

Computed Tomography and Magnetic Resonance features of focal hepatic steatosis and focal fatty sparing of the liver

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Authors: S. Palmucci, L. A. Mauro, G. Failla, A. Sigona, C. Trombatore, R. O. A. Siverino, G. Cappello, P. Milone, G. C. Ettore; Catania/IT
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Learning objectives

To describe the morphological features of focal hepatic steatosis and focal fatty sparing of the liver, evaluated on Multidetector Computed Tomography (MDCT) and Magnetic Resonance (MR).

Background

Focal hepatic steatosis and focal fatty sparing frequently occur in the liver; their typical distribution in the parenchyma, along the periportal spaces, in the pericholecystic regions and subcapsular areas ([Fig.1](#)) on page , has been well explained by a different vascular supply in these anatomic sites, due to [small perforating vascular branches and capsular veins](#) on page . From February 2008 to November 2010 we collected 17 MR and 17 MDCT exams with focal hepatic steatosis and focal fatty sparing of the liver.

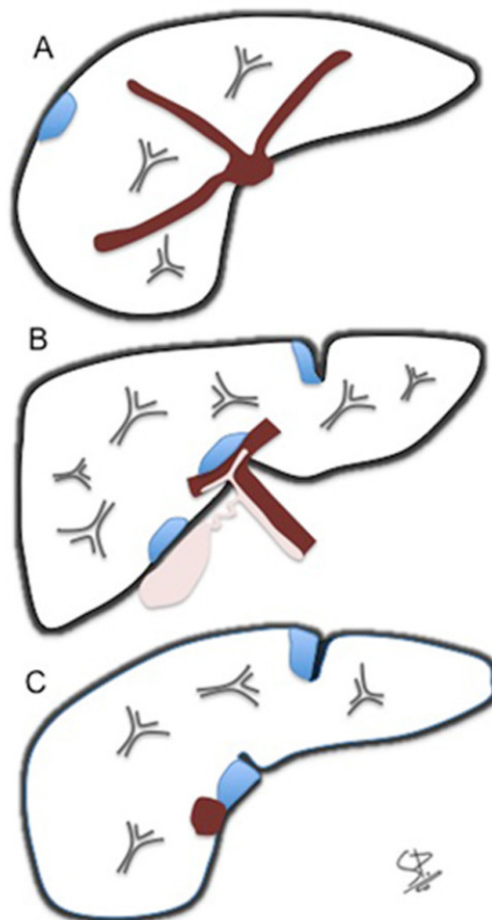


Fig.: 1. Typical distribution of focal hepatic steatosis and focal fatty sparing (blue areas) in the liver parenchyma.

References: S. Palmucci; Maternal-Child and Radiological Sciences Department, Radiology Institute, Catania, ITALY

"Small Systemic Veins Entering the Liver (SSVEL)"

The main vascular supply of the liver is performed by the hepatic artery and the portal vein (respectively 25-30% and 70-75% of liver perfusion). In addition, liver parenchyma has other sources of vascular provision, which are usually not visible in MDCT and MRI imaging.

Focal fatty infiltration and sparing may be explained by the presence of this accessory vascular supply, represented by the following small systemic veins entering the liver ([Fig.2](#)) on page : paraumbilical veins, cholecystic veins, parabiliary veins and capsular veins.

SSVEL	Sistemic ↔ Portal and Umbilical Connection	Liver segment or area
Superior veins of Sappey	Veins of median arcuate ligament of the diaphragm ↓ veins in the upper part of the falciform ligament	II-IV (adjacent the falciform ligament)
Inferior veins of Sappey	epigastric and cutaneous veins ↓ veins in the lower part of falciform ligament	II-IV (adjacent the falciform ligament)
Veins of Burow	epigastric veins ↓ umbilical veins	II-IV (adjacent the falciform ligament)
Cholecystic veins	cholecystic veins ↓ intrahepatic portal system	V segment (pericholecystic area)
Parabiliary veins	pyloric veins ↓ hepatic hilum	I-II-IV
Capsular veins	inferior phrenic vein ↓ intrahepatic portal system	II-IV-VII-VIII (sub-diaphragmatic areas)

Fig.: 2. This table shows the different small systemic veins entering the liver (SSVEL), and includes a schematic view of the connections between systemic, portal and umbilical vessels.

References: S. Palmucci; Maternal-Child and Radiological Sciences Department, Radiology Institute, Catania, ITALY

Paraumbilical veins (Fig.3) on page can be divided into three groups: the superior and inferior veins of Sappey and the veins of Burow.

The superior veins of Sappey drain the median arcuate ligament of the diaphragm and reach the convex surface of the liver through the upper part of the falciform ligament. The inferior veins of Sappey connect the epigastric and cutaneous veins with the portal system in the anteroinferior part of the liver parenchyma, and are located medially and laterally to the falciform ligament. This type of drainage may be one of the main causes of focal fatty infiltration around the falciform ligament.

The veins of Burow derive from epigastric veins and reach the umbilical veins without a direct connection with the intrahepatic portal system.

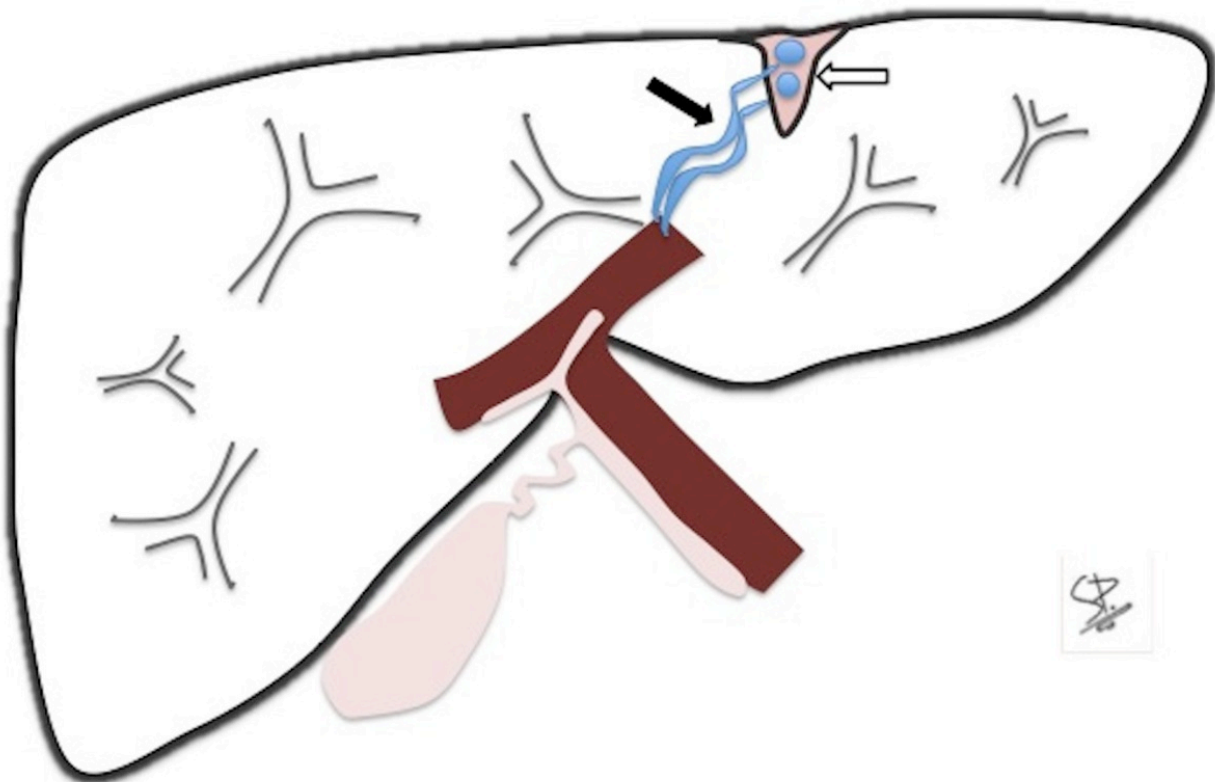


Fig.: 3. Connection between left portal system and paraumbilical veins (black arrow), next to the falciform legament (empty white arrow).

References: S. Palmucci; Maternal-Child and Radiological Sciences Department, Radiology Institute, Catania, ITALY

The **cholecystic veins (Fig.4)** on page are located around the gallbladder and are connected to the portal system by very small veins generally placed into the V segment or in the gallbladder fossa.

Cholecystic Veins

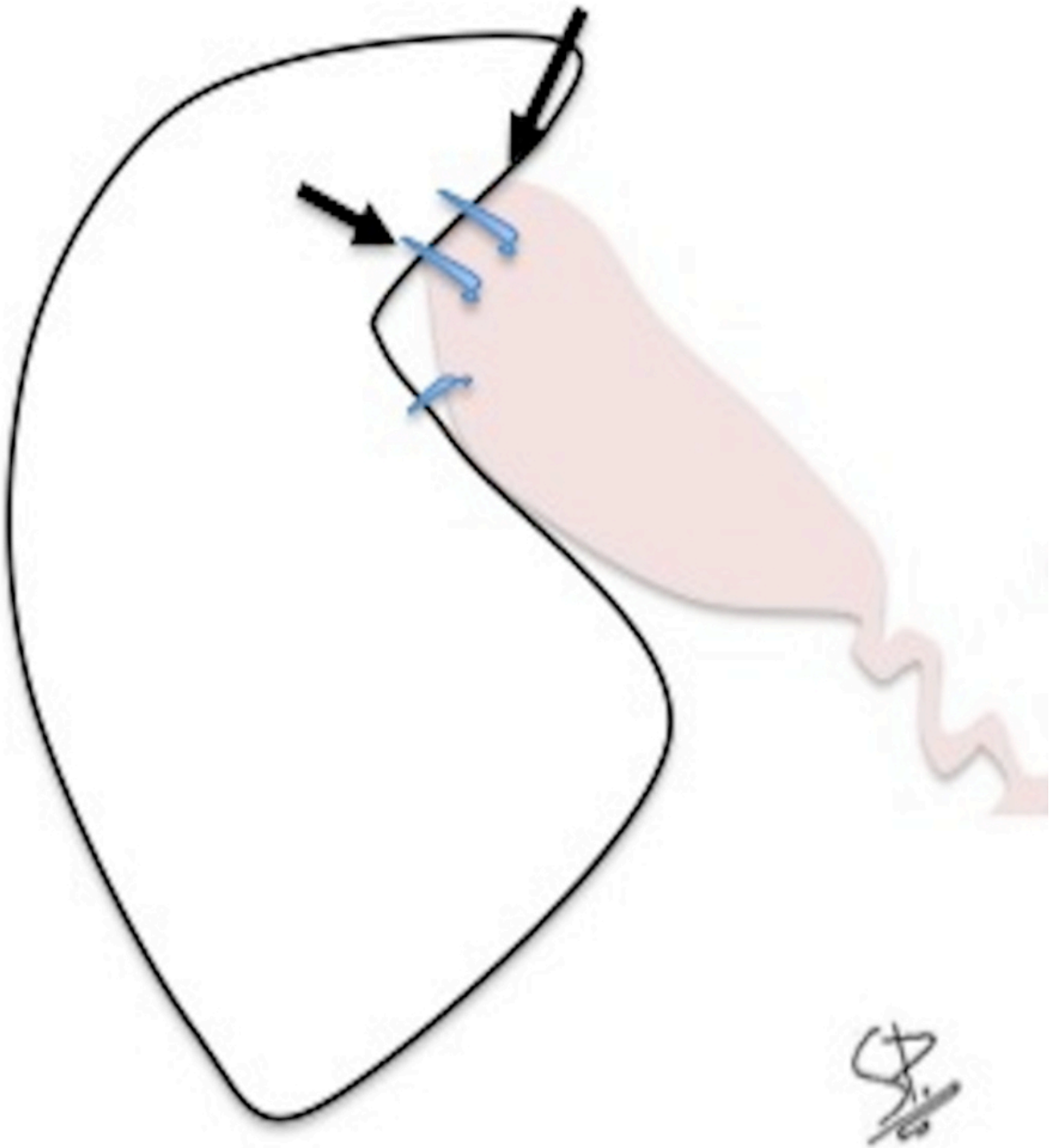


Fig.: 4. Representation of Cholecystic Veins. These small veins are located around the gallbladder and are connected to the intrahepatic portal system by very small veins (black arrows).

References: S. Palmucci; Maternal-Child and Radiological Sciences Department, Radiology Institute, Catania, ITALY

The **parabiliary veins** originate from pyloric veins around the head of the pancreas and duodenum. They reach the hepatic hilum through the hepatoduodenal ligament, and can be responsible for focal fatty infiltration in the dorsal part of the IV, II or I hepatic segments.

The **capsular veins** connect the inferior phrenic vein with the intrahepatic portal system. This "third inflow" may cause focal hemodynamic changes and, as a consequence, subcapsular steatosis.

Imaging findings OR Procedure details

Given the fact that focal hepatic steatosis and fatty sparing may mimic "true lesions" in the liver, it is important to know their typical imaging findings to easily characterize them.

In our exams, focal hepatic steatosis and focal fatty sparing are encountered in subcapsular regions ([Fig.1](#)) on page 10, along the pericholecystic region ([Fig.2](#)) on page 11 and periportal spaces ([Figg.3](#) on page 12 - [4](#)) on page 13. This typical localization is the first helpful mark to allow their identification and characterization; sites of localization are almost always dependent on the different vascular supplies discussed in the previous section.

Another main characteristic finding on MDCT and MR images is a "non-infiltrative pattern": they never cause alterations of the liver capsule, nor do they cause any bulging ([Fig.5](#)) on page .

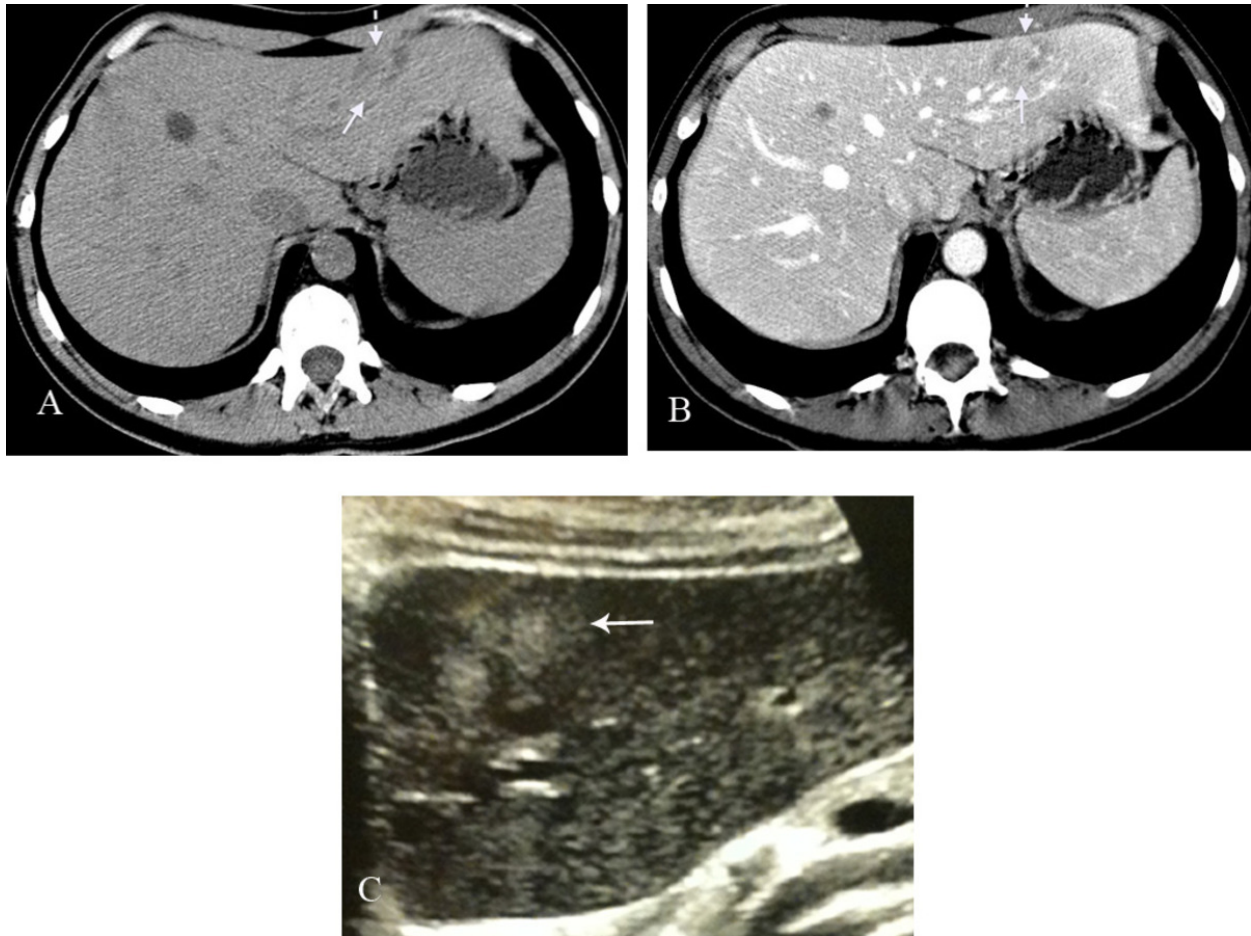


Fig.: 5. Patient with focal hepatic steatosis. Axial unenhanced multidetector computed tomography (MDCT) image (A). Axial enhanced MDCT image (B), portal phase. Ultrasound scan of left lobe liver (C). The figures show ill-defined hypodense areas (white arrows) in the left liver lobe, without any enhancement after contrast administration: they do not cause capsular bulging of the liver nor do they cause any mass effect on adjacent parenchyma (dotted arrows). In the follow-up, an ultrasonography exam confirmed the described areas in the left lobe.

References: S. Palmucci; Maternal-Child and Radiological Sciences Department, Radiology Institute, Catania, ITALY

In fact, in a previous study by Soyer et al., capsular retraction was found in 32% of patients with true lesions, against a mere 9% of patients with pseudolesions. In addition, in a review by Hamer et al. the absence of capsular bulging or mass effect on adjacent parenchyma has been reported as a significant item for fatty pseudolesions, enabling one to distinguish them from true masses. Moreover, although their shape is often poorly defined, images acquired after contrast administration do not show a distortion of the liver vessels (Fig.6) on page within the focal fatty area.

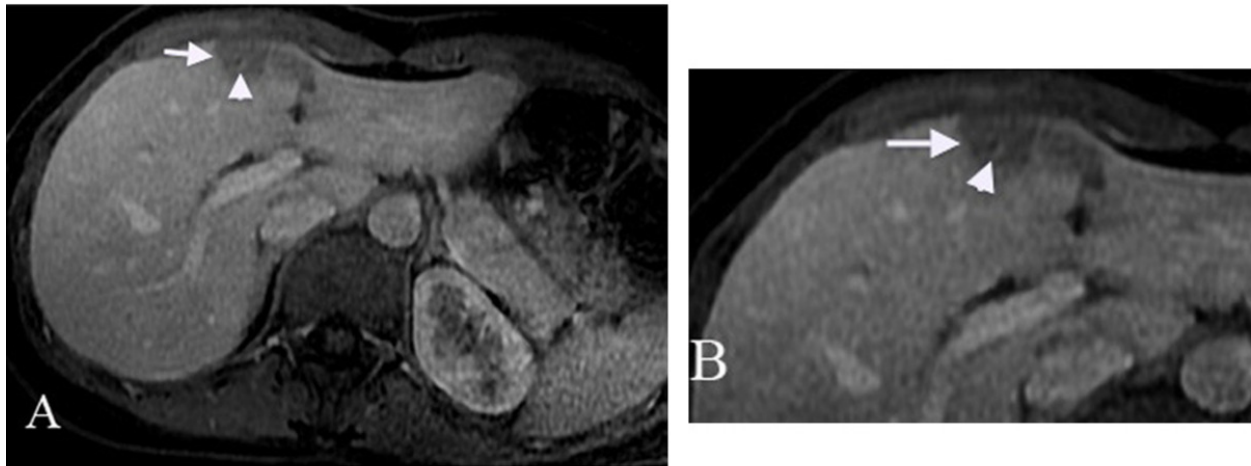


Fig.: 6. Chemical shift imaging technique, axial in and out-phase acquisitions (A and B respectively). T1-weighted out-of-phase image shows an inhomogeneous pattern of signal intensity in the liver parenchyma: there is a wide area of steatosis, which mainly involves the right lobe, whereas a fatty sparing area - suggested by the absence of any drop of signal - can be detected next to caudate lobe (B), adjacent to inferior cava vein (white arrow).

References: S. Palmucci; Maternal-Child and Radiological Sciences Department, Radiology Institute, Catania, ITALY

The chemical shift imaging technique allows a good differentiation of focal fatty areas on T1-weighted in and out-of-phase sequences (Fig.7) on page , due to the presence of equimolar amounts of water and lipid inside hepatic cells.

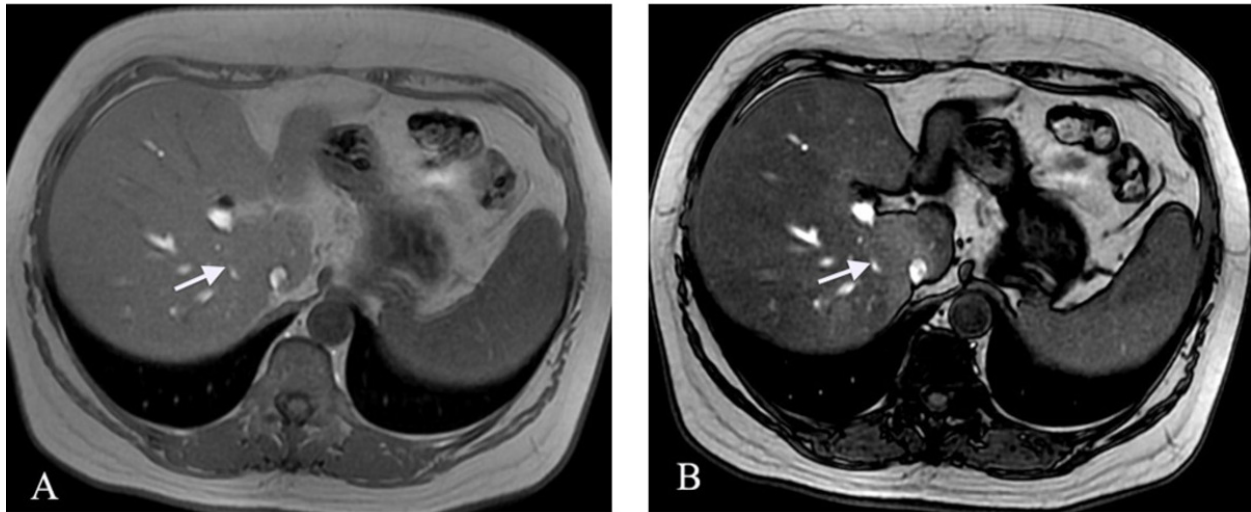


Fig.: 7. Patient with subcapsular focal hepatic steatosis; axial enhanced MR image (A), acquired in the portal phase after contrast administration; this image is shown in detail in figure B. A subcapsular hypointense area (white arrows in A and B) is depicted in the II and IV hepatic segments, adjacent to the falciform ligament. This subcapsular hypointensity does not swell the anterior liver contour: a vessel (arrowheads) flows through this region without any distortion.

References: S. Palmucci; Maternal-Child and Radiological Sciences Department, Radiology Institute, Catania, ITALY

On MR images, the signal drop off on T1-weighted out-of-phase acquisitions, due to intracellular fat content, is an important diagnostic tool to identify focal hepatic steatosis and focal fatty sparing (Fig.8) on page of the liver.

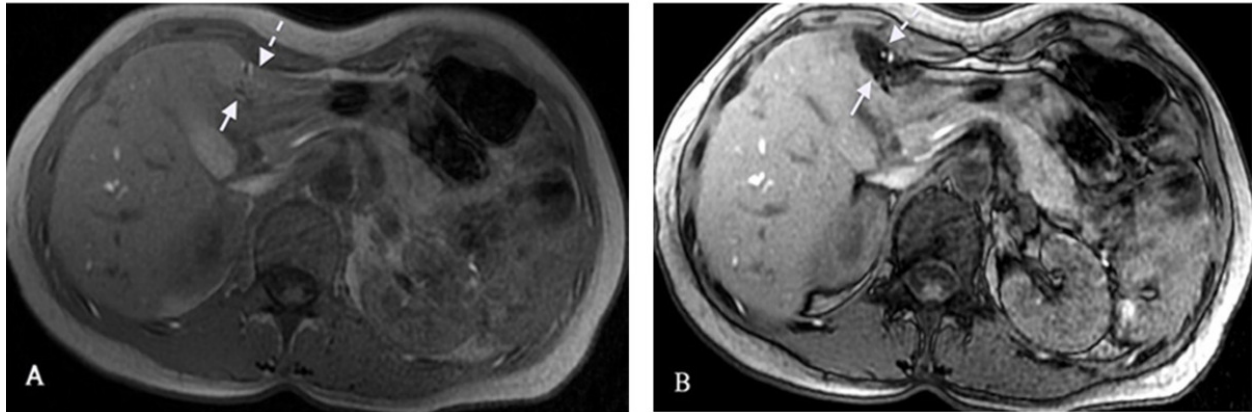


Fig.: 8. Subcapsular focal hepatic steatosis (same patient as in previous images); axial in-phase (A) and out-of-phase (B) T1-weighted MR acquisitions. The subcapsular focal hepatic steatosis (white arrows), adjacent to the falciform ligament, shows a typical signal drop in out-of-phase image (B), suggesting its intracellular content.

References: S. Palmucci; Maternal-Child and Radiological Sciences Department, Radiology Institute, Catania, ITALY

As a consequence, focal hepatic steatosis appears isointense or hyperintense to the liver parenchyma on in-phase images and loses the signal intensity on out-of-phase images. Conversely, on T1-weighted out-of-phase images fatty sparing is characterized by the absence of signal drop, in a uniformly fatty hypointense liver.

Images for this section:

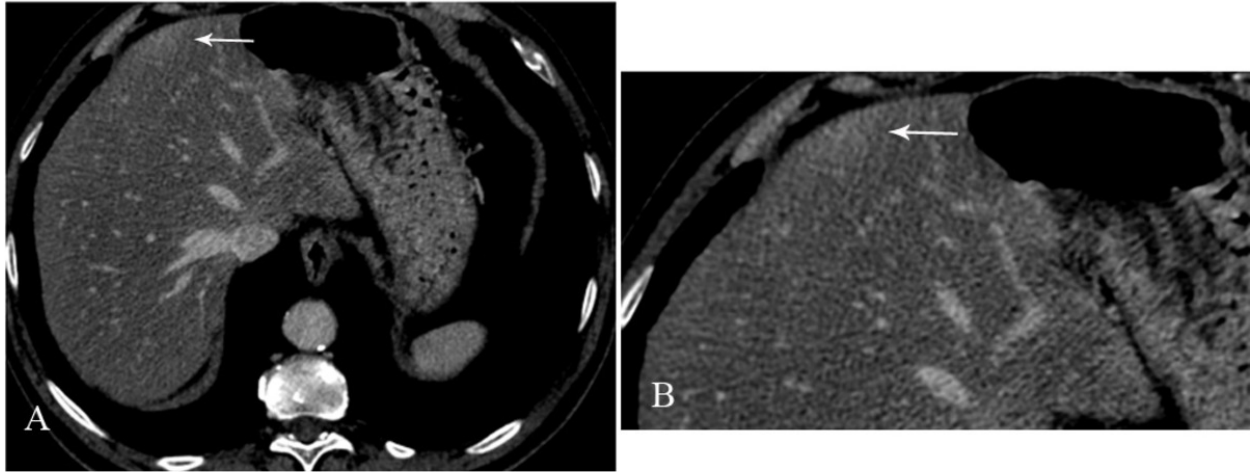


Fig. 1: Patient with subcapsular fatty sparing area. Axial enhanced MDCT images, portal phase (A-B). A small subcapsular hyperdense area is depicted (white arrow) in the liver (IV segment), and is due to a focal fatty sparing area in a diffuse liver steatosis.

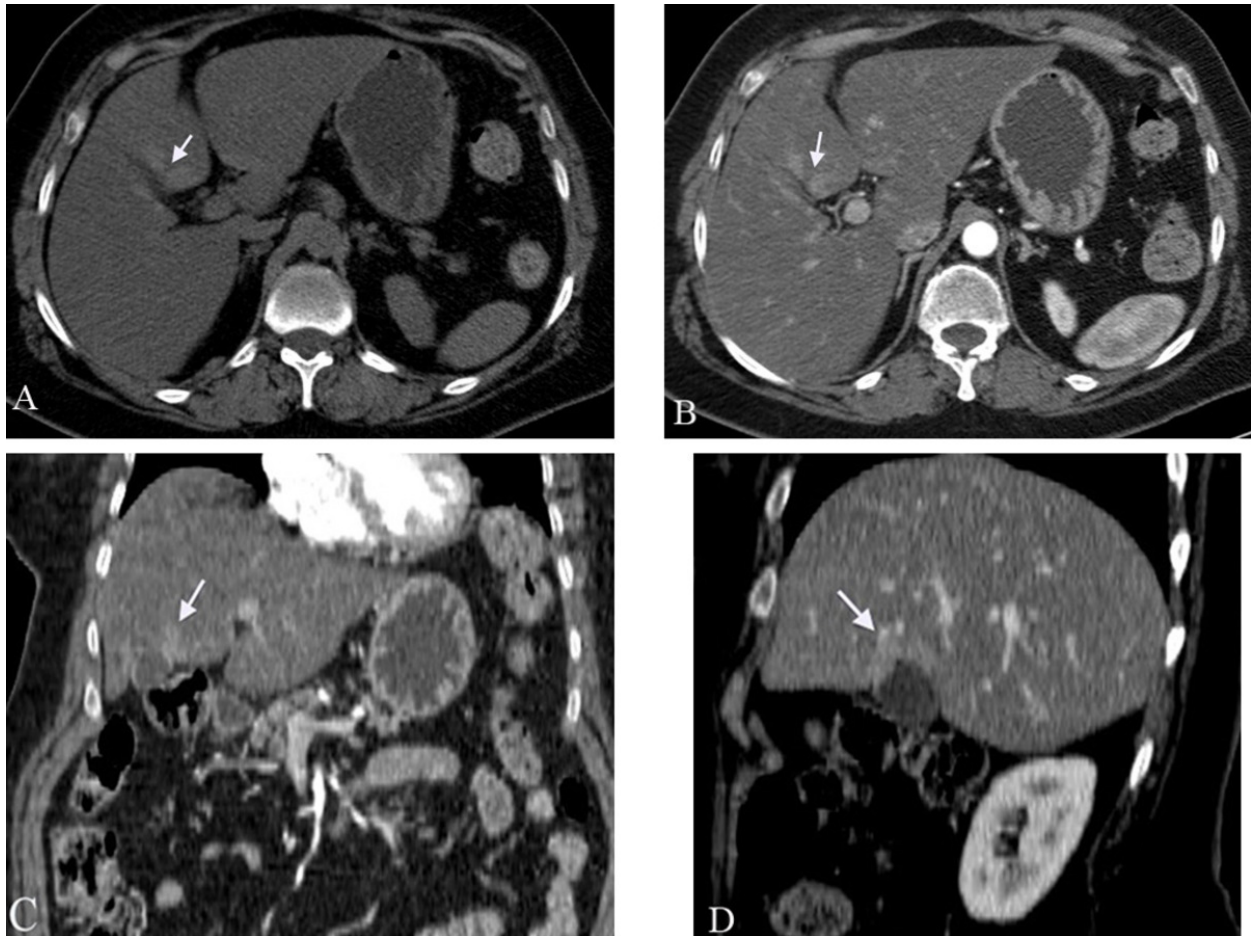


Fig. 2: Patient with typical peri-cholecystic fatty sparing area. Axial unenhanced MDCT image (A). Axial enhanced MDCT image, arterial phase (B); multiplanar reformatted MDCT image, arterial phase (C); multiplanar reformatted MDCT image, portal phase (D). In figure A liver steatosis appears as a diffuse low-attenuation of the liver parenchyma. An ill-defined area, with higher attenuation than surrounding parenchyma (white arrows in B, C, D), is depicted in the peri-cholecystic region, referred to as a fatty sparing area.

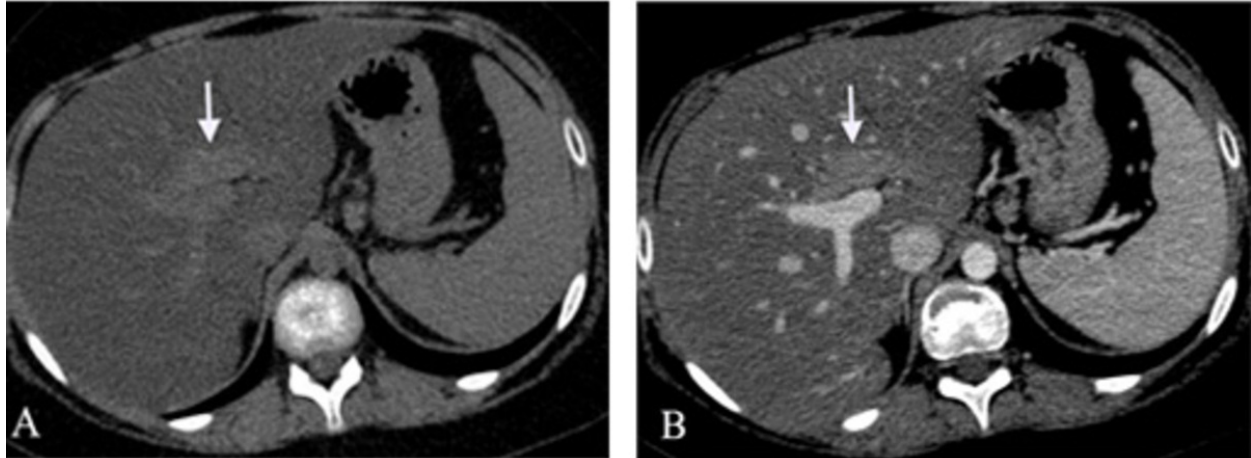


Fig. 3: Patient with typical periportal fatty sparing area. Axial unenhanced MDCT image (A); axial enhanced MDCT acquisition (portal phase). In figure A we observe a spread steatosis in the liver, revealed by a homogeneous low attenuation of the parenchyma; there is a fairly hyperdense area along the portal vessels, near the hepatic hilum (white arrow); this fatty sparing area is typically located in front of the portal bifurcation: its mildly increased attenuation does not change after contrast administration in the portal phase (B).

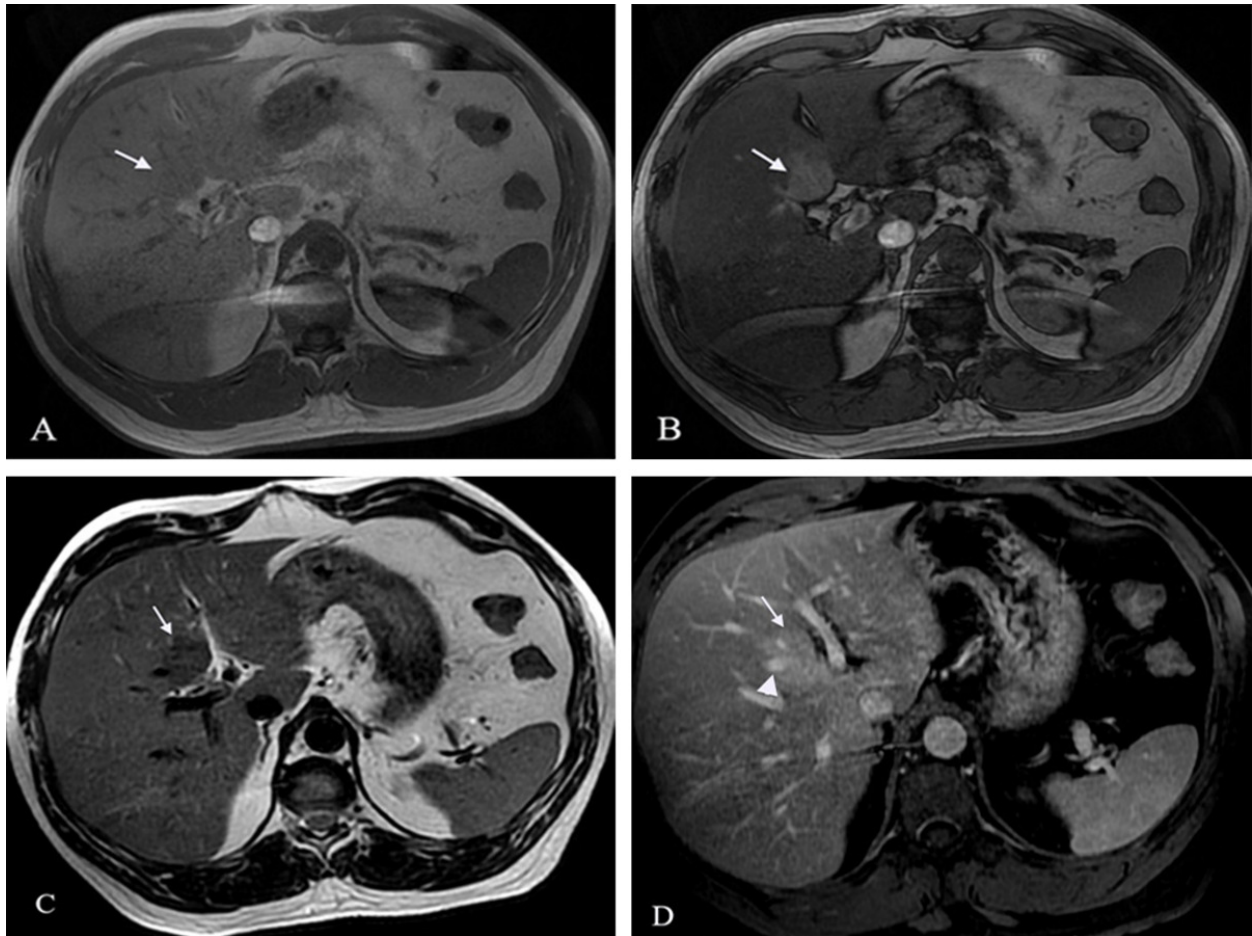


Fig. 4: Patient with typical periportal fatty sparing area. Axial T1-weighted in-phase and out-of-phase MR acquisitions (A and B respectively). Axial T2-weighted image (C) and T1-weighted enhanced image, acquired in the portal phase after contrast administration. Diffuse drop of signal of liver parenchyma in out-of-phase acquisition, except for a fatty sparing area in the IV segment (white arrow), located in front of the portal bifurcation, which remains slightly hyperintense. Axial T2-weighted image reveals moderate hypointensity of this fatty sparing area (white arrow); the portal phase shows two vessels (arrowhead) flowing through the fatty sparing area, suggesting the absence of any infiltrative pattern.

Conclusion

With reference to focal hepatic steatosis and focal fatty sparing, knowing their morphological features is important to make a differential diagnosis from other lesions, especially from metastases in oncological patients.

Personal Information

Stefano Palmucci, MD

spalmucci@sirm.org

Section of Radiological Sciences - Maternal-Child and Radiological Sciences Department

University Hospital "Policlinico-Vittorio Emanuele"

Catania - Italy

Letizia Antonella Mauro, MD

Imauro@sirm.org

Section of Radiological Sciences - Maternal-Child and Radiological Sciences Department

University Hospital "Policlinico-Vittorio Emanuele"

Catania - Italy

Giovanni Failla, MD

failla.giovanni@gmail.com

Section of Radiological Sciences - Maternal-Child and Radiological Sciences Department

University Hospital "Policlinico-Vittorio Emanuele"

Catania - Italy

Alessandra Sigona, MD

ariel833@hotmail.it

Section of Radiological Sciences - Maternal-Child and Radiological Sciences Department
University Hospital "Policlinico-Vittorio Emanuele"
Catania - Italy

Claudia Trombatore, MD
claudiatr84@libero.it

Section of Radiological Sciences - Maternal-Child and Radiological Sciences Department
University Hospital "Policlinico-Vittorio Emanuele"
Catania - Italy

Rita Olivia Anna Siverino, MD
ritasi84@hotmail.it

Section of Radiological Sciences - Maternal-Child and Radiological Sciences Department
University Hospital "Policlinico-Vittorio Emanuele"
Catania - Italy

Giuseppina Cappello, MD
giuseppina.cappello@gmail.com

Section of Radiological Sciences - Maternal-Child and Radiological Sciences Department
University Hospital "Policlinico-Vittorio Emanuele"
Catania - Italy

Pietro Milone, MD, Researcher
pietromilone@tin.it

Section of Radiological Sciences - Maternal-Child and Radiological Sciences Department
University Hospital "Policlinico-Vittorio Emanuele"
Catania - Italy

Giovanni Carlo Ettore, Professor

gc.ettore@policlinico.unict.it

Section of Radiological Sciences - Maternal-Child and Radiological Sciences Department

University Hospital "Policlinico-Vittorio Emanuele"

Catania - Italy

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