What is new in hepatocellular adenomas?

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Learning objectives

- Review the spectrum of different subtypes of hepatocellular adenomas, focusing on the imaging characteristics and pathologic features;

- To describe the appearance of pigmented hepatocellular adenomas and to discuss its clinical implications;

- To learn the new HCA molecular classification of hepatic adenomas and to understand how the various molecular subtypes impact on the clinical presentation of lesions.

Background

Hepatocellular adenomas are rare benign tumours of the liver. The incidence of hepatic adenomas has increased in the last several decades (incidence around 3/100,000), a trend that coincided with the introduction of oral contraceptives in the 1960s. In addition, adenomas are increasingly encountered as incidental findings in patients undergoing ultrasound, multiphasic computed tomography (CT), or magnetic resonance (MR) imaging for unrelated or nonspecific signs or symptoms. [1]

The majority of adenomas are solitary (70-80%), and typically occur in young women taking oral contraceptives . Multiple studies have demonstrated that the development of hepatic adenomas correlated with the dose and duration of hormonal therapy. Adenomas range in size from 1 to 20 cm. Multiple adenomas have been described in patients with glycogen storage diseases and hepatic adenomatosis. On histopathological analysis, hepatic adenomas contain well-differentiated hepatocytes lacking bile ducts or portal tracts.[1,6]

Most patients with solitary or no more than a few adenomas are asymptomatic and almost have normal liver function and no elevation of #-fetoprotein. Large adenomas may cause a right upper quadrant discomfort. The classic clinical manifestation of hepatic adenoma is spontaneous rupture or hemorrhage, leading to acute abdominal pain and possibly progressing to death. Large and multiple adenomas are more prone to spontaneous hemorrhage. The propensity to hemorrhage reflects the histologic characteristics of adenomas, which consist of well-differentiated hepatocytes lacking bile ducts or portal tracts with dilated sinusoids. These sinusoids are similar to thin-walled capillaries that are perfused only by peripheral arterial feeding vessels. The extensive sinusoids and

feeding arteries constitute the hypervascular nature of hepatocellular adenoma, and poor connective tissue support, also predisposes to hemorrhage within the adenoma. Because a tumor capsule is usually absent or incomplete, hemorrhage may extend into the abdominal cavity.[6,8]

Liver adenomatosis appears to be a different entity, they are not steroid dependent, but are multiple, progressive, symptomatic, and more likely to lead to impaired liver function, hemorrhage, and perhaps malignant degeneration.

Pigment deposition is rarely seen in hepatocellular adenomas. Several reports suggest that pigmented hepatocellular adenomas have increased risk of atypia and malignancy, especially in males, but these tumors remain incompletely understood.[3,5]

The prognosis of hepatic adenomas is not well established. However, they have been associated with malignant transformation, spontaneous hemorrhage, and rupture. [1,8]

Findings and procedure details

Imaging Features

Ultrasonography

The high lipid content of adenomas may contribute to the hyperechoic appearance of some of these lesions. Intratumoral hemorrhage can also result in increased echogenicity and heterogeneity, whereas other areas of hemorrhage may appear as hypoechoic or cystic areas.

Calcifications may be present in association with areas of necrosis and manifest as hyperechoic foci with acoustic shadowing.

Color Doppler US may demonstrate peripheral peritumoral vessels and intratumoral vessels that typically have a flat continuous or, less commonly, triphasic waveform (absent in the vessels within focal nodular hyperplasia).[1,6]

Computed Tomography

Multiphasic helical CT allows more accurate detection and characterization of focal hepatic lesions.

The degree of attenuation of the adenoma relative to underlying liver depends on the composition of the tumor and of the liver as well as on the phase of contrast material enhancement. CT may demonstrate a hypoattenuating mass due to the presence of intratumoral fat. However, because adenomas consist almost entirely of uniform hepatocytes, it is not surprising that most of the adenomas are nearly isoattenuating relative to normal liver on unenhanced, portal venous-phase, and delayed-phase images. In patients with fatty liver, adenomas are hyperattenuating at all phases of contrast enhancement and on unenhanced images as well. Hyperattenuating areas corresponding to recent hemorrhage can be noted. Old hemorrhage is seen as a heterogeneous, hypoattenuating area within the tumor.

At contrast material-enhanced CT, peripheral enhancement may be seen as reflecting the presence of the large subcapsular feeding vessels, with a centripetal pattern of enhancement. The enhancement usually does not persist in adenomas because of arteriovenous shunting.[7]

MR Imaging

Hepatocellular adenomas are typically bright on T1-weighted magnetic resonance images.

Adenomas are heterogeneous in appearance due to areas of increased signal intensity resulting from fat and hemorrhage and lowsignal-intensity areas corresponding to necrosis or old hemorrhage or calcifications.

Most of hepatocelular adenomas are predominantly hyperintense relative to liver on T2-weighted images;

One third of adenomas have a peripheral rim corresponding to a fibrous capsule.

After injection of a hepatocellular- specific contrast agent such as gadolinium benzyloxypropionictetraacetate (Gd- BOPTA) there is usually no substantial uptake; [2,10]

Gadoxetic acid-enhanced MR imaging features may suggest the subtype of HCA. The degree of OATP1B1/3 and MRP3 expression correlated with gadoxetic acid retention and washout, respectively, in the HBP.

The majority of HCAs show washout and appear hypointense to the

surrounding liver tissue, but few show incomplete washout or even retention of gadoxetic acid during the HBP. Some theories suggest that these different enhancement patterns could be related to the subtype of the adenoma and subsequently the amount and location of different transporter proteins, such as organic anion transporting polypeptide (OATP1B1/3) and multidrug resistance-associated protein 2 (MRP 2) and 3 (MRP3), which mediate the uptake and canalicular or basolateral excretion of gadoxetic acid during the HBP, respectively.[2]

HCA:

Hyperintense on T1-WI

- Haemorrhage
- Fat
- Sinusoidal dilatation

Capsule

- Delayed CE imaging

90% hypointense after Gd-EOB-DTPA

Subtypes:

Inflammatory hepatocellular adenoma

Fig. 1: Inflammatory hepatocellular adenoma. Axial T2-weighted fat-saturated imaging (A) and T1-weighted (B) MR images show a focal hepatic lesion that are slightly hyperintense on the T2-weighted image (arrows in A) and isointense to slightly hyperintense on the T1-weighted image (B). Axial T1-weighted in-phase (B) and out-of-phase images (C) show an important signal drop of the liver on the opposed-phase image -> Diffuse fat deposition. Contrast-enhanced T1-weighted MR images show the lesion with intense arterial phase enhancement (arrows in D), which persists into the portal venous phase and in the delayed phase (E and F).
References: - Coimbra/PT

- HCC risck low
- Peliosis, inflammatory cells
- Enhancement +++

- Heterogeneity
- Delayed enhancement

Fig. 2: Inflammatory hepatocellular adenoma. Focal hepatic lesion that are hyperintense on the T2 (A) and T1-weighted images (B). Contrast-enhanced T1-weighted MR images obtained in the arterial (C) and portal venous (D) phases show the lesion with arterial phase enhancement, which persists into the portal venous phase.
References: - Coimbra/PT

HNF-1#-mutated hepatocellular adenoma

Fig. 3: HNF-1#-mutated hepatocellular adenoma (HHCA)
References: - Coimbra/PT

- Low/null HCC risk
- Mostly fatty
- Enhancement +-

Fig. 4: HNF-1#-mutated hepatocellular adenoma in a 29-year-old woman. Axial T1-weighted in-phase (A) and out-of-phase (B) and T2-weighted (C) MR images depict a focal hepatic lesion that is mildly hyperintense on the T2-weighted image (arrows in C) and mildly hyperintense to the liver on the T1-weighted in-phase image (A), with signal loss on the out-of-phase image (B). Contrast-enhanced T1-weighted MR images obtained in the arterial (D) and portal venous (E) phases show that the lesion has mild enhancement in the arterial phase (arrow in D), which does not persist into the portal venous phase (E).
References: - Coimbra/PT

#-catenin type:

Fig. 5: #-catenin-mutated hepatocellular adenoma (#HCA). Axial T2-weighted MR image (A) shows a hyperintense focal lesion (S3), with focal and peripheral hyperintense areas on T1-weighted MR images (B, C, D, E, F, H) - intratumoral bleeding. Axial T1-weighted contrast-enhanced MR images show peripheral arterial enhancement in the arterial phase (E), with no washout in the portal venous phase (F).

Axial T1-weighted in-phase (B) and out-of-phase images (C) show a signal drop of the liver on the opposed-phase image -> Diffuse fat deposition.
References: - Coimbra/PT

- Central scar;
- Hyperintense signal on T2-weighted images;
- An persistence of contrast on the PV and delayed phases.

Table 1: MR imaging features
References: - Coimbra/PT

Differential Diagnosis

Because of the different therapeutic options, hepatocellular adenomas must be distinguished from other hypervascular lesions that occur in young adults (eg, fibrolamellar HCC, focal nodular hyperplasia, metastases).[7]

Fibrolamellar HCC

- Usually large, heterogeneous, and lobulated, with large, central, or eccentric scars and radiating fibrous septa.
- Calcifications are present in 40%-68% of tumors;
- Hypervascular areas are heterogeneous in all cases.
- Abdominal lymphadenopathy (65%);
- Vascular and biliary invasion;

Fig. 6: Fibrolamellar hepatocellular carcinoma (HCC). A - Axial unenhanced CT image shows large hypoattenuating mass (arrow) in right lobe. B - Axial CT image in arterial phase shows hypervascular enhancement within mass (arrow) and low-attenuation central scar (*) C - Axial CT image in portal venous phase shows mass is almost isoattenuating (arrow) compared with liver, and central scar (*) remains hypoattenuating.
References: - Coimbra/PT

Focal nodular hyperplasia

- A couple of HCAs can mimick focal nodular hyperplasias (gadoxetic acid retention in HBP), which can pose a diagnostic dilema. The higher T2-weighted signal intensity and the heterogeneous gadoxetic retention in the HBP may favor the diagnosis of a HCA.
- Does not undergo malignant degeneration nor is it likely to bleed.

- Almost always appears as a homogeneous, markedly hypervascular mass on arterial-phase images with a central scar that is hypoattenuating or hypointense on early contrast-enhanced images and hyperattenuating or hyperintense on delayed-phase images.
- The scar typically appears hyperintense on T2-weighted MR images.
- On unenhanced, portal venous-phase, and delayed-phase images, focal nodular hyperplasia is usually nearly isoattenuating or isointense relative to liver.

Fig. 7: 46-year-old woman with focal nodular hyperplasia (FNH) and HNF-1#-mutated hepatocellular adenoma (HHCA). FNH (arrow) almost always appears as a homogeneous, markedly hypervascular mass on arterial-phase images (D) with a central scar that is hypointense and HHCA appears with a moderate arterial enhancement. The scar typically appears hyperintense on T2-weighted MR images (A). On portal venous-phase (E) focal nodular hyperplasia is usually nearly isointense relative to liver and the HHCA show washout. HB phase gadoxetic acid-enhanced MR image shows that the FNH (arrow) is isointense to surrounding liver and HHCA is hypointense. A - Axial T2-weighted fat-saturated imaging; B - Axial T1-weighted imaging; C - Axial T1-weighted fat-saturated imaging; D, E and F - Contrast-enhanced T1-weighted MR images in arterial phase (D), portal venous phase (E) and HB phase gadoxetic acid-enhanced MR image.
References: - Coimbra/PT

Hypervascular metastases

- The differentiation of adenoma from hypervascular metastases may be difficult. Most hypervascular metastases (breast, thyroid, pancreatic islet cell tumor or renal carcinoma) are multiple and will manifest as lesions that are hypoattenuating or hypointense relative to normal liver on unenhanced, portal venous-phase, and delayed-phase images.
- Hypervascular metastases are usually hypointense on T1-weighted images and markedly hyperintense on T2-weighted images.
- Areas of fat and hemorrhage are rare in hypervascular metastases.

Black Adenomas:

Pigment deposition is occasionally seen in hepatocellular adenomas. Several reports suggest that pigmented hepatocellular adenomas have increased risk of malignancy, but these tumors remain incompletely understood. Pigmented adenomas are characteristically dark in color, due to the presence of pigment granules in hepatocyte cytoplasm resembling the pigment observed in patients with Dubin-Johnson syndrome. [3,5,9]

Fig. 9: Pigmented hepatocellular adenoma.
References: - Coimbra/PT

Fig. 8: Pigmented hepatocellular adenoma.
References: - Coimbra/PT

Fig. 10: Pigmented hepatocellular adenoma.
References: - Coimbra/PT

Fig. 11: #-catenin-mutated hepatocellular adenoma (#HCA). Dark pigment is present in the cytoplasm of the tumor cells and the pigment is positive by Pearls (A) and Fontana-Masson staining. The cytoplasm of the tumor cells is diffusely positive for C-reactive protein (B), and glutamine synthetase (C). The tumor cells show the focal nuclear expression of #-catenin.
References: - Coimbra/PT

Genotype-phenotype classification of the pigmented tumors showed different subtypes:

- HNF1# inactivated (48%);
- #-catenin activated (26%),
- Inflammatory (15%),
- #-catenin mutated inflammatory hepatocellular adenoma;

Fig. 12: Pigmented hepatocellular adenoma. On a T2-weighted MR images the pigmented HCA shows marked hypointensity and a intralesional signal drop on in-phase (B) image compared with out-of-phase image (C), which is consistent with intralesional siderosis. Contrast-enhanced T1-weighted MR images obtained in the arterial (D) and portal venous (E) phases show the lesion with arterial phase enhancement, which persists into the portal venous phase and delayed phase (F).
References: - Coimbra/PT

In conclusion, hepatocellular adenomas with lipofuscin pigment are a heterogeneous group of adenomas, with HNF-1# inactivation being the commonest genotype.

Molecular Classification

Hepatocellular adenomas are currently categorized into distinct molecular subtypes associated with different patients' risk factors for disease progression and pathology features of tumors:

- Inflammatory hepatocellular adenoma (JAK/STAT pathway activation) (50%);
- HNF-1#-mutated hepatocellular adenoma (HHCA; inactivating mutations of HNF1A12: that predispose to liver adenomatosis with >10 adenomas in the liver) (30-35%);
- #-catenin-mutated hepatocellular adenomas (#HCA) (10-15%);
- #-catenin mutated inflammatory hepatocellular adenoma (IHCA);
- Sonic hedgehog hepatocellular adenomas (shHCA; Sonic Hedgehog activation);
- Unclassified (7%).

Sonic hedgehog activation was identified in 4% of HCA previously unclassified. [4]

Mutations of cadherin-associated protein b1 (CTNNB1) exon 3, activating

b-catenin (#ex3HCA) and these tumors have an increased risk of malignant transformation in hepatocellular carcinoma. In contrast, mutations in CTNNB1 exon 7 or 8 are associated with a mild activation of the Wnt/b-catenin pathway without an increased risk of malignant transformation.

A subgroup of HCAs shared both inflammatory phenotype and activating mutations of the exon 3 of CTNNB1 (#ex3IHCA). [4]

The molecular classification could help to identify more precisely patients at risk for major complications in males and females.

Fig. 13: Molecular Classification
References: - Coimbra/PT

<u>Clinical Presentation and Risk Factors:</u>

- Patients with IHCAs and shHCAs demonstrated higher body mass índex and/or estrogen exposure cumulative.
- Androgen exposure was higher in bex3HCA with or without inflammation;
- HCAs arising on glycogenosis were never HHCA, but enriched in inflammatory HCA.

- Patients with liver vascular disease developed bex3HCA more frequently.
- Clinical symptoms at presentation were less frequent in patients with IHCA, in contrast they were more frequently diagnosed with abnormal laboratorial tests (raised g-glutamyltransferase and alkaline phosphatase);
- Liver adenomatosis was identified in patients enriched in males, glycogenosis and HHCA molecular subtype. However, the rate of complications, such as hemorrhage or malignant transformation, was the same whatever the number of adenomas .
- The classical cut-off at 5cm was associated with more frequent histologic hemorrhage in HCAs but not associated with symptomatic bleeding. shHCA was the unique subgroup associated with symptomatic bleeding.
- #catenin exon 3 nodules associated with a highest risk of malignant transformation.
- The androgen exposure due to male sex itself or androgen intake promotes CTNNB1 exon 3 mutations and consequently the malignant transformation of adenomas.

Patient features associated with malignant transformation in HCC:

- Male;
- CTNNB1 exon 3 mutations #ex3HCA or #ex3IHCA;
- Unique nodule;
- High alcohol intake;
- Fibrosis in non-tumor liverM;
- Diabetes type 2;
- Several features related to #-catenin activation (pseudoglandular formation, nuclear b-catenin, glutamine synthase expression, and cholestasis);
- Large tumor size.

Prognosis and Treatment

The prognosis of hepatic adenoma is not well established. [1,8,10]

The risks of hemorrhage and malignant transformation remain the primary clinical problems.

In males, the molecular classification is less useful due the global high risk of malignant transformation at baseline, justifying systematic surgery.

Then, molecular classification is more contributive in small tumors developed in females and would help to guide treatment in this population. In women, whatever the size of the

tumor, the presence of "at-risk HCA" was significantly associated with a higher rate of malignant transformation.

Only shHCA subtype was significantly associated with both histologic hemorrhage and symptomatic bleeding, whereas large size of the HCA was associated with histologic hemorrhage only, but not symptomatic bleeding. Consequently, adding molecular subtyping with histology and imaging data could help to better assess the risk of bleeding in clinical practice.[4]

Small HHCA were not associated with a risk of malignant transformation and patients could benefit from nonaggressive treatment and surveillance. [4]

In contrast, in small non-HHCAs, at magnetic resonance imaging, biopsy could be useful to confirm the diagnosis of HCA and exclude the diagnosis of focal nodular hyperplasia and identify molecular risk HCA that could benefit from surgery.[4]

Images for this section:

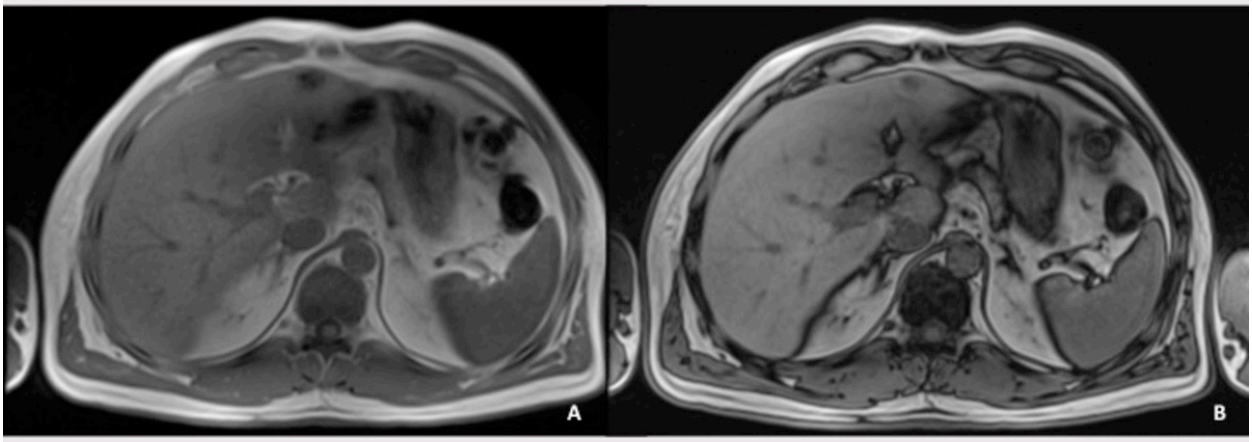

Fig. 1 - Pigmented hepatocellular adenoma with beta-catenin activation. There is a peripheral signal drop on in-phase (A) image compared with out-of-phase image (B), which is consistent with intralesional siderosis.

Fig. 9: Pigmented hepatocellular adenoma.

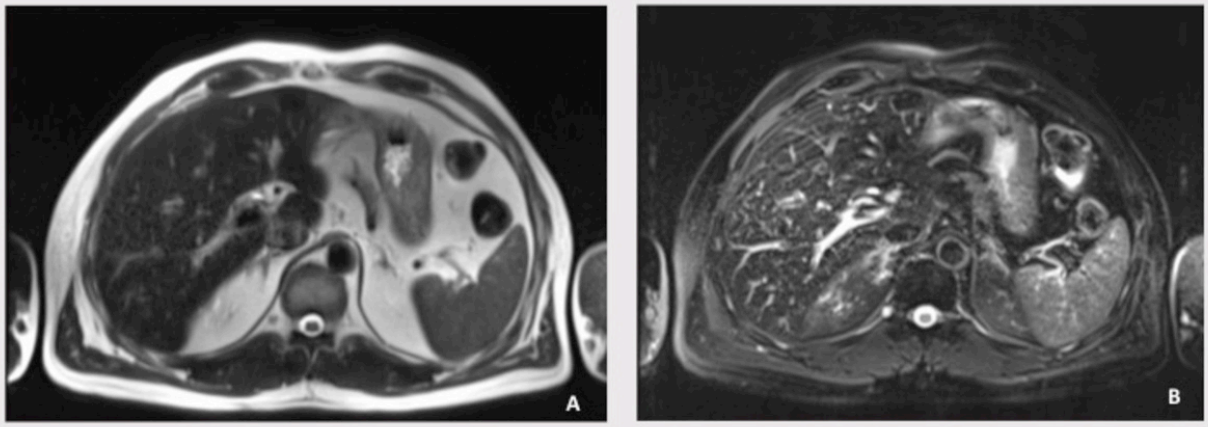

Fig. 2 - On a T2-weighted MR images (A, B), the pigmented hepatocellular adenoma with intralesional siderosis shows marked hyperintensity relative to the background liver.

Fig. 8: Pigmented hepatocellular adenoma.

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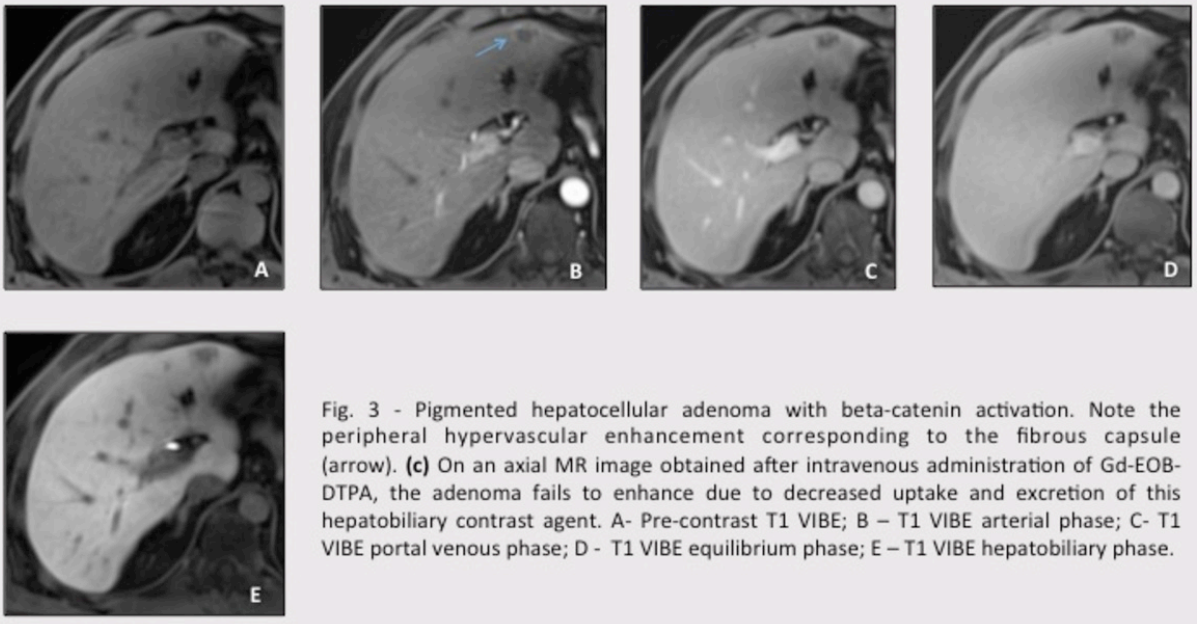

Fig. 3 - Pigmented hepatocellular adenoma with beta-catenin activation. Note the peripheral hypervascular enhancement corresponding to the fibrous capsule (arrow). **(c)** On an axial MR image obtained after intravenous administration of Gd-EOB-DTPA, the adenoma fails to enhance due to decreased uptake and excretion of this hepatobiliary contrast agent. A- Pre-contrast T1 VIBE; B – T1 VIBE arterial phase; C- T1 VIBE portal venous phase; D - T1 VIBE equilibrium phase; E – T1 VIBE hepatobiliary phase.

Fig. 10: Pigmented hepatocellular adenoma.

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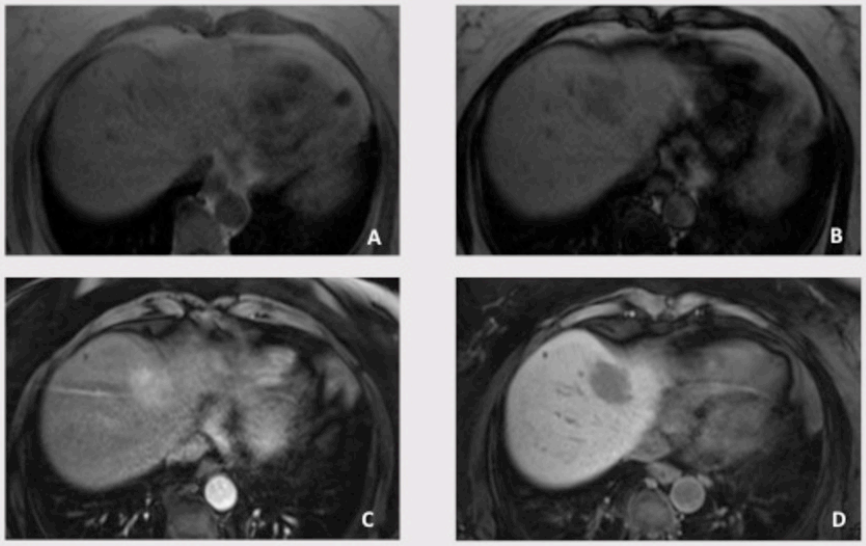

Fig. 4 - Hepatocellular adenoma with microscopic fat (A, B), which demonstrates hypervascular enhancement (C) without retention in the hepatobiliary phase (D), with Gd-EOB-DTPA. A- T1W1 in phase; B – T1W1 out of phase; C – T1 VIBE arterial phase; D – T1 VIBE hepatobiliary phase.

Fig. 3: HNF-1#-mutated hepatocellular adenoma (HHCA)

Subtype	T1 – weighted Gradient-Echo MR Images	T2 – weighted MR Images	Gadolinium-enhanced T1-weighted MR Images
Inflammatory hepatocellular adenoma	Isointense or midly hyperintense, without signal drop-off with use of chemical shift sequence	Hyperintense	Intense arterial enhancement that persists in the portal venous and delayed phases
HNF - 1α mutated hepatocellular adenoma	Isointense or hyperintense, with signal drop-off with use of chemical shift sequence	Isointense to slightly hyperintense	Moderate arterial enhancement with no persistent enhancement in the portal venous and delayed phases
B – Catenin mutated hepatocellular adenoma	Homogeneous or heterogeneous hyperintense signal intensity, depending on the presence of hemorrhage and/ or necrosis	Homogeneous or heterogeneous hyperintense signal intensity, depending on the presence of hemorrhage and/ or necrosis	Strong arterial enhancement that may or may not persist on the portal venous and delayed phases

Fig. 5 - MR Imaging Characteristics of Different Subtypes of Hepatocellular Adenoma

Table 1: MR imaging features

	HNF1α	Inflammation	β-catenin exon 7/8	β-catenin exon 3	Sonic Hedgehog	Unclassified
Molecular Definition	HNF1α inactivation	JAK/STAT activation	β-catenin activation	β-catenin activation	Sonic Hedgehog	No association
Risk Factors	HNF1α germline	Alcohol Obesity Glycogenosis	-	Androgen; Liver vascular disease	Obesity	No association
Clinical Presentation	Female; Liver adenomatosis	Older patient; Less pain; Asymptomatic; High GGT; Inflammatory syndrome	Unique tumor; Young patient	Malignant transformation; Male; Young patient; Unique Tumor;	Bleeding	No association
Histology	Tumor Steatosis; Microadenoma ; Less heamorrhage	Inflammatory infiltrate; Sinusoidal dilation; Dystrophic arteries; Non tumor steatosis	Heamorrhage; Cholestasis; Cytological atypia without malignant transformation	Cytological atypia; Cholestasis; Size > 5cm.	Heamorrhage; Non tumor steatosis;	No association

Fig. 13: Molecular Classification

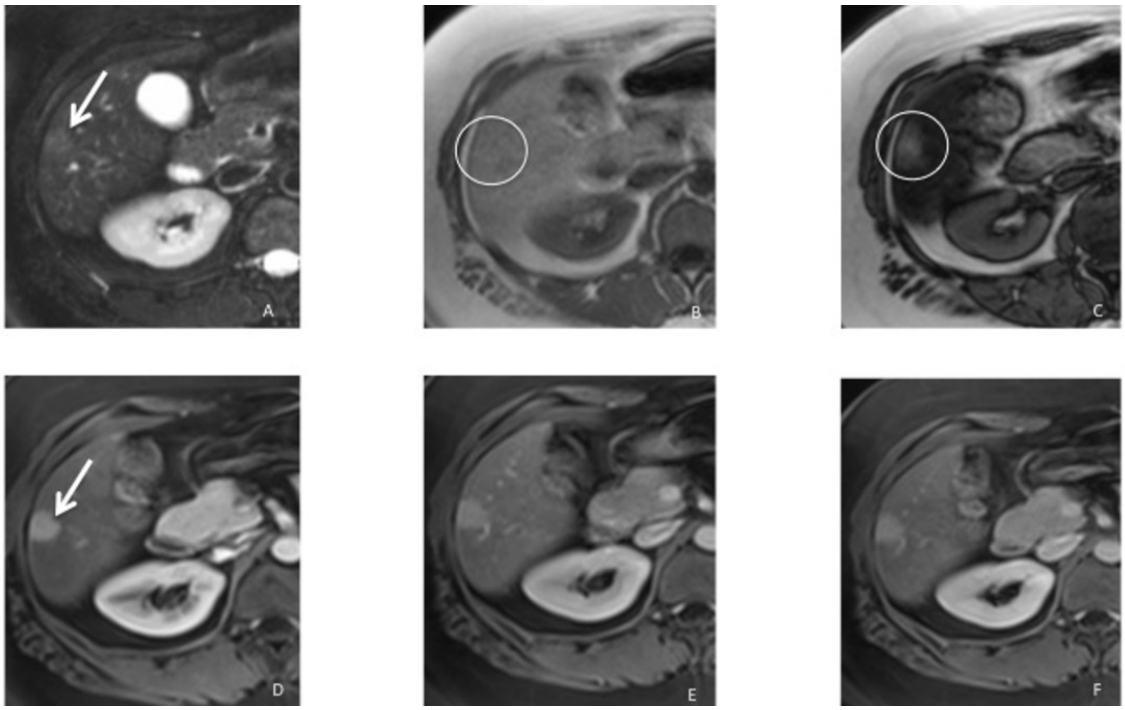

Fig. 1: Inflammatory hepatocellular adenoma. Axial T2-weighted fat-saturated imaging (A) and T1-weighted (B) MR images show a focal hepatic lesion that are slightly hyperintense on the T2-weighted image (arrows in A) and isointense to slightly hyperintense on the T1-weighted image (B). Axial T1-weighted in-phase (B) and out-of-phase images (C) show an important signal drop of the liver on the opposed-phase image -> Diffuse fat deposition. Contrast-enhanced T1-weighted MR images show the lesion with intense arterial phase enhancement (arrows in D), which persists into the portal venous phase and in the delayed phase (E and F).

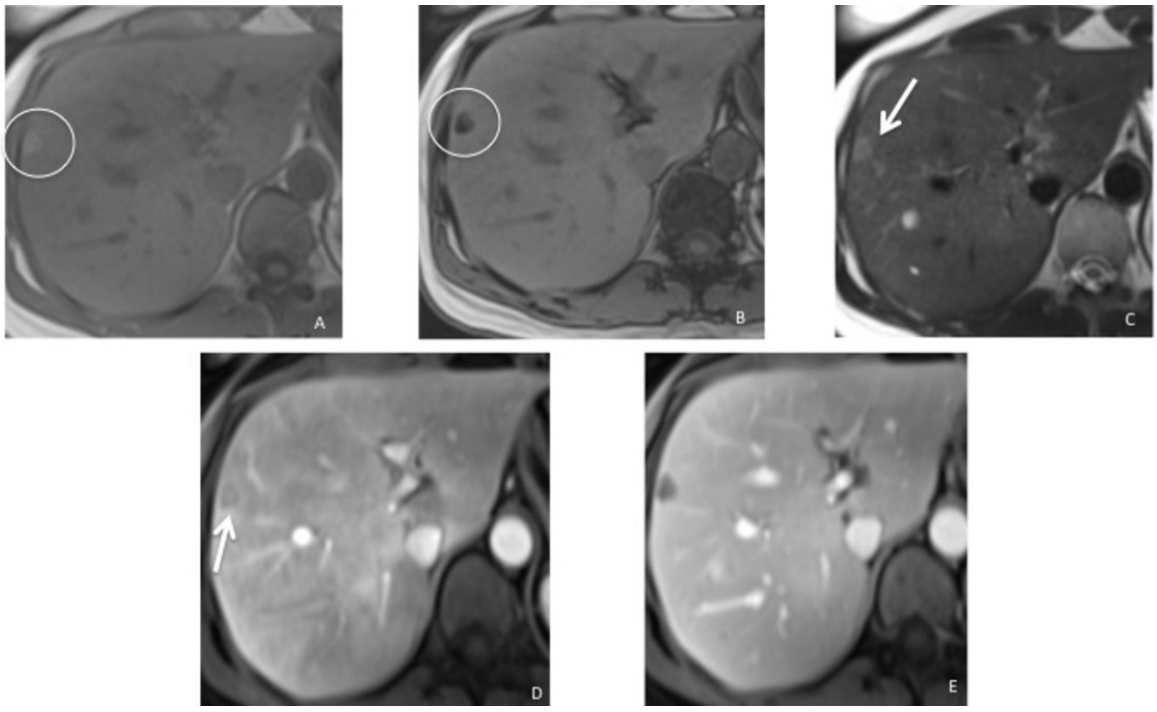

Fig. 4: HNF-1#-mutated hepatocellular adenoma in a 29-year-old woman. Axial T1-weighted in-phase (A) and out-of-phase (B) and T2-weighted (C) MR images depict a focal hepatic lesion that is mildly hyperintense on the T2-weighted image (arrows in C) and mildly hyperintense to the liver on the T1-weighted in-phase image (A), with signal loss on the out-of-phase image (B). Contrast-enhanced T1-weighted MR images obtained in the arterial (D) and portal venous (E) phases show that the lesion has mild enhancement in the arterial phase (arrow in D), which does not persist into the portal venous phase (E).

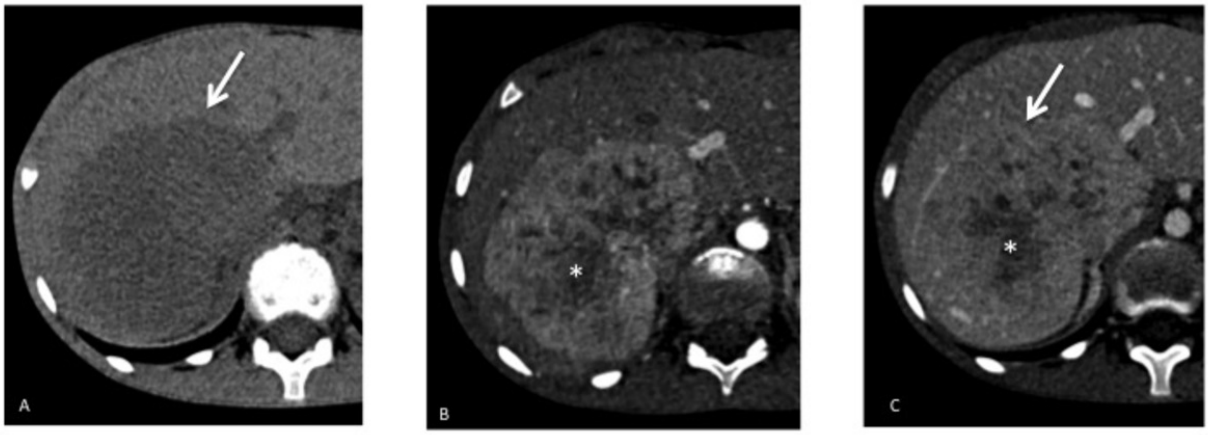

Fig. 6: Fibrolamellar hepatocellular carcinoma (HCC). A - Axial unenhanced CT image shows large hypoattenuating mass (arrow) in right lobe. B - Axial CT image in arterial phase shows hypervascular enhancement within mass (arrow) and low-attenuation central scar (*) C - Axial CT image in portal venous phase shows mass is almost isoattenuating (arrow) compared with liver, and central scar (*) remains hypoattenuating.

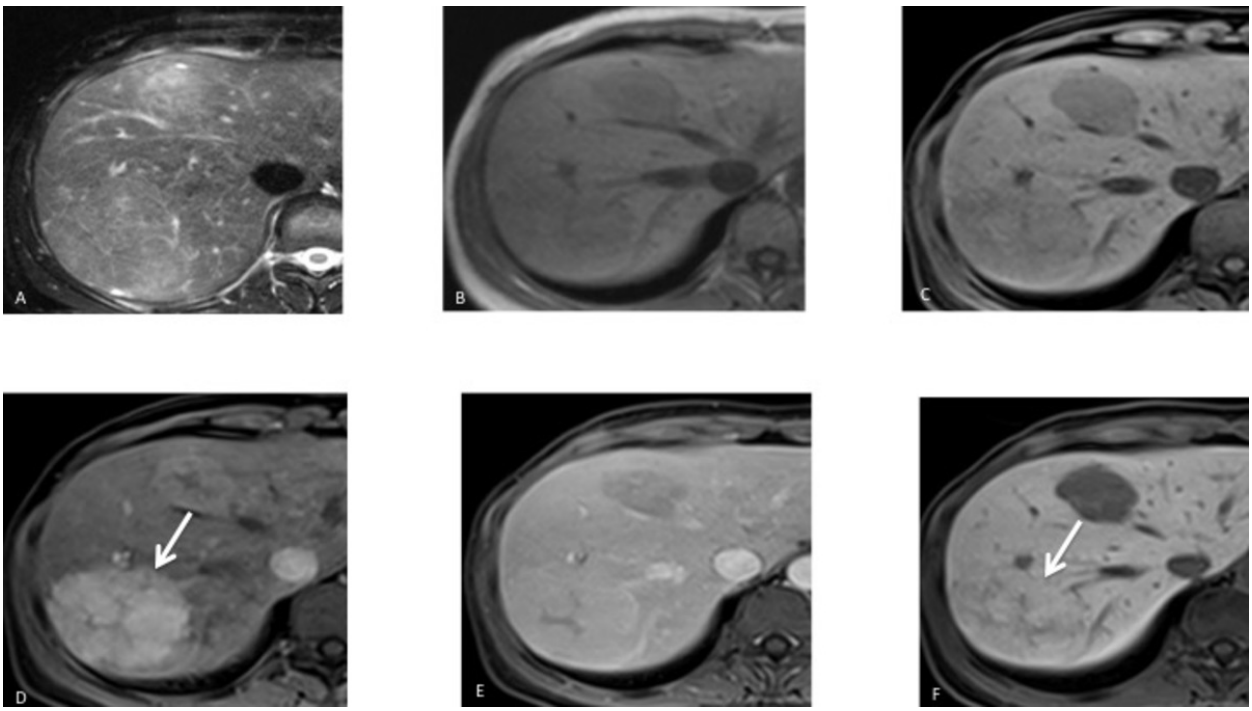

Fig. 7: 46-year-old woman with focal nodular hyperplasia (FNH) and HNF-1#-mutated hepatocellular adenoma (HHCA). FNH (arrow) almost always appears as a homogeneous, markedly hypervascular mass on arterial-phase images (D) with a central scar that is hypointense and HHCA appears with a moderate arterial enhancement. The scar typically appears hyperintense on T2-weighted MR images (A). On portal venous-phase (E) focal nodular hyperplasia is usually nearly isointense relative to liver and the HHCA show washout. HB phase gadoxetic acid-enhanced MR image shows that the FNH (arrow) is isointense to surrounding liver and HHCA is hypointense. A - Axial T2-weighted fat-saturated imaging; B - Axial T1-weighted imaging; C - Axial T1-weighted fat-saturated imaging; D, E and F - Contrast-enhanced T1-weighted MR images in arterial phase (D), portal venous phase (E) and HB phase gadoxetic acid-enhanced MR image.

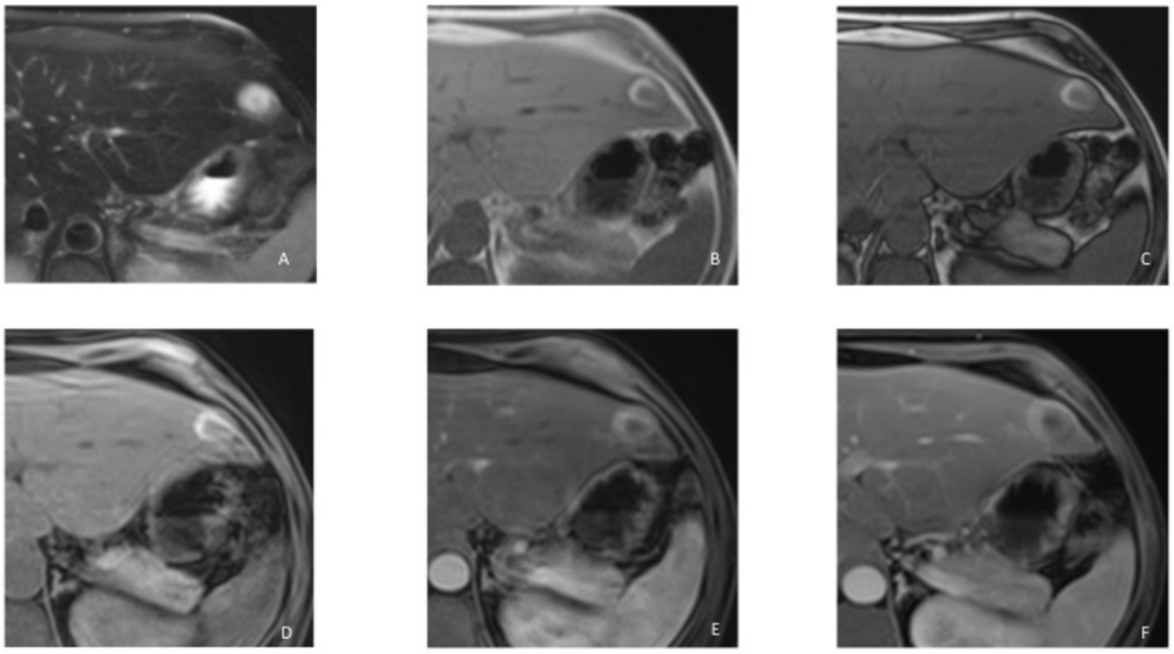

Fig. 5: #-catenin-mutated hepatocellular adenoma (#HCA). Axial T2-weighted MR image (A) shows a hyperintense focal lesion (S3), with focal and peripheral hyperintense areas on T1-weighted MR images (B, C, D, E, F, H) - intratumoral bleeding. Axial T1-weighted contrast-enhanced MR images show peripheral arterial enhancement in the arterial phase (E), with no washout in the portal venous phase (F). Axial T1-weighted in-phase (B) and out-of-phase images (C) show a signal drop of the liver on the opposed-phase image -> Diffuse fat deposition.

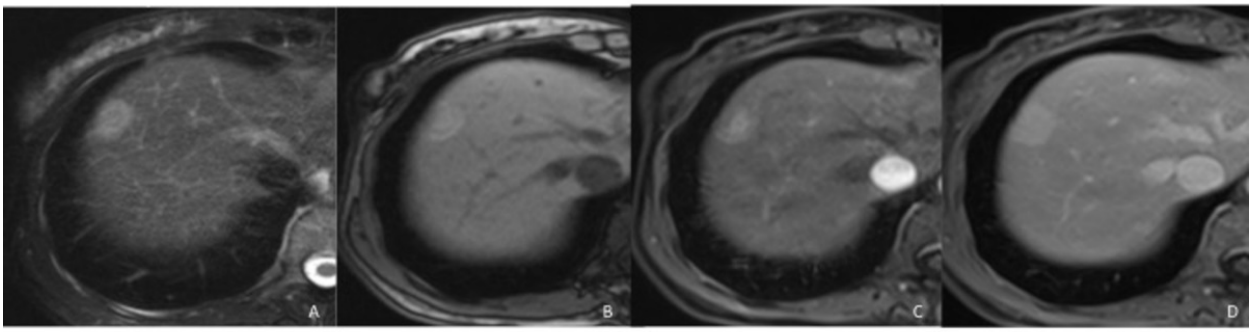

Fig. 2: Inflammatory hepatocellular adenoma. Focal hepatic lesion that are hyperintense on the T2 (A) and T1-weighted images (B). Contrast-enhanced T1-weighted MR images obtained in the arterial (C) and portal venous (D) phases show the lesion with arterial phase enhancement, which persists into the portal venous phase.

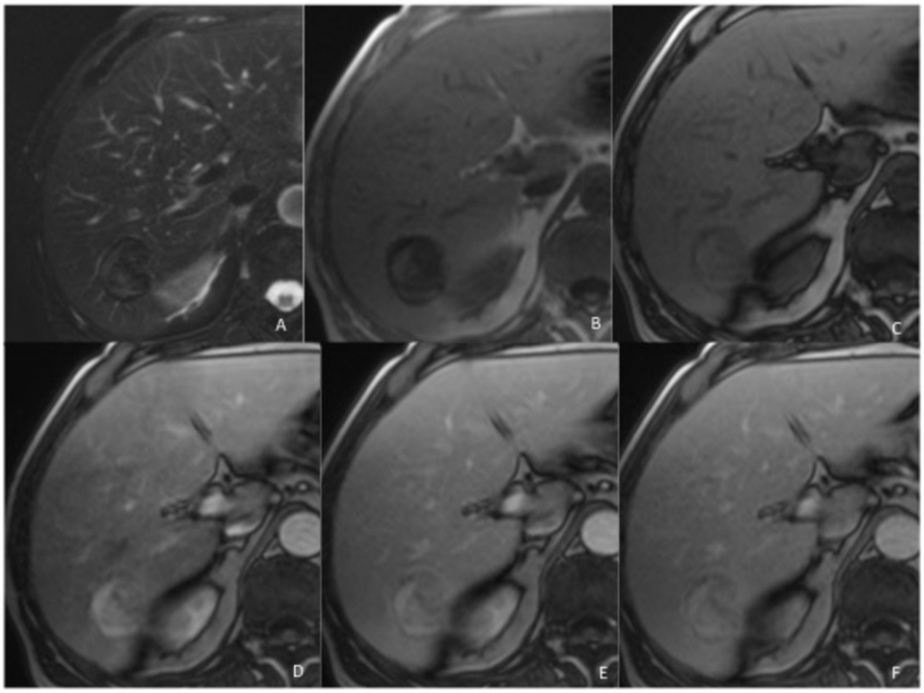

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Fig. 11: #-catenin-mutated hepatocellular adenoma (#HCA). Dark pigment is present in the cytoplasm of the tumor cells and the pigment is positive by Pearls (A) and Fontana-Masson staining. The cytoplasm of the tumor cells is diffusely positive for C-reactive protein (B), and glutamine synthetase (C). The tumor cells show the focal nuclear expression of #-catenin.

Conclusion

The purpose of this education exhibit is to present the state of the art of MRI in the diagnosis of hepatocellular adenoma and subtype characterization, with particular regard to pigmented hepatocellular adenomas and the new HCA molecular classification.

The new HCA molecular classification enabled to stratify patients according to the risk of complication, malignant transformation, and bleeding.

Personal information

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