Imaging Features of Hepatocellular Carcinoma with Bile Duct Tumor Thrombus: A Multicenter Study

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Abstract

Background: There are still difficult and challenging problems in diagnosis of hepatocellular carcinoma (HCC) with bile duct tumor thrombus (BDTT) before operation. This study aimed to analyze the imaging features of HCC with B1-B3 BDTT.

Methods: The clinicopathological data and imaging findings of 30 HCC patients with B1-B3 BDTT from three high-volume institutions were retrospectively reviewed. Eighteen patients underwent computed tomography (CT) scans and twelve patients underwent magnetic resonance imaging (MRI) scans before operation, respectively. The diagnosis of HCC with BDTT was confirmed by postoperative pathologic examination.

Results: According to Japanese classification, 5 patients were classified as B1 BDTT, 12 B2, 13 B3, and 82 B4, respectively. The HCC lesions were detected in all patients, and the localized bile duct dilation were detected in 28 (93.3%) patients. The BDTT was observed in all B3 patients and 3 B2 patients, but it was not observed in all B1 patients on CT or MRI. The BDTT showed relatively hypoattenuation on plain CT scans and T1W images, relatively hyperattenuation signals on T2W. The BDTT showed hyperattenuation at hepatic arterial phase with washout at portal venous phase. The localized biliary dilation showed no enhancement at hepatic arterial phase and no progressively delayed enhancement at portal venous phase, but it was more obvious at portal venous phase on CT.

Conclusions: The HCC lesions and the localized bile duct dilatation on CT or MRI scans are imaging features of HCC with BDTT, which might facilitate the early diagnosis for B1-B3 BDTT.

Background

Hepatocellular carcinoma (HCC) is the sixth most common cancer in men and the third most common cause of cancer death worldwide [1]. HCC often invades vascular, especially invasion of the portal vein, and forms tumor thrombus. HCC with bile duct tumor thrombus (BDTT) is uncommon with incidence between 0.53 to 12.9% [2–5].

Previous studies have attempted to explore the clinicopathological characteristics and surgical treatment of HCC with BDTT [6–10]. Hepatectomy have generally been considered the preferred treatment for HCC with BDTT. Therefore, accurate diagnosis and surgical treatment are important to improve survival. Both computed tomography (CT) and magnetic resonance imaging (MRI) have diagnostic value for HCC with BDTT and can evaluate the location of BDTT. According to the classification as proposed by liver cancer study group of Japan [11], BDTT was classified as B1-B4. There are still difficult and challenging problems in diagnosis of HCC with BDTT before operation. Few reports focusing on the CT or MRI features of HCC with B4 BDTT were available [12–17].

To the best of our knowledge, the imaging features of HCC with B1-B3 BDTT have not been reported in the literature. Thus, the purpose of our study is to analyze the CT or MRI characteristics of HCC with B1-

B3 BDTT to make a better understanding and early diagnosis of this disease.

Methods

Patient population

Because few HCC patients with BDTT have undergone surgical treatment in a single institution, this retrospective study recruited from three high-volume institutions in Fujian Provincial Hospital (Fuzhou, China), West China Hospital of Sichuan University (Chengdu, China) and the First Affiliated Hospital of Fujian Medical University (Fuzhou, China). From April 2010 to December 2019, totally 7753 HCC patients underwent surgical treatment in three institutions, and 112 patients were found having BDTT. The diagnosis of HCC and BDTT was confirmed by postoperative pathologic examination. Of these patients, 30 patients were classified as B1-B3 BDTT. The clinical data, imaging data and pathological reports of the 30 patients were recorded. The present study was approved by the institutional review boards of each institution.

Image acquisition

CT protocol

64 Slice multidetector CT scanner (Toshiba, Aquilion, Japan) were used. The imaging study was performed from the diaphragm to the iliac crest. The scanning parameters were as follows: section thickness, 5 mm; tube voltage, 120 kV; tube current, 250 mA and intersection gap of 5.0 mm. Using Nonionic contrast material (iopromide, Ultravist, Bayer Schering Pharma, Germany) as CT contrast agent, dose: 1.5 mL/kg, injection flow rate: 3-4.0 mL/s. After injection of contrast agent, hepatic arterial phase (HAP) and portal venous phase (PVP) scans were performed at 34-37s and 60-70s, respectively.

MRI protocol

MRI examinations was performed with 1.5 or 3.0 T MRI systems (Trio, Siemens Healthineers, Erlangen, Germany), using a torso coil. Transverse and coronal T1W scans were performed using the following sequences and parameters: Breath-hold T1-weighted fast low angle shot sequence: TR, 170 ms; TE, 2.30/3.67 ms; flip angle, 65°; matrix size, 256×205; Transverse T2W scan was performed using fat suppressed turbo-spin-echo sequence: TR, 2200 ms; TE, 103 ms; flip angle, 140°; matrix size, 320×106. The slice thickness was 5.0 mm, with 1.0 mm gap. All patients received power injector of 0.1 mmol/kg body weight of gadopentetate dimeglumine (Magnevist, Schering, Berlin, Germany) via the antecubital vein at a rate of 2 mL/s, serial dynamic contrast-enhanced scans were obtained on HAP (25–40 s), PVP (45–90 s) and equilibrium phase (2–5 min) after injection. Magnetic resonance cholangiopancreatography was performed in three patients using turbo spin echo sequence (slice thickness of 2.0 mm with 1.0 mm intersection gap).

Imaging analysis

The imaging findings of HCC with BDTT were retrospectively analyzed as follows: background liver, tumor size, capsule, the location of HCC lesions and BDTT, the precontrast density and contrast enhancement characteristics of HCC lesions and BDTT, vascular tumor thrombus, intrahepatic metastasis or satellite nodule, lymph node enlargement. Special attention was given to the presence or absence of biliary dilation. In comparison with background liver, the density of tumor and BDTT was divided as hypoattenuation, isoattenuation, or hyperattenuation in the precontrast, HAP and PVP. All images were retrospectively and blindly reviewed by two senior abdominal radiologists in consensus.

Pathology analysis

The HCC with BDTT were diagnosed based on the histopathologic findings and immunohistochemical results. Macroscopically, the location, size and capsule of HCC, presence of satellite nodule, necrosis or hemorrhage, vascular invasion, the location and appearance of BDTT, the location of biliary dilation were observed. The histological and differentiation of HCC lesions with BDTT, microvascular invasion, lymph node metastasis and liver cirrhosis were observed under microscope. The diagnoses and analyses were made by two experienced pathologists who were in consensus.

Results

Clinicopathological characteristics

According to Japanese classification, 5 patients were classified as B1 BDTT, 12 patients B2, 13 patients B3, and 82 patients B4 in the present study, respectively. The incidence of HCC with BDTT was 1.4% (112/7753), B1-B3 BDTT was account for 26.8% (30/112).

The clinicopathological characteristics of HCC patients with B1-B3 BDTT were listed in Table 1. Within this cohort, 23 of the patients were male and 7 were female. The mean age was 48.5 years with an age range of 23-76. The liver cirrhosis was found in 90.0% (27/30) of patients. Thirteen of them (27/30, 90.0%) were positive for alpha-fetoprotein (AFP) (>20 ng/dL). No patients were found having obstructive jaundice before operation. The primary tumors located at the left lobe (13 patients) or right lobe (17 patients). The average size of primary tumors was 7.4 (2.5-13.0) cm in diameter and the multiple hepatic lesions were observed in 19 patients (66.3%). Capsule formation was found in 25 patients (83.3%) and poor differentiation was observed in 18 patients (60.0%). Gross portal vein tumor thrombus (PVTT) (Vp2–Vp4) were detected in 14 (46.7%) patients. 26 (86.7%) patients underwent hemihepatectomy and 22 (73.3%) were patients with advanced HCC.

CT and MRI findings

18 patients received CT and 12 patients received MRI scans. The HCC lesions were detected in all patients, and the localized biliary dilation were observed in 28 patients. The BDTT was observed in all B3 patients and 3 B2 patients, but it was not observed in all B1 patients on CT or MRI. The BDTT in 13 B3

patients and 3 B2 patients showed relatively hypoattenuation on plain CT scans and T1W images, relatively hypoattenuation signals on T2W, hyperattenuation at HAP with washout at PVP.

One B1(Fig.1), nine B2 and eight B3 BDTT received CT scans. The HCC lesions showed relatively hypoattenuation on plain CT scan, hyperattenuation at HAP and hypoattenuation at PVP in all patients. The localized biliary dilation showed no enhancement at HAP and no progressively delayed enhancement at PVP, but it was more obvious at PVP.

Four B1, three B2 (Fig.2) and five B3 BDTT (Fig.3) received MRI scans. The HCC lesions and localized biliary dilation showed relatively hyperattenuation signals on T2W images and relatively hypoattenuation signals on T1W images. Early enhancement of HCC lesions at HAP with hyperattenuation signals were observed, but thickened and obviously enhanced bile duct wall were not observed in all patients. At PVP, HCC lesions showed hypoattenuation signals in nine patients and isoattenuation in three patients, and the localized bile duct dilation showed hypoattenuation signals in all patients. The localized biliary dilation was not observed in two B1 BDTT.

The imaging features of HCC with BDTT are summarized in Table 2. The primary tumor located at right anterior section (S5 and/or S8) in 12 patients, right posterior section (S6 and/or S7) in 5 patients, S2 and/or S3 in 13 patients.

Of the thirteen B3 patients, the tumor thrombus located at left hepatic duct in five patients, right hepatic bile duct in eight patients. Of five patients with B1 BDTT, the localized biliary dilation was observed in S2, S3, S5, S6, S8, respectively. Of twelve patients with B2 BDTT, the localized biliary dilation was observed in S2 (3 patients), S3(2 patients), S2 and S3(1 patients), S6 (1 patient), S8 (1 patient), S5 and S8 (2 patients), respectively. Of thirteen patients with B3 BDTT, the localized biliary dilation was observed in right hepatic liver (8 patients) and left hepatic liver (5 patients), respectively.

Discussion

Some studies found that the large lesions, capsule infiltration, poor differentiation, portal vein invasion and intrahepatic metastasis were more frequently observed in HCC patients with BDTT [5, 18, 19]. These differences suggested patients with BDTT had more particular infiltrative nature, which accounted for poorer prognosis than those without BDTT even after curative resection [7–9, 18–21]. Since the bile duct and portal vein are encapsulated in the same Glissonian sheath, tumors can easily involve both of them. About 46.7% patients with BDTT had gross PVTT and 73.3% were advanced stages in the present study. Anatomical liver resection may be more suitable for treating HCC patients with BDTT, because it can remove HCC lesions, BDTT and PVTT at the same time, and improve R0 resection.

Despite recent remarkable improvements in the imaging techniques, the diagnosis of HCC with BDTT is still challenging problems. The patients with B1-B3 BDTT usually have no specific clinical manifestations and do not develop obstructive jaundice. In addition, both clinicians and radiologists are mostly satisfied with the diagnosis of HCC, and lack sufficient awareness of the characteristics of BDTT. Of 34 HCC

patients with BDTT, none of 10 patients with B1-B3 BDTT and half of 24 patients with B4 BDTT was not diagnosed on preoperative CT or MRI scan before operation [2]. Ikenaga et al [18] reported preoperative diagnosis of BDTT was obtained in seven of 15 HCC patients with BDTT, but all of five patients with B1 and three of six patients with B3 BDTT were not diagnosed preoperatively. Only one patient with B3 BDTT and none of patients with B1-B2 BDTT were diagnosed before operation in our study. Therefore, distinctive image features of HCC with BDTT seem especially important to be recognized. HCC lesions and soft tissue masses in the biliary ducts are two typical features, which is the key for diagnosing HCC with B4 BDTT [15, 17]. Our results confirmed that HCC lesions and the localized bile duct dilation may be imaging features of patients with B1-B3 BDTT. Although the B1-B2 BDTT were not observed on CT or MRI scans, the localized bile duct dilation was defected in 93.3% patients and indirectly reflected the presence of BDTT in the study. The tumor invades the bile duct of subsegment, and then the bile duct dilation may not be found on imaging. For example, if the tumor locates in S8 and the tumor thrombus extends to the dorsal bile duct, but does not invade the confluence of dorsal and ventral bile duct, the localized biliary dilation of ventral segment is not observed on CT or MRI scans. When the tumor thrombus invades the confluence of dorsal and ventral bile duct, the localized biliary dilation of ventral subsegment is observed. As the tumor thrombus further extends to the confluence of the S5 and S8, the localized biliary dilation of S5 can be seen. The tumor thrombus further extends to the right hepatic duct, the biliary dilation of right posterior lobe also can be seen. Both CT and MRI have diagnostic value for HCC with BDTT, but MRI displays more detailed information for the diagnosis. The localized bile duct dilation can be seen in each phase of MRI, but it is more obvious at PVP on CT scans. Therefore, deeper understanding of different BDTT is the key to further improving the diagnosis preoperatively.

HCC with BDTT should be differentially diagnosed with intrahepatic cholangiocarcinoma (intraductal type). Both HCC with BDTT and intrahepatic cholangiocarcinoma have similar image features like intraductal neoplasm and upstream bile duct dilatation [17, 25]. Most BDTT show early enhancement at HAP and rapid wash out of contrast agent with hypointense signal at PVP [14, 26]. Intrahepatic cholangiocarcinoma usually manifests a narrowed bile duct with irregular wall thickening and progressively delayed enhancement at PVP [17]. Hepatic parenchymal mass and the T1W hyperintense signal on distal segment are valuable to distinguish BDTT from intraductal growing-cholangiocarcinoma [25]. The presence of liver cirrhosis, serum CA19-9 and AFP level are also supportive of the differential diagnosis. Another relatively rare disease, but also to be distinguished from BDTT, is the HCC compressing the intrahepatic bile duct. The latter can cause the intrahepatic bile ducts to dilate, the location of bile duct dilation is where the HCC compresses the bile duct. However, the bile duct dilation in HCC patients with BDTT is caused by tumor thrombus, not the tumor itself. The tumor and the dilated bile duct have a certain distance, rather than close to the dilated bile duct.

Several limitations to our study need to be acknowledged. Firstly, our study was relatively small sample size due to the rare incidence of these tumors. Despite this, our population is the largest one in the published studies. Second, because there was no jaundice, all patients did not receive MRCP before operation. Thus, to some extent our explanations for the imaging findings of B1-B2 BDTT might be considered speculative before operation.

Conclusions

In summary, pre-operative imaging diagnosis of HCC with BDTT remains a challenge. The HCC lesions and the localized bile duct dilatation on CT or MRI scans were commonly seen in HCC patients with BDTT. This imaging features is helpful in facilitating the early diagnosis for B1-B3 BDTT.

Abbreviations

HCC: Hepatocellular carcinoma, BDTT: bile duct tumor thrombus, CT: computed tomography, MRI: twelve patients underwent magnetic resonance imaging, HAP: hepatic arterial phase, PVP: portal venous phase, AFP: alpha-fetoprotein, PVTT: portal vein tumor thrombus.

Declarations

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Availability of data and materials

The datasets used and analyzed in our study are available from the corresponding authors upon reasonable request.

Authors' contributions

Conceived and designed the research: MLY, ZBZ. Data acquisition, data analysis: LMH, JYW, JYW, YNB, YGW. Drafting the manuscript: MLY, LMH, JYW. All authors read and approved the final manuscript.

Ethics approval and consent to participate

The study was approved by the Ethics Committee of Fujian Provincial Hospital, the Shengli Clinical Medical College of Fujian Medical University.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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Tables

Table 1 The clinicopathological feature of 30 HCC patients with type B1-B3 BDTT

Clinical information	Values
Age (years)	48.5(23-76)
Gender(male/female)	23/7
HBsAg(positive/negative)	29/1
Background liver (Nocirrhosis/Cirrhosis)	3/27
Child-pugh grade(A/B)	28/2

Total bilirubin (μmol/l)	15.9(10-25.7)
Albumin (g/L)	40.7 (29.7-50.8)
ALT(U/L)	51.8 (14-152)
AST(U/L)	60.8(17-286)
AFP (Positive/Negative)	27/3
Tumor number (Single/ Multiple)	11/19
Tumor size	7.4(2.5-13)
Capsule formation (Absent/ Present)	25/5
≥ hemihepatectomy (Yes/No)	26/4
Portal vein invasion (VP0/VP1/VP2/VP3/VP4)	16/0/4/10/0
Lymph node metastasis (Negative/ Positive)	27/3
Tumor differentiation (Well+Moderate/Poor)	12/18
TNM stage(□/□/□/□)	5/3/19/3

Table 2 Imagine findings of 16 HCC patients with type B1-B3 BDTT

Variables	Values		
NO.	Location of tumor	Location and type of BDTT	Dilation of bile duct
1	S5,S8,	RAHBD B2	S5,S8
2	S3	S3 B1	S3
3	S5	RAHBD B2	S8
4	S3	LLHBD B2	S2
5	S5,S8,	RAHBD B2	S5,S8
6	S2,S3	LHD B3	S4
7	S5	RHD B3	S6,S7,S8
8	S3	LLHBD B2	S2
9	S2,S3	LLHBD B2	S2,S3
10	S6S7	RHD B3	S5,S6,S8
11	S2	LLHBD B2	S3
12	S2,S3	LHD B3	S3,S4
13	S2	LLHBD B2	S3
14	S2	LHD B3	S3,S4
15	S5, S8	RHD B3	S5,S6,S7,S8
16	S2	LLHBD B1	S2
17	S5	RAHBD B2	S8
18	S6	S6 B1	No
19	S5	S5 B1	No
20	S3	LHD B3	S2,S4
21	S6	RPHBD B2	S6,S7
22	S2	S2 B2	S2,S3
23	S8	RAHBD B2	S5,S8
24	S8	RHD B3	S5,S6,S7
25	S8	VBD of S8 B1	DBD of S8
26	S5,S8	RHD B3	S6,S7,S8
27	S6	RHD B3	S5,S7,S8

28	S3	LHD	B3	S2,S4
29	S7	RHD	B3	S5,S6,S8
30	S5,S8	RHD	B3	S6,S7

Figures

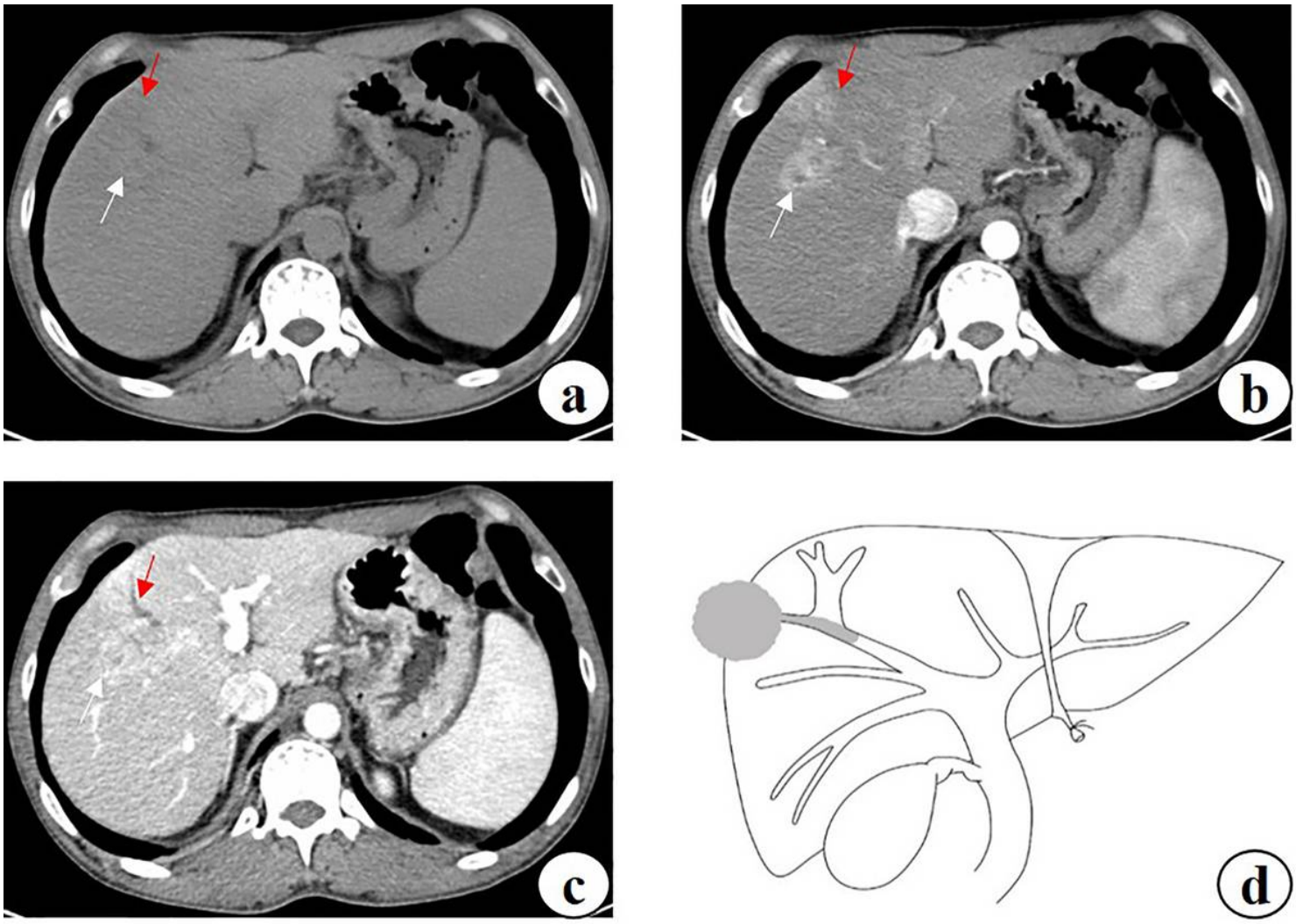

Figure 1

HCC with B1 BDTT. (a) The HCC lesion (white arrow) and the localized biliary dilation in S8 (red arrow) show hypoattenuation in plain CT image. (b) The HCC lesion has early enhancement (white arrow) in arterial phase, but enhancement of the localized biliary dilation (red arrow) was not observed. (c) The HCC lesion (white arrow) show hypoattenuation at portal venous phase, accompanied by dorsal bile duct dilation (red arrow) in S8. (d) HCC lesion, BDTT and bile duct dilation.

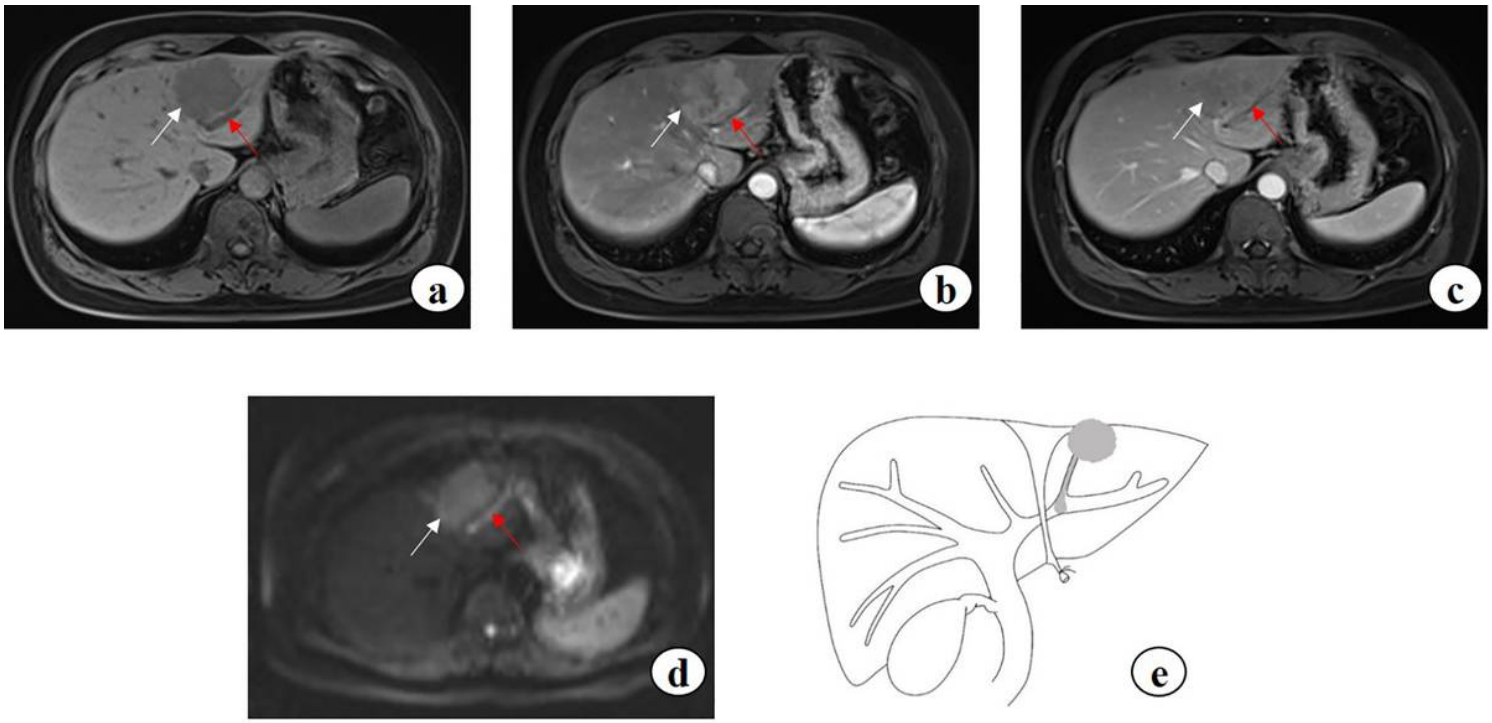

Figure 2

HCC with B2 BDTT. (a) HCC lesion (white arrow) in S3 and bile duct dilation (red arrow) in S2 show slightly hypoattenuation on T1W. (b) HCC lesion shows early enhancement (white arrow) at hepatic arterial phase, and (c) rapid washout of contrast material (white arrow) at portal venous phase, accompanied by the localized bile duct dilation in S2(red arrow). (d) HCC lesion (white arrow) in S3 and bile duct dilation (red arrow) in S2 show hyperintense on T2W. (e) HCC lesion, BDTT and bile duct dilation.

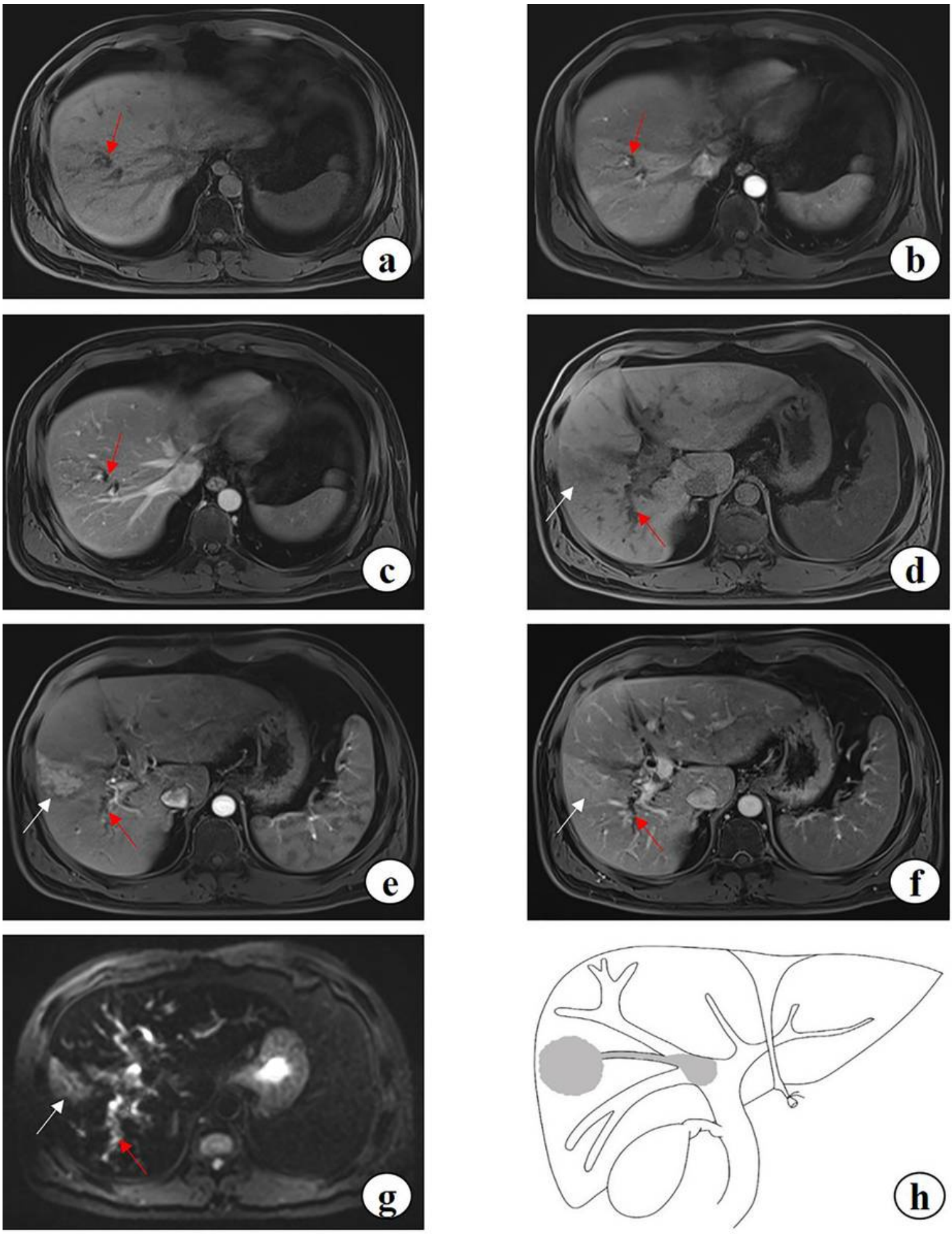
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Figure 3

HCC with B3 BDTT. (a-c) Bile duct dilation in S8 show slightly hypoattenuation (red arrow) on T1W (a), arterial phase(b) and portal venous phase(c). (d-f) HCC lesion locates in S5 and shows hypoattenuation (white arrow) in T1 phase(d), enhancement (white arrow) at arterial phase(e), and hypoattenuation (white arrow) at portal venous phase (f), accompanied by bile duct dilation (red arrow) in right posterior hepatic lobe. (g) HCC lesion (white arrow head) in S5, and bile duct dilation (white arrow) in right posterior hepatic lobe show hyperintense, the bile duct tumor thrombus in right hepatic duct show hypointense on T2W. (h) HCC lesion, BDTT and bile duct dilation.