Focal and diffuse hyperintense hepatic lesions on unenhanced T1-weighted MR images: a pictorial essay

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Learning objectives

The objective of this educational paper is to describe the most common focal and diffuse hepatic lesions with a characteristic hyperintense signal on T1-weighted images and to provide a differential diagnosis among them.
Background

Signal intensity of T1 weighted images is due to several factors such as the chemical composition of tissues, scanning sequences parameters and use of contrast medium [1].

T1 tissue relaxation time is the main factor that sets signal intensity in Magnetic Resonance Imaging (MRI) [1]. Normal liver tissue has a short T1 relaxation time [2]: for this reason, only lesions that contain T1-shortening elements appear relatively hyperintense.

Below are the most common causes of T1 hyperintensity:

- Due to its short T1 relaxation time, fat is the most common element that increases T1-weighted images signal in liver lesions [3]: *intracellular/microscopic fat* can be detected using MR sequences based on chemical shift (signal loss in out-of-phase images); *macroscopic fat*, instead may be detected using fat-suppressed T1-weighted images (no signal loss in out-images; india ink sign; hypointensity in fat-sat images) [4-5].

- **Proteins** in macromolecular compounds can bind water molecules, resulting in a restriction of motion and shortening of T1 relaxation time [6].

- Paramagnetic effect of *haemoglobin degradation product, copper* and *melanin* induce shorter T1 relaxation time in liver lesions [7-8-9].

- **Sinusoids dilatation** is associated with T1 hyperintensity, but the associated mechanism is not completely understood: probably it depends on increased blood viscosity or intra-sinusoid thrombosis [10].

Even when a liver lesion does not have a short T1 relaxation time, it may appear relatively hyperintense if the liver tissue has low signal because of *fibrosis, iron overload* or *edema* [11-12].

Lastly, **phase-encoded motion artifacts** should not be confused as real focal liver lesions. These artifacts, due to arterial pulsation, breathing or patient’s movement, manifest as faded T1 hyperintense area (*ghost artifact*).
Findings and procedure details

According to their histopathology liver lesions can be distinguished in benign and malignant.

Benign liver lesions

**Diffuse pathologies:**

- **Diffuse hepatic steatosis** ([Fig. 1 on page 7]): due to overload of triglycerides within the hepatocytes; may be distinguished an alcoholic- and nonalcoholic-disease. Since it is a benign lesion, there are no mass effect/infiltration of liver vessels and no contrast-enhanced areas of inhomogeneities. Out-of-phase T1w-images show loss of signal.

- **Wilson's disease:** an autosomal recessive disease that is characterized by increased intestinal copper uptake due to abnormal caeruloplasmin metabolism and subsequent deposition of copper and damage to various organs. Hepatic manifestations include fatty changes, cirrhosis and sometimes fulminant hepatic necrosis. Perihepatic fat layer and normal caudate lobe are typical features of Wilson's disease. In the context of cirrhotic liver parenchyma, multiple nodular lesions may be observed: T2-weighted images show hypointense signal and hyperintense on T1-weighted images, according to copper deposit paramagnetic effect. However, paramagnetic effect may be masked by high level of T1 signal of liver in advanced cirrhotic stages [13].

**Focal pathologies:**

- **Focal hepatic steatosis** ([Fig. 2 on page 7 - Fig. 3 on page 8]): may mimic a hepatic malignant lesion. Typical locations are adjacent to the falciform ligament, gallbladder fossa and liver hilum. It presents the same benign signs of diffuse pattern.

- **Complicated cyst** ([Fig. 4 on page 9]): cysts are normally hypointense in T1 weighted images and hyperintense in T2; they may show high T1 signal because of hemorrhage or suppuration.

- **Hematoma:** due to liver traumas or coagulation disorders, blood deposit is hyperintense in T1w images. Anamnesis, lesion morphology and the use of contrast medium are usually enough to raise a correct diagnosis.
• Regenerative hyperplastic nodules (*Fig. 5 on page 10*): generally related to chronic liver injury such as: alteration of tissue perfusion (portal hypertension or Budd-Chiari syndrome), toxic injury (alcohol) or viruses (HBV, HCV). These nodules are usually hypointense on T2 and T2* w images, and have variable T1 signal intensity. When they contain lipids they may show high T1 signal and signal loss in out-of-phase GRE images. Nodules present post-contrast enhancement similar to the adjacent liver both in arterial and hepatocellular phase [14].

• Hepatic Adenoma: generally arises in young women exposed to long-term oral contraceptive therapy. According to their histopathology [15] liver adenomas may be classified in:

1. HNF1-alfa mutated (*Fig. 6 on page 11 - Fig. 7 on page 12*): associated with fat-content multiple adenomas.
2. Beta-catenin mutated: associated with glycogen metabolism alteration, abuse of anabolic steroids and higher probability of malignant degeneration.
3. Inflammatory adenoma: the most common hepatic adenoma, usually hyperintense on T2w images. "Atoll sign" may be manifest.

Hepatic adenoma are in 59-77% of cases fat-containing, showing usually signal loss in out-of-phase images. Intralesional hemorrhage or sinusoid dilatation may be other causes of T1 hyperintensity.

• Atypical focal nodular hyperplasia (*Fig. 8 on page 13*): represents a hyperplastic process of liver parenchyma that shows an abnormal microarchitecture pattern. Typical FNH shows a central scar with radial arrangement of fibrous septa. Atypical lesions lack the central scar and may show T1 hyperintensity, which is related to copper accumulations due to bile duct obstruction, sinusoidal dilatation, hemorrhage (less frequent than in adenomas) or fat deposition (very uncommon) [10,16].

• Lipoma (*Fig. 9 on page 14*): very rare tumor consisting of mature adipose tissue. It shows T1 hyperintensity, india ink sign in out-of-phase images and signal loss in T1 fat-sat image [17].

• Angiomyolipoma (*Fig. 10 on page 15*): rare tumor formed by fatty cells, smooth muscle and blood vessels, that is associated with tuberous sclerosis. Contrast images show prolonged enhancement due to nonfatty components and sometimes an enlarged central vessel.

Malignant liver lesions

• HCC (*Fig. 11 on page 16*): generally hypointense on T1w-images and hyperintense on T2w-images. T1 hyperintensity may be present in
31-61% of tumors smaller than 3 cm due to intralesional presence of fat, copper, glycogen or proteins. Fat content is considered an important sign of transformation of premalignant lesion to HCC. Large tumors may show T1 high dishomogeneous signal because of hemorrhage or necrotic debris [18]. After chemoembolization the presence of lipiodol within the mass reduces T1 relaxation time resulting in hyperintensity. Radiofrequency ablation, also induces coagulative necrosis that causes T1 hyperintensity (Fig. 12 on page 17)

- **Metastasis**: normally are hypointense on T1w images and hyperintense in T2w- images. T1 high signal is related to hemorrhagic metastasis (Fig. 13 on page 17), mucinous carcinoma’s metastasis or melanoma metastasis [19].

- **Liposarcoma**: very rare malignant mesenchymal tumor. Fat content depends on grade of differentiation: low grade lesion has a high T1w images signal reflecting high fat content and shows contrast enhancement or local invasion evidence; high grade lesion shows low T1w signal because of the absence of fat and similar characteristics to other sarcomas. Most liver localizations of liposarcomas are indeed metastases of retroperitoneal or extremity liposarcomas [4,20].

**Rare entities**

Very uncommon lesions may appear hyperintense on T1 w images such as: adrenal rest tumor of the liver, focal intrahepatic extramedullary hematopoiesis, cystic teratoma, pseudolipoma of the Glisson’s capsule, xanthomatous lesions in Langheran’s cell histiocytosis and perihepatic endometriosis (Fig. 14 on page 18)[12].
Fig. 1: Diffuse hepatic steatosis on In-phase (a,c) and Out-of-phase T1-weighted images (b,d). Signal loss in out-of-phase acquisitions is pathognomonic of intrahepatic fat deposition.

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Fig. 2: Pericholecystic steatosis: in/out-of phase images (a,b); T2 fatsat images (c); 3D T1-weighted image with spatial fat saturation (d). Images show pericholecystic area (arrowheads) with signal loss in out-of phase T1w acquisition. Same area does not show enhancement after gadolinium injection (Fig. 3)

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Fig. 3: Pericholecystic fat deposit; same patient of Fig. 2. Gadolinium enhanced T1-weighted images do not reveal enhancement in the pericholecystic area of steatosis (arrowheads). Focal steatosis may depicted in any segment of hepatic parenchyma; more frequently, these areas are encountered in the pericholecystic regions, in the hepatic hilum, along portal vessels and adjacent to the falciform ligament.

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**Fig. 4:** Complicated Cyst: coronal (a) and axial (b) T1-weighted fat sat images that show a voluminous cyst (*) in Polycistic disease. Cyst shows high signal on T2 fat sat sequence (e) - with no changes observed on in- (c) and out-of-phase (d) images. Hyperintensity on T1-weighted images may be related to hemorrhage or proteinaceous contents.

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Fig. 5: Hyperplastic regenerative nodule: hyperplastic nodule (arrowhead) in cirrhotic patients may have high signal on T1-weighted images (a). Because of its benign nature, these lesions do not show enhancement after i.v. gadolinium injection (b,c,d) - also in the hepatobiliary phase (e).

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**Fig. 6:** Fat content adenoma: in- (a,c) and out-of-phase (b,d) images (white arrows) show signal loss - due to its intracellular fat content.

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Fig. 7: Fat content adenoma: (same patient of Figure 6) fat sat unenhanced-T1 weighted image shows hypointense lesion (white arrows) in the caudal part of V segment (a); this lesion has typical enhancement in the arterial phase (b). The adenoma become iso-intense in portal (c) and delayed (d) acquisitions.

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Fig. 8: Atypical FNH: due to its fat component, the lesion (arrowheads) shows signal loss in the out-of-phase T1-weighted image (b) - compared to the in-phase acquisition (a). Atypical FNH appears slightly hypointense on unenhanced T1-weighted fat sat acquisition (c). The lesion shows enhancement in the in arterial phase (d), with same signal of the rest of the liver in the delayed acquisition (e). Thanks to its connection with biliary system, the lesions is depicted isointense in the hepato-biliary phase (f), suggesting FNH.

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**Fig. 9:** Intrahepatic lipoma: lesion (arrowheads) is characterized by homogeneous high signal on T1-weighted image, due to presence of fat tissue in the hepatic parenchyma (a). Lipoma shows signal loss in the out-of-phase T1 weighted sequence (c) and in the 3D T1-weighted image acquired with spatial fat sat saturation (d). Since lipoma is a benign lesion - it does not show enhancement after gadolinium administration (e). (b: in-phase T1-weighted images).

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**Fig. 10:** Angiomyolipoma: this lesion (arrow heads) shows signal loss in the out-of-phase T1-weighted image (b), due to partial intracellular fat sat content; lesion appears hypointense on the unenhanced T1 acquisition (c), showing no enhancement in the arterial (d) and delayed phases (e). (f: T2 weighted image)
**Fig. 11:** Fat-HCC. The lesion (arrow heads) - located in the cranial part of the liver - is slightly hypointense in the T1-weighted acquired with in-phase echo-time (a). It shows inhomogeneous signal loss in the out-of-phase acquisition, due to intracellular fat content (b). The lesion is mild hyperintense in the T2-weighted sequence (c). Unenhanced T1-weighted acquisition shows a peripheral rim with high signal (d). Post-gadolinium images (from e to h) reveal a nodule located in the medial portion of the lesion, with hyper-enhancement in the arterial phase and wash-out in the portal and venous acquisitions.
**Fig. 12:** Coagulative necrosis: lesion (arrowheads) with high signal on T1 fat sat weighted image - in a HCC patient treated with radiofrequences ablation (a); fat sat T1-weighted image arterial phase (b); subtraction image (c) shows no contrast enhancement.
Fig. 13: Metastasis (melanoma): due to melanina content, melanoma's metastasis (arrowheads) are hyperintense on T1-weighted images (a,b,c). Melanina induces T1 relaxation time shortening, thus lesions do not show signal changes between in-phase and out-of-phase images (a,b). Because of their malignant nature, these lesions show enhancement after gadolinium i.v. administration (d: arterial phase; d: portal phase; f: delayed phase).

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**Fig. 14:** Perihepatic endometriosis: due to its hemorrhagic content, perihepatic endometriosis (arrowheads) may mimic a parenchymal lesion with hyperintense appearance on T1 weighted images. Axial (a) and coronal (b) T1 weighted fat sat images of perihepatic localization of endometriosis.

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Conclusion

MRI is a reliable diagnostic tool in recognizing focal and diffuse T1 hyperintense liver lesions and providing a characterization through their specific influence of T1 relaxation time.

Knowledge of the T1 signal characteristics of substances such as fat and hemoglobin degradation products allow the radiologist to perform a correct diagnosis.
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