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### **Transient hepatic intensity differences: part 1, Those associated with focal lesions.**

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# Transient Hepatic Intensity Differences: Part I, Those Associated with Focal Lesions

Stefano Colagrande<sup>1</sup>  
Nicoletta Centi<sup>1</sup>  
Roberta Galdiero<sup>2</sup>  
Alfonso Ragozzino<sup>2</sup>

**OBJECTIVE.** The purpose of our study was to evaluate, on MRI, transient hepatic signal intensity differences (THID) that have already been seen on CT as transient hepatic attenuation differences (THAD) and to show the range of appearance of such arterial phenomena, when associated with focal lesions, in an effort to correlate morphology, cause, and pathogenesis.

**CONCLUSION.** Hepatic arterial phenomena visualized on MRI should be known and recognized to avoid incorrect diagnoses and to improve the characterization of focal liver lesions because their shape can lead to an understanding of pathogenetic mechanisms.

**T**ransient hepatic attenuation differences (THAD) are areas of parenchymal enhancement visible during the hepatic artery phase on helical CT that are caused by the dual hepatic blood supply. In fact, there are compensatory relationships between two liver sources of blood supply so that arterial flow increases when portal flow decreases as a result of communication among the main vessels, sinusoids, and peribiliary venules that open in response to autonomic nervous system and humoral factors activated by liver demand for oxygen and metabolites.

Today hepatic arterial phase evaluation may also be easily performed on MRI, on which perfusion alterations can also be observed; we call these “transient hepatic signal intensity differences” (THID). Perfusion phenomena have been associated with many liver disorders [1–3]. This article intends to show the range of appearance on MRI of such arterial phenomena as has already been shown for helical CT [2], using the same comprehensive diagnostic organization that attempts to correlate morphology, cause, and pathogenesis.

The first ascertainable feature of transient hepatic intensity differences is an association with a focal liver lesion. Therefore, we have organized our article into two parts, the first regarding transient hepatic intensity differences associated with a focal lesion, and the second [4] concerning transient hepatic intensity differences without a focal lesion.

Focal lesions can determine two morphologic types of transient hepatic intensity differences through four pathogenetic mechanisms: directly by a siphoning effect (lobar multisegmental shape) or indirectly by means of portal hypoperfusion (sectorial shape) due to portal branch compression or infiltration, by thrombosis resulting in a portal branch blockade, or by flow diversion caused by an arteriportal shunt.

## Lobar Multisegmental Shape

Lobar multisegmental transient hepatic intensity differences occur when a hypervascular focal lesion, usually large and benign, or an abscess induces an increase in the primary arterial inflow, which leads to surrounding parenchyma hyperperfusion (the “siphoning effect”), in the absence of any demonstrable portal hypoperfusion. These signal intensity differences do not assume a triangular shape; nevertheless, a straight border (a clear line that separates arterial phenomena from adjacent parenchyma) may be present. A hypervascular tumor likely acts on the right or left hepatic artery, producing enhancement of the hepatic lobe containing the lesion [2, 5, 6] (Fig. 1).

Sometimes smaller tumors are active on the primary branch of the right or left hepatic artery, with segmental siphoning and signal hyperintensity only of the segment containing the tumor (Fig. 2); on the contrary, rarely, the contralateral segment in the hepatic lobe containing the tumor may show lower signal intensity than the opposite lobe (the tumor

**Keywords:** arterial phenomena, dynamic MRI, hemodynamics, liver, liver disease, liver perfusion abnormalities, MRI, THID, transient hepatic intensity differences

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<sup>1</sup>Department of Clinical Physiopathology, Section of Radiodiagnostics, University of Florence, Viale Morgagni 85, Florence 50134, Italy. Address correspondence to S. Colagrande (stefano.colagrande@unifi.it)

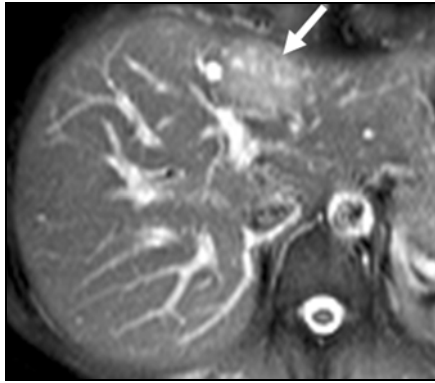
<sup>2</sup>Section of Radiodiagnostics, Ospedale SM Grazie Pozzuoli, Naples, Italy.

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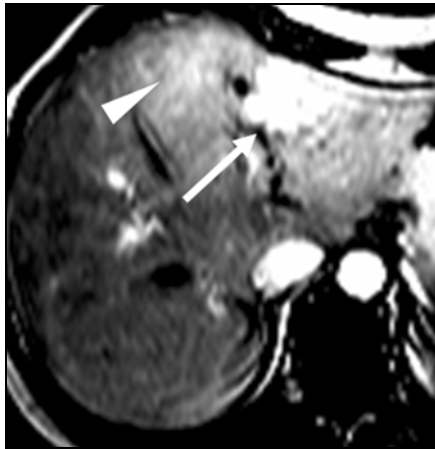
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## Transient Hepatic Intensity Differences



**A**

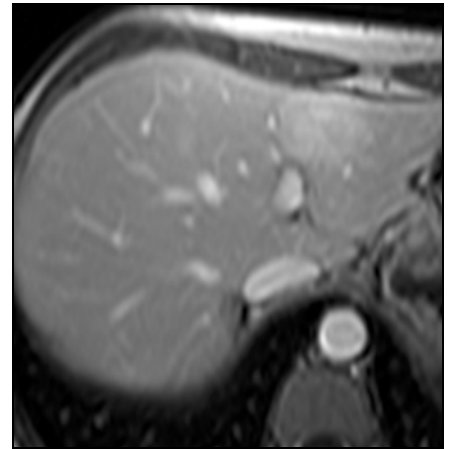
**Fig. 1**—34-year-old woman with fibronodular hyperplasia in left hepatic lobe determining homolateral lobar transient hepatic intensity difference (lobar siphoning effect). **A**, Axial T2-weighted MR image (TR/TE, 830/80) shows slightly hyperintense nodule (*arrow*) in left hepatic lobe. **B** and **C**, Axial gradient-echo T1-weighted gadolinium-enhanced arterial phase MR images (216/1.5) show rapid enhancement of lesion (*arrow*, **B**) and arterial phenomenon (*arrowheads*) of parenchyma in segments II–IV. **D**, Axial gradient-echo T1-weighted gadolinium-enhanced portal phase MR image (216/1.5) shows no parenchymal enhancement in segments II–IV.



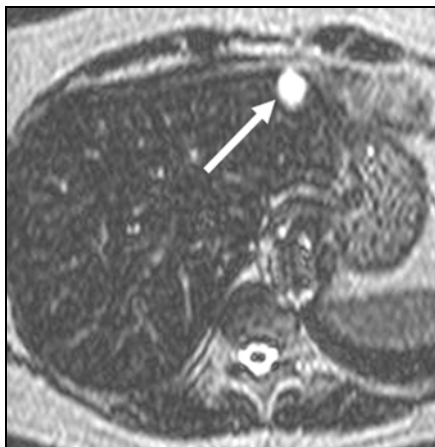
**B**



**C**



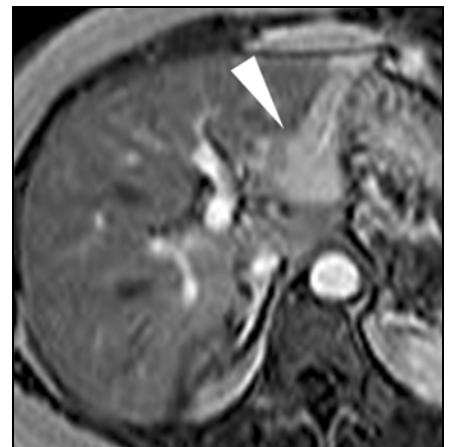
**D**



**A**



**B**



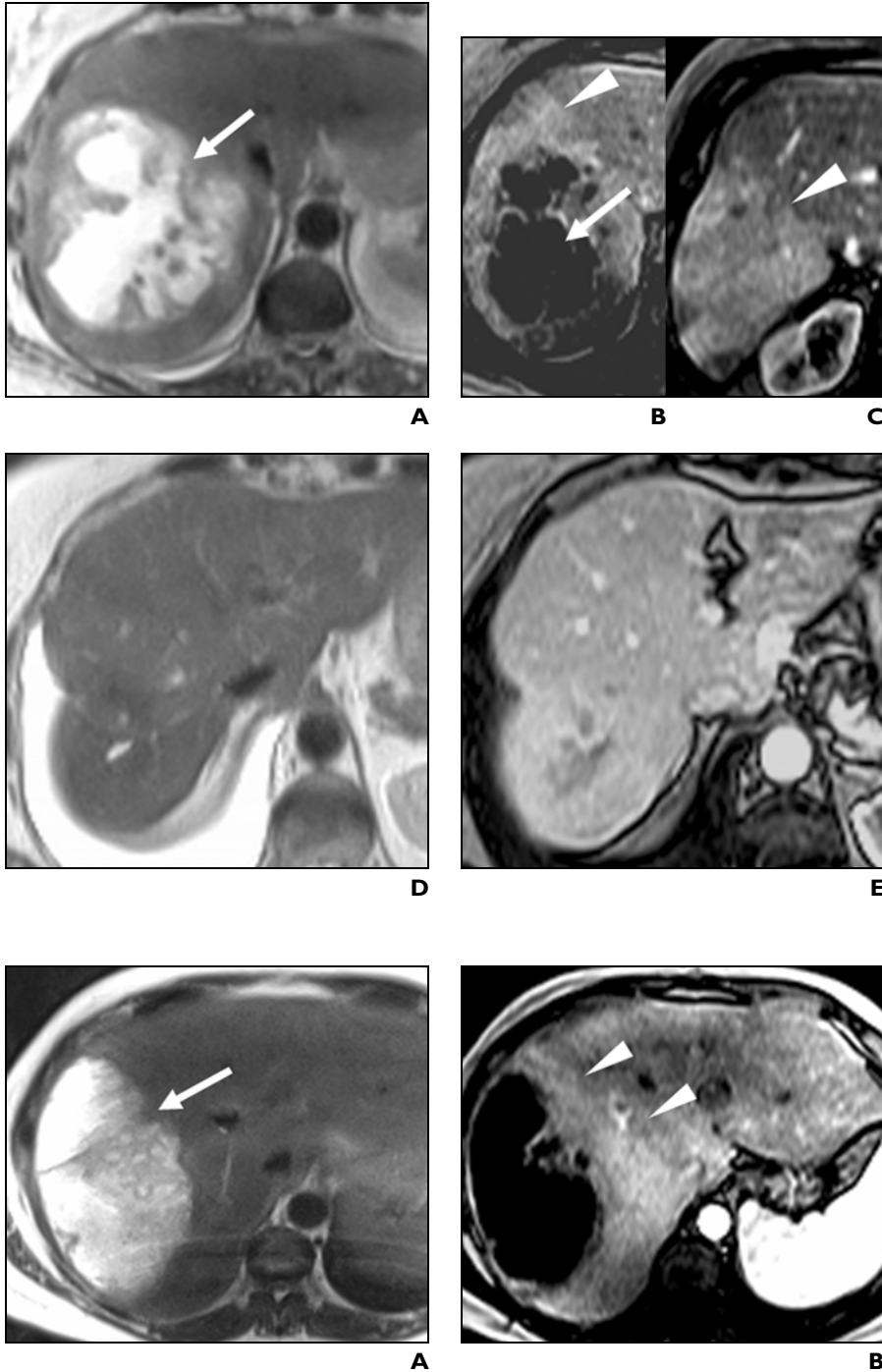
**C**

**Fig. 2**—58-year-old woman with hemangioma in left hepatic lobe inducing homolateral transient hepatic intensity difference in liver segment II (segmental siphoning effect). **A**, Axial T2-weighted MR image (TR/TE, 862/320) shows highly hyperintense nodule (*arrow*) in left hepatic lobe, segment II. **B** and **C**, Axial gradient-echo T1-weighted gadolinium-enhanced arterial phase MR images (216/1.5) show enhanced lesion (*arrow*, **B**) and arterial phenomenon (*arrowheads*) involving only parenchyma of segment II.

“steals” blood flow from the ipsilobar contralateral segment) [6]. Biochemical mediators, if any, are unknown; increased arterial

inflow and consequential hyperenhancement may simply be caused by arterial vascular bed enlargement due to tumor (“sump effect”). In

that way, the remainder of the healthy parenchyma surrounding the tumor can also receive a greater blood supply than usual [5, 6].



**Fig. 3**—58-year-old woman with inflammatory pseudotumor in right hepatic lobe causing homolateral lobar transient hepatic intensity difference (lobar siphoning effect). **A**, Axial T2-weighted MR image (TR/TE, 12,000/84) shows large hyperintense mass (arrow). **B** and **C**, Axial gradient-echo T1-weighted gadolinium-enhanced arterial phase MR images (146/2) show hypointense pseudotumor (arrow, **B**) and arterialization (arrowheads) surrounding lesion. **D** and **E**, Axial T2-weighted (12,000/82) (**D**) and axial gradient-echo T1-weighted (**E**) gadolinium-enhanced arterial phase (146/2) MR images obtained 3 months after **A** and **B** show pseudotumor size reduction (**D**) and consequent disappearance of arterial phenomenon (**E**). Note right pleural effusion.

**Fig. 4**—43-year-old man with large subcapsular liver abscess in right lobe producing homolateral lobar transient hepatic intensity difference (lobar siphoning effect). **A**, Axial T2-weighted MR image (TR/TE, 12,000/84) shows large hyperintense lesion (arrow). **B**, Axial gradient-echo T1-weighted gadolinium-enhanced arterial phase MR image (146/2) shows hypointense mass and lobar arterialization (arrowheads) in surrounding parenchyma.

In cases of phlogistic lesions (Figs. 3 and 4), inflammatory mediators spread in the parenchyma around the lesion and induce hyperemia and then the sump effect. In addition, the same generic benign histologic type can be associated with lobar transient hepatic intensity differences if the nodule is large so that the sump effect occurs, or associated with sectorial arterialization, if the

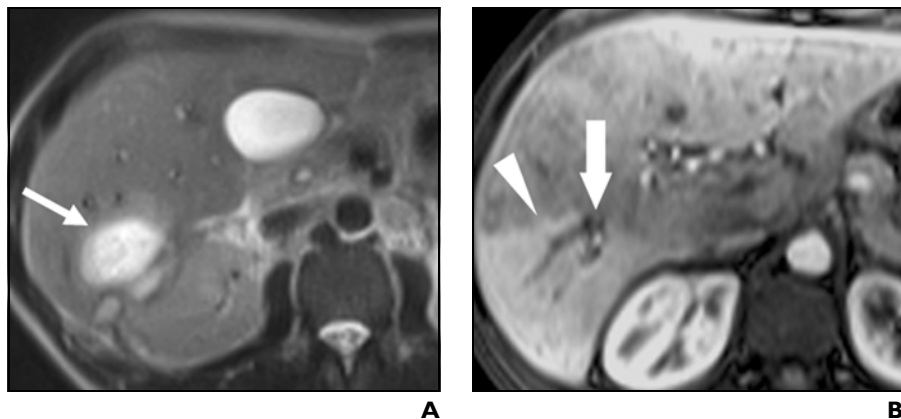
lesion is small and induces portal hypoperfusion (by means of compression, an arteriportal shunt, or portal thrombosis). This phenomenon may occur not only in abscesses (Figs. 4 and 5) but also in association with angiomas or other focal benign lesions. Finally, when the causal focal lesion becomes smaller, vanishing of the siphoning effect can be observed as well (Fig. 3).

**Sectorial Shape**

Sectorial transient hepatic intensity differences follow portal vessel dichotomy and appear as triangular areas, with at least a straight border that is a result of the strict relationship between the portal hypoperfused area and the arterial reaction.

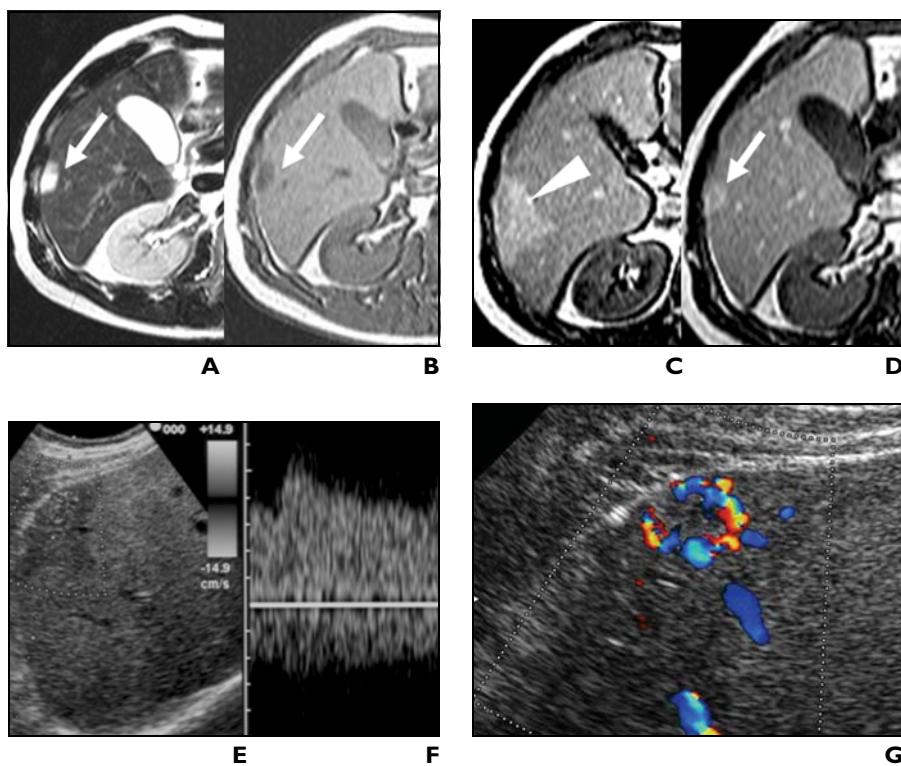
Sectorial transient hepatic intensity differences are associated not only with benign or

## Transient Hepatic Intensity Differences



**Fig. 5**—32-year-old man with liver abscess in right lobe that is inducing sectorial wedge-shaped transient hepatic intensity difference.

**A**, Axial T2-weighted MR image (TR/TE, 12,000/84) shows hyperintense lesion (*arrow*) in segment V. **B**, Axial gradient-echo T1-weighted gadolinium-enhanced arterial phase MR image (146/2) shows sectorial arterial phenomenon (*arrowhead*) in right hepatic lobe due to portal hypoperfusion secondary to portal branch thrombosis (*arrow*) induced by abscess.



**Fig. 6**—50-year-old man with small round hemangioma beneath Glisson's capsule in right hepatic lobe and intralésional arterioportal shunt producing sectorial wedge-shaped arterial phenomenon.

**A and B**, Axial T2-weighted (TR/TE, 830/80) (**A**) and axial gradient-echo unenhanced T1-weighted (216/1.5) (**B**) MR images show right hepatic lobe nodule (*arrow*) that is strongly hyperintense in **A** and hypointense in **B**. **C and D**, Axial gradient-echo T1-weighted iron-oxide-enhanced arterial phase (**C**) and 10-minute delayed phase (**D**) MR images (216/1.5) show wedge-shaped arterial phenomenon (*arrowhead*, **C**) of parenchyma and late enhancement of hemangioma (*arrow*, **D**). **E–G**, Axial sonogram (**E**), pulsed-wave Doppler sonogram (**F**), and color Doppler sonogram (**G**) (all obtained with convex, 3.5-MHz probe) show sectorial hypoechoic area (spare in fatty liver) that has same location and shape as sectorial arterial phenomenon (**E**), arteriovenous pulsed waves (**F**), and color pattern flow (**G**) inside hemangioma, indicating an intralésional shunt.

malignant ( $\approx 70\%$ ) tumors [7] but also with liver abscesses, probably due to portal hypoperfusion as well as to the spread of inflammatory mediators [8] (Fig. 5). They can be either wedge- or fan-shaped [2], depending on where the associated focal lesion is situated.

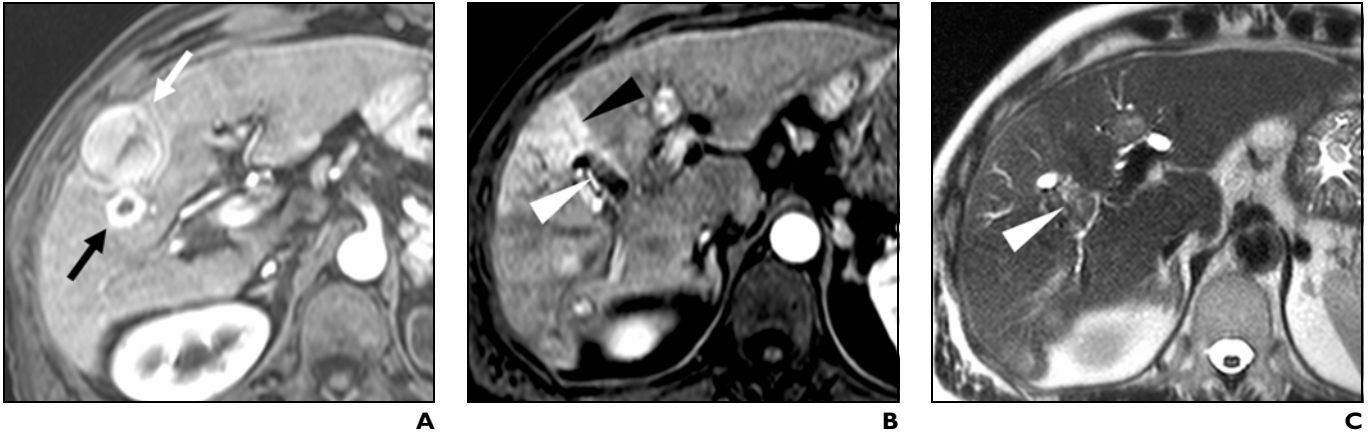
When an associated focal lesion is inside the arterial phenomenon, centrally or laterally positioned, and induces an arterioportal shunt (Fig. 6) or portal thrombosis (Fig. 7), the sectorial transient hepatic intensity difference is wedge-shaped. Because arterioportal shunts occur frequently in hepatocellular carcinoma, arterial phenomena associated with this malignancy

are common; nevertheless, they are more frequently caused by large tumors inducing portal vein invasion or thrombosis.

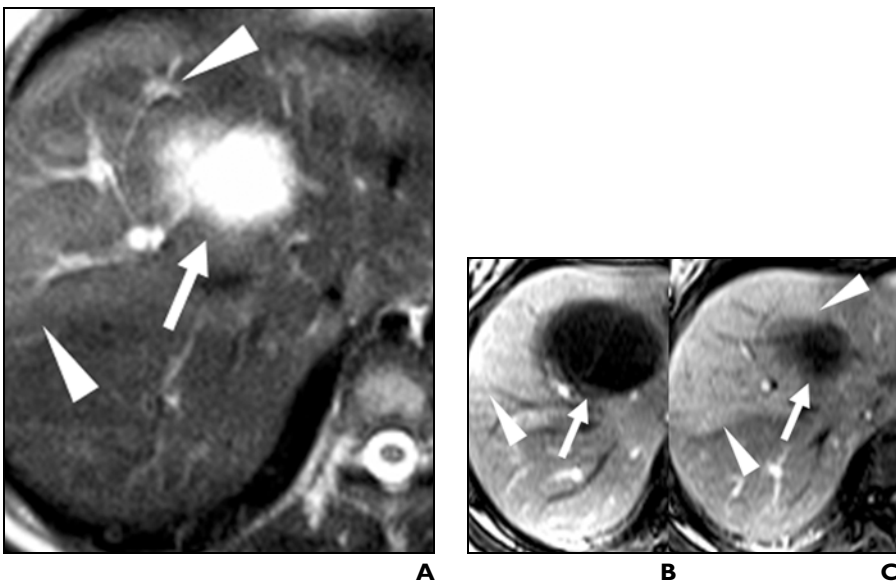
In nodular lesions smaller than 3 cm, the incidence of arterioportal shunts is higher in angiomas (21%) than in hepatocellular carcinomas (4%) [1, 9]. In these cases, shunts lead to a mixing of venous low-pressure and arterial high-pressure blood and then to portal flow diversion, with relative portal hypoperfusion of contiguous parenchyma and an arterial reaction. If persistent, these changes can determine focal metabolic alterations, resulting in a triangular area, fatty in normal liver or spare in fatty liver (Fig. 6).

When an associated focal lesion is situated at the apex of the arterial phenomenon and causes portal compression (Fig. 8) or portal branch infiltration (Fig. 9), the sectorial transient hepatic intensity difference is fan-shaped; this shape is the type most frequently linked to malignancies (Fig. 10).

Transient hepatic intensity differences are not usually seen on T2-weighted images, probably because no changes occur in the amount of free water in the area of the differences [7]; however, when arterial reaction is intense, a slight T2 hyperintensity can be observed (Figs. 7, 8, and 10).



**Fig. 7**—65-year-old man with liver cirrhosis and hepatocellular carcinoma causing sectorial wedge-shaped transient hepatic intensity difference induced by portal thrombosis secondary to tumor.  
**A**, Axial gradient-echo T1-weighted gadolinium-enhanced arterial phase MR image (TR/TE, 146/2) shows strongly enhancing nodule (*white arrow*) and related satellite (*black arrow*).  
**B**, Axial gradient-echo T1-weighted gadolinium-enhanced arterial phase MR image (146/2) shows arterialization (*black arrowhead*) and portal thrombosis (*white arrowhead*).  
**C**, Axial T2-weighted MR image (12,000/82) confirms portal thrombosis (*arrowhead*) and shows slight signal intensity changes in triangular area of arterial phenomenon due to small increase in amount of free water.



**Fig. 8**—27-year-old woman with echinococcus cyst and sectorial fan-shaped transient hepatic intensity difference.  
**A**, Axial T2-weighted MR image (TR/TE, 12,000/84) shows hyperintense round cyst (*arrow*) and slight hyperintensity of liver parenchyma at site of arterial phenomenon (*arrowheads*), probably due to increase in amount of free water.  
**B and C**, Axial gradient-echo T1-weighted gadolinium-enhanced arterial phase MR images (TR/TE, 146/2) show lesion (*arrows*) positioned at apex of fan-shaped arterial phenomenon (*arrowheads*) caused by portal compression.

Sometimes arterial phenomena have no clear explanation. When the arterial phenomenon is due to portal hypoperfusion caused by a focal lesion, the diameter of the causal lesion is not related to the area (size) of the arterial phenomenon; then a small lesion can cause a wide area of arterialization. As a consequence, a sectorial arterial phenomenon may sometimes be the only warning sign of a hidden nodular lesion that is not detectable for size or contrast reasons and yet causes portal compression. In these cases, arterialization may herald an underlying abnormality and precedes the MRI or CT detection of the

nodular lesion (Fig. 9). The latter possibility must be considered whenever a sectorial arterialization has no other explanation [10].

Finally, arterial phenomena not connected to a focal liver lesion, due to cirrhosis, to an arterioportal shunt, or to a small portal branch thrombosis, may have a round appearance and might mimic a hypervascular nodule, making diagnosis difficult [1] (see part 2 of this article [4]).

**Conclusion**

As happens at CT, arterial phenomena are visualized more and more often on MRI because of the shorter acquisition time. These

phenomena should be known and recognized to avoid an incorrect diagnosis and to improve the characterization of focal liver lesions because their shape can lead to understanding of the pathogenic mechanisms.

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## Transient Hepatic Intensity Differences



**A**

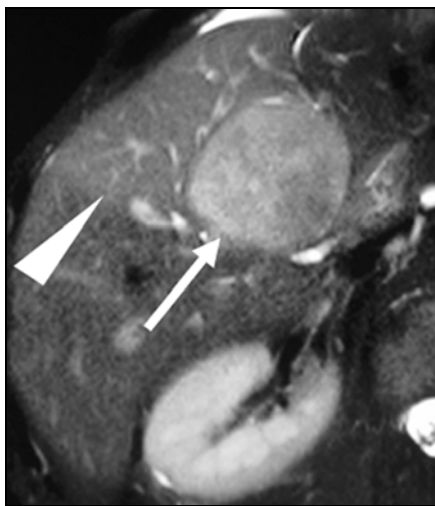


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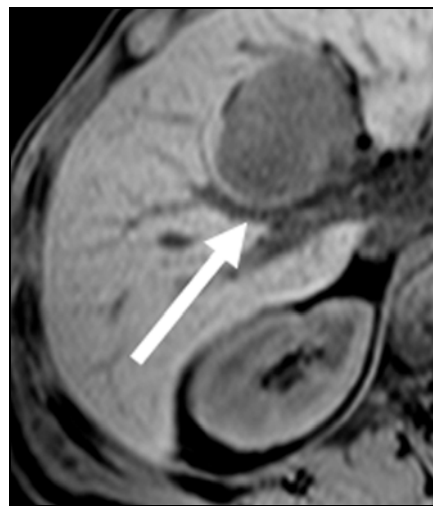
**Fig. 9**—56-year-old man with sectorial fan-shaped transient hepatic intensity difference associated with cholangiocellular carcinoma.

**A**, Axial iodinated contrast-enhanced arterial phase helical CT image shows sectorial arterial phenomenon (*arrowheads*) apparently not associated with focal lesion.

**B**, Axial gradient-echo T1-weighted gadolinium-enhanced arterial phase MR image (TR/TE, 216/1.5) obtained 3 months after **A** shows small hypointense focal lesion (*arrow*) at apex of fan-shaped arterial phenomenon, causing portal branch infiltration and subsequent portal hypoperfusion.



**A**



**B**



**C**

**Fig. 10**—59-year-old man with large hepatic intraparenchymal metastasis from colon carcinoma and correlated sectorial fan-shaped transient hepatic intensity difference.

**A**, Axial T2-weighted MR image (TR/TE, 12,000/84) shows hyperintense parahilar nodule (*arrow*) with associated slight signal intensity change (*arrowhead*) due to small increase in amount of free water. Note small fluid collection near Glisson's capsule.

**B and C**, Axial gradient-echo fat-suppressed T1-weighted unenhanced (146/2) (**B**) and axial gradient-echo fat-suppressed T1-weighted gadolinium-enhanced arterial phase (146/2) (**C**) MR images show wide fan-shaped arterial phenomenon with straight border (*arrowhead*, **C**) due to hypointense neoplastic lesion at its apex (*arrow*), causing portal compression. Note how segment III is also slightly enhanced. Although this transient hepatic intensity difference could look like lobar type because of distribution, this arterial phenomenon is undoubtedly sectorial because lesion, being hypodense and hypoenhancing, causes portal compression and not a primary increase in arterial flow.

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### FOR YOUR INFORMATION

The reader's attention is directed to part 2 accompanying this article, titled "Transient Hepatic Intensity Differences: Part 2, Those Not Associated with Focal Lesions," which begins on page 160.