



Hepatic hemangioma with arteriportal shunt: Prevalence and lesion characteristics based on DSA, CT and MR imaging



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ABSTRACT

Purpose: To evaluate the prevalence and lesion characteristics of hepatic hemangioma (HH) with arteriportal shunt (APS) based on digital subtraction angiography (DSA), computed tomography (CT) and magnetic resonance (MR) imaging.

Method: Ninety-eight consecutive patients with 189 HHs who underwent DSA, CT and/or MR imaging of the liver between January 2014 and December 2017 were included. The diagnosis of APS was established by DSA. APS was categorized as peripheral and central shunt based on imaging findings. The incidence and appearance of APS on DSA were compared with those on CT/MR images. Eleven possible lesion characteristics associated with APS were compared between HHs with and those without APS. Multiple logistic regression modeling was used to identify the independent lesion characteristics associated with APS.

Results: APS was diagnosed in 103 (103/189, 54.5%) HHs on DSA, of which 96 lesions appeared as peripheral APS and 7 appeared as central. In contrast, APS was detected only in 57 HHs (57/103, 55.34%) on CT/MR imaging, of which 50 (50/96, 52.08%) appeared as peripheral APS while 7 (7/7, 100%) appeared as central. Lesion size ($P < .001$), enhancement rapidity ($P = .031$), and vascularization degree ($P < .001$) were found to be significant independent imaging characteristics associated with APS.

Conclusions: APS can occur in HH with high frequency. DSA was superior to CT/MR imaging in detection of APS, particularly for the peripheral APS. Lesion size, enhancement rapidity and vascularization degree were associated with the presence of APS.

1. Introduction

Hepatic hemangioma (HH) is the most common benign hepatic tumor. It is frequently found incidentally on noninvasive imaging modalities, including ultrasonography, computed tomography (CT) and magnetic resonance (MR) imaging [1]. Digital subtraction angiography (DSA) was used only during the transcatheter therapy of HH. Although most HHs are asymptomatic and do not require active treatment, transcatheter arterial embolization (TAE) has emerged as an alternative treatment for large hemangiomas that produce symptoms and have risk of complications [2,3]. Through injection of sclerosing or embolic agent into the tumor-feeding artery, TAE can significantly reduce HH lesion size and control the tumor-related symptoms [2,3]. We performed TAE for HH and showed similar results, but we also found that the efficacy of TAE may be impaired in some HHs, particularly in lesion associated

with arteriportal shunt (APS).

APS is a well-recognized atypical adjacent abnormality associated with HH [4]. Anatomically, APS refers to abnormal communications between the hepatic arteries and the portal vein, through which the high-pressure arterial blood can reflux into a low-pressure portal vein branch [5]. This entity is mainly associated with hepatic malignancy but can also be observed in a wide variety of pathologic conditions [5]. In HH, APS was first observed with angiography by Itzchak et al in 1974 [6]. Subsequent studies using CT, MR imaging or DSA have shown that APS was not uncommon in HH and correlated with the speed of intratumoral contrast material enhancement [7–9]. Although the APS seems no longer a challenge to HH diagnosis, with the introduction of TAE in the treatment of HH, it is necessary to better understand the prevalence, performance and lesion characteristics of HH with APS based on imaging modalities. However, little report is available

Abbreviations: APS, arteriportal shunt; HH, hepatic hemangioma; TAE, transcatheter arterial embolization

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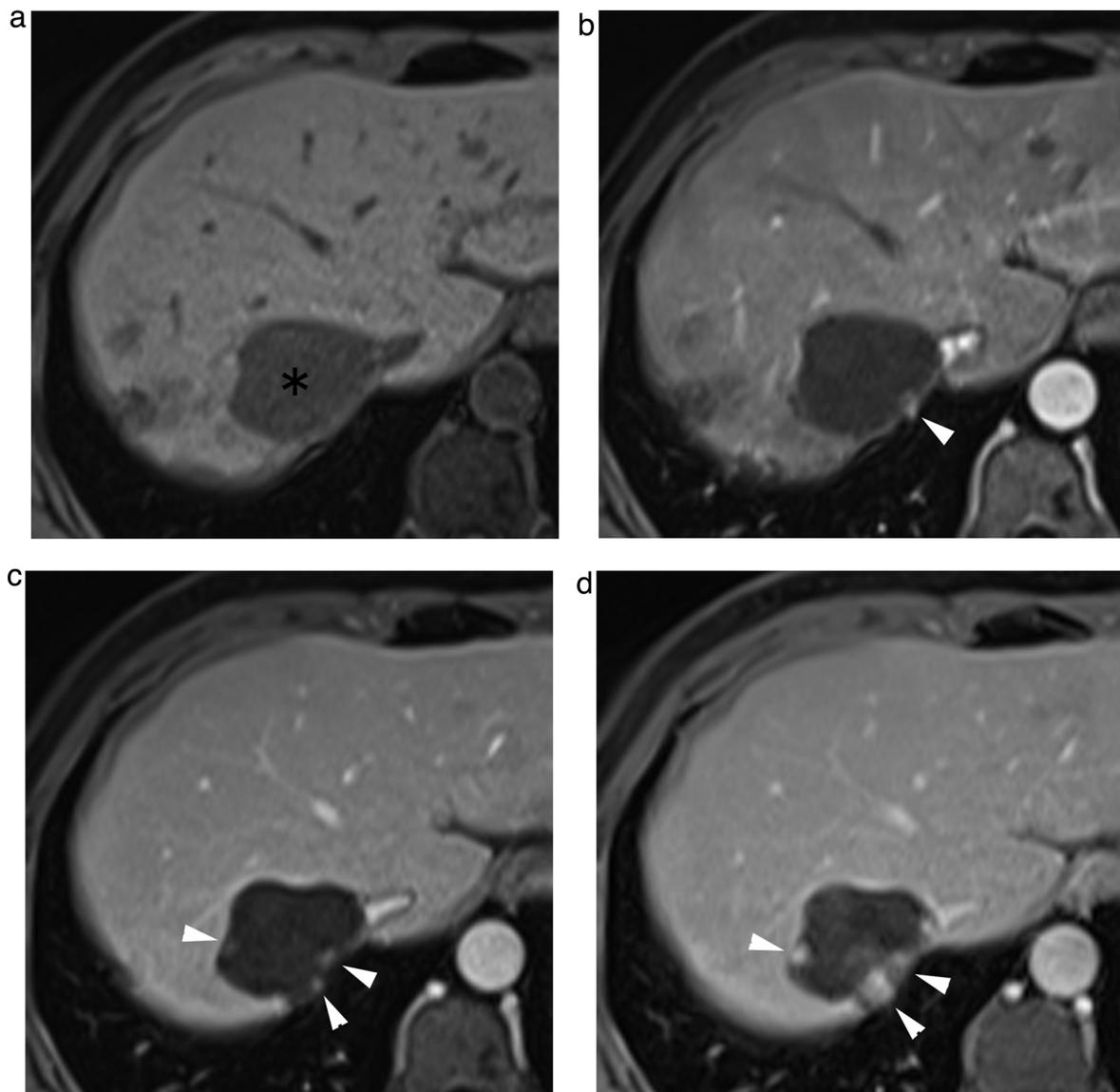


Fig. 1. HH with type I dynamic enhancement appearance. (a) No-enhanced axial MR T1W image shows a low-signal lesion (asterisk) in segment VII. (b,c,d) Multiphase contrast-enhanced T1W images show peripheral tiny enhancing dots in the periphery of the lesion during the hepatic arterial (arrowhead, b) and portal venous phase (arrowhead, c) followed by nodular infilling during the parenchymal phase (arrowhead, d).

regarding the comparison of the HH-related APS between angiography and cross-sectional imaging. Furthermore, the lesion characteristics associated with APS have not yet been adequately studied. Therefore, we performed a single center retrospective study to address these issues.

2. Materials and methods

2.1. Patients

The institutional review board of Wuhan Union Hospital, Huazhong University of Science and Technology (Wuhan, China) approved this study and waived informed consent. We used our radiology department's information system to identify consecutive patients who had a

diagnosis of HH confirmed by dynamic contrast enhanced CT and/or MR imaging and who underwent DSA as a component of transcatheter therapies for HH at our institution from January 1, 2014 and December 31, 2017. Patients were excluded from the study if they had concomitant disorders that can develop APS, such as hepatocellular carcinoma, hepatic cirrhosis, hepatic trauma, hepatic arterio-venous malformations, or had undergone prior liver needle, biopsy, locoregional surgical and interventional therapies. Inclusion criteria for performing TAE in our institution were as follows: (1) HH lesion size ≥ 10 cm; (2) lesion size < 10 cm with progressive enlargement or the tumor-related pain.

All hemangioma lesions of the included patients were reviewed by two radiologists (Yong Wang and Bin Liang, with 12 and 11 years of experience, respectively) in consensus. The imaging data included DSA,

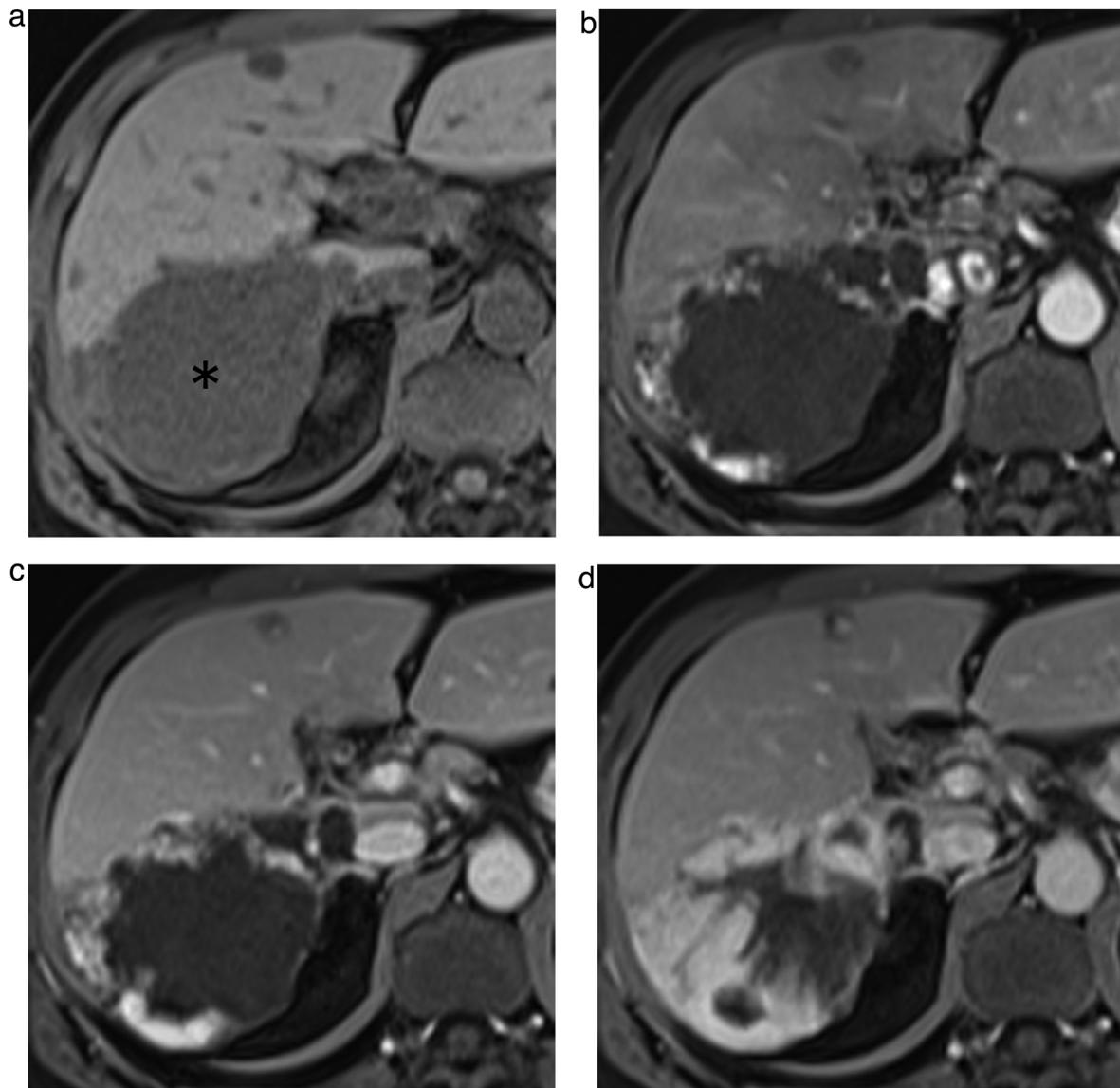


Fig. 2. HH with type II dynamic enhancement appearance. (a) No-enhanced axial MR T1W image shows a low-signal lesion (asterisk) in segment VI and VII. (b,c,d) Multiphasic contrast-enhanced T1W images show typical appearance of enhancement with initial peripheral nodular high signal vessel signal followed by progressive infilling of the lesion.

dynamic contrast-enhanced CT and MR images.

2.2. Diagnosis of HH

The diagnostic criteria of HH were based on characteristic imaging findings. At multiphasic contrast-enhanced CT, a lesion was considered to be a hemangioma if it had the following features: (a) tiny enhancing dots in the hepatic arterial and portal venous phase; (b) early peripheral nodular discontinuous contrast enhancement, isoattenuation relative to the aorta during the hepatic arterial phase and centripetal fill-in enhancement during the portal venous phase; or (c) early homogeneous enhancement during the hepatic arterial phase, persistent enhancement during the portal venous phase, and isoattenuation relative to enhancing intrahepatic vessels [10].

At MR imaging, a hemangioma was diagnosed if a lesion showed

high signal intensity on T2-weighted images and dynamic enhancement pattern on gadolinium-enhanced T1-weighted images similar to that on CT images (Figs. 1–3).

2.3. CT and MR imaging

Abdominal CT examinations were performed by using a 128-section multidetector CT scanner (Somatom Definition AS, Siemens Healthcare GmbH, Erlangen, Germany) and a dual source 64-cross-sectional CT scanner (Somatom Definition, Siemens Healthcare GmbH, Erlangen, Germany). All patients received an unenhanced and dynamic contrast-enhanced CT images. Hepatic arterial phase, portal venous phase and parenchymal phase scanning was performed 25–30 seconds, 50–70 seconds and 3 min, respectively, after commencing the injection of contrast medium after a intravenous bolus of 100-mL nonionic contrast

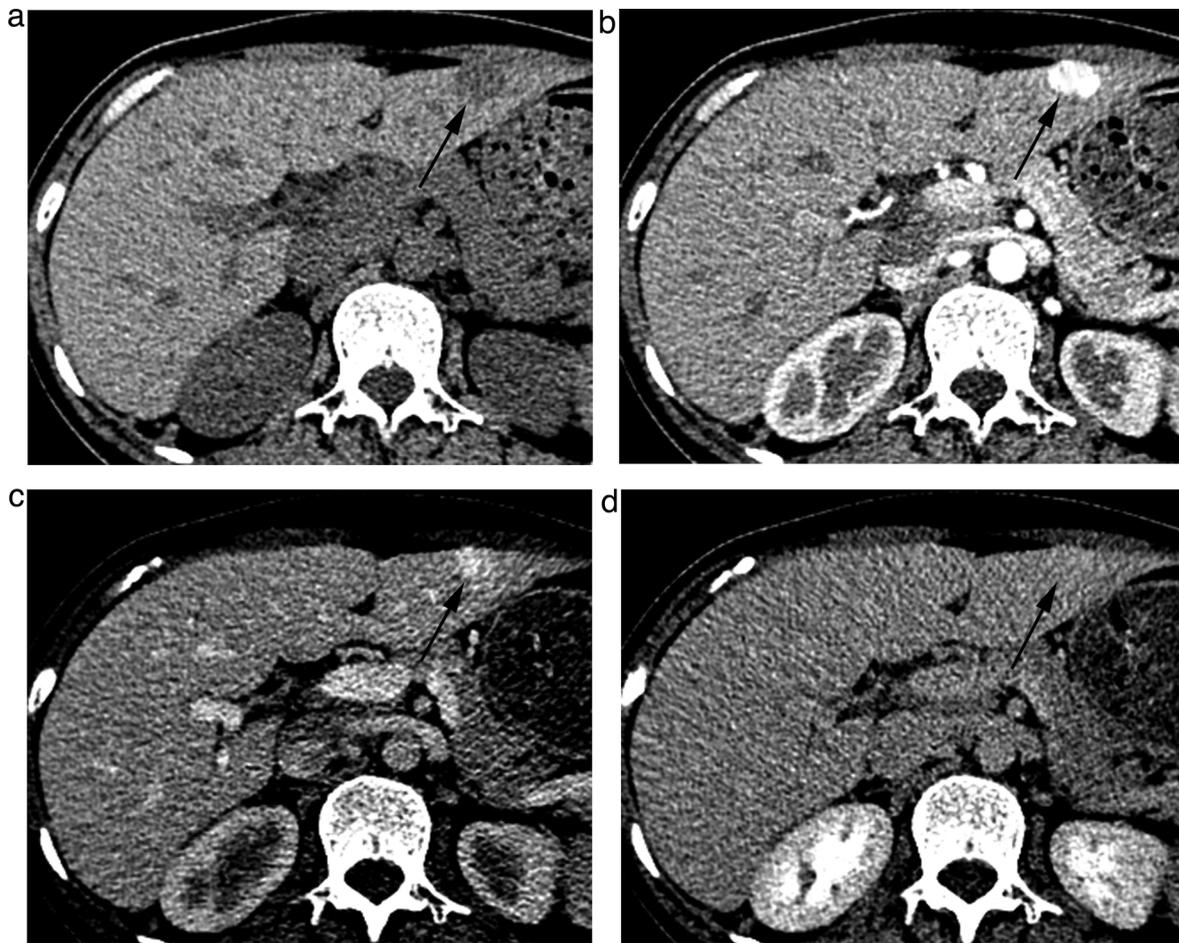


Fig. 3. HH with type III dynamic enhancement appearance. (a) No-enhanced axial CT image shows a low-density lesion (arrow) in segment III. (b,c,d) Multiphasic contrast-enhanced CT images show early homogeneous enhancement of the lesion during the hepatic arterial phase (arrow, b), persistent enhancement during the portal venous phase (arrow, c), and isoattenuation relative to enhancing liver parenchyma during the parenchymal phase (arrow, d).

agent (Iohexol Injection, 300 mgI/ml, Beilu pharmaceuticals, Beijing, China) at a rate of 3 ml per second with a power injector (Stellant, Medrad Pittsburgh, PA, USA). For some atypical HH lesions, delayed phase scan 5–8 minutes post injection was optional.

Abdominal MR examinations were performed with a 1.5-T unit (Magnetom Avanto, Siemens Healthcare GmbH, Erlangen, Germany) using a phased-array torso coil. The protocol included an axial T2-weighted fat-suppressed turbo spin-echo sequence with respiratory triggering, nonenhanced T1-weighted fast low-angle shot sequence with out-of-phase and in-phase imaging and dynamic contrast-enhanced T1-weighted sequence with three-dimensional fat suppression during a single breath hold. Intravenous contrast material, gadopentate dimeglumine (Magnevist®; Bayer, Berlin, Germany), was administered by a power injector (Spectris Solaris® EP, Medrad Pittsburgh, PA, USA) as a bonus of 0.1 mmol per kilogram of body weight at 2 ml/sec and a subsequent 20-ml normal saline flush in all patients. The arterial, portal venous, equilibrium, and if necessary, delayed phases began at 25 s, 50–60 seconds, 3 min, and 5–8 minutes after injection, respectively.

2.4. Criteria for diagnosing APS on CT and MR images

The following imaging findings comprised the criteria for the diagnosis of APS on CT and MR imaging: (a) an area (often peripherally located or subcapsular) of early nodular or wedge-shaped or irregularly shaped parenchymal enhancement with straight margins adjacent to the hemangioma, with or without internal branching structures, during the hepatic arterial phase (Figs. 4a, b, 5 a, b) [7,8,10]; (b) segmental or lobar parenchymal enhancement adjacent the hemangioma, with or without early opacification of large portal branches, during the hepatic arterial phase (Figs. 6a and b). The former APS was defined as peripheral APS, while the latter was defined as central APS.

2.5. DSA and interventional therapy of HH

DSA procedure was performed using Siemens Artis Zee DSA system (Artis Zee, Siemens, Erlangen, Germany). Patients underwent selective or superselective angiography of the celiac artery, common hepatic artery, proper hepatic artery, and right or left hepatic artery with 5-F visceral catheter and 2.7-F coaxial microcatheter system. The contrast medium used included iohexol 350 mg I/ml (Omnipaque; Shanghai Nycomed Pharmaceutical, Shanghai, China) and iopromide 370 mg I/

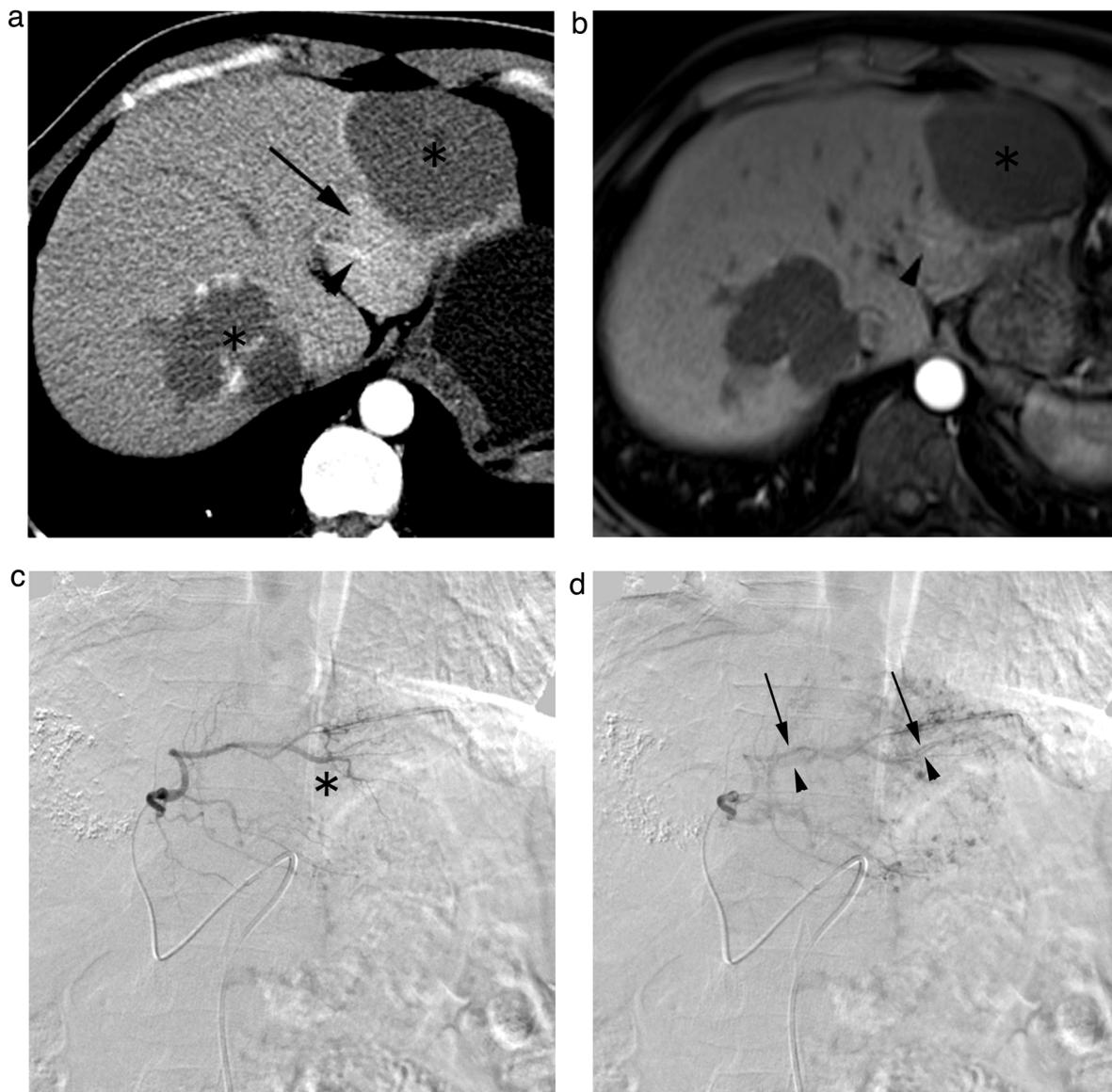


Fig. 4. Peripheral APS associated with a HH. (a,b) Contrast-enhanced axial CT (a) and MR T1W image (b) obtained during hepatic arterial phase show two HH lesions (asterisk) in segment II and VII. Note that the early irregularly shaped parenchymal enhancement (arrow) adjacent to the HH in segment II with internal branching structure (arrowhead), which is consistent with the peripheral APS. (c) Selective left hepatic arteriogram obtained during the early arterial phase confirms the hypervascular tumor (asterisk). (d) Arteriogram obtained during the late arterial phase shows early filling of small portal branches (arrowhead), parallel to the accompanying hepatic artery (arrow), which is termed as parallel track sign.

ml (Ultravist; Schering AG, Berlin, Germany). The flow rate was 5 ml/sec and a total dose of 15 ml contrast was injected for the common hepatic artery DSA, 4 ml/sec flow and total dose of 10 ml for the proper hepatic artery, and 3 ml/sec flow rate and total dose of 8 ml for the right or left hepatic artery. Subtraction images of the early arterial phase, late arterial phase, capillary phase, portal venous phase, and post-portal venous phase were acquired with pulse imaging mode (3–6 frames/sec), injection delay, and density compensation.

After angiography, patients underwent chemoembolization with infusion of the mixture of bleomycin (Tianjin Taihe Pharmaceuticals, Tianjin, China) and Lipiodol followed by embolization with gelatin sponge. In the case of significant APS, embolization of the shunt with large-size embolic particles was performed prior to the bleomycin/Lipiodol mixture administration.

Lipiodol mixture administration.

2.6. Criteria for diagnosing APS on DSA images

On DSA, the diagnostic criteria of APS were: (a) early filling of small branches of the portal vein, parallel to the accompanying hepatic artery - parallel track sign (Fig. 4d); (b) early appearance of a tubular vascular structure suggestive of a branch of the portal vein during the arterial phase and subsequent focal parenchymal contrast enhancement around the vascular structure (Fig. 7d); (c) early opacification of large portal veins and/or their inflow branches, particularly seen in patients with prominent hepatofugal blood flow draining into portal veins (Fig. 6d) (9). These features of APS are usually appreciated in the late arterial

Table 1
Definitions of the eleven variables.

Terms	Definition
Size of lesion	The largest diameter of lesion on the portal venous phase of CT/MR images: Small: ≤ 3 cm; Medium: 3–5 cm; Large: 5–10 cm; Huge/markedly enlarged: ≥ 10 cm
Location of lesion	The exact hepatic lobe or ≥ 2 lobes (CT/MR).
Component of lesion	Homogeneous or heterogenous lesion according to the CT/MR appearance.
Dynamic enhancement pattern	The enhancement appearance on CT/MR (10) <ul style="list-style-type: none"> • Type I: tiny enhancing dots in the hepatic arterial and portal venous phase • Type II: early peripheral nodular discontinuous contrast enhancement during the hepatic arterial phase and centripetal filling-in during the portal venous phase • Type III: early homogeneous enhancement during the hepatic arterial phase and persistent enhancement during the portal venous phase
Rapidity of enhancement	The extent of intratumoral enhancement CT/MR (7) <ul style="list-style-type: none"> • Rapid: more than 50% of the lesion • Slow: equal to or less than 50% of the lesion
Portal vein involvement	Compression, encasement or invasion of a large intrahepatic portal vein (proximal to the third branch of the portal vein) by hemangioma (CT/MR)
Hepatic vein involvement	Compression, encasement or invasion of a large hepatic vein by hemangioma (CT/MR)
Gallbladder fossa involvement	Compression, encasement or invasion of the gallbladder fossa by hemangioma (CT/MR)
Porta hepatis involvement	Compression, encasement or invasion of the porta hepatis by hemangioma (CT/MR)
Hepatic capsular involvement	Direct contact of the hepatic capsule by hemangioma (CT/MR images)
Vascularization degree	The angiographic appearance of the lesion on DSA [11] <ul style="list-style-type: none"> • Hypervascular: lesion was markedly enhanced at both celiac and selective angiography • Moderate vascularity: lesion was non-enhanced or mildly enhanced at celiac angiography and moderately or markedly enhanced at selective angiography • Hypovascular: lesion was not detected at both celiac and selective angiography

phase, despite earlier display in higher shunt flows. The first two findings were defined as peripheral APS, while the third one was defined as central APS.

2.7. Statistical analysis

Lesion characteristics of HH with APS were evaluated by using a logistic regression in uni- and multivariate analyses. Eleven variables of lesion characteristics defined in Table 1 were compared between lesions with and those without APS by using univariable statistics: including the Pearson χ^2 test, the Wilcoxon rank sum test and the Fisher exact test. Candidate variables with *P* values of 0.1 or less at univariate analyses were included in multivariable logistic regression analysis to examine the significance of each lesion characteristics for others. All statistical analyses were performed by using software (SPSS, version 24.0; SPSS Inc., Chicago, Ill). A *P* value less than 0.05 was considered to indicate statistically significant differences.

3. Results

3.1. Patient and lesion characteristics

A total of 98 patients with hemangioma were included in this study. Twenty-one patients were included in whom at least one HH lesion was equal to or larger than 10 cm in size, and 77 patients were included due to progressive enlargement of hemangioma or the tumor-related pain. This cohort consisted of 65 females and 33 males. The mean age was 47.89 ± 9.21 year (mean \pm standard deviation). DSA examination was performed in all the patients. In contrast, pre-procedure CT was done in 72 patients; MR imaging was done in 22; and both CT and MR imaging were done in 4.

One hundred and eighty-nine HH lesions were detected in the 98 patients. Fifty-one patients had single lesion, while 47 patients had multiple lesions (range, 2–7 lesions). The lesion size measured 5.36 ± 4.24 cm (range, 1.0–18.3 cm). One hundred and sixty-eight HH

lesions developed in a single lobe, while 21 lesions grew across bi- or multi-lobes. The other characteristics of HH and adjacent abnormality are listed in Table 2.

3.2. Incidence of APS in HH based on DSA, CT and/or MR imaging

Based on the diagnostic criteria with DSA, APS was identified in 103 HH lesions, with a diagnosis rate of 54.5% (103/189 lesions). Central APS was diagnosed in 7 lesions, accounting for 6.8% of APS; while peripheral APS was diagnosed in 96 lesions, accounting for 93.2%. Of the 96 peripheral APS, 43 appeared as early filling of small branches of the portal vein and 53 as early filling of tubular vascular structure and subsequent focal parenchymal contrast enhancement around the vascular structure.

On CT/MR images, APS was detected only in 57 HHs, with a detection rate of 30.2% (57/189) of the whole lesions. Based on APS diagnosis established by DSA, CT/MR imaging yielded a true-positive rate of 55.3% (57/103 lesions). Central APS was detected in 7 HH lesions, with a true-positive rate of 100% (7/7 lesions) (Fig. 6); while peripheral APS was observed only in 50 lesions, with a true-positive rate of 52.08% (50/96 lesions) (Figs. 4, 5 and 7). It was noted that of the 50 lesions with peripheral APS, 5 (10%) showed internal branching structures which represented early opacification of portal branches during the hepatic arterial phase in the early peritumoral enhancement areas (Figs. 4a and 4b). On the other hand, all the 7 lesions with central APS showed early opacification of large portal branches during the hepatic arterial phase (Figs. 6a and 6b).

3.3. Lesion characteristics of HH with APS

Univariate analysis revealed that all the 11 parameters were significant factors in terms of presence of APS in HHs (Table 2). At multivariate analysis however, size of lesion (*P* < .001; odds ratio, 3.391; 95% confidence interval [CI]: 2.174, 5.290), rapidity of enhancement (*P* = .031; odds ratio, 4.063; 95% CI: 1.139, 14.491) and

Table 2
Univariate analysis of lesion characteristics associated with APS.

Variable	HH with APS (n = 103)	HH without APS (n = 86)	P Value
Largest size of lesion			< .001 ^b
≤ 3 cm	21(20.4)	45(52.3)	
3–5 cm	19(18.4)	21(24.4)	
5–10 cm	47(45.6)	15(17.4)	
≥ 10 cm	16(15.5)	5(5.8)	
Location of lesion			.007 ^c
Caudate lobe	1(1.0)	1(1.2)	
Left lateral lobe	30(29.1)	26(30.2)	
Left medial lobe	7(6.8)	9(10.5)	
Right anterior lobe	13(12.6)	17(19.8)	
Right posterior lobe	33(32.0)	31(36.0)	
≥ two lobes	19(18.4)	2(2.30)	
Component of lesion			.004 ^a
Homogeneous	72(69.9)	75(87.2)	
Heterogeneous	31(30.1)	11(12.8)	
Dynamic enhancement pattern			< .001 ^b
Type I	1(1.0)	7(8.1)	
Type II	91(88.3)	78(90.7)	
Type III	11(10.7)	1(1.2)	
Rapidity of enhancement			.031 ^a
Slow	85(82.5)	80(93.0)	
Rapid	18(17.5)	6(7.0)	
Vascularization degree			< .001 ^b
Hypovascular	0(0)	19(22.1)	
Moderate vascularity	68(66.0)	61(70.9)	
Hypervascular	35(34.0)	6(7.0)	
Portal vein involvement			< .001 ^a
Absent	72(69.9)	79(91.9)	
Present	31(30.1)	7(8.1)	
Hepatic vein involvement			.002 ^a
Absent	75(72.8)	78(90.7)	
Present	28(27.2)	8(9.3)	
Gallbladder fossa involvement			.002 ^a
Absent	87(84.5)	84(97.7)	
Present	16(15.5)	2(2.3)	
Porta hepatis involvement			.001 ^a
Absent	82(79.6)	82(95.3)	
Present	21(20.4)	4(4.7)	
Hepatic capsular involvement			.009 ^a
Absent	8(7.8)	18(20.9)	
Present	95(92.2)	68(79.1)	

^a Calculated with the Pearson Chi-square test.
^b Calculated with the two independent sample Wilcoxon rank sum test.
^c Calculated with the Fisher exact test.

vascularization degree (P < .001; odds ratio, 9.074; 95% CI: 3.568, 23.076) showed significance after adjustment for other variables (Table 3). APS was prone to occur in large (5–10 cm, > 10 cm in size), rapidly enhanced and hypervascular HH lesions.

4. Discussion

Although the APS in HH has been previously investigated on different imaging modalities [7–9], to our knowledge this is the first study to report the prevalence and risk factors of APS on DSA, CT and MR imaging in a large cohort. Our results demonstrated that APS develops in HHs with high frequency, and DSA was superior to CT/MR imaging in the detection of APS, particularly for peripheral APS. Furthermore, the presence of the APS bore statistically significant relationship to

lesion size, enhancement rapidity and vascularization degree.

The prevalence of APS associated with HH has been investigated in previous studies. Kim et al. [7] determined the incidence of APS in patients with HH on two-phase CT and found that APS was detected in 25.7% (28/109 lesions) of hemangiomas. Similarly, in another study, Jeong et al. [8] examined HH with dynamic MR imaging and found that 19% (32/167 lesions) of HHs had APS. The authors concluded that APS was not uncommon in HH at cross-sectional images. In contrast, Ouyang et al. [9] investigated frequency of APS in 30 patients with HH by using DSA alone and found that the incidence rate of APS was as high as 73.3% (22/30 patients). However, the study by Ouyang et al. [9] analyzed the presence of APS only based on patients, not based on lesions. In this study, we examined HH based on lesions with multi-imaging modalities and showed a 54.5% (103/189 lesions) incidence of APS with DSA, which was higher than that with CT/MR imaging (30.2%, 57/189 lesions). These data suggest that APS occurs in HH and with high frequency, and DSA is superior to CT/MR imaging in the identification of APS. In addition, according to the DSA features described previously [9], we classified the APS into central and peripheral categories and found that the latter was the principal manifestation of APS associated with HH. We believe that the distinguishing of central APS from peripheral APS is of some clinical significance as the central APS may produce clinical problem such as portal hypertension and heart failure and therefore require positive treatment [12]. In addition, although the central APS was embolized with large-size embolic particles prior to the injection of bleomycin/Lipiodol mixture in this study, the effect of central APS on the therapeutic efficacy of chemoembolization for HH awaits the result of further study.

On the basis of the diagnosis of APS with DSA, we found that the true-positive rate of APS with CT/MR imaging was 55.35% (57/103 lesions) and that all APSs missed on the cross-sectional images were peripheral APSs diagnosed on DSA. This is possibly due to differences in characteristics between CT/MR imaging and DSA. CT and MR clearly demonstrate a marked deficit in spatial resolution compared to angiography, leading to limited capability for visualizing small portal branches within the liver. Previous and our study have demonstrated that only a minority of small portal branches can be displayed within early peritumoral enhancement area on cross-sectional imaging [7]. This also further explains why there was a lower detection rate for peripheral APSs which were associated with smaller caliber of portal branches compared to the central APSs which were more readily displayed due to their larger portal vein branches. In addition, CT and MR imaging can be affected by relatively longer period of intravenous contrast material injection and intermittent image acquisition periods, which impairs the optimal opacification of transient peritumoral enhancement and hence hinders the detection of small APS [8]. Nevertheless, concerning this finding, it appears that CT and MR images, which show transient parenchymal enhancement, have limited capacity to demonstrate peripheral APS in HH.

From the multiple logistic regression analysis, we found that APSs are more likely to occur in larger-sized (5–10 cm, > 10 cm in size), rapidly enhancing and hypervascular HH lesions. Previous analyses of lesion characteristics of HH with APS focused on enhancement rapidity and lesion size on dynamic CT and MR images and found that the temporal peritumoral enhancement representing the presence of APS was related to the speed of intratumoral contrast material enhancement but not to the lesion size [7,8]. Although little information is available regarding the effect of lesion vascularization on presence of APS by using DSA, colour Doppler sonography has recently found that HHs with intratumoral blood flow were accompanied with peritumoral



Fig. 5. Peripheral APS associated with a rapidly enhancing HH. (a,b) Contrast-enhanced axial MR T₁W images obtained during hepatic arterial phase shows a rapidly enhancing HH (asterisk, a) in segment V. Note that the early peritumoral enhancement (arrow, a) and opacification of portal vein branches (arrowhead, b) distal to the lesion, which is comparable to the peripheral APS. (c) Selective common hepatic arteriogram obtained during the early arterial phase confirms the hypervascular tumor (asterisk), supplied by branches of the right hepatic artery. (d) Arteriogram obtained during the late arterial phase shows the early opacification of portal vein branches (arrowhead) distal to the lesion, which is consistent with the peripheral APS.

hepatofugal portal flow, which suggested lesion vascularization had a relationship with the APS [13]. Our findings are in agreement with the previous results that APS have a positive correlation with the enhancement rapidity and vascularization degree. Yamashita et al. [14] correlated pathological findings of HH with CT enhancement patterns and found that HHs with slow fill-in had relatively large vascular spaces whereas those with rapid enhancement had small vascular channels and comparatively large interstitium. Taken together, it is reasonable to believe that the APS in HH is probably caused by rapid flow in the smaller intratumoral vascular spaces [5]. In contrast, our results showed a positive relationship of APS to lesion size, which is different

from the aforementioned studies [7,8]. This may be due to the difference in the criteria for the diagnosis of APS. In this study, we used DSA to diagnose APS and showed a diagnosis rate of up to 54.5%; whereas earlier studies detected APS with cross-sectional imaging and showed relatively low detection rates (19%–25.7%). This diagnostic discrepancy is likely to affect the results of APS-related lesion characteristics analysis. Another possible interpretation is that the lesion sizes of our cohort are larger than those of the previous studies.

Our study had several limitations. First, we did not have pathological results to correlate them with imaging findings. Although the typical findings on CT and MR images are sufficient for HH diagnosis and

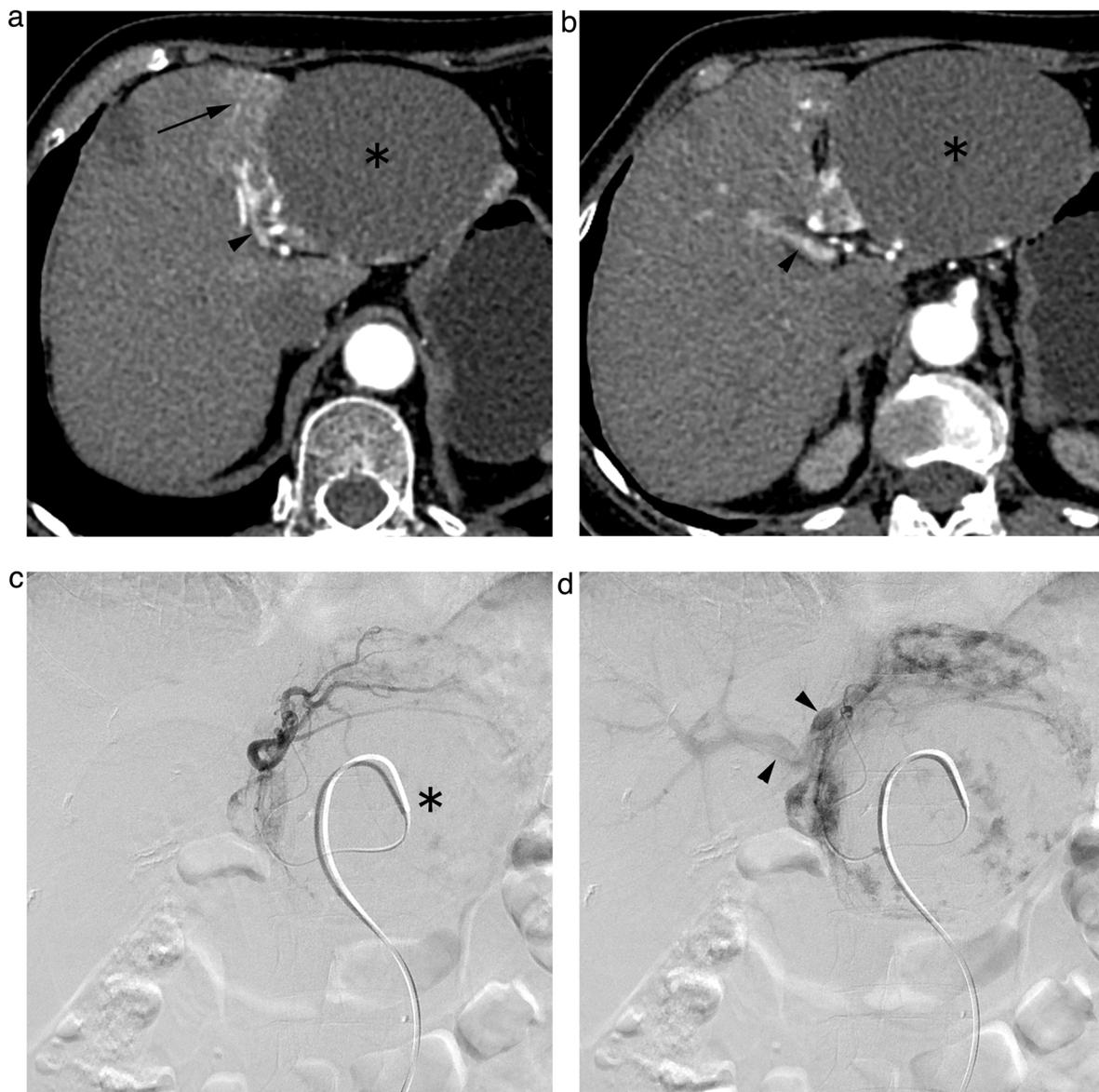


Fig. 6. Central APS associated with a large HH. (a,b) Contrast-enhanced axial CT images obtained during hepatic arterial phase show a large HH (asterisk) in segment II and III. Note that the early peritumoral parenchymal enhancement (arrow, a) and opacification of the left (arrowhead, a) and right portal trunk (arrowhead, b), which is comparable to the central APS. (c) Selective common hepatic arteriogram obtained during the early arterial phase confirms the hypervascular tumor (asterisk), supplied by branches of the left hepatic artery. (d) Arteriogram obtained during the late arterial phase shows the early opacification of the left and right portal trunk (arrowhead), which is consistent with the central APS.

the patients with common entities that can cause APS were excluded from analysis, the presence of early peritumoral parenchyma enhancement in hemangioma still presents a diagnostic challenge due to the association of peritumoral enhancement with a wide variety of pathologic conditions. Second, all 98 patients underwent DSA examination, whereas not all the patients received both CT and MR imaging. This disparity may present a limitation in the comparison of the radiological features on DSA and the cross-sectional images. Third, this is a retrospective study and is inevitably subject to biases affecting such studies.

In conclusion, our results suggest that APS occurred in HH with high

frequency and that DSA had a greater detection rate for the HH-related APS compared to CT and MR imaging. These findings imply that the detection of APS on CT and MR imaging is somewhat limited and that the actual prevalence of APS in HH is much higher than that seen on the cross-sectional images. Besides enhancement rapidity and vascularization degree, we also found a significant relationship of the presence of APS to lesion size. Given the fact that large HHs can produce symptoms and require treatment, special attention should be paid to the APS in the large HHs. Additional study is underway to evaluate the effect of APS on therapeutic efficacy of TAE for HH.

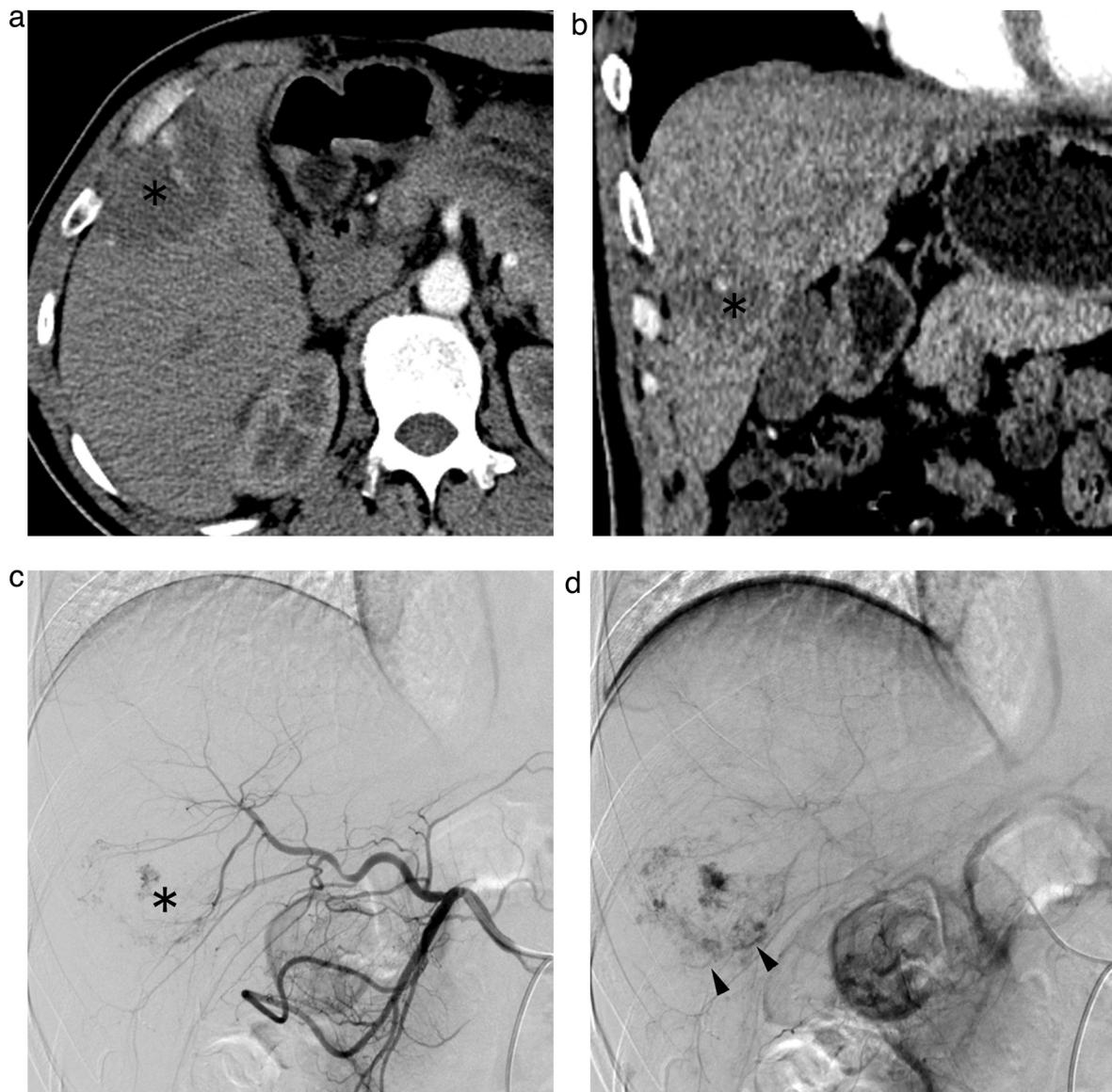


Fig. 7. Peripheral APS associated with a HH. (a,b) Contrast-enhanced axial (a) and coronal (b) CT images obtained during the hepatic arterial phase show a HH lesion (asterisk) in segment V, without any early enhancement of the peritumoral parenchyma. (c) Selective common hepatic arteriogram obtained during the early arterial phase confirms the HH (asterisk) in the right liver lobe. (d) Arteriogram obtained during the late arterial phase shows the early opacification of a tubular vascular structure (arrowhead) around the lesion, which is suggestive of a portal vein branch.

Table 3
Multivariate analysis of lesion characteristics associated with APS.

Risk Factor	b	SE χ^2	Wald	Odds Ratio	95% CI	P Value
Largest size of lesion	1.221	0.227	29	3.391	2.174–5.290	< .001
Rapidity of enhancement	1.402	0.649	4.671	4.063	1.139–14.491	0.031
Vascularization degree	2.205	0.476	21.443	9.074	3.568–23.076	< .001

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Declaration of Competing Interest

None.

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None.

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