



ICONOGRAPHIC REVIEW / *Gastrointestinal imaging*

## Hepatic haemangioma: Common and uncommon imaging features

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### KEYWORDS

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CT-scan;  
Atypia

**Abstract** The haemangioma, the most common non-cystic hepatic lesion, most often discovered by chance, may in certain situations raise diagnostic problems in imaging. In this article, the authors first demonstrate that the radiological appearance of the hepatic haemangioma, in its typical form, is closely related to three known histological sub-types. They then show that certain atypical features should be known in order to establish a diagnosis. They also observe the potential interactions between the haemangioma, an active vascular lesion, and the adjacent hepatic parenchyma. Finally, they discuss the specific paediatric features of hepatic haemangiomas and illustrate the case of a hepatic angiosarcoma.

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Detected by chance in most cases, the hepatic haemangioma is the most common non-cystic hepatic lesion. The incidence may reach 20% according to several autopsy series [1].

In its typical form, the haemangioma is well known and does not raise diagnostic problems in imaging. However, certain “variants” and “atypias” may complicate the diagnosis.

The hepatic haemangioma is a benign vascular lesion. In the vast majority of cases, it is non-evolving and does not require treatment or monitoring.

Histologically, it is a mesenchymal lesion consisting of blood-filled vascular cavities of different size, surrounded by a simple layer of flat endothelial cells, supported by a fibrous connective tissue [2].

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The purpose of this iconographic review is to call to mind the classic radiological aspects of the hepatic haemangioma and the different atypical forms that this lesion may have. The potential interactions between the haemangioma and the adjacent hepatic parenchyma are also discussed. Finally, the specific paediatric features of hepatic haemangiomas are presented, illustrated by the case of a hepatic angiosarcoma.

## Typical aspects

In its typical form, three histological sub-types have been described: the capillary haemangioma, the cavernous haemangioma and the sclerosing haemangioma, united by a common lesional continuum.

## Cavernous haemangioma

The cavernous haemangioma is the most common histological sub-type and corresponds to the classic semiological description of the haemangioma in imaging.

This is a lesion consisting of large vascular spaces with a central cavernous zone, all the larger with a voluminous haemangioma, and not very extensive connective tissue [3].

In general, this typical appearance is observed in lesions less than 3 cm in diameter [4]. The outlines are sharp, well defined.

In the sonograph, it is a hyperechogenic, homogenous lesion presenting a posterior acoustic enhancement. According to Yu et al., there is a correlation between the echogenicity of the lesion, its internal architecture and its haemodynamic behaviour [5]. Therefore, the hyperechogenicity of cavernous haemangiomas seem to be related to the great many interfaces between the vascular spaces and the fibrous stroma as well as to the slower blood flow in the large vascular spaces [5].

In unenhanced CT, the density of the lesion is the same as that of the vessels.

In MRI, the lesion presents an homogenous and high intensity signal on T2-weighted images (similar to that of the cerebrospinal fluid), a low intensity signal on T1-weighted images and the absence of restriction of the apparent diffusion coefficient (ADC), with mean values much higher than that of the hepatic parenchyma and ranging from  $1.69 \times 10^{-3} \text{ mm}^2/\text{s}$  ( $\pm 0.34 \times 10^{-3} \text{ mm}^2/\text{s}$ ) [6] to  $2.36 \times 10^{-3} \text{ mm}^2/\text{s}$  ( $\pm 0.48 \times 10^{-3} \text{ mm}^2/\text{s}$ ) [7] according to the gradient values (b) used (Fig. 1a).

The enhancement kinetics is slow. Classically, a nodular peripheral enhancement is observed, as well as late, progressive, centripetal, full and persistent filling [3]. This enhancement kinetics is also observed by contrast sonography and is reproducible and specific (Fig. 1b) [8]. At the arterial time, "bridged" contrast enhancement crossing the lesion may be associated with early peripheral contrast enhancement (Fig. 1c) [8].

According to Yamashita et al. [9], the haemodynamic behaviour of haemangiomas depends on their inner structure and, in particular, on the size of the vascular spaces. Therefore, in cavernous haemangiomas, the diameters of the vascular spaces are significantly smaller in the early

peripheral zones of enhancement in clusters compared with the central zone of progressive centripetal filling [9].

## Capillary haemangioma

Also known as fast-flow haemangioma, the capillary haemangioma presents small vascular spaces and extensive connective tissue [3]. This form accounts for 16% of all haemangiomas [4]. It often consists of small lesions. According to Hanafusa et al. [10], 42% of these haemangioma are under 1 cm in diameter.

In sonography, these haemangioma are most often hypoechogenic and homogenous. This hypoechogenicity seems to be related to a predominant fibrous stroma as well as to a fast blood flow within the reduced vascular spaces, leading to less reverberation of the echoes [5]. In the color doppler, it is possible to observe an intra-lesional flow [4].

In CT, these small haemangiomas appear to be slightly hypodense without injection. The density is similar to that of the aorta but they may also be isodense and therefore sometimes not detectable [11].

The enhancement kinetics is rapid. An early, intense, homogeneous contrast is observed "by flash", similar to the aortic enhancement in the arterial phase [1]. Late, this enhancement follows that of the aorta, without washing [1], in particular distinguishing the haemangioma from hepatocellular carcinoma and certain hypervascular metastases. According to Yamashita et al., this enhancement dynamics is related to the presence of small vascular spaces, the size of which is similar to that of the peripheral zones of cluster enhancement [9].

In MRI, these small lesions also present an homogenous and high intensity signal on T2-weighted images as well as a contrast kinetics similar to that seen in X-ray computed tomography with a uniform and rapid enhancement [12].

The association with an arteriportal shunt is common. In this case, there is a transient perilesional enhancement (Fig. 2) [13].

## Sclerosed haemangioma

Certain haemangioma may degenerate with an extensive fibrosis beginning at the centre of the lesion at the origin of the obliteration of the vascular spaces. This is also called a thrombosed or hyalinised haemangioma [14].

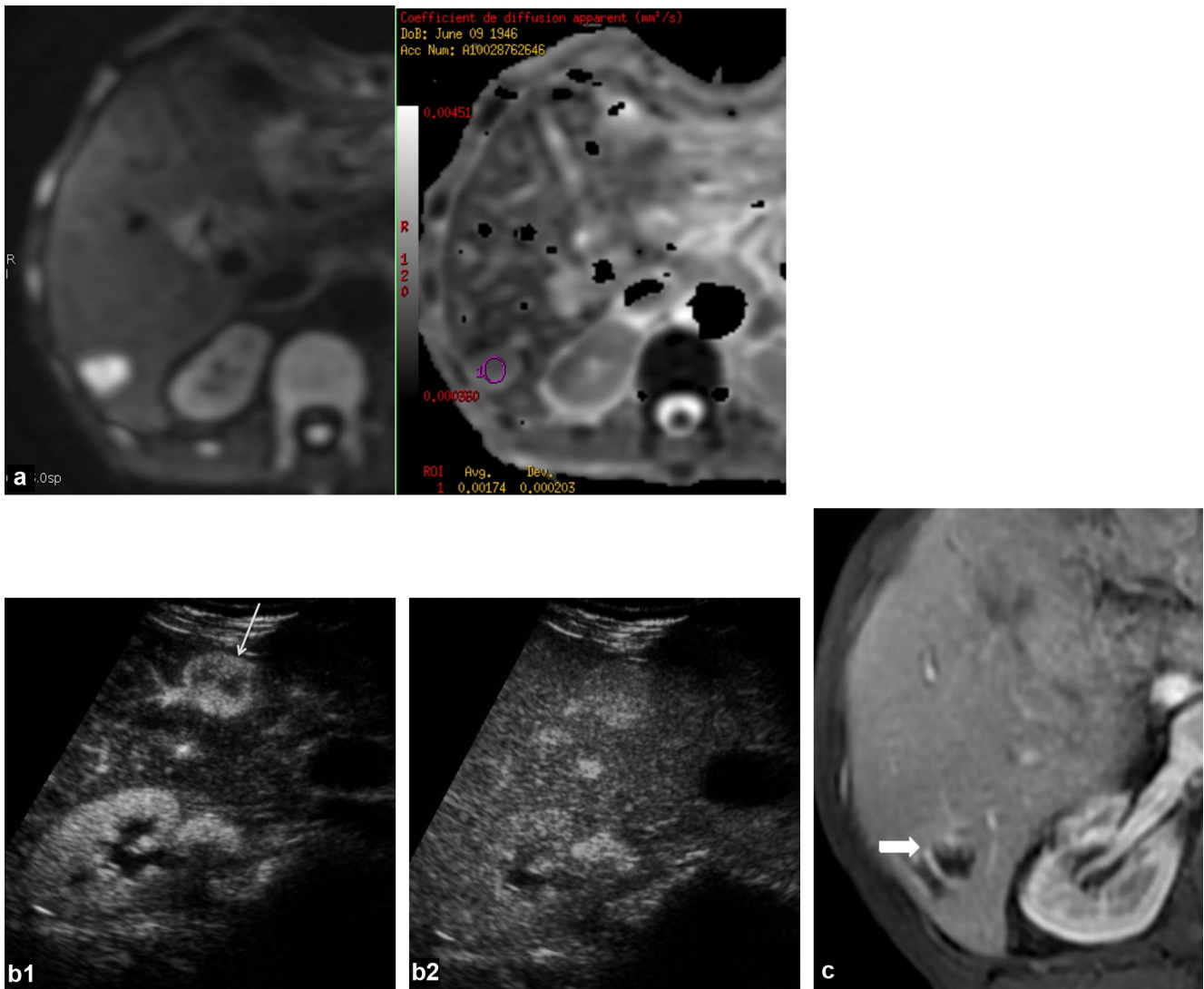
The criteria indicating the diagnosis of sclerosed haemangioma are the geography map appearance associated with a reduction in the volume of the hepatic parenchyma with capsular retraction. Lesional heterogeneity may also exist with the presence of cystic, haemorrhagic or fibrous patches [14].

In sonography, it consists of a globally heterogeneous lesion with hypoechogenic zones that may correspond to sclerotic zones in histology [14].

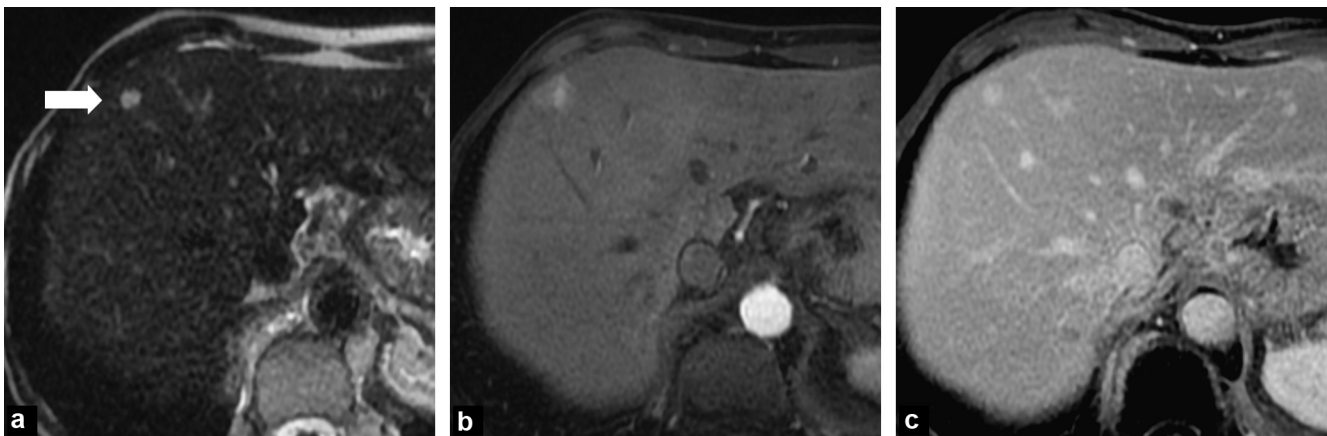
In CT, focal nodular focal patches are observed that are more spontaneously hypodense than the rest of the lesion, also corresponding to sclerotic zones [15].

In MRI, on T2-weighted images, signal is heterogeneous, the zones of central sclerosis appear in hypointense.

The enhancement kinetics is slow with a peripheral nodular enhancement, similar to the cavernous haemangioma, but with full and very late progressive filling. In fact, the



**Figure 1.** Cavernous haemangioma. a: MRI axial section, diffusion-weighted image with b800 (on the left) and ADC map (on the right): persistent homogenous hyperintense signal without restriction of absence of restriction of the apparent diffusion coefficient ( $1.74 \cdot 10^{-3} \text{ mm}^2/\text{s}$ ); b: contrast sonography: peripheral contrast enhancement at arterial time (b1) with progressive centripetal filling (b2); c: MRI axial section dynamic T1-weighted image during hepatic artery phase after gadopentate dimeglumine administration: peripheral contrast enhancement with “bridged” contrast enhancement crossing the lesion (also visible on Fig. b).



**Figure 2.** Capillary haemangioma. a: axial T2-weighted MR image: homogenous hyperintense centrimetric lesion; b: axial dynamic T1-weighted MR image during hepatic arterial phase: “flash” contrast enhancement associated with transitory less intense and poorly defined perilesional enhancement; c: MRI axial section dynamic-weighted image during portal venous phase: persistence of the homogeneous lesional contrast enhancement and disappearance of the perilesional enhancement.

enhancement of the central scar, sometimes obtained several hours after the injection, is inconstant. Sometimes, sclerosed haemangiomas may not be enhanced at all, even late. In addition, there is classically an early transient perilesional enhancement (Fig. 3). The diagnosis, cautiously suggested by the imaging, most often remains histological [14].

Table 1 presents the different characteristics of cavernous, capillary and sclerosed haemangiomas.

## Variants and atypies

### Giant haemangioma

It consists of a cavernous haemangioma measuring over 4 cm in diameter [4]. These haemangiomas may be the seat of thrombosis, liquefaction and fibrosis. A cystic cavity or central calcifications may appear. Internal septa are classically

observed. The edges are regular without loss of parenchymateous volume or capsular retraction.

In sonography, this lesion seems to be heterogeneous.

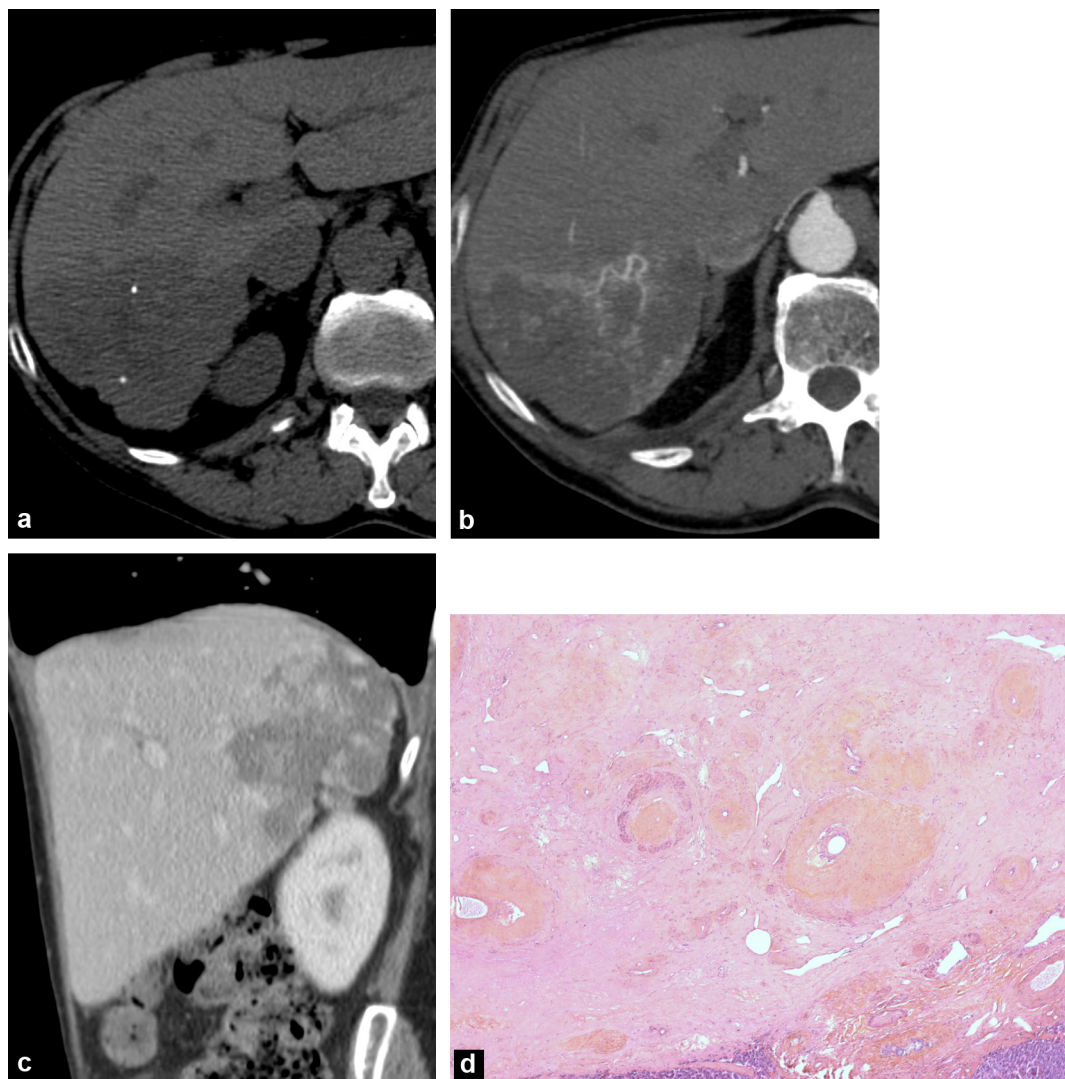
In CT-scan, a heterogeneous, more hypodense central zone may be seen.

In MRI, the hyperintensity on T2-weighted images may be modified by the presence of a hypointense central scar.

The enhancement kinetics of giant haemangiomas is slow but identical to that of cavernous haemangiomas with nodular peripheral enhancement followed by centripetal filling that often remains incomplete [4] (Fig. 4).

### Multiple haemangiomas

This consists of several haemangiomas distributed in the hepatic parenchyma. The haemangiomas are multiple in 10% of the cases and most often present a typical appearance in imaging [4].



**Figure 3.** Sclerosed haemangioma. a: Unenhanced CT, axial section: central punctiform calcifications evocative of pheboliths. Also note the retraction of the opposite hepatic capsule; b: Contrast-enhanced CT during arterial phase, axial section: early perilesional enhancement in ring, visualization of an afferent artery; c: Contrast-enhanced CT during portal venous phase, sagittal section: progressive centripetal filling; d: microscopic appearance with low magnification ( $\times 40$ ) showing a proliferation of vessels with thick walls containing endoluminal thrombi, separated by a connective tissue presenting hyaline rearrangements.



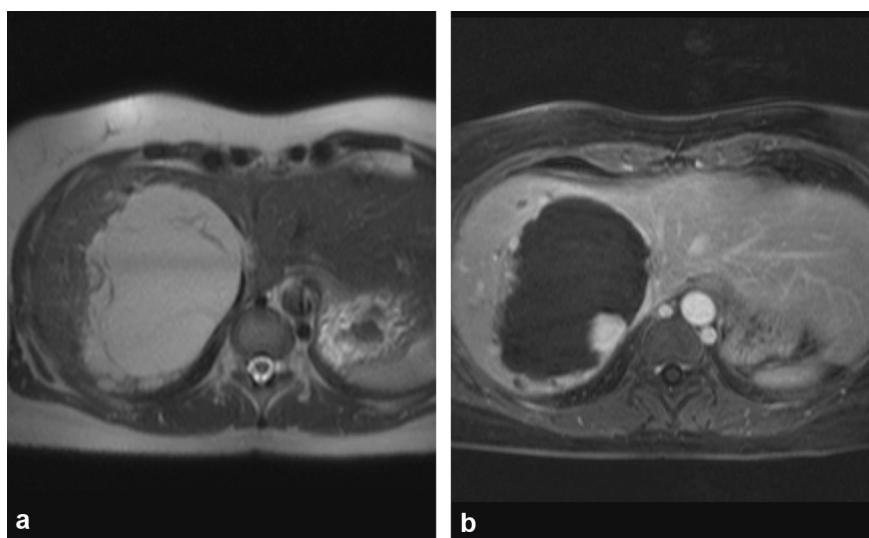
**Table 1** Characteristics of the typical appearance of the three histological forms of hepatic haemangiomas.

	Capillary haemangioma	Cavernous haemangioma	Sclerosed haemangioma
Histological composition	Reduced vascular spaces Extensive connective tissue	Large vascular spaces Not very extensive connective tissue	Extensive beginning fibrosis at the centre of the lesion
Size	Small size (in general < 1 cm)	Lesion < 3 cm = typical appearance Giant > 4 cm	Average size (3.7 cm on the average)
Morphology	Nodular, homogenous	Well defined, internal septa	Geography map appearance, central scar, capsular retraction, punctiform calcifications
Echogenicity	Hypoechogetic, homogenous Posterior enhancement	Hyperechogetic, homogenous Posterior enhancement	Heterogeneous, hypoechogetic centre
MRI: T2-weighted images	Hyperintense, homogenous	Hyperintense, homogenous	Heterogeneous, peripheral hyperintense, central hypointense
Kinetics of the contrast enhancement	Rapid flow, early enhancement, intense, in flash, persistent at late time	Slow flow, nodular peripheral enhancement, progressive and complete centripetal filling	Slow flow, peripheral nodular enhancement, very late homogeneous filling (3 h)
Perilesional environment	Transitory zone of perilesional enhancement, arteriportal shunt		Enhancement in perilesional ring, capsular retraction

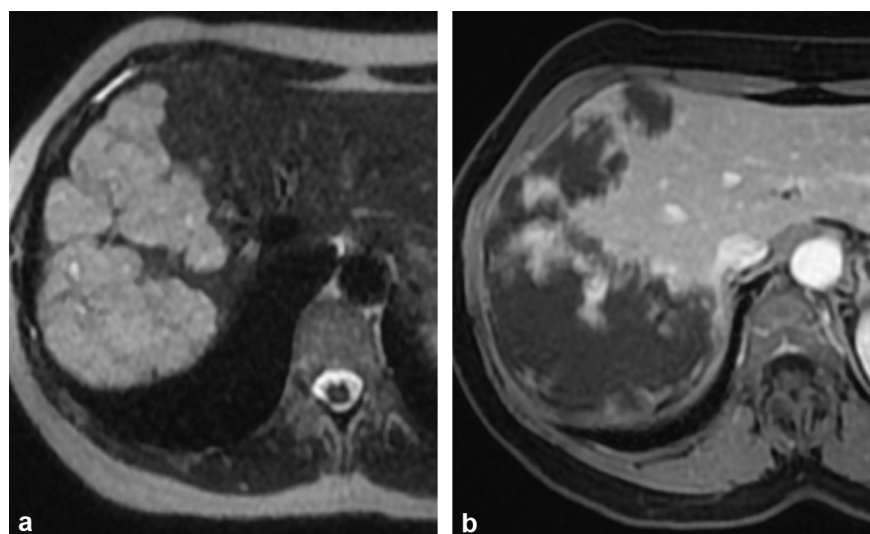
## Haemangiomatosis

Haemangiomatosis is rare and asymptomatic in the adult. It is more often found in the child where it may be associated with congestive heart failure [4]. The lesions are large, poorly defined, confluent, replacing almost all of the hepatic

parenchyma. In the sonograph, the masses are poorly delimited and difficult to distinguish from the adjacent hepatic parenchyma. In MRI, the T1 and T2-weighted signal remains characteristic. The typical peripheral enhancement may be absent and only late acquisitions are informative (Fig. 5) [4].



**Figure 4.** Giant haemangioma. a: MR axial T2-weighted image: presence of internal septa; b: dynamic T1-weighted MR axial image during portal venous phase: nodular peripheral enhancement.



**Figure 5.** Haemangiomas. a: axial T2-weighted MR image: large confluent masses in heterogeneous hyperintense and presenting polylobulated contours; b: axial T1-weighted dynamic MR image at portal venous phase: nodular peripheral enhancement.

### Pedunculated haemangioma

The pedunculated haemangioma is a very rare lesion with extrahepatic development. It is well delimited, encapsulated, attached to the liver by a thin pedicle that is not always visible in imaging [16]. Multi-plane reformatting may be of use to confirm the hepatic origin of the lesion (Fig. 6). The literature provides several cases of volvulus, a specific complication of pedunculated haemangioma, revealed by an acute abdominal picture complicated with necrosis or haemorrhage [16].

### Calcified haemangioma

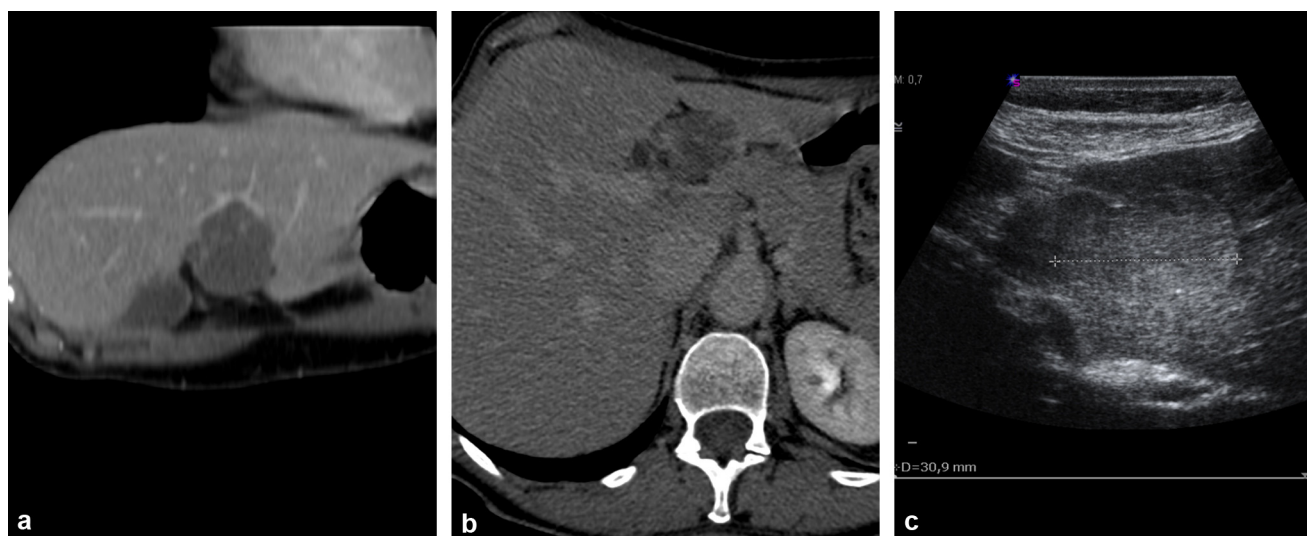
Calcifications are rare, punctiform and are either central or peripheral. They attest to the presence of phleboliths, most

often visible in sclerosed haemangiomas or giant reshaped haemangiomas (Figs. 3 and 9) [4].

### Associated adjacent parenchymateous anomalies

#### Haemangioma with arterioportal shunt

The association of a haemangioma and an arterioportal shunt is reported with an incidence of up to 26% [13]. This association is significantly more common with haemangiomas with fast enhancement kinetics [13], that is, capillary haemangiomas. The presence of transient perilesional enhancement is significantly more common with a small haemangioma (<2 cm) than with a HCC of the same size [17].



**Figure 6.** Pedunculated haemangioma of the round ligament. a: Contrast-enhanced CT during portal venous phase, coronal section: well defined lesion with slight enhancement; b: Contrast-enhanced CT during the equilibrium phase, axial section: seemingly extrahepatic lesion with mass effect on the left lobe; c: sonography: hyperechogenic, heterogeneous appearance with posterior enhancement.

With color doppler, it is possible to visualise the afferent arterial flow and the efferent portal flow.

After injection, this shunt provokes the early opacification of the adjacent portal branches (Fig. 7) [18].

### Haemangioma associated with focal nodular hyperplasia

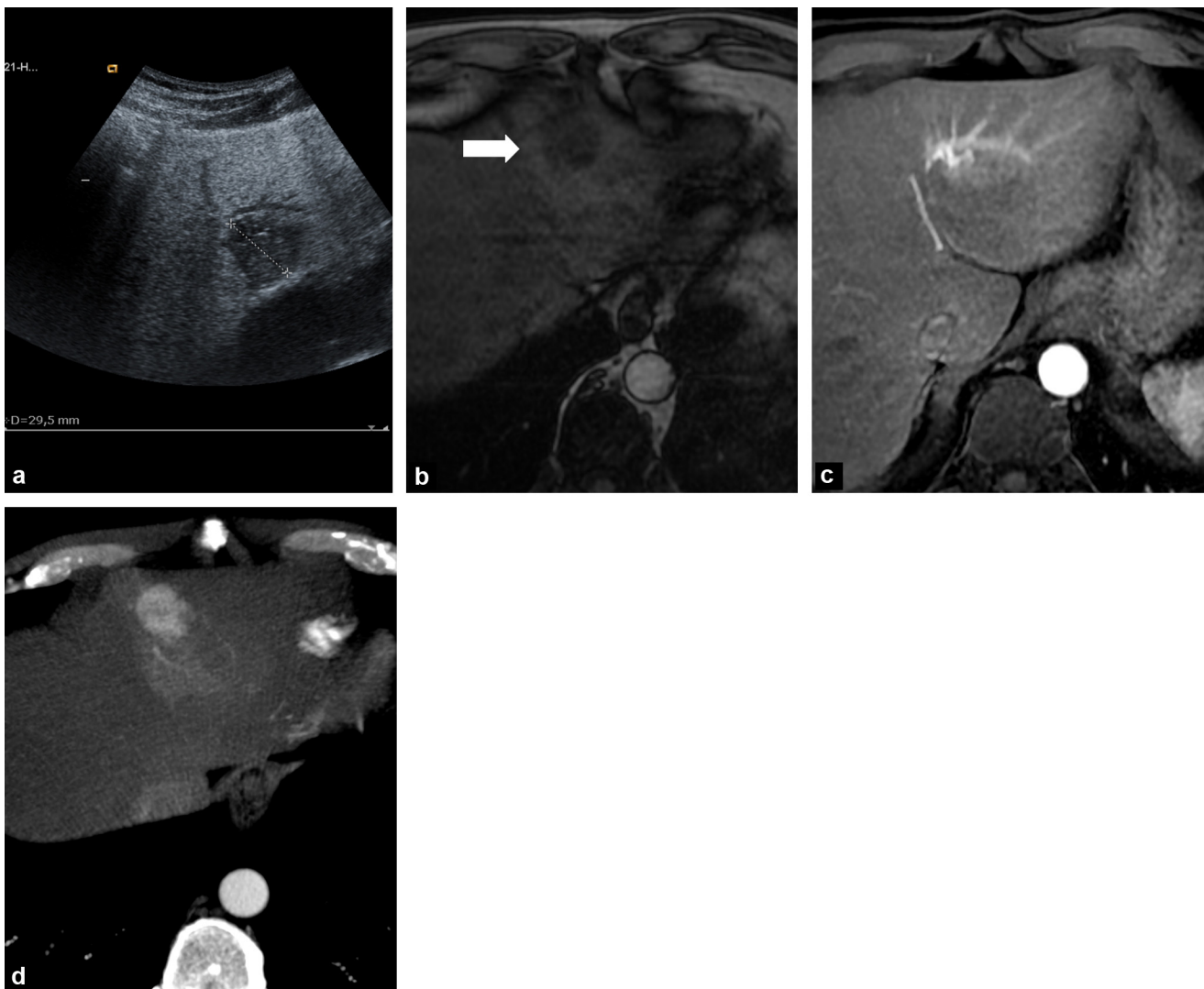
This consists of a relatively common association involving about 20% of all patients with focal nodular hyperplasia [19]. This association is also more often noted in cases of multiple focal nodular hyperplasia and in case of treatment with oral contraceptives [20]. In fact, nodular and focal hyperplasia is a hyperplastic response to the focal increase in the arterial flow potentially generated by a haemangioma [4]. When these lesions maintain a typical appearance in imaging, the diagnosis does not require histological proof (Fig. 8) [4].

### Haemangioma with capsular retraction

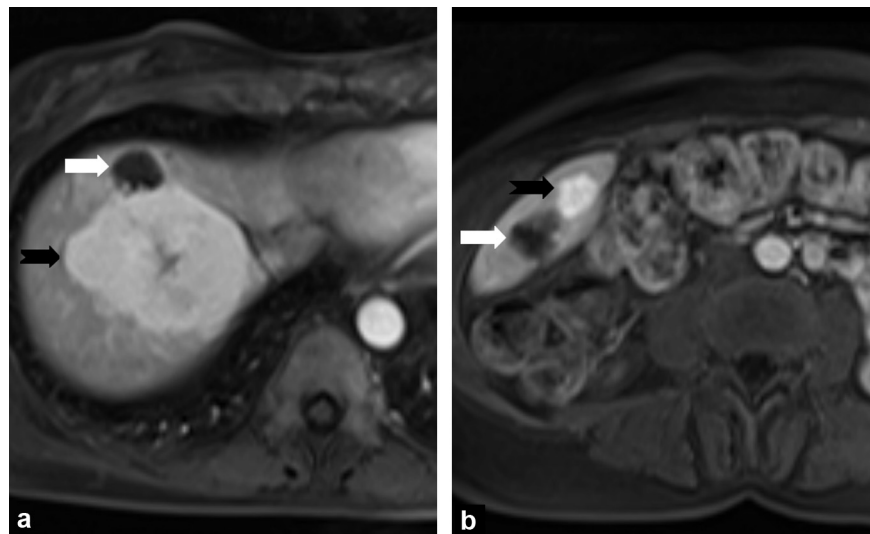
Although often associated with malignant tumours, hepatic capsular retraction is not formally a criterion indicative of malignancy. In fact, it is present when there is a marked focal fibrous stroma reaction as in the sclerosed haemangiomas. This fibrous reaction is the cause of a progressive reduction in the size of the haemangioma resulting in a capsular retraction that is all the more marked if the lesion is sub-capsular [21]. In the series by Doyle et al., 70% of the sclerosed haemangiomas presented capsular retraction or concavity (Figs. 3 and 9) [14].

### Haemangiomas and dilation of the bile duct

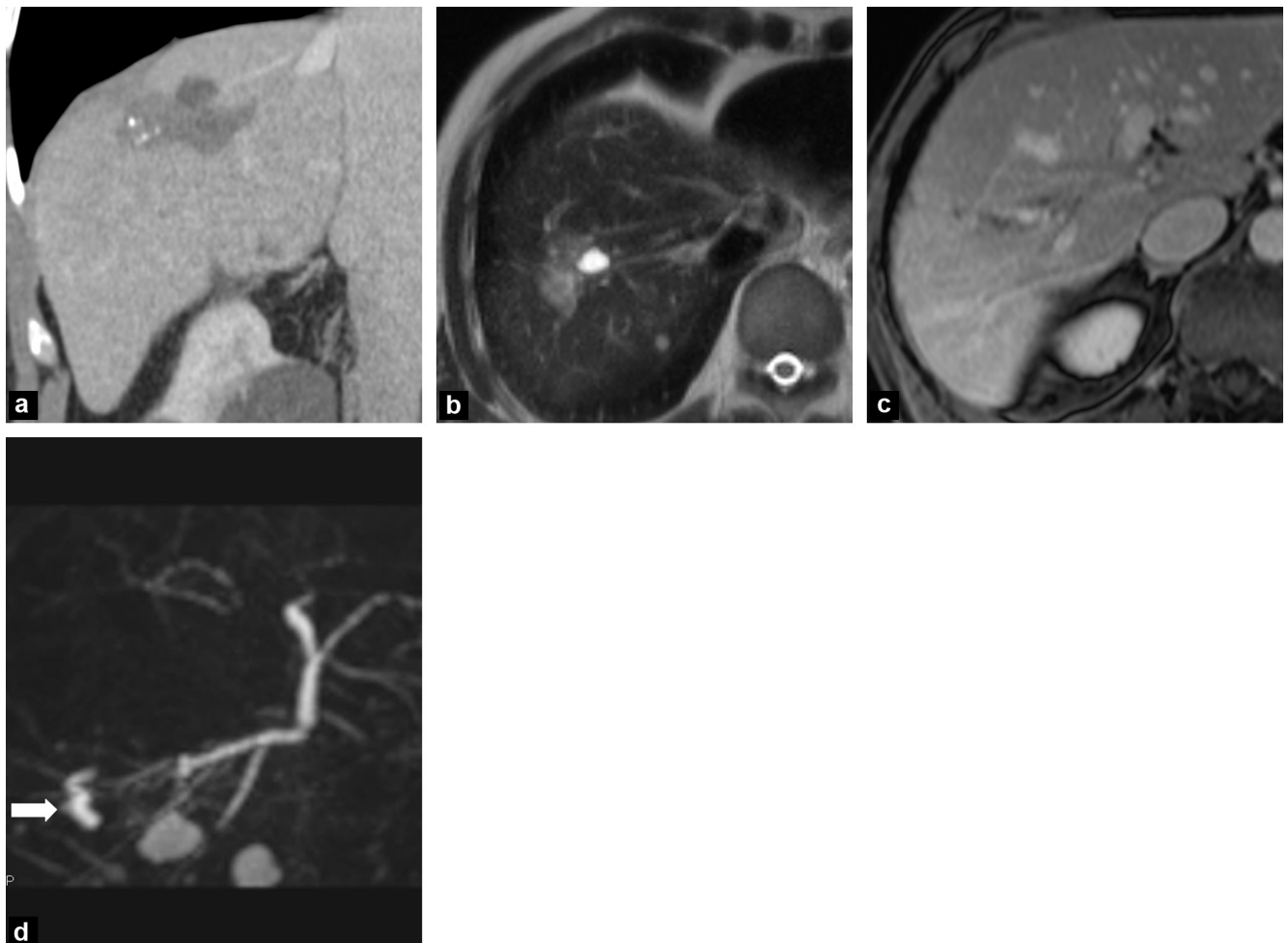
A haemangioma may exceptionally induce the dilation of the bile duct, in particular if it is located in segment IV or



**Figure 7.** Haemangioma with arterioportal shunt and hepatic steatosis. a: sonography: hypoechoic, heterogenic nodular lesion on a hyperechoic hepatic parenchyma related to diffuse steatosis; b: Out-of-phase T1-weighted MR image: zone free of perilesional fat overload with drop in the signal of the rest of the hepatic parenchyma; c: dynamic T1-weighted image during arterial phase: visualization of the afferent artery and early opacification of a left segmental portal branch attesting to the presence of the shunt; d: Contrast-enhanced CT during arterial phase: axial section: early and intense lesional enhancement and perilesional parenchymateous enhancement.



**Figure 8.** Haemangiomas (white arrows) and focal nodular hyperplasias (black arrows). a: Axial dynamic T1-weighted MR image during arterial phase: haemangioma of segment V next to a large FNH presenting a central scar; b: Axial dynamic T1-weighted MR image during arterial phase, same patient: on the underlying sections, there is a second haemangioma of segment VI next to a second FNH.



**Figure 9.** Sclerosed haemangioma at the origin of dilation of the segmental bile ducts upstream. a: Contrast-enhanced CT during portal venous phase, coronal section: punctiform central calcifications and capsular retraction; b: axial T2-weighted MR image: heterogeneous hyperintense with presence of a large area of liquid rearrangement; c: Axial dynamic T1-weighted MR image during portal phase: dilation of the intrahepatic bile ducts upstream from the lesion; d: bili-MRI 3D sequence, reconstruction in the axial plane: dilation of the intrahepatic bile ducts of VII (arrow) upstream from the compression exerted by the lesion.



close to the hepatic hilum [22]. This compressive appearance, classically found in all malignant tumours, should call into question the diagnosis of haemangioma, outside of a perfectly characteristic appearance (Fig. 9) [22].

## Haemangiomas and diffuse liver disease

### Haemangioma and hepatic steatosis

Hepatic steatotic infiltration is known to modify the typical appearance of focal hepatic lesions. In sonography, the haemangioma may appear slightly hyperechogenic, isoechogenic or often hypoechogenic (Fig. 7) with respect to the steatotic liver [23]. It may maintain a posterior acoustic enhancement [4].

In unenhanced CT, the lesion may be hyperdense or isodense and not visible [4].

In MRI, the hyperintensity on T2-weighted images is not modified by the hepatic steatosis.

The enhancement kinetics in CT or MRI is not modified by the hepatic steatosis [24].

A perilesional zone is often observed that is free of fat infiltration. This is due to the arterioportal shunt with preferential arterial vascularisation. This appearance is often seen with fast circulation haemangiomas [25]. In sonography, this zone is visible by a perilesional hypoechogenic ring [25]. In CT-scan, it is seen in the form of annular hyperdensity in spontaneous contrast. In MRI, this zone does not present a drop in signal on out-of-phase T1-weighted image (Fig. 7) [17,25].

### Haemangioma and cirrhosis of the liver

The detection and lesional characterisation of cirrhosis of the liver may be a problem [26]. In fact, the progression of cirrhosis of the liver may induce a reduction in the size of haemangiomas. They become more fibrous and more difficult to recognise radiologically and histologically [26]. Capsular retraction may occur [21,26].

In sonography, the dysplastic nodule and hepatocellular carcinoma may come in the form of a hyperechogenic nodule similar in appearance to a haemangioma [24].

Due to liver perfusion disorders related to the fibrosis, the lesional enhancement may be perturbed in CT-scan [26]. The MRI may, in this case, be superior, allowing visualisation of the more characteristic enhancement profiles [26]. The T2 sequences and diffusion-weighted also increase the

sensitivity of the examination although the high intensity signal on T2-weighted images [17].

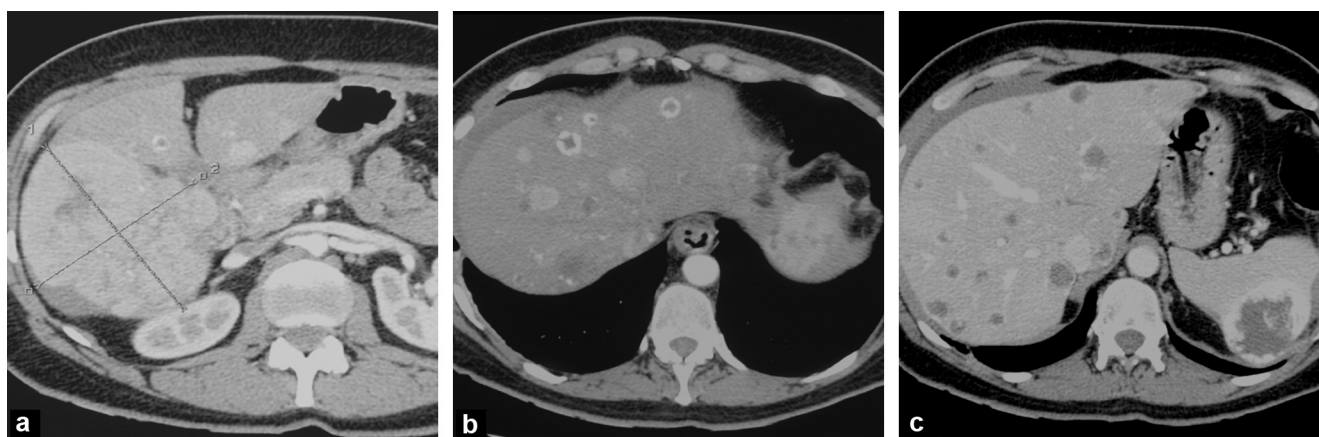
## Special cases

### Infantile hepatic haemangiomas

Comprising almost 90% of the hepatic vascular anomalies in the child [27], infantile hepatic haemangiomas may present themselves in the form of an asymptomatic abdominal mass or induce serious, possibly life-threatening, complications (congestive heart failure due to the association with large arterioportal shunts; Kasabach-Merrit syndrome: coagulopathy due to the intra-lesional platelet sequestration; severe hypothyroidism; anaemia and haemoperitonitis on spontaneous rupture) [28]. The study by Kassarian et al. [29], in a series of 55 infantile hepatic haemangiomas, found 40% to be of solitary form and 60% of multifocal form. Congestive heart failure is the complication that determines the need for treatment (corticoids, embolisation or liver transplant), carried out for 76% of the children [29]. Although they do not reveal an imaging criterion predictive of death, the presence of a shunt (arterioportal, arterio-venous or porto-venous), present in 36% of the children, predisposes a failure in the medical treatment [29].

### Hepatic angiosarcoma: a rare differential diagnosis

If in most cases, all of the imaging techniques help distinguish haemangiomas from hypervascular metastases and HCC, the differential diagnosis between the two latter lesions and angiosarcoma may turn out to be impossible since percutaneous biopsies are counter-indicated due to the potentially deadly risk of haemorrhage [30]. A primitive rare hepatic sarcoma (about 1% of all primitive hepatic tumours), the angiosarcoma is a malignant tumour consisting of fusiform or pleomorphic cells developing in the opening of pre-existing vascular spaces [2]. There are often metastatic lesions at the time of the diagnosis, preferentially located at the spleen and lungs. Four lesional forms are observed attesting either to multiple nodules, or a predominant voluminous mass, or a mixed form associating a dominant mass and nodules, or diffuse micronodular infiltration (Fig. 10). An intra-intestinal haemorrhage may be observed leading, in MRI, to a hyperintensity on T1-weighted images and a liquid-liquid level in T2-weighted images by deposit of haemosiderin [30].



**Figure 10.** Hepatic angiosarcoma. a: Contrast-enhanced CT during arterial phase, axial section: dominant mass of segments V and VI, early and heterogeneous enhancement; b: Contrast-enhanced CT during arterial phase, axial section: multiple associated nodular lesions disseminated in the hepatic parenchyma with contrast enhancement either annular and intense or homogeneous; c: Contrast-enhanced CT during portal venous phase, axial section: secondary splenic location and intra-peritoneal effusion.

## Conclusion

Although, in the vast majority of cases, the radiological diagnosis of hepatic haemangioma is clearly established, certain semiological atypies may make the diagnosis difficult. The presence of a lesional continuum between the capillary, cavernous and sclerosed haemangiomas may account for the radiological variants. The haemangioma appears as an active vascular lesion in interaction with its immediate environment that may be at the origin of arterioportal shunts, perilesional zones free of steatotic infiltration, focal nodular hyperplasias, compressions on the bile ducts and hepatic capsular retraction.

## Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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