Transient hepatic attuenation differences and focal liver lesions:Sump effect due to primary arterial hyperperfusion

Transient Hepatic Attenuation Differences and Focal Liver Lesions: Sump Effect Due to Primary Arterial Hyperperfusion

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Objective: To analyze at computed tomography (CT) examination the "sump effect," a particular type of transient hepatic attenuation differences, related primarily to an increase in arterial flow without any accompanying decrease in portal flow.

Methods: We retrospectively evaluated all biphasic upper abdomen CT examinations (1283 in 807 patients) performed from the year 2003 to the year 2006 and selected and organized those with at least 1 transient hepatic attenuation differences. Of these, we enrolled patients with lobar/multisegmental arterializations surrounding focal lesion(s), without CT portal hypoperfusion signs, in the study group. We assessed histology, number, site, diameter, and volume of causing focal lesion(s); site, extension, and attenuation of arterial area; greater visibility of feeding artery branches ipsilateral to causal focal lesion; and presence of aberrant left hepatic artery. Thirty patients with normal liver represented the control group.

Results: Fifteen of the 99 patients with transient hepatic attenuation differences presented with sump effect. In our series, this phenomenon was always related to hypervascular inflammatory and benign lesion(s) with overall average diameter of 8 ± 4 cm inscribed in arterial area. Attenuation of arterial enhanced areas were significantly higher than the contralateral parenchyma and control patients' parenchyma, with frequent hypertrophy of ipsilateral arterial feeding branches and/or aberrant left hepatic artery visibility.

Conclusions: Siphonage seems to be primary hyperperfusion area determined by arterial bed enlargement, induced by inscribed large hypervascular inflammatory/benign lesion(s).

Key Words: hepatic perfusion abnormalities, hypervascular focal liver lesion(s), liver arterial phenomena, transient hepatic difference(s)

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Transient hepatic attenuation differences (THADs) are areas of parenchymal enhancement, visible only or mainly during the late hepatic artery phase on spiral computed tomography (CT) that are related to the dual hepatic blood supply. Most arterial phenomena connected to focal liver lesions have a sectorial shape and are well known in literature as compensatory reactions to portal hypoperfusion.[1,2]

However, some less frequent perfusion abnormalities seem to be caused by primary arterial flow increment. These particular perfusion abnormalities are called "sump effect or siphoning phenomenon"[3–5] and defined as arterial phenomena involving all or almost all segments of 1 hepatic lobe, associated with hypervascular hepatic lesions. Although rarer than those related to portal hypoperfusion, these primary arterial phenomena are becoming increasingly common, owing to widespread diffusion of dynamic CT, and may represent potential diagnostic pitfalls. Sump effect has been described in case report(s),[3] pictorial essay(s),[5–10] and review(s),[2,4] but to our knowledge, no case series about this arterial phenomenon has been published.

The aim of this retrospective study was therefore to evaluate imaging features at CT examination in a series of patients with siphoning effect.

MATERIALS AND METHODS

Patient Population

We evaluated, on digital archives, images of all biphasic upper abdomen CT examination from February 2003 to February 2006 (1283 examinations of 807 patients) and selected those with at least 1 transient hepatic attenuation differences. This group was composed of 99 of 807 patients (16% overall arterial phenomena incidence), in which also available data by ultrasound (US) examination (written reports and/or images on hard copies or digital archives) were considered.

All examinations were performed because of clinical indications according to standard procedure; all patients gave their written consents after being informed about possible risks of x-rays and contrast medium injection. Ethical committee approval and patient consent for this retrospective study were not required because patient privacy was maintained, and patient care was not impacted.

Imaging

Spiral CT examinations were performed using a Somatom Plus (Siemens, Erlangen, Germany) scanner. Entire liver parenchyma was scanned cephalocaudally within a single breath hold (scanning time, 18–22 seconds): section thickness, from 5-mm pitch 1.5 to 8-mm pitch 1 (reconstruction, 4.5 mm); matrix, 512 × 512; 170 to 220 mAs; and 120 kV. Unenhanced scan was followed by intravenous administration of 1.5 mL/kg body weight of nonionic iodinated contrast material (Ultravist 370; Schering, Berlin, Germany) by automatic injector (Envision CT; MedRad, Pavia, Italy) at the rate of 3 mL/s. Hepatic artery and portal vein phase scans started 30 and 75 seconds, respectively, after the beginning of bolus.

Ultrasound examinations were achieved using Astro MP echograph (Esaote; Ansaldo, Genova, Italy), equipped with wide angle convex 3.5 MHz probe, pulse waves, and power Doppler module.

Image Analysis

All the images of the study group were reviewed and reassessed by 2 radiologists, both experienced in liver imaging for more than 15 years and unaware of the diagnosis; disagreement was resolved by consensus. Transient hepatic

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TABLE 1. Classification of THADS

Type	Focal Lesion	Morphology/Pattern	Pathogenesis
Sectorial	Yes	Wedge- or fan-shaped	Secondary to portal hypoperfusion: benign or malignant focal lesions
	No	Wedge-shaped	Secondary to portal hypoperfusion: portal or hepatic vein thrombosis and arterial-portal shunt
Lobar	Yes	Lobar multisegmental, not sectorial	Primary hyperperfusion: arterial hyperperfusion associated with the presence of a large benign focal liver lesion without signs of portal hypoperfusion
Polymorphous	No	Various shape and size, marginal or central, without a clear straight border sign	Secondary to portal hypoperfusion: parenchymal injuries, anomalous arterial supply or venous drainage, inflammation, and interventional treatment outcome
Diffuse	No	Patchy	Secondary to portal hypoperfusion: right-sided heart failure, Budd-Chiari syndrome
		Central-peripheral	Secondary to portal hypoperfusion: protein vein thrombosis, cirrhosis
		Peribiliary	Secondary to portal hypoperfusion: biliary tree dilation

Sectorial—arterialization area follows the portal dichotomy with triangular shape and at least 1 "straight border" sign (ie, a clear separation line from the normally attenuating parenchyma) due to the strict connection between the portal hypoperfused area and arterial reaction.

Lobar—they involve all or almost all the segments of a hepatic lobe and are usually caused by a primary increase in arterial inflow due to a large benign lesion (sump effect or siphoning phenomenon), they involve all or almost all the segments of one hepatic lobe following arterial distribution and they are usually caused by a primary increase in arterial in-flow due to a large benign lesion (sump effect or siphoning phenomenon) arterial distribution.

Polymorphous—they usually do not follow the portal dichotomy and show various shapes, without a straight border sign, in relation to the cause, such as aberrant blood supply, inflammation, extrinsic compression, or percutaneous treatment outcome.

Diffuse—when a total or subtotal obstruction involving the entire hepatic parenchyma occurs. They show 3 different patterns on the basis of the portal obstruction site: patchy (postsinusoidal blockade with trans-sinusoidal system opening), central peripheral (presinusoidal-intrasinusoidal obstacle with peribiliary shunt opening), or peribiliary (peribiliary shunt obstruction).

attenuation differences were classified (Table 1) as previously published.[1,5,10]

Accordingly, "sump effect or siphoning phenomenon" was defined lobar/multisegmental arterial phenomena surrounding hypervascular lesion(s),[2,4,5,10] without portal hypoperfusion and straight border signs. The latter is a typical feature of sectorial THAD and is defined as a clearly definite separation line of arterial area with respect to uninvolved parenchyma, consequence of blocked flow in dichotomic portal tree.[5,10]

In the present study, only selected cases with sump THAD are analyzed, although all the other transient hepatic attenuation differences types detected in our series were revised and classified too[5,10] but not discussed at this time, as judged not relevant cases.

Arterial phenomena hyperattenuation compared with normoattenuating parenchyma was assessed at CT examinations. Densitometry (Hounsfield Units [HU]) was evaluated by 2 sets of measurements through regions of interest, performed at the same section level in hyperattenuating areas and contralateral nonhyperattenuating parenchyma during hepatic artery phase. The same attenuation measurements in the equivalent sites were obtained at unenhanced scan and during portal vein phase as well. Regions of interest were manually defined in each image as reproducibly as possible (approximately 400 mm^2) and were placed to avoid large blood vessels and borders.

The mean and SD were calculated for each set of measurements and compared with a third set of measurements performed during hepatic artery and portal vein phase on 30 control patients, without past or present clinical/imaging evidence of hepatoportobiliary pathology, which were submitted to CT examinations to study retroperitoneal vessels. This control group represented a reference standard measurement of attenuation and enhancement for comparison purposes. All CT/US examinations were inspected to search for flow blockade due to thrombosis or encasement (so defined when lumen vanish into the mass) in main portal branches, which could cause arterial compensatory reaction involving 1 lobe or multiple segments. Signs of arterial inflow increment and anatomical variants were sought in every CT examination. We recorded whether hepatic artery primary division branch homolateral to sump effect was hypertrophic, so arbitrarily defined when at least 50% more in diameter than the contralateral one. We also investigated the visibility of aberrant left hepatic artery (ALHA), defined as an arterial branch not originating from common hepatic artery (23%–25% of cases)[11] but from a different vessel, mainly from left gastric artery. The maximum diameter of every lesion was

TABLE 2. Details of Patients With THADs and Related Focal Liver Lesions

THAD Type	Patients	Without FL	With FL (Malignant-Benign)
Sectorial	60	15	45* (36–9)
Sump	15	0	15† (0–15)
Polymorphous	14	11	3‡ (1–2)
Diffuse	10	10	0
Total	99	36	63 (37–26)

The table summarizes all THADs found, to clarify relative percentages among various types and causal focal lesion.

*Sixteen hepatocarcinoma, 4 cholangiocarcinoma, 16 metastases (2 hypervascular), 6 hemangioma, 1 focal nodular hyperplasia (FNH), and 2 abscesses. All benign lesions always a diameter less than 25 mm.

†Five hemangioma, 5 FNH, and 5 abscesses—all with diameter equal or more than 25 mm.

‡One hepatocarcinoma and 2 hemangioma with diameter less than 25 mm.

FL indicates focal fesion.

TABLE 3. Details of the Study Group

Patient No.	Age/Sex	Focal Lesion(s)					THAD		Arterial Feeding	
		No. and Segment Site	Diameter, mm	Volume, cm^3	Histology	Diagnosis Confirmation	Involved Segments	Sump/Contralateral Area Attenuation, HU	Primary Artery Branch Hypertrophy	ALHA Visibility
1	74/M	1: II and III	100	522	Abscess	Biopsy/drainage	II–IV and VIII	134/96	Yes	Yes
2	34/F	1: III	35	22	FNH	Biopsy/follow-up	II–IV	150/104	Yes	Yes
3	50/F	1: II and III	90	380	Hemangioma	Follow-up	I–IV	128/90	Yes	Yes
4	27/F	1: III	100	522	FNH	Biopsy	II and III	116/70	Yes	Yes
5	46/M	1: II and III	60	113	Abscess	Surgery/follow-up	II and III	146/92	Yes	Yes
6	36/F	1: II	30	14	FNH	Surgery	II–IV and VIII	112/84	No	No
7	51/M	1: V-VIII	140	1434	Hemangioma	Follow-up	V–VIII	102/84	No	No
8	23/M	1: VII and VIII	80	268	Abscess	Biopsy/drainage	IV, V, VII, and VIII	120/92	Yes	No
9	38/F	3: III, IV, and VIII	30, 25, 30	36	Hemangioma	Follow-up	I–IV and VIII	94/84	No	No
10	37/M	1: II	70	179	Hemangioma	Follow-up	II–IV	98/74	Yes	Yes
11	61/M	2: II and III	25, 45	56	Hemangioma	Follow-up	II–IV	125/92	Yes	Yes
12	28/F	1: III	35	22	FNH	Biopsy/follow-up	I–IV	130/88	Yes	No
13	59/F	1: V-VIII	115	795	Pseudotumor	Biopsy/follow-up	V–VIII	104/90	Yes	No
14	43/M	1: V-VIII	190	3584	Abscess	Biopsy	V–VIII	103/86	No	No
15	34/F	1: IV	70	179	FNH	Follow-up	VI and VII	140/92	No	No

Hemangiomas were confirmed by a 12- or 24-month follow-up with magnetic resonance/US. FNHs were histologically proven (4/5 patients) and/or confirmed by magnetic resonance with gadolinium chelate liver-specific contrast agent (MultiHance [gadobenate dimeglumine]; Bracco, Milan, Italy) administration (2/5 patients). Pseudotumor hepatis was histologically proven; 3 pyogenic abscesses were drained, and tuberculoma received surgical treatment.

F indicates female; M, male.

measured, and volume was calculated (assuming each focal lesion as a sphere). Whenever more than 1 focal lesion was present, the sum of volumes of single focal lesions was considered, and equivalent sphere diameter and overall average diameter were calculated. Correlation between causal lesion dimension (volume [cm^3]) versus sump effect area (number of hepatic segments involved, according to the numbering system of Couinaud) was evaluated. Statistical analysis was performed using the package Medcalc version 9.1, and correlation coefficient (r) and significance level (p) were calculated.

RESULTS

Among the selected 99 patients, we have found the following: 60 (60.60%) sectorial, 15 (15.15%) siphoning, 14 (14.14%) polymorphous, and 10 (10.10%) diffuse THAD(s) (Table 2). Patients with sump effect (7 men and 8 women; age, 23–74 years [average, 42 ± 14 years]) are the study group and presented in 4 of 15 cases with fever, raised inflammation markers, and abdominal pain (Table 3). All patients of study group received at least 1 CT and 1 US, for a total of 40 examinations.

Focal Lesions

Sixty-three (63.6%) of 99 patients with transient hepatic attenuation differences of our series presented at least 1 focal lesion (malignant in 37 cases and benign in 26) (Table 2). Lesions associated with sump effect were always benign (left-sided in 11 patients and right-sided in 4) and inscribed in arterial area (Table 3): 5 patients had hemangioma(s) (Fig. 1), 5 FNH (Fig. 2), and 5 abscess(es) (Fig. 3). Diagnosis confirmation was obtained as reported (Table 3). Thirteen patients of 15 had only 1 focal lesion connected to arterial phenomenon, and 2 of 15 patients showed more than 1 lesion (patients 9 and 11). Single focal lesion diameters varied from 25 to 190 mm (average, 70 ± 44 mm). Associated lesion volume (considering sum of single focal lesion volumes in those patients who had more than one) varied from 14 to 3584 cm^3 (average, 542 ± 925 cm^3). Maximum diameter was 3 cm or greater in patients with 1 focal lesion (13/15); the overall average diameter, calculated on the basis of equivalent sphere diameter for patients 9 and 11, was 8 ± 4.3 cm.

Transient Hepatic Attenuation Difference

Mean parenchymal attenuation was homogeneous at unenhanced scan (45 ± 9 HU). During hepatic artery phase, mean parenchymal attenuation was 120 ± 18 HU in siphoning areas, 88 ± 8 HU in contralateral nonhyperattenuating areas, and 90 ± 7 HU in control patients. During portal vein phase, attenuation of THAD area equaled again to that of uninvolved liver parenchyma (127 ± 12 HU), except in 2 cases of great hemangiomas (patients 3 and 7), in which portal enhancement of arterialized area remained slightly higher than that in contralateral nonhyperattenuating parenchyma (Fig. 1). Arterialization areas were always multisegmental (mainly 3 or more segments) and homolateral to the lesion(s) (Table 3), with undefined but appreciable border. No correlation between causal lesion volume and number of hepatic segments involved by sump effect was found ($r = 0.0380$; $P = 0.8931$). At CT examinations, 10 (66.6%)

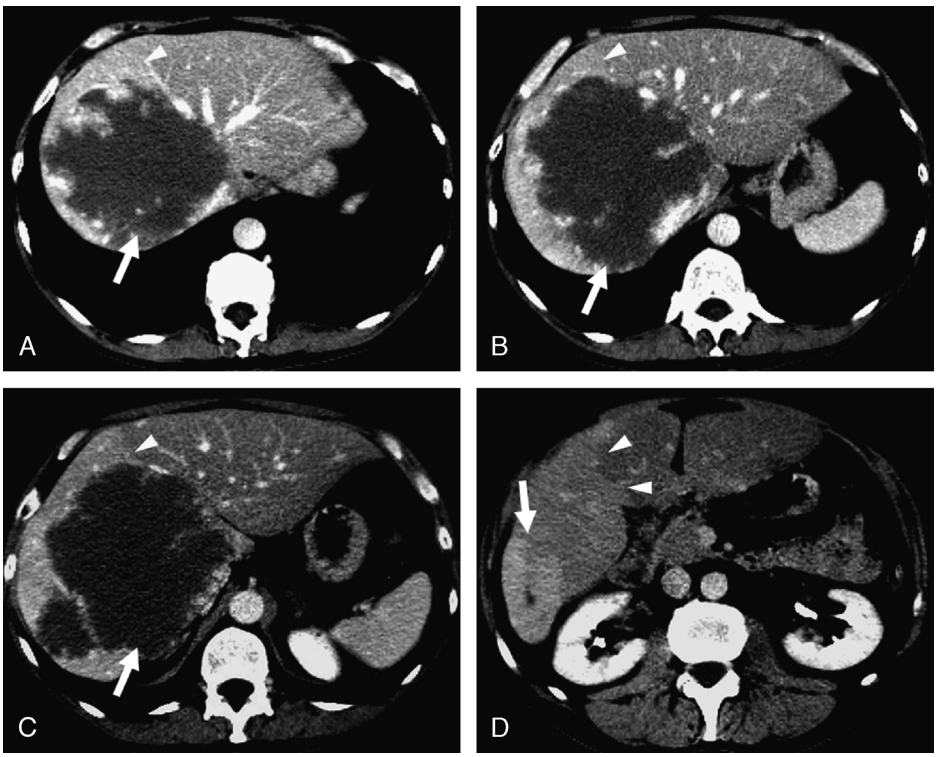


FIGURE 1. A 51-year-old man (number 7 of our series) with siphoning transient hepatic attenuation differences in the right liver lobe caused by large hemangioma. A–C, Artery phase CT scans show large hypoattenuating hemangioma (arrows) in segments VI, VII, and VIII sited within arterial phenomenon involving the entire right lobe (arrowheads). D, Portal vein phase CT scan shows, in a caudal scan, hyperattenuating hemangioma (arrow). Associated arterial phenomenon is still visible (arrowhead), maybe owing to long-duration capability of hemangioma to draw blood flow (slow fill-in phenomenon) or to compression on medial hepatic vein.

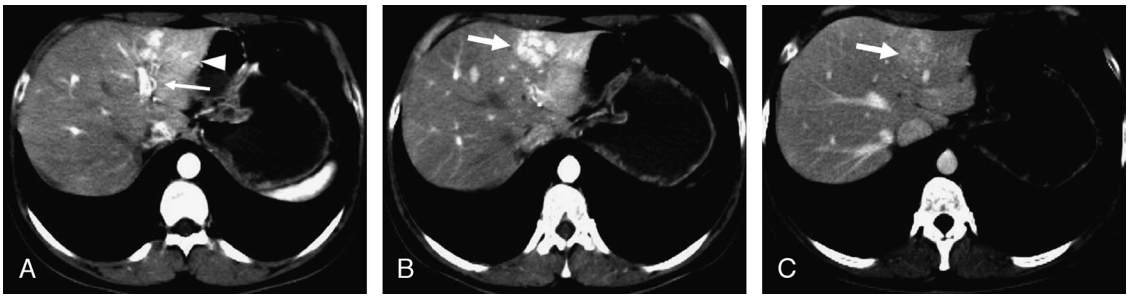


FIGURE 2. A 34-year-old woman (number 2 of our series) with sump effect transient hepatic attenuation differences in the left hepatic lobe determined by FNH. A–B, Artery phase CT scans show rapid enhancement of focal lesion (arrow) and concomitant large arterial phenomenon involving segments II and III (arrowhead). Note visibility of ALHA (thin arrow). C, Portal vein phase CT scan shows slight hyperattenuation of FNH (arrow) and no portal enhancement of surrounding parenchyma.

of 15 patients showed a primary division branch, homolateral to focal lesion, which was more evident than the contralateral branch (Fig. 3). Aberrant left hepatic artery was detected in 7 patients (7/15 [46.4%]), all with associated lesion in the left hepatic lobe (7/11 [63.6%]) (Fig. 2). None of the patients with sump effect showed any evidence of main portal branch thrombosis or encasement at CT/US examinations.

DISCUSSION

Hepatic perfusion disorders connected to focal liver lesions[2–5,9,10] can be differentiated as those caused by portal flow decrease and those exclusively due to primary arterial flow increase.

Arterial phenomena secondary to portal hypoperfusion are well known and usually have sectorial morphology with at least a straight border,[1,2,4,5,10] occurring as compensatory reactions to portal flow decrease, through arterial vasodilation induced mainly by adenosine.[12] Such diminish is due to portal branch encasement, by thrombosis resulting in a portal branch blockade or by flow diversion caused by an arterioportal shunt.[1,2,4,5,10]

Sump effect arterialization conversely is not recognized as well as sectorial THADs. It is defined "primary"—as caused by arterial supply increase not secondary to portal flow decrease and associated with hypervascular lesions.[2–5,10] In accordance with the indexed literature, in our series, sump effects were connected to hypervascular nodules without any sign of portal hypoperfusion (Fig. 4).

In the presented cases, causal lesions were always inscribed within arterial siphoning areas and large (overall average diameter, 8 ± 4.3 cm). However, we did not find a correlation between focal lesion volume and number of hepatic arterial enhanced segments: usually involved segments are I/II to IV or V to VII with or without segment VIII. Such multisegmental/lobar shape and the constant absence of the straight border sign support the hypothesis that causal lesion acts at the level of hepatic artery primary division branch and then with a connection to arterial vessels rather than to portal dichotomy.

Although siphonage and steal (this second term is sometimes associated with siphonage) have been shown,[3,8] or reported to be in association also with malignant hepatic nodules,[9] in our series, it was connected only with benign lesions without any evidence of main portal branch thrombosis or encasement (Figs. 1–3). In fact, in our patients, malignant lesions, even if hypervascular nodules, resulted related with sectorial THAD in 36 out of 37 patients (Table 2). The explanation of the exclusive association of sump effect with benign focal lesion(s) could be that even if a great hypervascular hepatic malignancy can induce arterial flow increase, tumor large enough to provoke parenchymal hyperperfusion determines in all probability a portal involvement too, with consequent hypoperfusion and arterial reaction. This can happen in portal thrombosis due to hepatocellular carcinoma or encasement by metastases, in particular if malignant lesions are situated near the hepatic hilum.[10] Then, in our opinion, siphonage-steal associated with malignant nodules previously

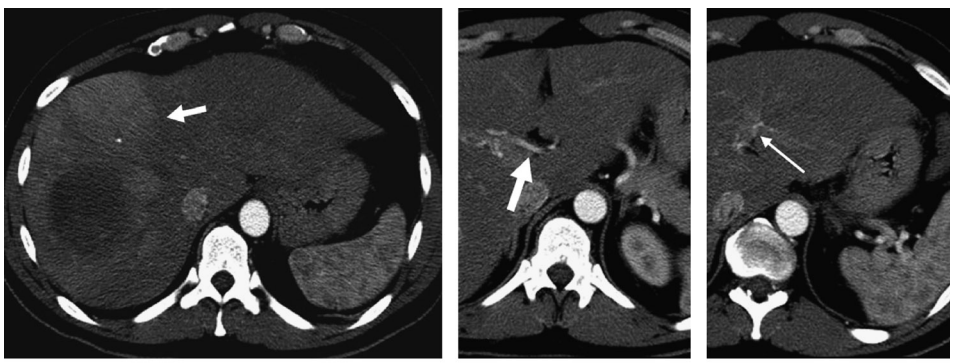
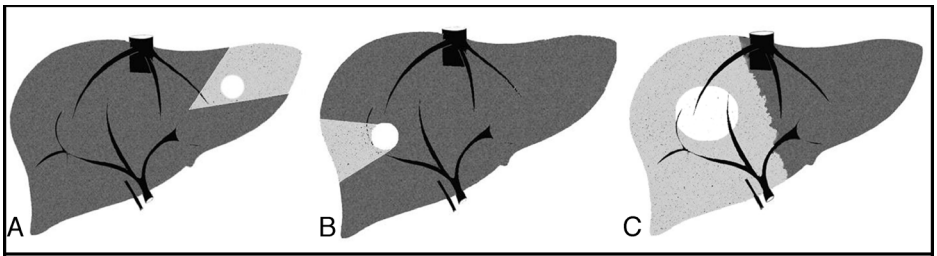


FIGURE 3. A 23-year-old man (number 8 of our series) with sump effect transient hepatic attenuation differences in the right liver lobe caused by pyogenic abscess. A, Artery phase CT scan reveals lobar transient parenchymal enhancement surrounding abscess endowed (arrow). B, Artery phase shows additional right primary artery branch (arrow) more conspicuous than the contralateral one (thin arrow).



THAD	SECTORIAL (A, B)	SUMP (C)
morphology	wedge (A) or fan (B) following portal dichotomy	lobar/multisegmental related to hepatic artery subdivision
straight border sign	yes	not evident
portal flow	reduced or blocked	not demonstrably modified
mediators	adenosine, vasopressin etc.	not identified
related focal lesion	prevalently malignant but also benign	benign
site focal lesion	inscribed in wedge, at the apex in fan	inscribed in siphoning area

FIGURE 4. Sectorial versus sump THADs confrontation plan.

quoted could be considered at least partially secondary to portal hypoperfusion, rather than true exclusively primary.

Attenuation measurements lead us to consider sump effects as real hyperenhancing areas (120 ± 18 vs 88 to 90 HU of reference parenchyma). No differences in parenchymal attenuation mean values were found at unenhanced scan, and therefore, hyperattenuation during hepatic artery phase could not be attributed to precontrast hyperattenuation. Moreover, at portal vein phase, THAD area attenuation equaled again to that of uninvolved liver parenchyma, except in 2 cases of large hemangioma (Fig. 1). The slight portal hyperattenuation of the latter cases is probably due to the ability of hemangioma to draw blood flow up to equilibrium phase (slow fill-in phenomenon).

In case of inflammatory events, hyperemia can be caused by bradykinin/histamine release; on the contrary, to our knowledge, in case of sump effect related to benign lesions, mediators, if any, have not been clearly identified.[5,10,13] So siphonage may be mainly due to arterial flow increment caused by vascular bed enlargement, as suggested by hypertrophy of hepatic artery primary division branch homolateral to the lesion (66.6%) (Fig. 3) and by frequent detection of ALHA (Fig. 2), in our series, more prevalent (46.4%) than that described in the literature (23%–25%)[11] in particular if only patients with lesion in the left liver lobe were considered (63.6%). Aberrant left hepatic artery could be supposed to sustain primary arterial supply increase to the left hepatic lobe, (in particular, segments II/III), especially if a large homolateral hypervascular lesion is present.[5]

As a consequence, in our experience, sump effect hyperattenuation area seems to be determined by an increment of arterial blood, and then due to an increased early inflow rich of undiluted contrast agent.

The sump effect has been quoted in the international literature in association with the steal phenomenon.[2,3,8] This term, sometimes misinterpreted,[13] was first used to describe a particular type of siphonage originating at the hepatic artery primary division branch. In this case, the ipsilobar contralateral segment to that containing the hypervascular tumor may receive less contrast agent and consequently appear less hyperattenuating than the contralateral lobe that does not contain the tumor.[3] In our series, there was no evidence of steal phenomenon, as intended above.

It is important to recognize the limitations of our study. Although our series is the largest reported on the sump effect up to now, nevertheless, our number of patients is relatively small, therefore requiring a retrospective analysis. Moreover, we cannot know the incidence of the sump effect. When taking into consideration the same benign focal lesion, with identical volume and intraparenchymal site, we cannot ascertain whether and how many times it determines a primary arterial supply increase and thus the sump effect. Future prospective studies on a larger series would be helpful in determining the siphoning phenomenon incidence and in confirming its features.

In conclusion, in our series, siphonage seems to be a real primary arterial hyperperfusion area with undefined borders, determined by the enlargement of artery vascular bed. It is induced by large benign hypervascular or inflammatory focal lesion(s) inscribed in arterial area without any sign of main portal branch involvement.

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