 128 bpm, and blood pressure was 124/76 mmHg. Her abdomen was soft, with mild tenderness on the right upper quadrant, and no rebound tenderness or muscle rigidity. The height of the patient was 158 cm and weight of the patient was 55 kg.

Laboratory examinations

Laboratory examinations showed that the patient's procalcitonin was 0.20 ng/mL (reference range: < 0.05 ng/mL), C-reactive protein was 142.25 mg/L (reference range: < 8.0 mg/L), white blood cell count was 12.6 × 10^9/L [reference range: (3.5-9.5) × 10^9/L], carbohydrate antigen 19-9 was 95.5 U/mL (reference range: < 43.0 U/mL), and hepatitis B surface antibody was negative. Alpha fetoprotein, carcinoembryonic antigen and liver function were normal.

Imaging examinations

An abdominal computed tomography (CT) scan conducted in the emergency department showed a mixed low-density

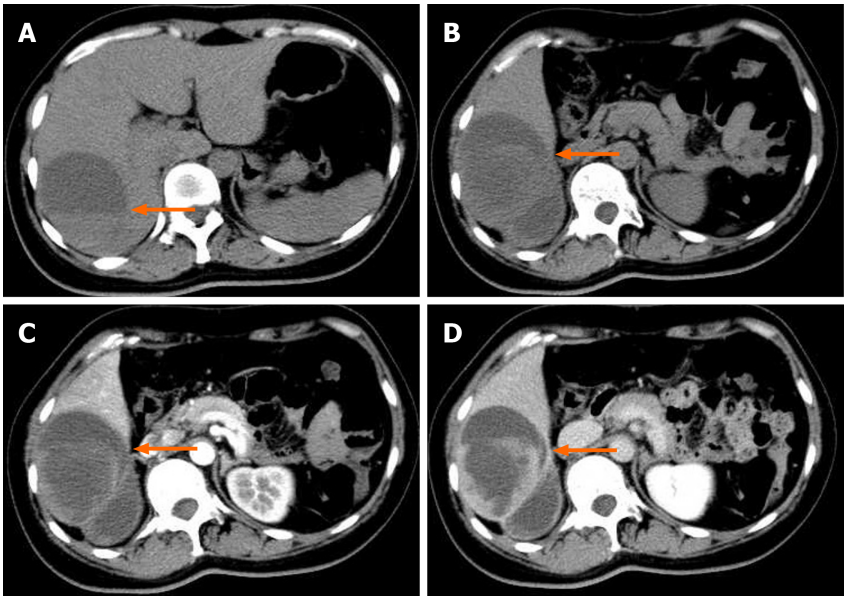

Figure 1 Computed tomography of the upper abdomen before surgery. A mixed low-density lesion (6.5 cm × 9.7 cm × 9.6 cm) can be seen in the right lobe of the liver, within which septations and fluid levels are visible. The enhanced scan showed significant enhancement of the lesion margin and septation (the arrow shows the lesion). A: Plain computed tomography scan; B: Plain computed tomography scan; C: The arterial phase of the enhanced scan; D: Delayed phase.

lesion (6.5 cm × 9.7 cm × 9.6 cm) in the right lobe of the liver, with visible septations and fluid levels. The edges and septations of the lesion were significantly enhanced on the enhanced scan, and our primary consideration was liver abscess (Figure 1).

MULTIDISCIPLINARY EXPERT CONSULTATION

Magnetic resonance imaging (MRI) showed a massive abnormal signal lesion (6.5 cm × 9.7 cm × 7.0 cm) in the right lobe of the liver, with septations and fluid levels. The lesion showed heterogeneous hypointensity on T1-weighted imaging (T1WI) and heterogeneous hyperintensity on T2WI. Enhanced MRI revealed that the edges and septations of the lesion were significantly enhanced during the arterial phase, and the enhancement partially fused and filled toward the center of the lesion during the portal phase and delayed phase. Diffusion-weighted imaging showed a hyperintense signal and the corresponding apparent diffusion coefficient map showed a decreased signal intensity in parts of the wall of the lesion (Figure 2A-H).

Combined with the findings of MRI, the patient was considered to have an intrahepatic malignant tumor with intratumoral bleeding. Further ultrasound-guided percutaneous liver biopsy was performed to confirm the nature of the lesion. Microscopic examination revealed that the tumor was composed of spindle-shaped cells, arranged in a woven pattern, and irregular cell nuclei and frequent mitotic images were observed (Figure 3A). Immunohistochemistry showed positive results for vimentin, smooth muscle cell markers [smooth muscle actin (SMA)], desmin, and Ki-67 (approximately 30%). All others were negative: S-100, SATB2, CD117, ER, CD10, PR, CK7, DOG-1, STAT6, Melan-A, and HMB45 (Figure 3B-D). Considering the immunohistochemistry findings, a mesenchymal-origin tumor was initially suspected. In order to exclude other primary lesions or metastases, a whole-body positron emission tomography-CT scan was conducted and the examination results showed an irregular low-density lesion in the right lobe of the liver with increased uptake and a maximum standardized uptake value of 13.9, and no evidence of distant metastasis (Figure 2I).

FINAL DIAGNOSIS

Combined with preoperative evidence, a primary hepatic mesenchymal malignancy was considered.

TREATMENT

The patient's hepatic lesion was diagnosed as a malignant tumor and obvious distant metastasis and surgical contraindications were excluded. Therefore, partial hepatectomy was performed.

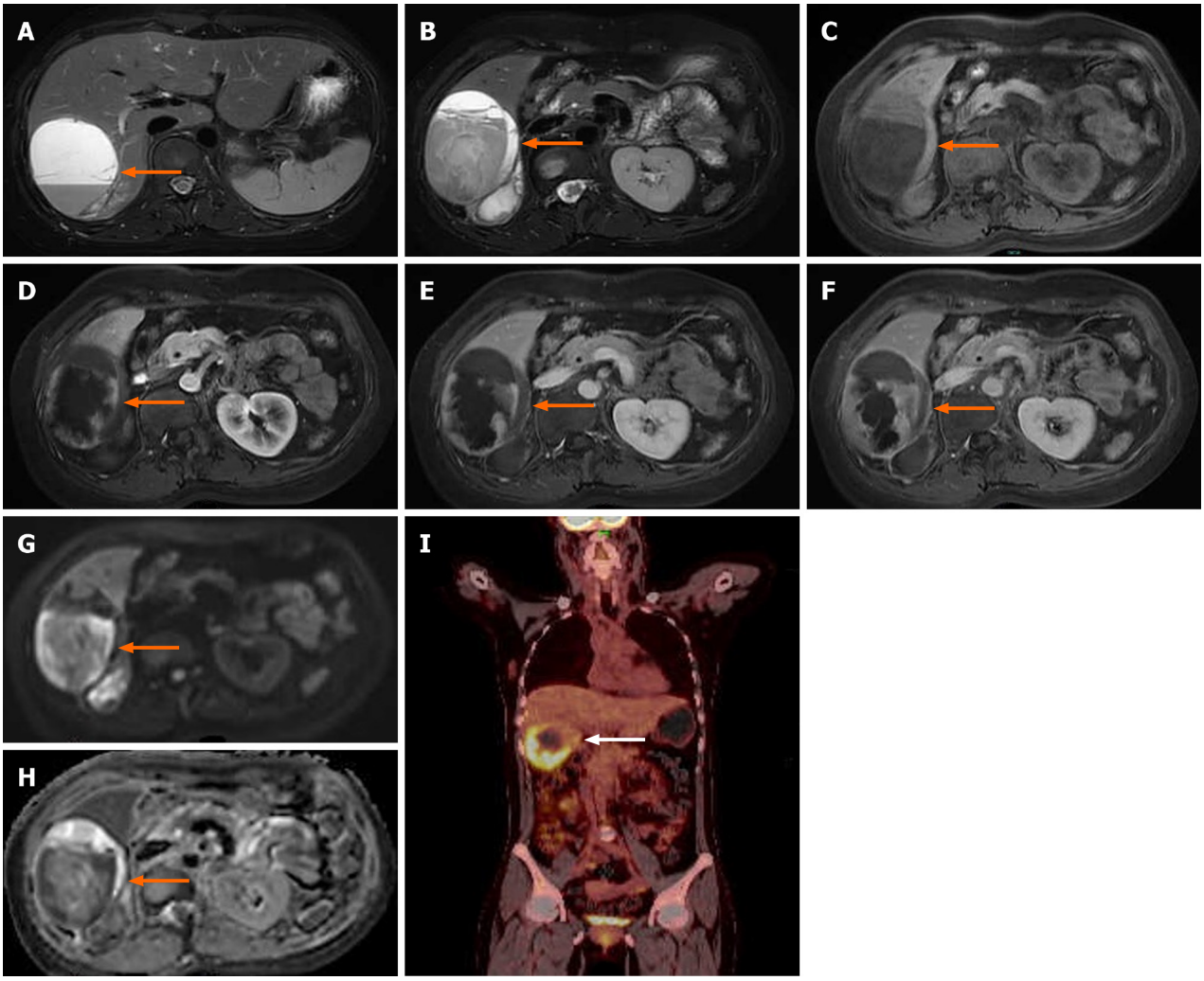

Figure 2 Magnetic resonance imaging of the upper abdomen and positron emission tomography-computed tomography. Magnetic resonance imaging showed a massive abnormal signal lesion in the right lobe of the liver, with septations and fluid levels. The lesion showed heterogeneous hypointensity on T1-weighted imaging and heterogeneous hyperintensity on T2-weighted imaging. The edges and septations of the lesion were significantly enhanced during the arterial phase, and the enhancement partially fused and filled toward the center of the lesion during the portal phase and delayed phase. Diffusion-weighted imaging showed a hyperintense signal and the corresponding apparent diffusion coefficient map showed a decreased signal intensity in parts of the wall of the lesion (the arrow shows the lesion). The positron emission tomography-computed tomography scan showed an irregular low-density lesion in the right lobe of the liver with increased uptake (the arrow shows the lesion). A and B: T2-weighted imaging; C: T1-weighted imaging; D: Diffusion-weighted imaging; E: Apparent diffusion coefficient; F: The arterial phase of the enhanced scan; G: The portal vein phase; H: Delayed phase; I: Positron emission tomography-computed tomography.

OUTCOME AND FOLLOW-UP

During the operation, an irregular solid-cystic tumor (10 cm × 9 cm) was found in the right posterior lobe of the liver. The tumor appeared to have a capsule, with a white-yellow appearance on the cut surface. The cystic part of the tumor was characterized by reddish fluid and old blood clots (Figure 4). Microscopic examination revealed that the tumor exhibited large areas of map-like necrosis, spindle-shaped tumor cells arranged in bundles, cellular atypia was noticeable, and mitotic images were extremely common (Figure 5A). The liver capsule was involved, no definite tumor thrombus was seen in the vessels, and the liver resection margin was negative. Immunohistochemistry revealed vimentin+, SMA+, desmin+, h-cald-, CD117-, CD34-, BcL-2-, STAT6-, DOG-1-, S-100-, MyoD1-, Melan-A-, HMB45-, Myogenin-, Ki-67+ (approximately 75%) (Figure 5B-D). The final diagnosis was PHL. The patient recovered well after surgery, and she is currently receiving AD (doxorubicin liposome 53 mg intravenous infusion *via* micro-pump 16 hours, dacarbazine 675 mg intravenous drip d1-2) chemotherapy, with a 21-day cycle. No recurrence or metastasis has been observed.

DISCUSSION

Primary hepatosarcoma is a rare malignant tumor, accounting for approximately 2% to 3% of hepatic malignant tumors. The most common primary hepatic sarcoma is angiosarcoma, with leiomyosarcoma accounting for only about 12%[3]. It

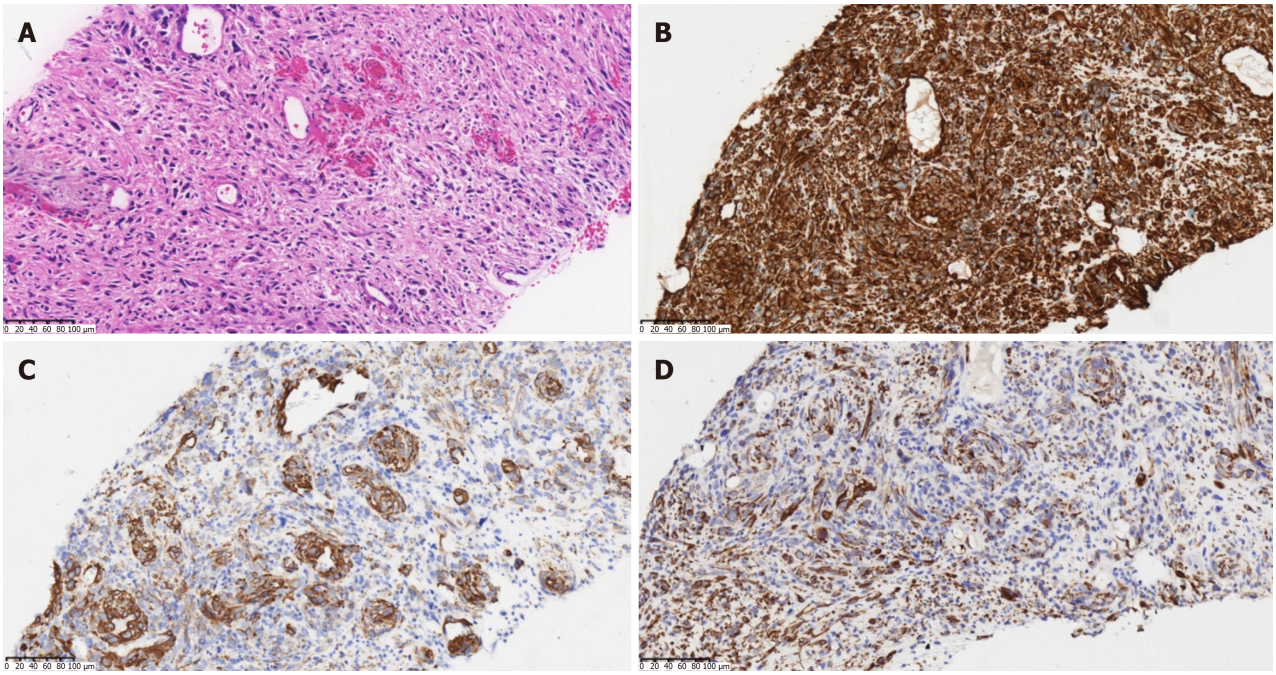

Figure 3 Pathology of the biopsy. A: The tumor was composed of spindle-shaped cells, arranged in a woven pattern, and irregular cell nuclei and frequent mitotic images were observed (hematoxylin and eosin staining, × 200); B: Vimentin (+) (hematoxylin and eosin staining, × 200); C: Anti-smooth muscle actin (+) (immunohistochemical staining, × 200); D: Desmin (+) (immunohistochemical staining, × 200).

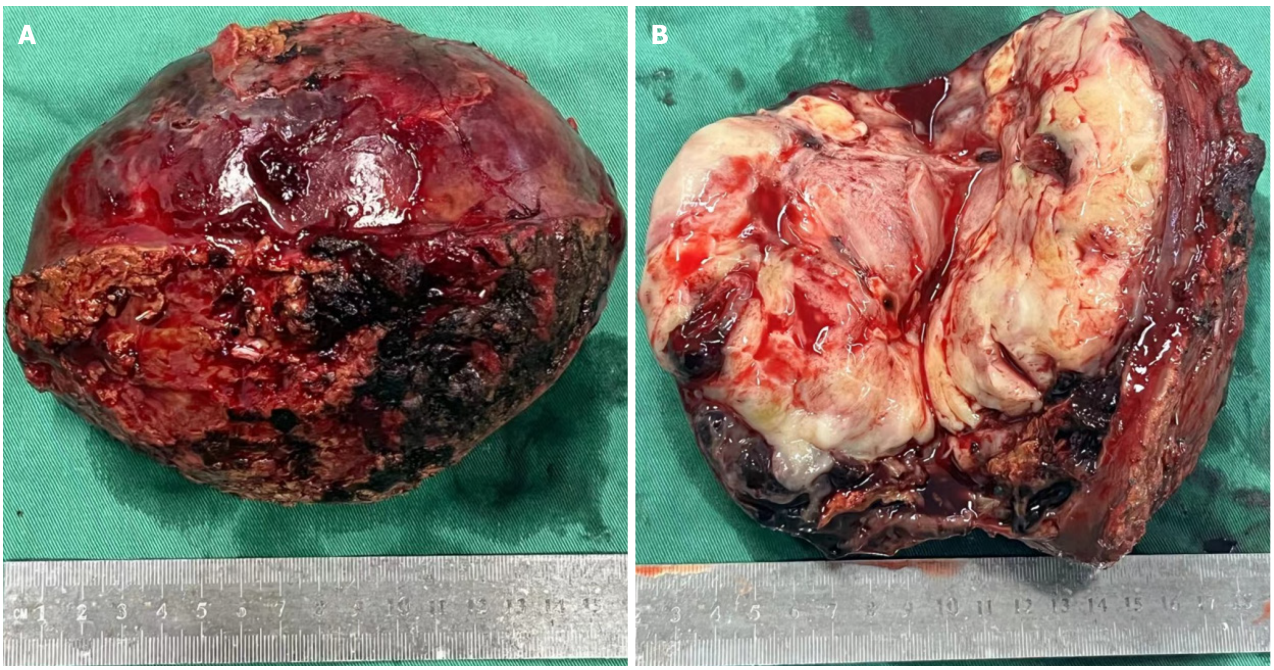

Figure 4 Gross specimen after surgery. The tumor appeared to have a capsule, with a white-yellow appearance on the cut surface. The cystic part of the tumor was characterized by reddish fluid and old blood clots. A: Gross appearance of the tumor; B: Transverse view of the tumor.

has been reported that PHL may originate from the intrahepatic blood vessels, bile ducts, or ligaments[1]. The pathogenesis remains unclear, and the potential causative factors may include Epstein-Barr virus infection, acquired immunodeficiency syndrome, a history of immunosuppression, polycystic kidney disease, renal transplantation, Hodgkin's lymphoma and so on[4,5].

Patients with PHL often have no obvious symptoms in the early stages and may develop abdominal pain, jaundice, anorexia, nausea and vomiting after tumor enlargement. Fever may be the primary manifestation in very few patients due to liquefaction and necrosis in the tumor[6]. PHL originating from the hepatic vein may lead to Budd-Chiari syndrome due to the significant space-occupying effect of a large tumor[7]. Additionally, serum tumor markers such as alpha-fetoprotein and carcinoembryonic antigen are usually normal in patients with PHL, making early diagnosis cha-

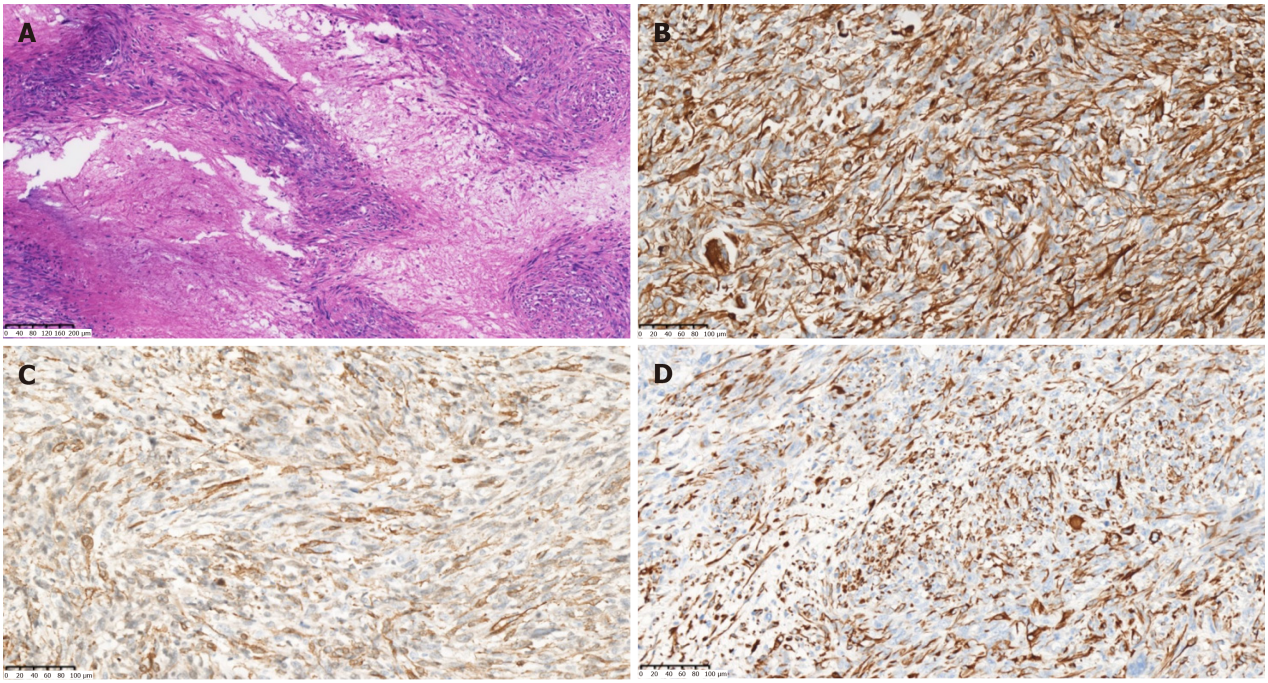

Figure 5 Postoperative pathology. A: Large areas of map-like necrosis were found in the tumor. The tumor cells were spindle-shaped and in a bunch-like arrangement, with obvious cell atypia, varying sizes, and frequent mitotic images (hematoxylin and eosin staining, × 100); B: Vimentin (+) (hematoxylin and eosin staining, × 200); C: Anti-smooth muscle actin (+) (immunohistochemical staining, × 200); D: Desmin (+) (immunohistochemical staining, × 200).

llenging, and around 40% of cases already have metastasized at the time of diagnosis[8]. Our patient presented with fever and abdominal pain as the main symptoms, without potential causative factors or abnormal serum tumor markers, raising suspicion of an association with bleeding and infection in the tumor.

Ultrasonography usually shows a hypoechoic or heterogeneous echogenic lesion[1]. CT findings generally describe a large, well-defined, heterogeneous low-density lesion with internal and peripheral enhancement, or a cystic lesion with enhanced thick walls. During the portal phase and delayed phase, enhancement may either wash out or partially fuse and fill toward the center of the lesion. In some cases, enhancement begins during the delayed phase[9,10]. Combined with pathology, it was found that the low-density areas on CT scans do not solely represent liquefied necrosis, and a large proportion was still tumor parenchyma, which could be scattered with old hemorrhage[11]. In addition, MRI characteristically displays homogeneous or heterogeneous hypointensity on T1WI and hyperintensity on T2WI, and the enhancement patterns are similar to CT scans[12]. In our patient, CT revealed a mixed low-density lesion with septations and fluid levels, and enhancement in the edges and septations of the lesion after intravenous contrast administration. Fever and leukocyte elevation were also present, resembling liver abscess. However, the following two points distinguished the lesion from liver abscess. First, the lesion in this case lacked perilesional edema, which is a typical imaging feature of liver abscess. Second, MRI showed no significant diffusion restriction in the area of hyperintensity on T2WI, while diffusion restriction was evident in the edges and septations of the lesion, which was notably enhanced after intravenous contrast administration. Thus, the possibility of malignancy was considered. The patient was diagnosed with mesenchymal malignant tumor by liver biopsy. After completing positron emission tomography-CT to exclude other primary lesions and metastases, partial hepatectomy was performed, and postoperative pathology confirmed PHL.

The varied and non-specific imaging characteristics of PHL necessitate reliance on histopathology and immunohistochemistry for accurate diagnosis. Histopathology usually shows intersecting bundles of spindle-shaped cells with hyperchromatic nuclei and mitotic figures. The diagnosis is further confirmed by a positive immunohistochemical reaction for SMA, desmin and vimentin, as well as a negative reaction for S-100 protein, CD31, CD34, CD117, DOG1, HMB45, Melan-A, and CK[9,13]. The histopathological and immunohistochemical characteristics in this patient were consistent with reports in the relevant literature.

At present, there is no consensus on the standardized treatment strategies for PHL, but almost all reports suggest that surgical resection is currently the most effective treatment[14]. Negative tumor margins and a tumor diameter less than 10 cm are major favorable prognostic factors[15], so early detection and curative resection are important for PHL patients. Hilar lymph node metastasis is rare, and there is a lack of relevant research on whether regional lymph node dissection should be performed routinely during the operation[15]. For early-stage patients without metastasis, radiofrequency ablation appears to be an acceptable candidate for consideration[16]. The application of liver transplantation for PHL remains controversial[17]. Adjuvant therapy following curative surgery is still in the exploratory phase[18,19]. Due to the aggressive metastatic potential of PHL, adjuvant therapies such as radiotherapy and chemotherapy are recommended following surgical resection[2]. For advanced-stage patients ineligible for surgical intervention, systemic chemotherapy is commonly employed. Chemotherapy regimens are mostly for soft tissue sarcomas, and anthracycline-based chemotherapy regimens are recommended[20]. Irinotecan also shows potential in inhibiting PHL growth[21]. Some studies on combined radiotherapy have shown limited efficacy[22]. Interventional treatments such as transcatheter arterial che-

moembolization and portal vein embolization appear to be safe and effective methods for PHL; however, there are limited sample sizes in current research, necessitating further evidence[7,23]. Moreover, the application of targeted therapy and immunotherapy in PHL is undergoing preliminary investigation and has achieved certain clinical efficacy[21, 24].

The prognosis of PHL is poor, with a median overall survival of 19 months, and the 1-, 2- and 5-year survival rates are approximately 61%, 41%, and 14%, respectively[15]. Early curative surgery can improve prognosis significantly, and disease-specific survival rate at 5 years after R0 resection can reach 67%[2]. Advanced PHL is prone to metastasis, with the lungs being the most common site of metastasis, followed by the pleura/thorax/diaphragm, kidneys and bones[15].

CONCLUSION

We report a rare case of PHL masquerading as liver abscess, which is very confusing. When there is a thick wall and rich blood supply in the hepatic lesion with a large proportion of uneven low-density areas, PHL should be considered. MRI should be performed to assist evaluation, and percutaneous liver biopsy is feasible to confirm the diagnosis. Early radical surgery can improve the prognosis.

FOOTNOTES

Author contributions: Tu CY and Zhou QY contributed equally to the manuscript; Tu CY and Zhou QY designed the study, they are the co-corresponding authors of this article; Wu FN completed the first draft of this manuscript and performed the experiments and data collection; Zhang K, Lv XL, and Guo JQ were involved in data collection; Zhang M provided pathological findings; Zhou QY revised the manuscript. All authors have read and approved the final version of the manuscript.

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