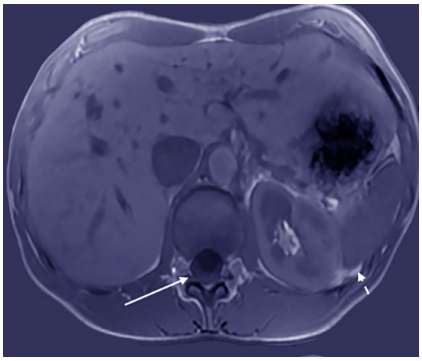
Mistakes in imaging hepatic lesions and how to avoid them

Katja De Paepe

Incidental liver lesions are increasingly found due to the incremental use of cross-sectional imaging. They encompass a large group of benign and malignant lesions, and the combined use of different imaging modalities is often required to make an accurate diagnosis. It is of utmost importance for clinicians and radiologists to be familiar with each imaging modality's strengths and limitations and be aware of common pitfalls that can confound the correct interpretation of findings.

The following article will discuss eight common mistakes in the interpretation and acquisition of radiological images. Recommendations on avoiding these mistakes will be based on clinical experience and literature where possible. As MRI plays an essential role in the characterisation of liver lesions, a standard MRI protocol with a brief explanation of the sequences has been added for reference (figures 1 and 2).



Mistake 1 Overestimating the ability of US to detect liver lesions in cirrhotic patients

Patients with cirrhosis and non-cirrhotic HBV-positive patients are at high risk of developing hepatocellular carcinoma (HCC).[1] Surveillance for HCC in these target populations has shown an improved detection of early HCC, increased curative treatment rates, and improved overall survival.[2]

Ultrasonography (US) at six-month intervals remains the imaging modality of choice due to its easy accessibility, low cost, and non-invasive nature. Although the sensitivity of US to detect HCC amounts to an acceptable 84%, this decreases considerably to 47% for early HCC. [3]

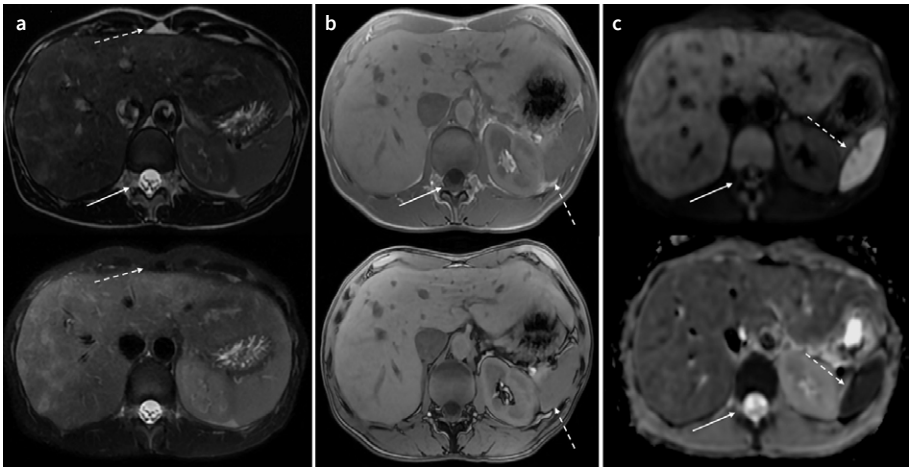


Figure 1 | Standard non-contrast MR sequences.(**a**) On T2-weighted images water will be bright (hyperintense), hence the high signal intensity of the cerebrospinal fluid (CSF). On fat-saturated T2 images the hyperintense signal of fat is suppressed (dashed arrow, prehepatic fat). T2 is the main sequence for the assessment of anatomy and pathological processes. (**b**) On T1, water is hypointense (arrow). T1 in-phase (IP, top figure) and out-of-phase (OOP, bottom figure) are routinely acquired. On the OOP images fat-water interfaces are hypointense, giving rise to the "Indian ink artefact" which refers to the black outlining of the organs (dashed arrow). T1 allows for the detection of specific entities such as blood, melanin, high protein content, microscopic fat and iron. (**c**) Diffusion-weighted images (DWI) are fat suppressed T2-weighted images which are optimised to highlight those structures and processes in which the diffusion of water molecules on a microstructural level is restricted (top figure), for instance tumour, while suppressing all background signal (arrow). Note that also some normal organs such as the spleen (dashed arrow) are hyperintense on DWI. From the DWI, apparent diffusion coefficient (ADC) maps can be calculated. Signal intensity is inversely related to the level of diffusion restriction, i.e. diffusion restriction will be dark (dashed arrow) and vice versa. DWI is an essential sequence in oncological imaging.

Furthermore, other factors such as body mass index and heterogeneity of the liver parenchyma can also significantly limit HCC detection (figure 3), resulting in surveillance failure in possibly up to 20% of patients.[4]

The Liver Imaging Reporting and Data System (LI-RADS) is a validated classification system of HCC and offers standardised terminology across disciplines. They have developed a three-point US visualisation score defining the quality of the US, ranging from no limitations in the assessment to severe limitations.[5] In the last category, a significant decrease in the detection rate of HCC and other imaging modalities should be considered. An increasing number of centres are applying an abbreviated MRI (AMRI) protocol in a subset of patients in whom US is deemed inadequate for HCC detection. AMRI consist of a few selected sequences allowing liver lesions detection. Protocols, including administering a contrast agent, attained a higher sensitivity (84-95%) to detect HCC compared to non contrast studies (62-86%).[6]

In summary, it is crucial to recognise that US detection of HCC may be compromised in patients who are obese or have difficulty holding their breath and in those with very fatty or heterogeneous livers. Alternative imaging modalities should be considered in these patients.

Mistake 2 Getting tricked by focal fat infiltration in the liver

Hepatic steatosis is estimated to be present in 20-30% of the Western population. [7] Fatty livers are present with highly variable patterns of fat distribution. Fat can diffusely infiltrate the liver,

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Katja De Paepe Affiliate assistant professor at Cambridge, is an abdominal radiologist with a special interest in gastrointestinal and hepato-pancreatico-biliary imaging at the. Department of Radiology, Cambridge University Hospitals, Hills Rd CB20QQ Cambridge UK.
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Correspondence to: katja.depaepe@nhs.net
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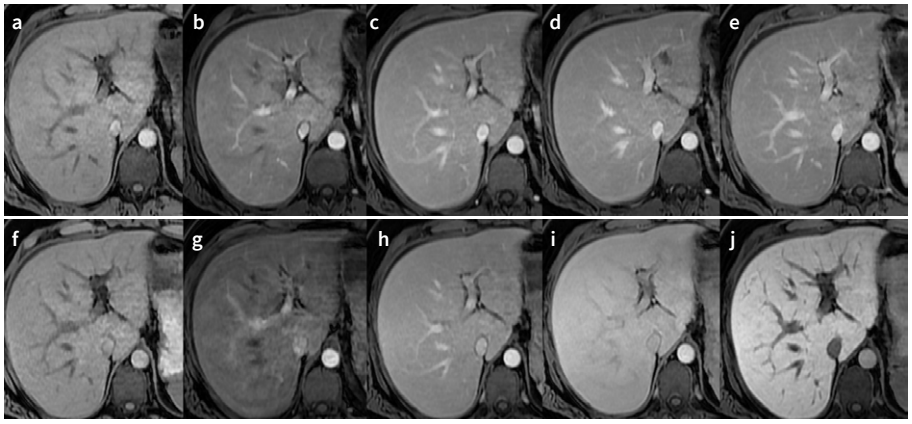

Figure 2 | Contrast-enhanced MR sequences with an extracellular and hepatocyte-specific IV contrast agent. Top row: (**a**-**d**) depict the different phases of MRI using an extracellular IV contrast, which is excreted by the kidneys. The protocol consists of a pre-contrast (**a**), arterial phase (**b**), portal venous (**c**), and two delayed venous phases (**d** and **e**). Note that in on all contrast-enhanced images the vessels are hyperintense relative to the background liver. Bottom row: (**f**-**j**) shows the phases of an MRI with hepatocyte-specific contrast. Pre-contrast (**f**), arterial (**g**) and portal venous phase (**h**) are similar to the top row. However, in the transitional phase (**i**), contrast is taken up by hepatocytes rendering the background liver parenchyma hyperintense compared to the vessels. In the hepatobiliary phase (**j**) contrast will have been excreted in the bile ducts. The latter sequence offers the best contrast between liver lesions and the normal background liver and has therefore the highest lesion detection sensitivity.

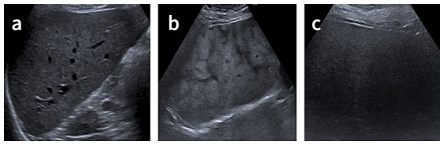

Figure 3 | Variability of ultrasound appearances of the liver. Normal ultrasound (US) appearances of the liver (**a**) with homogenous liver parenchyma and regular liver contours. However, lesion detection may be hampered when the background liver is heterogeneous (**b**) or when the penetration of US waves is limited due to the presence of marked hepatic steatosis (**c**). Other patient-related factors such as body habitus and inability of the patient to hold their breath can further limit the exam.

but other patterns, such as geographical or nodular fat infiltration or sparing, can also be found. Focal fat deposits with a mass-like or nodular appearance that can mimic focal solid liver lesions and pose a diagnostic conundrum.

Common regions of focal fat accumulation are near the gallbladder, the falciform ligament, the posterior segment four and the anterior segment. [1] Focal fat deposition in these areas has been attributed to variations in the venous supply and drainage.[8] Alcohol abuse, diabetes, hypoxia, or drug-induced changes have also been proposed as causes of focal fat deposition. [9]

More atypical presentations of focal fat are frequently found in US and CT. Diagnosing focal fat can be difficult on these imaging modalities, and an additional MRI for further lesion characterisation is often mandated. Two helpful imaging features are 1) the lack of mass effect on the blood vessels and bile ducts and 2) a geographical shape (figure 4). The latter means that the lesion has irregular contours, resembling the contours of a continent on a map, rather than being mass-like. However, MRI can more definitely solve whether a lesion is real or reflects focal fat. All routine MRI livers include in-phase (IP) and out-of-phase (OOP) T1 images (figure 2), which can demonstrate the presence of microscopic fat or iron.

The liver parenchyma has the same signal intensity in normal livers on both IP and OOP images. However, in hepatic steatosis, triglycerides accumulate within the hepatocytes, resulting in increased intracellular fat, which will be apparent as regions of signal loss on the OOP compared to the IP images. Several hepatic lesions, such as hepatocellular carcinoma or hepatic adenomas, may contain fat. Correlation with other MRI sequences such as diffusion-weighted images (DWI) and contrast-enhanced sequences can help arrive at the proper diagnosis, as focal fat – unlike primary or secondary liver lesions – will not demonstrate diffusion restriction or enhancement.

Ultimately, focal liver fat is highly prevalent and can be present in peculiar patterns, sometimes mimicking sinister lesions. Familiarity with classic areas of focal fat infiltration and typical imaging features can frequently obviate an additional MRI. MRI can quickly and confidently confirm the presence of focal fat in atypical cases.

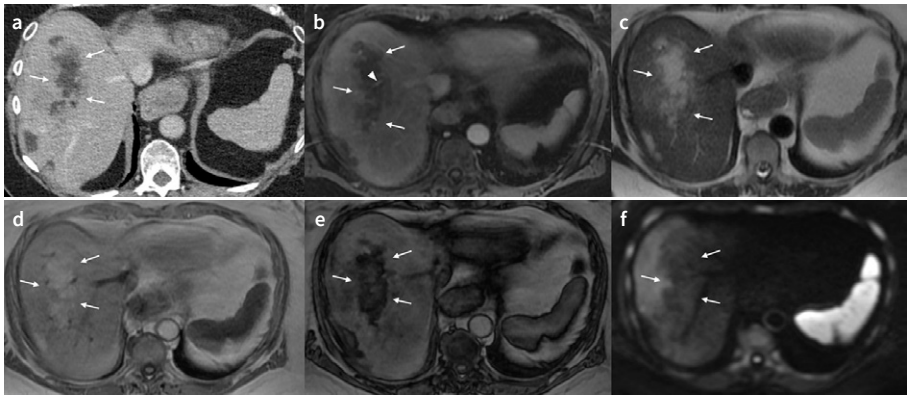

Figure 4 | Focal fat simulating a liver lesion. (**a**) CT showed numerous geographical areas (arrows) which were also demonstrated on MRI. (**b**) On the contrast-enhanced MRI sequences, vessels course through the area and the 'lesion' is not enhancing. On T2 (**c**) and T1 IP (**d**) the area is hyperintense and shows a signal drop on the T1 OOP images (**e**) in keeping with fat. DWI (**f**) is a fat-suppressed sequence and as such the fatty area is hypointense.

Mistake 3 Presuming routine CT can detect hypervascular lesions

Hypervascular primary and secondary liver lesions are less common and easily missed on routine cross-sectional imaging if not optimised for their detection.

An increased arterial supply is a well-established feature in hepatocellular carcinoma (HCC), and arterial hyperenhancement (APHE) is arguably the most important criterion for diagnosing HCC. This is also illustrated in the LI-RADS diagnostic table, [10] where an observation without APHE can, at most, be categorised as a probable HCC but never as a definite HCC. However, to allow for an accurate assessment of APHE, precise timing of the arterial phase is imperative. During the early arterial or angiographic phase, contrast pools in the aorta and large arteries and has not reached the solid organs. Consequently, the capillaries of the HCC are not yet filled with contrast, and APHE may not be evident, particularly in smaller lesions. The optimal timing to detect APHE in both primary and secondary liver lesions is in the late arterial phase (figure 5), where contrast is present within the portal vein but not in the hepatic veins.

CT is the working horse for staging and monitoring disease recurrence or progression in oncological patients. CT of the abdomen and pelvis is generally acquired in the portal venous phase to allow for the best assessment of the solid abdominal organs. However, there are certain cancer types that characteristically give rise to hypervascular metastases, such as renal cell carcinoma (RCC), neuroendocrine tumours (NET) and melanoma. Moreover, these tumours metastasise to relatively uncommon sites such as the pancreas and subcutaneous tissues, which

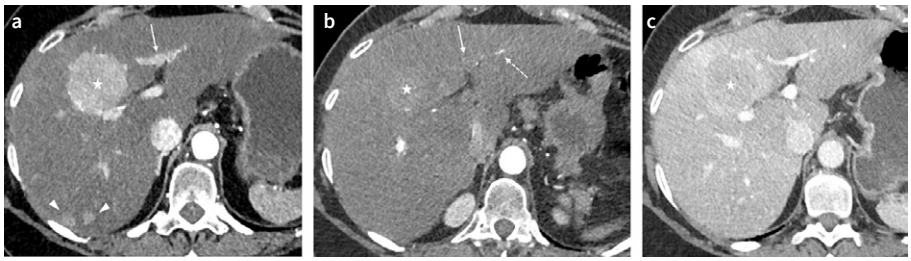

Figure 5 | Presentation of hypervascular liver metastases in a patient with a neuro-endocrine tumour. (**a**) CT in the late arterial phase depicts a large liver lesion in segment 4/5 (star) and multiple small lesions in the posterior liver (arrowheads). (**b**) In the angiographic phase, there is opacification of intrahepatic artery branches (dashed arrow) but not of the portal vein (arrow). The index lesion is faintly visible, but the remaining liver metastases are undetectable. They are not visible on the portal venous phase CT images either (**c**).

are notoriously difficult to detect. Therefore, multiphase CT/MRI, including an arterial phase, should be the standard for assessing these hypervascular tumour types.

To conclude, routine portal venous phase CT can underestimate the disease burden in hypervascular tumours. The addition of an appropriately timed arterial phase will improve lesion detection both in CT and MRI.

Mistake 4 Scanning too early after locoregional treatment of a liver lesion

Liver metastases and primary liver tumours are widely managed with locoregional therapy. These can roughly be divided into three main categories: ablative techniques, trans-arterial treatments, and stereotactic body radiotherapy (SBRT).[11,12]

Thermal ablation techniques such as radiofrequency (RFA) or microwave ablation (MWA) are usually reserved for solitary smaller lesions. Lesions are destroyed by applying extreme heat, causing coagulation necrosis, resulting in typical post-treatment changes (figure 6). Arterial hyperenhancement in keeping with post-treatment inflammation around the ablation site or haemorrhage within the ablated lesion can persist for at least three months[13] and hamper accurate treatment response assessment.

Trans-arterial treatment involves selective catheterisation of the hepatic artery branches supplying the liver lesion, after which embolic agents (TACE) or without chemotherapy (TAE) are administered. Alternatively, radioactive Yttrium-90 particles can be injected, referred to as Selective Internal Radiotherapy (SIRT). Like RFA/MWA, residual arterial enhancement related to inflammation or granulation tissue can be seen after treatment. These changes tend to be more florid and persist long after SIRT, requiring a post-treatment period of at least three-six months to allow for adequate assessment (figure 7).

SBRT can be a valuable palliative treatment option in selected patients with colorectal liver metastases or HCC. Thanks to developments in radiotherapy planning, high doses of radiotherapy can be administered to specific lesions without causing excessive damage to the surrounding liver parenchyma. Nevertheless, post-treatment liver changes are extensive and evolve from acute inflammation to chronic changes over at least twelve months. A paradoxical increase in lesion size can be seen in the early phase (< three months).

After locoregional therapy of a liver lesion, post-treatment changes can persevere for a considerable time, rendering early treatment response assessment unreliable. A conservative approach is advisable in case of an ambiguous response, and reintervention should be reserved for those showing certain residual or progressive diseases.

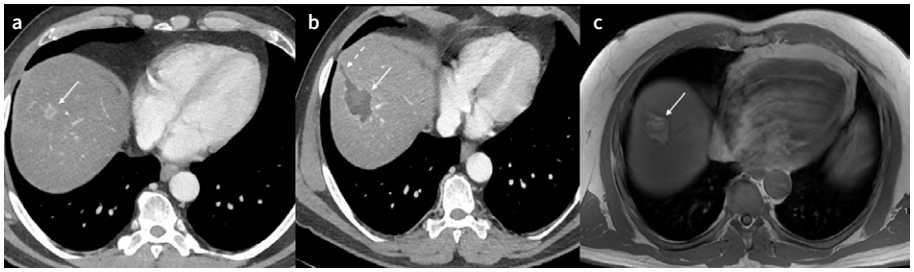

Figure 6 | Satisfactory post-treatment appearances of a liver metastasis treated with radiofrequency ablation (**a**) The pre-treatment CT shows a small liver metastasis in segment 8 (arrow). (**b**) Post-treatment CT shows a large hypodense area covering the liver metastasis. Also note the residual needle track (dashed arrow). (**c**) On MRI, the ablation cavity typically returns high signal, due to high protein content and (sometimes) blood products, with hypointense rim. Hypointense nodularity arising in the rim would be highly suspicious for recurrent disease.

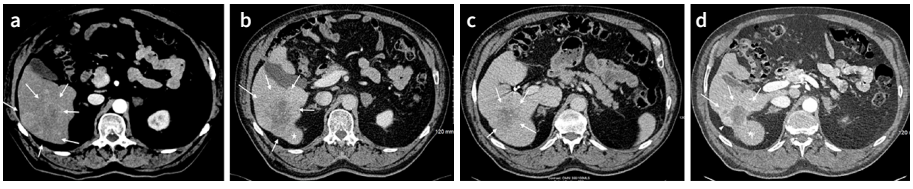

Figure 7 | Post-treatment appearances of a hepatocellular carcinoma treated with SIRT. (**a**) The pre-treatment CT demonstrates a large ill-defined and faintly hypervascular lesion occupying segment 6 (arrows). (**b**) On the first post-SIRT CT after 3 months, the liver lesion hasn't changed significantly in size or appearance. (**c**) On the subsequent CT performed 7 months post-treatment, there is clear shrinkage of the liver reflecting partial response to treatment. (**d**) A CT performed 8 months later demonstrates further size decrease of the lesion. However, there is reappearance of a lateral nodule with arterial hyperenhancement (arrowhead) in keeping with recurrent viable disease. A posterior nodule has increased in size (star) but is not enhancing and most likely related to treatment-related remodelling of the liver.

Mistake 5 Assuming every lesion in the liver is of hepatic origin

Extrahepatic lesions arising from organs or tissue near the liver may mimic genuine hepatic lesions due to their mass effect or infiltrative behaviour. Familiarity with typical presentation patterns of common confounding lesions and awareness of the appropriate clinical context is vital.

Peritoneal metastases are by far the most frequent extrahepatic lesions involving the liver. Due to the compartmentalisation of the abdomen by peritoneal ligaments and the directional flow of peritoneal fluid, tumour deposits typically precipitate in the suitable subphrenic space, liver surface and hepatorenal recess. [14] Usually, multiple lesions cover the liver surface, infiltrating the liver capsule and potentially underlying liver parenchyma. The presence of a primary tumour, ascites and metastatic disease at other sites are useful clues. However, in some cases, no primary mass is present, such as in primary peritoneal carcinoma or pseudomyxoma peritonei. [15] Of note, not all liver surface lesions are malignant, as endometriosis can also present as a focal subcapsular liver deposit. [16] Therefore, this diagnosis should always be considered in premenopausal women with haemorrhagic subcapsular liver lesions.

As the liver occupies the entire right upper quadrant, not surprisingly, growing lesions arising from right renal and adrenal origin will often compress or involve the adjacent liver. Rarer entities to bear in mind are nerve sheath tumours and lymphoma. The first and most crucial step is to carefully assess the lesion's shape and its extension beyond the

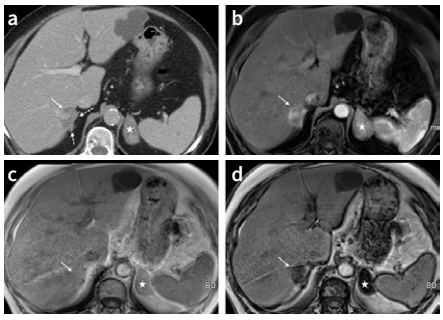

Figure 8 | A benign adrenal lesion mimicking a liver metastasis. (**a**) The staging CT of this patient with a pancreatic cancer shows a lesion in the posterior liver (arrow) in proximity of the right adrenal (dashed arrow). It demonstrates enhancement on the (**b**) contrast-enhanced MR images but also a signal drop on the (**c**) T1 out-of-phase images compared to (**d**) T1 in-phase images in keeping with intracellular fat. These imaging features would not fit with a pancreatic liver metastasis but are consistent with a benign adrenal lesion such as an adrenal myelolipoma. Also note the lipid-rich adenoma in the left adrenal gland (star).

liver. Next, one needs to look at the intralesional imaging characteristics. A purely fluid-containing lesion will most often reflect a renal cyst, whilst the presence of fat within the lesion is highly suggestive of a benign adrenal lesion such as an adenoma or myelolipoma (figure 8).[17,18]

In conclusion, distinguishing a true liver lesion from an extrahepatic lesion involving/compressing the liver is important as the treatment plan is generally different. Usually, the extrahepatic lesions are located in the periphery of the liver, and the vast majority will have imaging appearances uncharacteristic of true liver lesions.

Mistake 6 Mislabelling a haemangioma as a malignant lesion and vice versa

Hepatic haemangiomas are very common benign vascular lesions and may be prevalent in up to 20% of the adult population.[19] A typical haemangioma has a high signal on T2-weighted images and demonstrates discontinuous peripheral nodular enhancement with centripetal filling-in in the delayed venous phase (figure 9). Rapid-filling, giant and sclerosing haemangiomas are considered atypical haemangiomas and can be challenging to differentiate from malignant lesions. [20]

Rapid-filling haemangiomas comprise 42% of small haemangiomas (< 1 cm). In the arterial phase, the entire lesion has a homogeneous enhancement, resembling hypervascular metastasis. DWI is not reliable due to the small size of the lesion, but persistent enhancement in the delayed venous phase may be a helpful imaging feature of benignity. In oncological patients with suspected liver lesions, an MRI liver with a hepatocyte-specific contrast agent is often performed because of its high sensitivity (93%) and specificity (95%) to detect liver metastases. [21] Compared to the standard extracellular contrast, excreted by the kidneys, hepatocyte-specific contrast is taken up by the hepatocytes and at least partially excreted via the bile ducts. An important pitfall is the pseudo-washout sign in rapid filling haemangiomas. [22] A haemangioma does not contain hepatocytes and will appear hypointense compared to the background liver (figure 10), possibly mimicking a sinister lesion. An MRI with extracellular contrast should be performed in these cases to exclude metastatic disease and avoid unnecessary surgery. PET/CT is a proper problem-solving method if diagnostic doubts persist, given that the lesion is large enough (at least 5-10 mm).

Giant haemangiomas appear very heterogeneous and can become quite large. The presence of the typical haemangioma e nhancement pattern allows for the diagnosis. However, if a lesion shows continuous rim enhancement and/or heterogenous T2 signal, one should remain cautious and include cholangiocarcinoma or metastasis in the differential diagnosis.

To summarize, uncommon presentations of common benign liver lesions can mimic metastases. If only an MRI with hepatocyte-specific contrast has been performed, it is highly desirable to get an additional MRI with extracellular contrast. PET/CT is also a valuable complementary imaging tool depending on the tumour type. Biopsy of possibly resectable liver metastases is not recommended because of seeding risk.

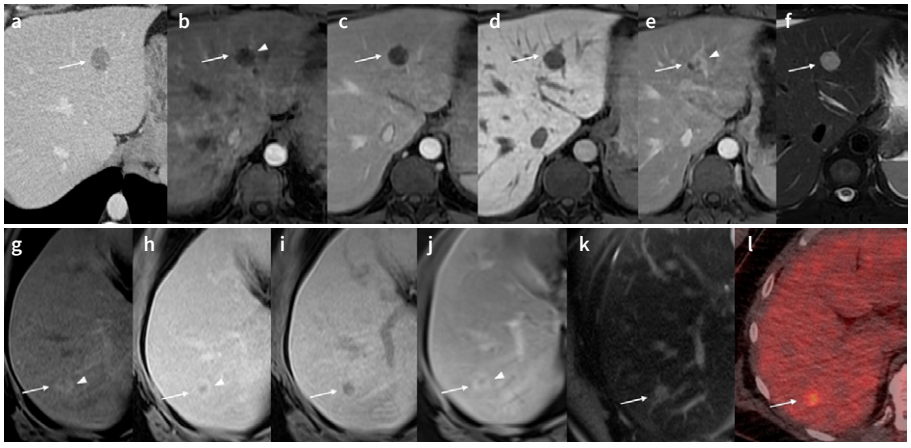

Figure 9 | Example of two colorectal cancer patients with a suspicious looking lesion on MRI with hepatocyte-specific contrast. Top row: (**a**) CT shows a relatively well-defined hypodense liver lesion (arrow) in a patient with T4 colon cancer and peritoneal metastases. (**b**) The MRI with hepatocyte-specific contrast shows a hypovascular lesion in the arterial phase with subtle nodularity in the margin (arrowhead) and (**c**) portal venous phase. (**d**) It remains hypointense in the hepatobiliary phase suggestive for a metastasis. (**e**) A subsequent MRI with extracellular contrast shows centripetal filling of the lesion and (**f**) on T2 it appears very well-defined and markedly hyperintense in keeping with a haemangioma. Bottom row: (**g**) On the MRI with hepatocyte-specific contrast performed in a patient with a T2 caecal tumour, a 10 mm lesion was found in segment 6 (arrow). It was hypervascular in the arterial phase with a target appearance in the (**h**) portal venous and (**i**) hepatobiliary phase, possibly reflecting pseudo washout in a flash filling haemangioma. (**j**) The additional MRI with extracellular contrast shows no filling in of the lesion, but a persistent target appearance (arrowhead). (**k**) On T2, it is relatively ill-defined, moderately hyperintense and heterogeneous. These imaging features are suggestive of a metastasis rather than a haemangioma. (**l**) This was confirmed on the PET/CT, where it shows FDG-uptake.

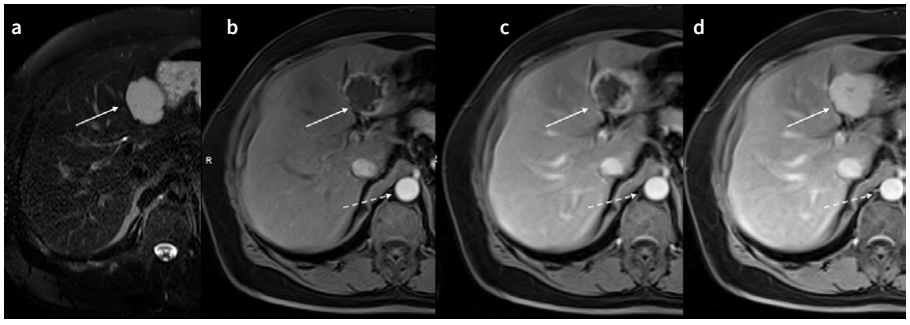

Figure 10 | Typical appearances of a haemangioma (**a**) A typical haemangioma (arrow) is well-defined and is slightly less T2 hyperintense than the cerebrospinal fluid. (**b**) In the arterial phase it demonstrates discontinuous nodular peripheral enhancement and will show gradual filling on subsequent venous phase (**c** and **d**). In all contrast-enhanced phases (**b-d**), its signal intensity is similar to that of the aorta (dashed arrow).

Mistake 7 Not recognising the necrotic/mucinous liver metastasis

Colorectal cancer is the main source of hepatic metastatic disease with 25-30% of patients developing liver metastases during the course of their disease. Although the majority of metastases will be solid, a subset may be necrotic or

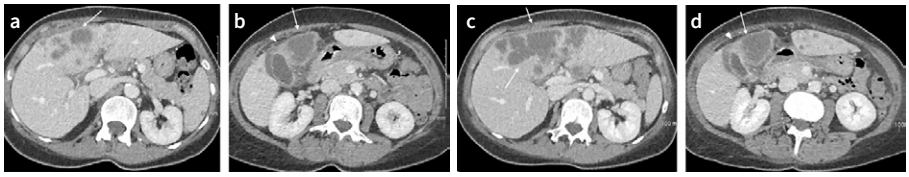

Figure 11 | Liver metastases simulating liver abscesses in a septic patient The patient presented with right upper quadrant pain, high fever and elevated CRP. (**a**, **b**) The CT shows multiple hypodense areas (arrow) with variable size around a thickened gallbladder, suggestive of acute cholecystitis complicated with liver abscesses. (**c**, **d**) A CT performed 1 week later shows significant worsening of the liver findings and the patient was referred to US for drainage of the presumed collections. However, US didn't show any fluid-filled collections and fine needle aspiration was performed instead which showed metastatic disease from a gallbladder cancer, which in hindsight was recognisable as a hyperdense area involving the gallbladder wall (arrowhead in **b** and **d**).

mucinous. Rare causes of mucinous metastases are pancreatic, ovarian or thyroid cancer. Mucinous metastases consist of >50% of mucin and – similar to necrotic lesions - will have imaging features resembling simple fluid, thus simulating benign entities such as haemangiomas or cysts.[23]

Conversely, (micro)abscesses can simulate necrotic/mucinous liver metastases. The classic scenario is that of a patient with a septic clinical picture in whom liver lesions are detected (figure 8) on cross-sectional imaging performed in search for a source of infection. This can be further complicated when the septic patient has a known primary tumour and/or biliary stent, for instance in pancreatic cancers.

In both settings, careful review of the clinical history is of utmost importance. From an imaging perspective, the development of new cystic lesions or the presence of a continuous enhancing or diffusion restricting rim in cystic liver lesions are features in favour of a malignant lesion. Nevertheless, conventional cross-sectional imaging may not be able to provide a definitive diagnosis and PET/CT and some occasions biopsy may prove useful.

In general, the differentiation between necrotic/mucinous metastases and benign liver lesions can be arduous. Providing sufficient clinical information in the imaging request and highlighting the history of a mucinous primary cancer is highly recommended if this information was not to be readily available to the reporting radiologist.

Mistake 8 Diving straight into the contrast enhanced images without looking at the anatomical/pre-contrast images first

Tempting as it may be, analysing the contrast-enhanced images prior to looking at the anatomical sequences may complicate accurate lesion characterisation. Assessment of disease response after local treatment of a liver lesion can be hampered in the presence of blood products or calcifications on MRI and CT. On pre-contrast CT, blood (and calcifications) will be hyperdense, whilst on MRI, blood products or proteinaceous lesions will appear T1 hyperintense (figure 11). If only the contrast-enhanced images were to be reviewed, one might erroneously conclude that a lesion is showing enhancement. Simply measuring the lesion density on pre- and post-contrast sequences, particularly on CT, can easily establish whether a lesion is truly enhancing. In addition, MRI has the advantage of creating subtraction images, which are the visual representation of the subtraction of the pre-contrast from the contrast-enhanced images, allowing for the revelation of areas of enhancement that might have been obscured otherwise.

Siderotic regenerative and dysplastic nodules are prevalent findings in cirrhotic patients. Due to the increased iron content, these will return high signal intensity on pre-contrast T1 images. [24] The distinction between dysplastic nodules and HCC relies on the presence of arterial hyperenhancement. Again, a review of the arterial phase images in isolation may give the impression that the lesion is enhancing. As a result, observations will be incorrectly upstaged and may trigger unnecessary biopsies or treatment (figure 12).

To sum up, including a pre-contrast sequence in the standard CT protocol and careful review of the pre-contrast images will prevent benign findings from being misinterpreted as a disease.

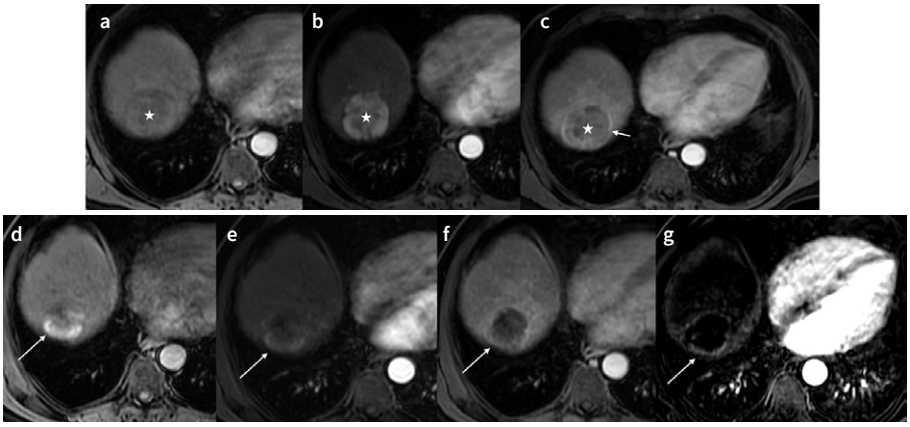

Figure 12 | Post-treatment haemorrhage simulating arterial hyperenhancement in a patient with HCC Top row: (**a**) MRI shows a lesion in the liver dome (star) with (**b**) arterial hyperenhancement, (**c**) washout and a capsule (arrow) Bottom row: (**d**) On the post-treatment pre-contrast MR image a T1 hyperintense area is seen which reflects blood products. (**e**) The signal persists in the arterial phase but not clearly in the (**f**) portal venous phase. (**g**) On the images subtracting the pre-contrast and arterial phase images, no nodular enhancement is seen to suggest residual viable disease.

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Your imaging hepatic lesions briefing

UEG Week

- 'Current imaging approach to incidental lesions in the healthy liver: Key diagnostic recommendation' session at UEG Week Virtual 2021 [https://ueg.eu/library/current-imaging-approach-to-incidental-lesions-in-the-healthy-liver-key-diagnostic-recommendation/247918]
- 'MRI and/or CT' session at UEG Week 2019 [https://ueg.eu/library/mri-and-or-ct/211667]
- 'MRI in HCC' session at UEG Week 2019 [https://ueg.eu/library/mri-in-hcc/211675]
- 'MRI in NAFLD / NASH' session at UEG Week 2019 [https://ueg.eu/library/mri-in-nafld-nash/211674]

Standards and Guidelines

- European Association for the Study of the Liver. EASL Clinical Practice Guidelines: Management of hepatocellular carcinoma [published correction appears in *J Hepatol*. 2019 Apr;70(4):817]. *J Hepatol*. 2018;69(1):182-236. [https://ueg.eu/library/easl-clinical-practice-guidelines-management-of-hepatocellular-carcinoma/175934]
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- Dietrich CF, Nolsøe CP, Barr RG, Berzigotti A, Burns PN, Cantisani V, et al.. Guidelines and Good Clinical Practice Recommendations for Contrast Enhanced Ultrasound (CEUS) in the Liver – Update 2020 – WFUMB in Cooperation with EFSUMB, AFSUMB, AIUM, and FLAUS. Ultraschall in der Medizin – *European Journal of Ultrasound* 2020;41(05):562–85. [https://ueg.eu/library/guidelines-and-good-clinical-practice-recommendations-for-contrast-enhanced-ultrasound-ceus-in-the-liver-update-2020/248729]