Clinical and Imaging characteristics of primary hepatic sarcomatoid carcinoma and sarcoma: a comparative study

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Abstract

Background

Primary hepatic sarcomatous carcinoma (PHSC) and primary hepatic sarcoma (PHS) are rare malignancy with frequent overlap in the clinic and radiology. There has never been a comparative study of these tumors for the restricted cases. The purpose of our study was to analyze the clinical and imaging features of PHSCs and PHSs, with an emphasis on the particularities and similarities by comparing the two tumors.

Methods

We retrospectively analyzed the clinical and imaging features of 39 patients with pathologically proven PHSCs (n = 23) and PHSs (n = 16) from three university centres over a 9-year period from 2010–2019. Univariate analyses were performed to determine the consistent and distinctive features.

Results

The background of chronic hepatitis or cirrhosis was observed with a high frequency in both of PHSCs (73.7%) and PHSs (62.5%). Tumors with a diameter more than 10 cm were significantly more common in PHSs than PHSCs (p = 0.043) and cystic masses were more detected in PHSs (P = 0.041). Both of PHSCs and PHSs mainly present hypovascularity (73.7% vs 82.6%). The ring hyper enhancement on the arterial phase (AP) and wash out were more frequently seen in PHSCs and the iso-hypo enhancement on the AP followed persistent or progressive enhancement were more commonly detected in PHSs (all, p < 0.05).

Conclusion

PHSC and PHS generally present as mass lesions with hypovascularity. The ring hyper enhancement on the AP and wash out favor the diagnosis of PHSC. The large size greater than 10 cm, cystic lesion, iso-hypo persistent or progressive enhancement pattern might suggest the possibility of PHSs.

Introduction

Primary hepatic sarcomatous carcinoma (PHSC) and primary hepatic sarcoma (PHS), are rare malignancies accounting for only 0.2% (1) and 1% (2) of primary malignant liver tumors, respectively. These tumors present with overlapping features clinically, radiologically and morphologically, making it a challenging task to diagnose accurately.

Sarcomatous carcinoma is defined as a tumor containing an intimate mixture of carcinomatous (either hepatocellular or cholangiocellular) and sarcomatous elements. Sarcomatous change in hepatocellular

carcinoma (HCC) or intrahepatic cholangiocarcinoma (ICC) is defined as “sarcomatous HCC (S-HCC)” or “sarcomatous ICC (S-ICC)” in the World Health Organization (WHO) classification (3). This entity is differentiated from a true hepatic sarcoma such as undifferentiated embryonal sarcoma (UES), leiomyosarcoma (LS), malignant solitary fibrous tumor (SFT), epithelioid sarcoma (ES) and other interstitial tumors deriving from the liver. It should be diagnosed as sarcomatous carcinoma when the sarcomatous component is predominantly composed of spindle cells, but the epithelial cells are still morphologically, immunohistochemically, and ultrastructurally identifiable (1). However, how to accurately identify the two tumors pathologically is still a challenge for pathologists (4–7). Clinically, these tumors are usually asymptomatic until they become significantly large at the time of diagnosis (5, 8). Moreover, these tumors have frequent overlap in the imaging appearances (8–10). Therefore, the preoperative diagnosis is challenging. However, some characteristic clinical and imaging features may exist that can suggest a specific diagnosis. To the best of our knowledge, the current literature about these tumors was limited to either case reports or small case series (11–15) and no reports were available on the comparison of these two tumors except a mention by Mani, H (16). Although the diagnosis is difficult, there still exist some characteristic clinical and imaging features that can suggest a specific diagnosis. Our research aims to explore the clinical and imaging features that can aid to differentiate PHSCs from PHSs.

Methods

Patients

We retrospectively reviewed the patients with PHSC and PHS proven pathologically according to the World Health Organization (WHO) definition in 2000, from three university centres between January 2011 and April 2019. For the inclusion of the subject,all the evaluated lesions did not have any prior treatment. In PHSC, one patient with preoperative intervention by transcatheter arterial chemoembolization (TACE) and two patients with liver metastasis from extrahepatic origin of SC were excluded. In the PHS group, sarcomas of vascular origin including epithelioid hemangioendothelioma (n = 7), angiosarcoma(n = 8), and Kaposi sarcoma(n = 1) were excluded for their relative specificity in the imaging or clinical characteristics. Our study included PHSCs (n = 23, 11 S-HCCs, 4 S-ICCs, 1 S-HCC–CC, 7 unclassified) and PHSs (n = 16, 1 UES, 2 SFTs, 2 ES, 3 LSs, 8 unclassified sarcomas). The clinical materials (demographic characteristics, laboratory data, clinical symptoms and prognosis), imaging findings and pathology results were reviewed. The request and approval for the study protocol was obtained from the Institutional Review Board of each hospital.

Imaging

CT techniques

Twenty-one patients with PHSC and all the patients with PHS were instructed to finish the examinations on the Computed tomographic (CT) scanner (LightSpeed VCT 64, GE Healthcare, Waukesha, Wisconsin, USA) with the following parameters as bellow: tube voltage, 120 kV; tube current, 189–200 mA; matrix, 512 × 512 mm; and section thickness 5 mm. All patients underwent dynamic three phases scanning including hepatic arterial phase (HAP) (25–40 s), portal venous phase (PVP) (45–90 s) and equilibrium phase (EP) (2–5 min) which were obtained after the bolus injection of contrast agent with lopromide (Ultravist 370, Bayer Schering Pharma, Berlin, Germany) at a dose of 1.5 mL/kg and rate of 3 mL/s.

Mri Techniques

Nine patients with PHSC and four patients with PHS were instructed to finish the examinations with 3.0T whole-body MRI systems (Trio, Siemens Healthineers, Erlangen, Germany) with an 8-channel phased array body coil. The parameters of T1-weighted fast low angle shot sequence were mentioned as below: TR/in phase: TE, 170/2.30; out-of phase TE, 3.67 ms; matrix size, 256 × 205; flip angle, 65°. The three-dimensional volumetric interpolated breath-hold examination (3D-VIBE) sequence was obtained before (pre-contrast) and after the injection of contrast agent (Gd-BOPTA, MultiHance, Bracco Pharma, Italy) at a rate of 2 ml/s. The serial dynamic contrast-enhanced scans including HAP, PVP and EP were collected at the time of 25–40 s, 45–90 s and 2–5 min.

Image Analysis

All the images were retrospectively assessed by two abdominal radiologists with more than 7 years of experience in hepatic imaging. When there is disagreement in the assessment of the images, the two readers need to reassess them together.

For morphological lesion assessment, the following items were evaluated: 1. The location (right lobe, left lobe), 2.Size (> 10 cm, ≤ 10 mm), 3.Contour (round, lobulated or irregular), 4. Margin (sharp and indistinct), 5. Liver surface contour (retraction, smooth, bulging), 6.The presence of capsule appearance, hemorrhage, and perfusion alteration, 7.The cystic lesion (The cystic lesion was evaluated based on the predominant parts (75%) of the tumor with cystic changes without any enhancement), 8.The presence of vascular invasion, intrahepatic metastasis and extrahepatic metastasis.

AP enhancement was classified according to the categorizations provided by Rimola et al with modifications (17): 9.non-ring enhancement include the global enhancement that hyperenhancement involving > 75% of the lesion and the nodular enhancement that hyperenhancement involving 25–75% of the lesion; ring enhancement include the peripheral enhancement that hyperenhancement involving 25–75% of the lesion and rim enhancement that rimlike hyperenhancement involving < 25% of the lesion), and iso-hypointensity/density. 10.The vascularity of the whole tumor (lesions with heterogeneous enhancement were evaluated based on the predominant parts more than half of the entire tumor) (17), 11.Dynamic pattern of enhancement (washout, progressive or persistent enhancement).

Statistical Analysis

The continuous variables including the age of patients and the diameter of tumors were expressed as mean ± SD, and the differences between PHSC group and PHS group were conducted using the independent t-test. The categorical variables were compared using -Fisher's exact. P□0.05 was considered to indicate a statistically significant difference. All statistical analyses were performed with the software SPSS® version 23.0 (IBM, Armonk, NY, USA).

Results

Patient Characteristics and clinical background

The PHSC cohort included 23 patients (20 men, 3 women; median age, 56 years; range, 32–77 years), and the PHS cohort consisted of 16 patients (11 men, 5 women; median age, 58 years; range, 22–75 years). The diagnosis proved pathologically was got after surgical resection (15 PHSCs, 6 PHSs) or biopsy (8 PHSCs, 10 PHSs). The clinical data of the patients with PHSC and PHS were summarized in Table 1. There were not significant difference in the tumor markers including AFP, CEA and CA1-99. The majority of patients in both groups were middle-aged men (87.0% vs 68.8%) with the background of liver cirrhosis (73.7.0% vs 62.5%). The most common complaints in PHS and PHSC were abdominal discomfort (43.8% vs 30.4%) and in PHSCs, 34.8% of these patients were detected incidentally in their routine check up for the hepatitis or some other disease. Vascular invasion (56.5% vs 25.0%), intrahepatic metastasis (40.9% vs 25.0%), and extrahepatic metastasis (57.1 vs 37.5%) tended to be more commonly seen in PHSCs than PHSs. In the PHSC group, 13 patients underwent surgery, 5 of them were combined with TACE or RFA (radiofrequency ablation),4 patients received interventional therapy and one patient underwent liver transplantation. 64.7% (11 of 17) of PHSC patients progressed or died between 1–16 months. In the PHS group, 7 patients underwent surgery and 7 received interventional therapy. 76.9% (10 of 13) of PHS patients progressed or died between 1–26 months.

Table 1
Clinical characteristics of the study patients with PHSC and PHS

Variable	PHSC (n = 11)	PHS (n = 11)	P value
Age (y)*	56 (32–77)	58 (22–75)	0.276
Male: female ratio	20: 3	11: 5	0.235
Liver hepatitis cirrhosis	14 (73.7)	10 (62.5)	0.716
Clinical manifestions	11 (52.4)	11 (73.3)	0.500
Tumor markers			
AFP	9 (47.4)	5 (33.0)	0.495
Ca19-9	5 (35.7)	2 (14.3)	0.385
CEA	1 (6.7)	0	0.999
Vascular invasion	13 (56.5)	4 (25.0)	0.099
Intrahepatic metastasis	9 (40.9)	4 (25.0)	0.490
Extrahepatic metastasis	12 (57.1)	6 (37.5)	0.325
Note. Unless otherwise specified, data are numbers of patients, with percentages in parentheses, *AFP* a-fetoprotein, *CA 19 − 9* carbohydrate antigen 19 − 9, *CEA* carcinoembryonic antigen.			
*Data are medians, with ranges in parentheses.			

The morphologic features and accompanying findings of PHSC and PHS

Table 2 summarizes the morphologic characteristics and accompanying findings of PHSCs and PHSs. The PHSCs and PHSs occurred more frequently from the subcapsular area in the right hepatic lobe (69.6% vs 75.0%) with a sharp margin. The tumor greater than 10 mm was more commonly seen in PHSs than PHSCs (P = 0.043). The PHSCs and PHSs mainly showed smooth (47.8% vs 37.5%) or bulging surface (43.5% vs 56.3%) and retraction of the capsule was rare. The capsule appearance occurred in around half of theses patients and 54.5% (6 of 11) of capsule in PHSCs was not complete. Although there was no significant difference, the hemorrhage was more common in PHSs than PHSCs (50.0% vs 26.1%)(Fig. 1)

Table 2
The morphologic features and dynamic enhancement characters of PHSC and PH

Variable	PHSC (n = 23)	PHS (n = 16)	P value
Right lobe	16 (69.6)	12(75.0)	0.999
Tumor diameter	72.6 ± 38.9	94.8 ± 44.4	0.823
≥10 cm	5 (21.7)	9 (56.3)	0.043
10 cm	18 (78.3)	7 (43.8)	
contour			0.999
round	16 (69.6)	10 (62.5)	
Lobulated	2 (8.7)	2 (12.5)	
irregular	5 (21.7)	4 (25)	
margin			0.444
sharp	19 (82.6)	11 (68.8)	
indistinct	4 (17.4)	5 (31.3)	
Liver surface contour			0.786
retraction	2 (8.7)	1 (6.3)	
smooth	11 (47.8)	6 (37.5)	
bulging	10 (43.5)	9 (56.3)	
Capsule appearance	11 (47.8)	6 (37.5)	*0.743*
Hemorrhage	6 (26.1)	8 (50.0)	0.179
Perfusion alteration	11 (47.8)	9 (52.2)	0.748
Cystic mass	4 (17.4)	8 (50.0)	0.041
Note. Data are numbers of lesions, with percentages in parentheses.			

Comparison Of Enhancement Characteristics Of Phsc And Phs

Table 3 summarizes the enhancement characteristics of PHSC and PHS. There was significant difference in the AP enhancement (P = 0.027). Ring hyper-enhancement was more commonly seen in PHSCs than PHSs (60.1% vs 18.8%). Of the 14 patients in PHSCs with ring hyper enhancement on AP, 7 patients demonstrated rim enhancement and the others showed peripheral enhancement (Fig. 2, 3). The iso-hypo

enhancement was more frequently detected in PHSs (Fig. 4) than PHSCs (56.3% vs 30.4%). Pattern of persistent and progressive enhancement was observed in both of PHSCs and PHSs, especially in the PHSs, but wash out was more commonly seen in PHSCs (p = 0.017) (Fig. 5). For the vascularity of the whole tumor, PHSCs and PHSs were predominantly hypovascularity (78.3% vs 87.5%) and the cystic mass was more commonly seen in the PHSs (Fig. 1) than the PHSCs (50.0% vs 17.4%,p = 0.041). Notably, no or minimal enhancement with a nearly complete cystic appearance existed in PHSs in our study (Fig. 1).

Table 3
The dynamic enhancement characters of PHSC and PHS

Vascularity*[1]			**0.999**
Hypervascularity	5 (21.7)	3 (17.6)	
Hypovascularity	18 (78.3)	14 (82.3)	
AP enhancement			0.027
Ring hyperintensity	14 (60.1)	3 (18.8)	
Non-ring hypertensity	2 (8.7)	4 (25.0)	
Iso- or hypointensity	7 (30.4)	9 (56.3)	
Dynamic pattern			0.017
Wash out	12 (52.2)	2 (12.5.0)	
Persistent or progressive	11 (47.8)	14 (87.5)	
Note. Data are numbers of lesions, with percentages in parentheses.			
*[1] The whole tumor was evaluated according the predominant parts more than 50%.			

Discussion

In our study, we did not find any significant difference between the two tumors in the background of liver cirrhosis and the tumor markers such as AFP, CEA and CA1-99. Being different from previous reports that patients with PHSs had no evidence of hepatitis or cirrhosis (8), Ten (62.5%) of 16 PHSs were positive for hepatitis or cirrhosis (18). Fourteen (73.7%) of 19 PHSCs had the background of liver cirrhosis, similar to previous reports that liver hepatitis virus infection might have relationship with the occurrence of PHSCs (5, 19). For PHSs, most of the laboratory data were not helpful in diagnosis (11, 15). Less than half of the patients with PHSCs were positive for AFP, which is lower than that in the ordinary HCC.

Similar to previous study (3, 12, 13, 20), the PHSCs and PHSs demonstrate hypovascularity probably for hemorrhage, necrosis, fibrous tissue or myxoid degeneration (21–24). However, the AP enhancement and dynamic enhancement pattern were significantly different. The current study concluded that PHSCs

mainly showed ring hyper-enhancement on the AP, followed a washout on the later phase. It was reported that the diverse tissue compositions of PHSC determine its enhancement pattern (6). The PHSCs, especially the S-HCCs, were characterized by the peripheral viable cancerous tissue (viable cells, higher microvascular densities and relatively less fibrous tissue) and central necrosis. The sarcomatous component consists of poorly differentiated cells that grow rapidly with the neovasculature unable to adequately supply the fast-growing malignant cells, resulting in necrosis. The PHSs generally present iso- or hypo enhancement on the AP and persistent or progressive enhancement on the later phase, similar to previous studies (12, 25). The myxoid degeneration and the loose arrangement of the cells in PHSs could expand the extracellular space and the contrast agent in the extracellular space were accumulated gradually and expurgated slowly, leading to hypo-iso continuous or progressive enhancement.

Our study demonstrated the cystic mass was commonly seen in PHSs ($P < 0.05$). In current study, there were PHSs displaying as nearly complete cyst-like masses nearly without any enhancement simulating benign tumors, which were never seen in PHSCs. There had been an emphasis on cystic-like appearance in PHSs, which was mainly attributed to the varying degrees of myxomatous change (11, 12, 14, 26, 27). Hemorrhage also played a role in the cystic appearance, which was reported more frequently seen in PHSs than some other rare liver malignant tumors and attributed by rupture of the tumor for the serpiginous vessels (10, 11). In previous studies, there was often extensive hemorrhage in PHS creating a huge cyst mass so that the underlying tumor was obscured and misdiagnosed as a hematoma, abscess or cystic tumor (28), which also occurred in our study. Although there was not statistical difference in the current study, hemorrhage was more commonly seen in PHSs than PHSCs (50.0% vs 26.1%). Besides, the tumor larger than 10 cm in PHSs was significantly more detected than PHSCs ($P < 0.05$). It was reported that solid or cystic manifestations were different stages of PHSs and as the tumor grew, necrosis increased,tending to result in a cystic appearance. In summary, cystic lesions occurred more often in the PHSs and it might help us distinguish PHS from PHSCs.

The capsule invasion (19), vascular invasion or thrombosis, intrahepatic metastasis and lymph node metastasis were more prevalent in PHSCs and in our study the vascular invasion in PHSCs was close to significantly more common than the PHS ($P = 0.099$). The PHSCs were highly aggressive, and the presence of SC were considered to be closely related to the more invasive tumor biology, more common metastasis, low resectability and frequent postoperative recurrences (19, 29, 30). By contrast, the PHSs usually involved the adjacent anatomic structures, and vascular invasion, metastases and lymph node involvement were less common (9, 28).

These tumors should also be differentiated from other liver masses (18). The ring hyper-enhancement of PHSCs may mimic those of ICCs (31). The elevated CA19-9 levels, bile duct dilation around the lesion and capsule retraction may be helpful for the differentiation of these lesions (32). The global avid enhancement with washout and elevated AFP levels help us to differentiate the HCC from PHS and PHSC (18, 33, 34). When the PHSs displaying as nearly complete cyst-like mass as seen in our study, they should be distinguished from other cyst-like lesions such as hydatid cyst, abscess, biliary cyst and adenoma. It was reported in studies that a cyst-like PHS could be frequently misdiagnosed as a hepatic

cyst (8, 14, 26). However, the presence of feeding vessels, the findings of hemorrhage and the abrupt increase in its size should alert us to the diagnosis of PHS (11, 28).

We should acknowledge several limitations to our study. First, for the retrospective study, it was technically unworkable to make the imaging-pathology match slice-by-slice. Second, the relatively small sample size had its internal disadvantages, but it was inevitable for the rare incidence of the tumors. Third, there wasn't a recognized international standard for the evaluation of the cystic tumors.

In conclusion, the PHSC and PHS generally presented as a large subcapsular hypovascular mass. The ring hyperenhancement, wash out and more common vascular invasion favored the diagnosis of PHSC. The large mass with a diameter more than 10cm,iso-hypo intensity/density on AP and pattern of persistent or progressive enhancement might alert us to the possibility of PHS. In spite of the presence of these meaningful diagnostic features, there were not specific for the diagnosis and differential diagnosis of PHSC and PHS. However, the absence of characteristic imaging manifestations of primary hepatic tumors should remind us of the possibility of these tumors.

Abbreviations

PHSC
Primary hepatic sarcomatous carcinoma
S-ICC
Sarcomatous intrahepatic cholangiocarcinoma
S-HCC
Sarcomatous hepatocellular carcinoma
S-HCC–CC
Sarcomatous combined hepatocellular and cholangiocarcinoma
PHS
primary hepatic sarcoma
UES
undifferentiated embryonal sarcoma
LS
leiomyosarcoma;
SFT
solitary fibrous tumor
HAP
Hepatic arterial phase
PVP
Portal venous phase
EP
Equilibrium phase
AFP

Alpha-fetoprotein
CA 19–9
Carbohydrate antigen 19–9
CEA
carcinoembryonic antigen
CT
Computed tomography
MRI
Magnetic resonance imaging

Declarations

Availability of data and materials

Original data and material can be available from the corresponding author if necessary.

Ethics declarations

This retrospective study was conducted with the approval of the Ethics Committee of the Capital medical University Affiliated Youan hospital Zhengzhou University Affiliated Henan Cancer Hospital. Southeast University Affiliated Xuzhou Central Hospital. Written informed consent was obtained from the patients or their parents before MRI or CT.

Consent for publication

Not applicable

Competing interests

The authors declare that they have no competing interest.

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Contributions

DIS: data acquisition and analysis, manuscript preparation and writing. LM:collection of clinical data, manuscript editing. JS: data acquisition and analysis. JC: pathological data analysis .The others: collected and review the data. All authors read and approved the final manuscript.

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Figures

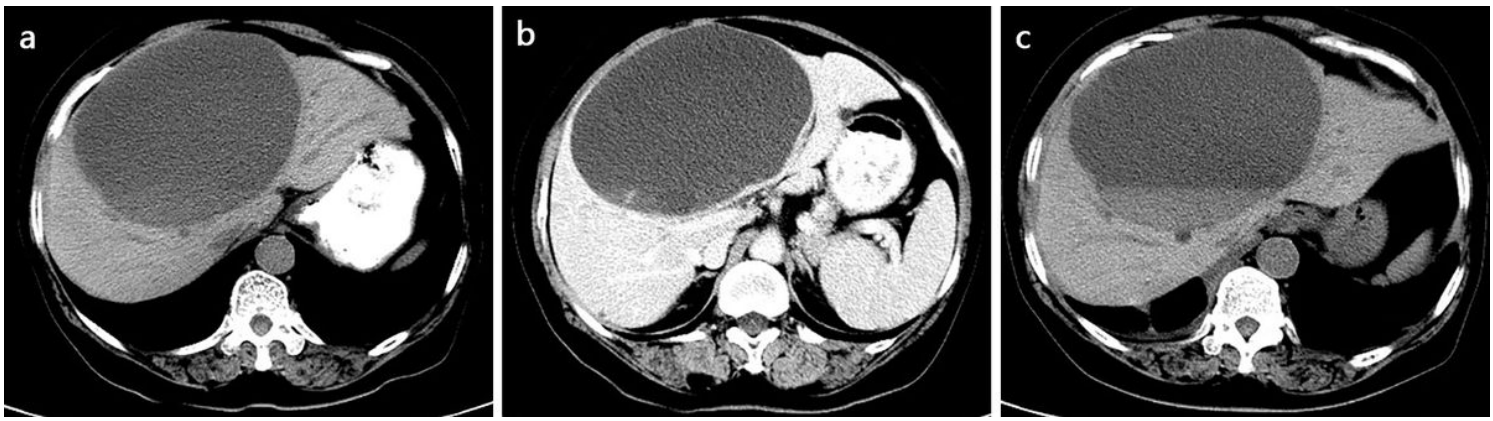

Figure 1

Undifferentiated sarcoma in a 66 year-old man. A large complete cyst-like mass with fluid-fluid level (a) presents minimal enhancement (b) and one month later, the mass grows up and the fluid-fluid level becomes obvious, consistent with hemorrhage (c).

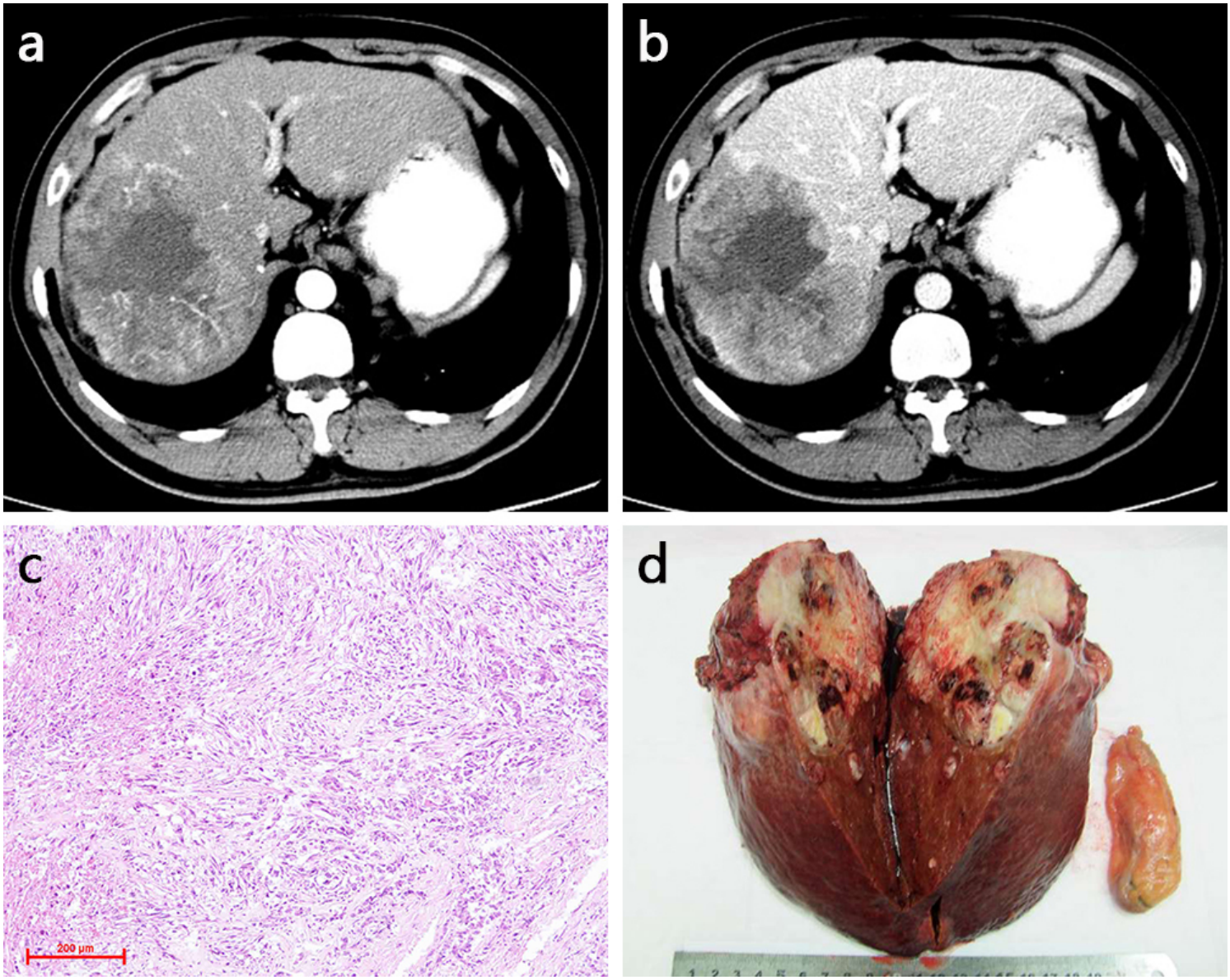

Figure 2

Sarcomatous hepatocellular carcinoma in a 32 year-old man. The contrast-enhanced dynamic CT axial images exhivit the mass hyper peripheral enhancement on the AP (a) and washout on the PVP (b). H & E stain shows some neoplastic cells with pleomorphism(c). The bisected specimen displays a large solid tan mass with necrosis and a satellite lesion (d).

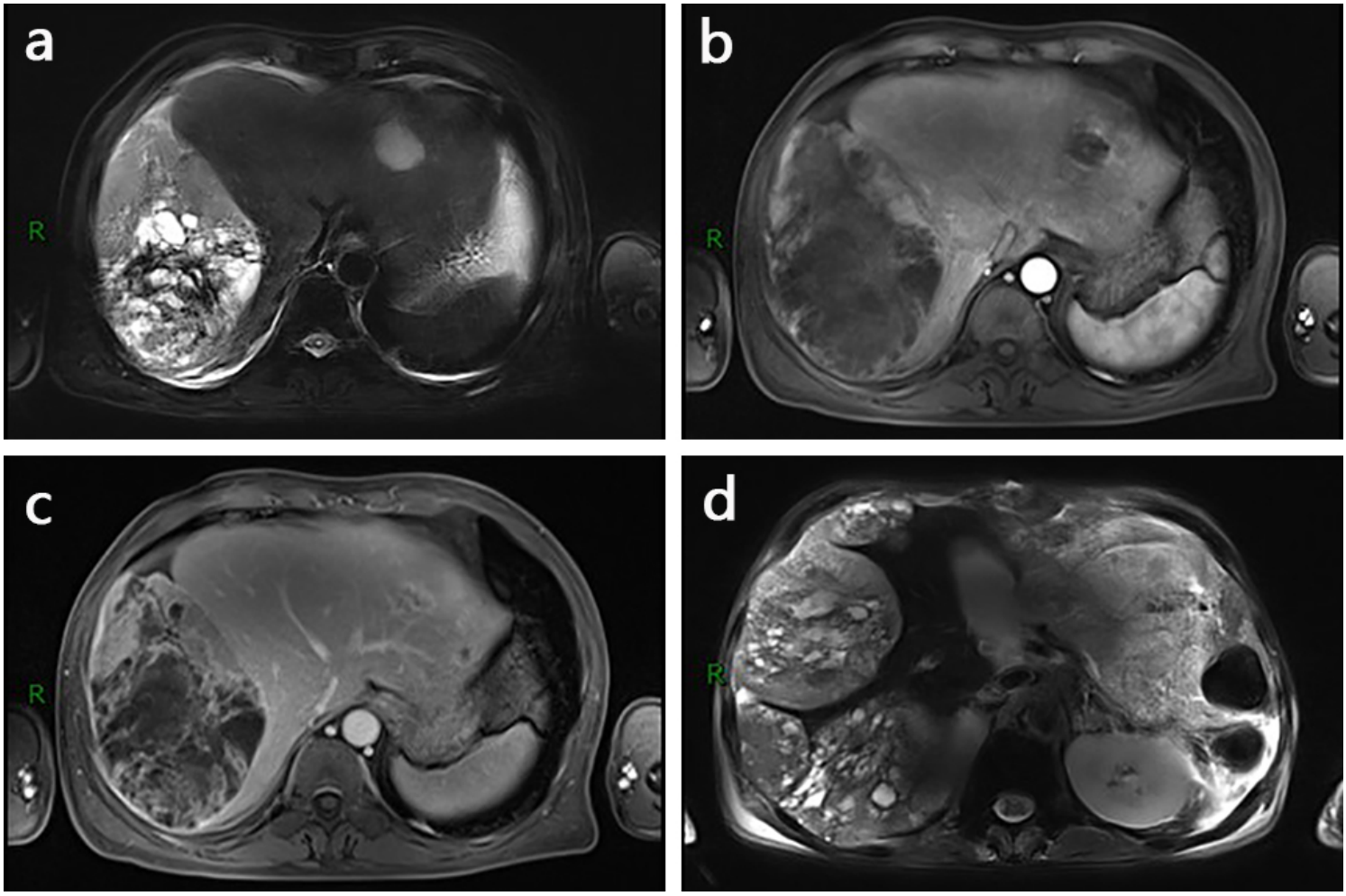

Figure 3

Sarcomatous intrahepatic cholangiocarcinoma in a 55 year-old man. T2-weighted TSE BLADE sequence presents a protruding bulging mass with multilocular cyst-like changes and hemorrhage (a). Dynamic gadoxetic acid–enhanced MR images show hyper irregular peripheral enhancement on the AP (b) followed by peripheral wash out and centrally progressive enhancement with septa on the later phase(c). The mass recurs one month after surgery (d).

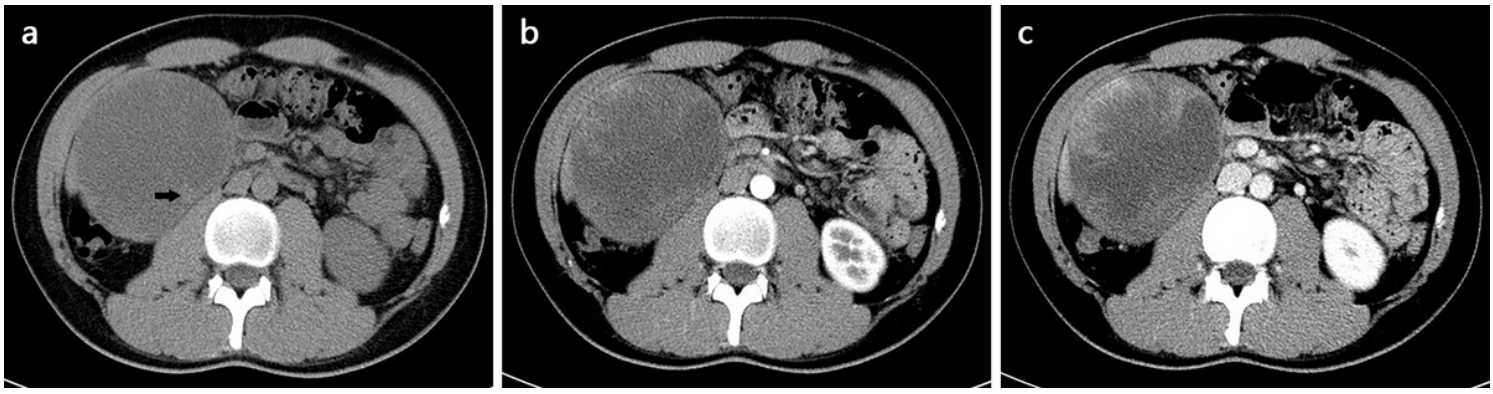

Figure 4

Hepatic undifferentiated embryonal sarcoma in a 22 year-old man. Axial CT image shows a protruding bulging mass in hepatic segment VII with a large cyst-like area (a). Contrast-enhanced dynamic CT axial images present the mass hypo-enhanced peripherally on the AP (b) and centripetally in the PVP (c).

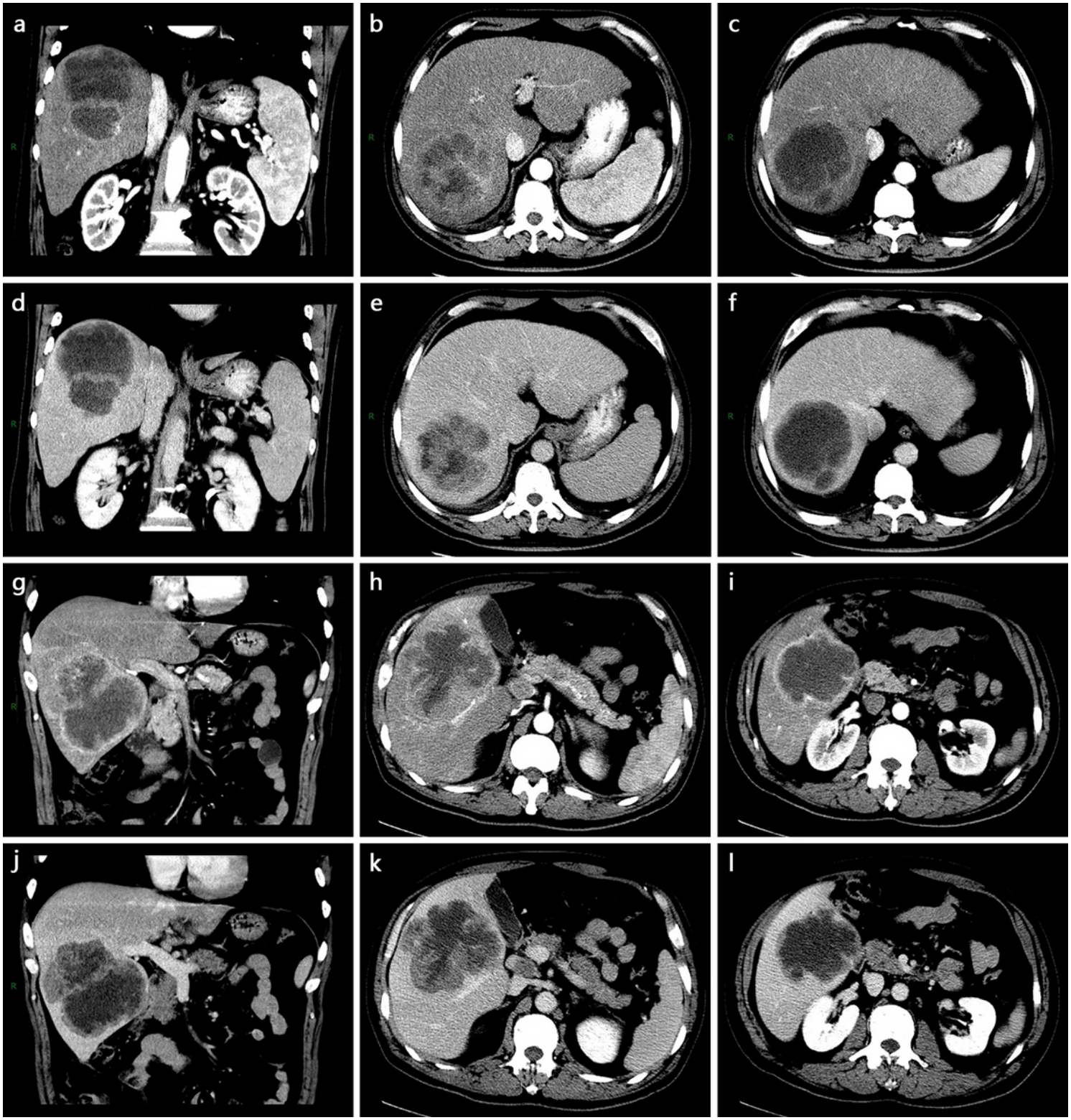

Figure 5

Hepatic leiomyosarcoma in a 62 year-old man (a-f) and sarcomatous hepatocellular carcinoma in a 56 year-old man (g-l). On the contrast-enhanced dynamic CT coronal and axial images, hepatic

leiomyosarcoma (a-c) exhibits hypoenhancement on the AP and persistent or progressive enhancement into the center on the later phase (d-f). The sarcomatous hepatocellular carcinoma presents obvious peripheral enhancement on the AP (g-i) and wash out later (j-l).