Hepatic nodules in cirrhosis

Poster No.: A-366
Congress: ECR 2012
Type: Invited Speaker
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Keywords: Liver, Oncology, Education, CT, MR, Contrast agent-intravenous, Decision analysis, Cirrhosis, Neoplasia, Tissue characterisation
DOI: 10.1594/ecr2012/A-366

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Learning objectives

Learning objectives:

- To understand particularities of CT and MR imaging technique in liver cirrhosis.

- To consolidate knowledge of CT and MR imaging appearance of regenerative nodules, dysplastic nodules and hepatocarcinoma in liver cirrhosis.

- To become familiar with the differential diagnosis in liver cirrhotic focal lesions considering the various enhancement patterns.

- To discuss the importance of clinical, biochemical information and follow up of small nodules by the same imaging modality that may be helpful in differential diagnosis of these lesions.

Main

BACKGROUND

Liver cirrhosis is one of the most common causes of mortality in people aged 45-65 years after heart disease and cancer (1,2).

Definition. Cirrhosis is a morphologic alteration of the liver that involve the entire organ, characterized by complete destruction and replacement of its normal architecture by extensive fibrosis and nodular regenerative areas formation. Cirrhotic liver contains regenerative nodules and may also contain dysplastic nodules as well as hepatocellular carcinoma (HCC) (1, 34,35).

Etiology. Cirrhosis etiology is variable and can be usually identified by the patient's history combined with serological and histological investigation. Alcoholic liver disease and viral chronic hepatitis (HVC, HVB are the most common causes. Others possible causes in adults are represented by primary biliary obstruction, secondary biliary obstruction, autoimmune hepatitis and in children Wilson disease and biliary atresia. Rare causes are represented by haemochromatosis, glycogen storage disease, galactossema, cryptogenic cirrhosis, parasitic cirrhosis (1,2).
**Pathways of carcinogenesis.** According to the currently used terminology, the stepwise sequence of events (regenerative nodule to adenomatous hyperplasia to atypical adenomatous hyperplasia to early HCC to early advanced HCC) can be translated as follows: regenerative nodule, low-grade dysplastic nodule, high-grade dysplastic nodule, small HCC, and large HCC (1-5, 39, 41, 42).

**Diagnosis.** Generally diagnosis of liver cirrhosis is made by histological examination of a liver biopsy while imaging methods are used to determine the anatomical distribution of the disease and to evaluate the cirrhotic nodules characteristics (1,3).

**Ultrasonography** is the first imaging method providing important informations about hepatic architecture, is inexpensive, and widely available. Atrophy of the right lobe and hypertrophy of the left and especially caudate lobes are typical signs. Nodularity and increased echogenicity of the liver are often found in cirrhosis. Doppler ultrasonography of portal-vein and central-vein diameters and velocities are useful screening tests for portal hypertension and vessel patency. The sensitivity and specificity of ultrasonography to detect hepatocellular in cirrhotic liver is lower than that of CT or MRI, and the malignant potential of nodular lesions should be confirmed by helical CT or MRI (3,7). Contrast ultrasonography, harmonic imaging, and power doppler improve the detection of hepatocellular carcinoma by visualisation of abnormal intratumoral vessels (8,9).

**Multislice CT** and **MRI** are used to define the severity of cirrhosis by determining spleen size, ascites, vascular collaterals and to evaluate the liver structure abnormalities, especially in cases where tumoral or vascular lesions are suspected (10, 18-22, 23-29, 33). MRI was found to be better than helical CT in determining hepatic fat and iron content in liver steatosis and haemochromatosis, as well for detecting small hepatocellular cancers (1-2 cm size) using hepatobiliary contrast agents (8-22, 36-39,40, 44-46). Screening for HCC is one of the most important tasks in the following of patients with cirrhosis (8, 40, 44-47).

Current American Association for the Study of Liver Diseases (*AASLD*) and European Association for the Study of the Liver (*EASL*) guidelines recommend at least one screening per year for hepatocellular carcinoma in patients with cirrhosis using imaging with ultrasonography, triphasic CT, or gadolinium-enhanced MRI. Serum #-fetoprotein, which was an integral component of previous screening algorithms, is no longer recommended because of its poor sensitivity and specificity (43,48).

**Table nr.I. The sensitivity and specificity of US CT and MRI (3)**

<table>
<thead>
<tr>
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<th>US</th>
<th>CT</th>
<th>MRI</th>
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<tbody>
<tr>
<td>Se</td>
<td>57%</td>
<td>68%</td>
<td>81%</td>
</tr>
<tr>
<td>Sp</td>
<td>97%</td>
<td>93%</td>
<td>88%</td>
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New MRI developments are represented by diffusion weighted sequences (54), perfusion MR imaging to evaluate intratumoral angiogenesis and MR spectroscopy to assess liver steatosis (15-17).

**Complications.** Cirrhosis and end stage chronic liver disease (CLD) are prone to develop serious complications including hepatocellular carcinoma (HCC), variceal bleeding, ascites, spontaneous bacterial peritonitis, and encephalopathy (1,2, 37, 43).

**Prognosis.** The natural history of cirrhosis depends on both the cause and treatment of the underlying cause. Yearly rates of decompensation are 4% for viral hepatitis C and 10% for viral hepatitis B, and incidence of hepatocellular carcinoma is 2-7% per year. Once decompensation has occurred in all types of liver disease, mortality without transplantation is as high as 85% over 5 years (1,2).

**Treatment.** If HCC is detected, many treatments are available that depend on tumour size, tumour number, and local expertise. Most patients with cirrhosis and HCC will not tolerate liver resection or have microscopic satellite lesions, and the best option for cure is liver transplantation. The Milan criteria are used as a guideline in most liver centres worldwide, suggested that the mortality and recurrence of hepatocellular carcinoma is acceptable if liver transplantation is done for either a single tumour smaller than 5 cm in diameter, or no more than three tumours with the largest is less than 3 cm in diameter with no extrahepatic manifestations and no vascular invasion. Alternative treatments for patients who do not meet the Milan criteria for surgical resection or transplantation are radiofrequency ablation, chemoembolisation, alcohol ablation, and cyberknife radiotherapy, serving also as a bridge to transplantation (48-51).

**PURPOSE**

The aim of this work is to present our last 10 years of experience regarding CT and MR imaging of benign and malignant nodules developed in liver cirrhosis based on 1407 patients explored in our radiology department, focused on the CT and MRI techniques, semiology of cirrhotic nodules (regenerative, dysplastic, hepatocarcinoma), their structural changes in time and the multidisciplinary team management of cirrhotic nodules.

**PATIENTS POPULATION**

1407 patients, hospitalized in the Gastroenterology and Hepatology Clinics of Fundeni Clinical Institute, with confirmed liver cirrhosis and hepatocellular nodules with uncertain origin, visualized on ultrasound, aged between 12 and 88 years (mean age 59 years), M/F: 2/1, were explored in the last 10 years, by CT (1088 patients) or by MRI (319 patients) in Fundeni Radiology and Imaging Department. The incidence of
patients with liver cirrhosis hospitalized in Fundeni Clinical Institute compared with other gastrointestinal pathologies is synthesized in the Fig.no. The etiology and the incidence of different types of liver cirrhosis in Fundeni Clinical Institute are presented in the Fig. 1 on page 12.

Typical presentation. In our study group patients presented: weight loss (55%), anorexia (22%), abdominal pain (65%), hepato-/ splenomegaly (40%). AFP was increased in 28% of patients.

TECHNIQUES

Multislice CT evaluation of cirrhotic nodules involved a nonenhanced and enhanced triphasic CT during the arterial, portal and equilibrium/delayed phases to visualize early hypervascular lesions and to analysis the washout curve. A total of 100 ml of nonionic contrast material was injected into an antecubital vein at a rate of 3 ml/s with a power injector. The scan delay for the arterial phase (AP) was between 15 and 25 seconds; a 45 to 55 second scan delay was used for the portal venous phase (PVP) acquisition. For the delayed phase (late phase-LP) images were obtained at 180 seconds after the start of contrast material iv. administration (Fig. 2 on page 25).

MRI evaluation of the cirrhotic liver included unenhanced conventional sequences (T1, T2w, T2*-important in the evaluation of iron content (Fig. 3 on page 36), chemical shift artifact sequences- T1 in/out of phase-important to delineate liver steatosis or intralesional lipomatous content (Fig. 4 on page 38) in combination with contrast enhanced multiphase T1 dynamic 2D and 3D acquisitions after iv. administration of liver-specific contrast agents including the hepatobiliary phase (HBP- Fig. 5 on page 35). In our department, in about 85% of cases we have used mixed extracellular and hepatocellular agents (Gd-BOPTA-82% and Gd-EOB-DTPA-3%) and in 15% of cases superparamagnetic iron oxide (SPIO) particles (between 2002-2006). Up to 5% of the administered dose of Gd-BOPTA (0.1 ml/kg) is selectively taken up by functioning hepatocytes and excreted into the bile by the multispecific organic ion transporter shared by bilirubin, so in all cases evaluated with Gd-BOPTA we have performed a hepatobiliary phase T1 at 90-120 minutes after the start of the contrast injection (Fig. 6 on page 33).

In cases evaluated with Gd-EOB-DTPA the hepatobiliary phase was realized 20 minutes after the contrast injection, because about 50% of the total injected dose is taken up by the functioning hepatocytes (Fig. 7 on page 31). We have used Gd-BOPTA to evaluate liver vessels (hepatic artery, portal vein, hepatic veins) and the abnormalities in portal hypertension (Fig. 8 on page 32) or to delineate different thrombotic process involving the portal structures (intra-/ extrahepatic), hepatic veins or inferior vena cava (IVC). Hepatic signal intensity change at superparamagnetic iron oxide (SPIO)-enhanced MR imaging is mediated by phagocytic activity, dependent on the number of sinusoidal Kupffer cells (KCs) per unit volume of hepatic parenchyma and individual KC function. SPIO is strongly paramagnetic and shortens both T1 (Fig. 9 on page 30) and T2 (Fig. 10 on page 31).
for several hours after administration; the effect is more dominant on T2-marked reduction intensity on normal liver tissue. 85% of the iv. injected dose (1.4 ml pre-filled syringe for patients over a weight than 60 kg and 0.9 ml for patients with a body weight lower than 60 kg) is taken and stored by KC (compartmentalization) and 15% is phagocytized by others cells of RES. SPIO produce: a shortening of the T2 relaxation time and cause distortion of local magnetic field (susceptibility effects); a signal loss particulary on T2 and T2*wi. The decrease of signal is significant in the early and accumulation phases, the signal intensity is displayed on T2 and T2* wi, up to 10 minutes after contrast injection(Fig. 11 on page 39). In 0.3 % of cases we have used double contrast-enhanced MRI in which SPIO particles and a gadolinium chelate were administered sequentially in the same examination (Fig. 12 on page 37). The use of the two contrast agents increased the conspicuity of hepatic tumors (19,20,22). Only in two cirrhotic patients we have performed liver diffusion weighted imaging and ADC map in other medical center (Fig. 13 on page 34).

**IMAGING AIMS** in chronic liver disease are to ass:

- the dysmophy of the liver (left lobe hypertrophy, caudate lobe hypertrophy, right lobe and segment IV atrophy)
- the liver CT attenuation and MR signal abnormalities
- the presence of liver nodules: solitary, multiple, infiltrative form
- the detection and characterization of cirrhotic nodules
- the extension of HCC
- the follow up
- the portal hypertension
- the anatomical variants: liver vascularity and biliary tree

**IMAGE ANALYSIS**

**CT.** After contrast injection, the attenuation of the hepatocellular nodules were classified as hyperattenuation, isoattenuation, and hypoattenuation, compared with the surrounding liver parenchyma on arterial phase, portal venous phase, and equilibrium phase images. In portal venous phase and equilibrium phase images, each lesion was evaluated for the presence of washout, defined when any part of the lesion that was hyperattenuating on arterial phase images had a corresponding hypoattenuating area relative to the adjacent liver parenchyma on portal venous phase or equilibrium phase images. Morphologic and structural changes of the liver, spleen, of the portal venous system and the others abdominal structures were also evaluated.

**MRI.** Using unenhanced MR images we have obtained morphologic information's regarding liver and lesion pattern, signal intensity characteristic in T1, T2, T2 *, chemical shift artifact sequence, velocity of growth (capsule, pseudocapsule, edema) and internal lesion structure (homogeneous, heterogeneous, presence of necrosis, fat, hemorrhage,
central scar). T2 wi served as a very sensitive sequence for detection of focal liver lesions. Single shot FSE with long and short TE served for characterizing lesions (solid versus nonsolid). Arterial phase was crucial for detection of hypervascular lesions. On enhanced MR images we have analyzed tumor dynamics with evaluation of different patterns of enhancements and functional information's (hepatocytes/ Kupffer cells).

**IMAGING FINDINGS**

From the total number of 1407 cirrhotic patients (Fig. 14 on page 40):

- **280** had "uncomplicated" liver cirrhosis (20% of cases),
- **578** patients had liver nodular regenerative cirrhosis and portal hypertension (41% of cases),
- **112** patients had regenerative and dysplastic nodules (8% of cases) and
- **437** patients (31% of cases) had hepatocellular carcinoma: -multiple tumors in 278 cases and -single tumor in 159 cases. Tumor size varied between 1 cm to 18 cm.

In early cirrhotic forms CT findings are cvasi normal (1,2).

**CT.** In moderate and advance cirrhotic disease imaging was dominate by irregular nodular contours and surface of the liver; heterogenous liver parenchyma with nodules of varing size (24, 26, 27) and fibrotic bands; nodule (s) with increase iron content appeard in CT evaluation hyperdense; after contrast injection: heterogeneous enhancement of the liver parenchyma.

**MRI:** T1-wi show hypointense fibrotic changes; on T2-wi, the inflammatory fibrotic tissue appears as increased signal (Fig. 15 on page 12).

Relation between signal intensity of hepatic nodules in cirrhotic liver and histology is systematized in table below (34,37,52):

<table>
<thead>
<tr>
<th>Histology</th>
<th>Signal intensity T1</th>
<th>Signal intensity T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regenerative nodule</td>
<td>Iso/ hyperintense</td>
<td>Isointense</td>
</tr>
<tr>
<td>Siderotic nodule</td>
<td>Hypointense</td>
<td>Hypointense ++</td>
</tr>
<tr>
<td>Dysplastic nodule</td>
<td>Hyperintense</td>
<td>Hyopintense</td>
</tr>
<tr>
<td>Small HCC (&lt;2cm)</td>
<td>Hyperintense</td>
<td>Variable signal</td>
</tr>
<tr>
<td>Large HCC</td>
<td>Variable signal</td>
<td>Variable signal</td>
</tr>
</tbody>
</table>
Hyperintense

**Regenerative nodule (RN).** *CT.* RN with increase iron content appeared hyperdense; after contrast injection: heterogeneous enhancement of the liver parenchyma, in the early phase, and homogeneous enhancement in the late phase (Fig. 16 on page 13).

*MRI.* RN have a low signal intensity on T2 and variable signal intensity on T1 (sometimes slightly hyperintense- fat, or hypointense because of the tendency to accumulate iron. RN enhance to the same degree as the adjacent liver or show slightly less enhancement (34,35,38). Images acquired during the hepatocellular phase showed that all regenerative nodules have similar signal intensity compared to the surrounding liver parenchyma, which gives to the liver a homogeneous appearance (Fig. 17 on page 14).

**Dysplastic nodule (DN)** have variable appearances on MR images. Their signal intensity characteristics overlap with those of regenerative nodules and well-differentiated hepatocellular carcinoma (34,35). On T2-weighted, low-grade dysplastic nodules have low signal intensity relative to adjacent liver (see Fig.15); high-grade dysplastic nodules have a slightly higher signal intensity. T1-weighted images are not helpful because both low- and high-grade dysplastic nodules display variable (low, intermediate, or high) signal intensity. On gadolinium MR images low-grade dysplastic nodules typically are indistinguishable from regenerative nodules and high-grade dysplastic nodules are indistinguishable from well-differentiated hepatocellular carcinomas (35,36).

Hepatobiliary phase (HBP) is very important in the characterization of cirrhotic nodules. RN appeared isointense T1 in the HBP (RN contain normal small biliary ducts). DN may appear hyperintense T1 (DN contain altered small biliary duct) (38, 36).

CT is less sensitive than MRI in a correct delineation and differential diagnosis between RN, DN and HCC (Fig. 18 on page 16, Fig. 19 on page 15, Fig. 20 on page 17), in all these uncertain cases, histology after liver biopsy remains the gold standard.

**Small hepatocarcinoma.** CT/ MR findings, in our study (35% of cases), were represented by a nodular lesion (less than 3 cm) iso-/ slightly hypodense to surrounding liver tissue on nonenhanced scans/ discretely to moderate hyperintense on T2 wi, iso-/ hypointense on T1 wi. After contrast material injection using a multiphase CT/ MRI acquisition the nodule (s) showed rapid enhancement in the arterial phase and rapid passage of the contrast agent (Fig. 21 on page 19, Fig. 22 on page 18) simmilar of those findings published in the litterature (28-30, 32). In practice we can be confronted with atypical issues, in which the tumoral nodul in HBP is iso-/ or even discretely hyperintense to the liver parenchyma (Fig. 23 on page 20).
Relation between vascularization of a cirrhotic nodule and the possibility to differentiate by using contrast injection a regenerative/dysplastic nodule from a small HCC is summarised in the table below (17,30):

<table>
<thead>
<tr>
<th>Histology</th>
<th>Signal intensity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regenerative and dysplastic nodules</td>
<td>Hypovascular (96% cases)</td>
</tr>
<tr>
<td>Displastic nodul with malignant degeneration</td>
<td>Hypo-/ hypervascular</td>
</tr>
<tr>
<td>Small hepatocarcinoma</td>
<td>Hiyervascular (in general)</td>
</tr>
<tr>
<td>Large hepatocarcinoma</td>
<td>Hypervascular (90-95% cases)</td>
</tr>
</tbody>
</table>

Large hepatocarcinoma were considered in cases with tumoral lesions larger than 3 cm in diameter (43% in our patients group). CT and MRI current aspects were represented by a mosaic pattern of the tumor: variable CT attenuation and signal intensities on T1, T2w due to the presence of necrotic areas, of fatty infiltration, cooper protein or hemorrhage (Fig. 24 on page 22, Fig. 25 on page 21). Intratumoral calcifications are rare (Fig. 26 on page 23). Heterogeneous enhancement were observed in arterial and later phases. Tumor (pseudo) capsule in encapsulated HCC: hypointense T1, T2; hyperintense T2 capsule thickness>4 mm (see Fig.23,24). Satellite nodules were presents in 38 % of large HCC cases (Fig. 27 on page 24).

Multicentric hepatocarcinoma was observed in 18% of all the HCC evaluated in our study (Fig. 28 on page 26).

Diffuse/infiltrative hepatocarcinoma was present in 9% of all HCC cases explored in our department (Fig. 29 on page 27, Fig. 30 on page 28).

In HBP, HCC, were in 96% of all the tumoral cases, hypointense relative to the surrounding liver parenchyma in T1-w images.

Extracapsular extension and/or vascular invasion (portal vein, hepatic veins, IVC) was present in 32% of HCC (large and diffuse types- Fig. 31 on page 41). In extensive portal thrombosis we have observed the development of a portal cavernoma.

#Tumoral thrombus/ thrombosis: enhancement during arterial phase and filing defect during later phases (see Fig.25, 28, 30).

DIFFERENTIAL DIAGNOSIS

Differential diagnosis must be done with other hypervascular liver tumors such as (52,53):
- focal nodular hyperplasia: more pronounced contrast enhancement; central enhancing scar; uptake of hepatobiliary contrast agent in the late phase;

- adenoma: occurs in healthy liver patients after hormone use; hemorrhage is common;

- hemangioma: high-signal intensity on T2-wi, progressive and persistent filling up of the contrast agent;

- hypervascular metastasis: usually multiple, smaller focal nodules;

- cholangiocarcinoma: capsular retraction; late contrast enhancement (after 10 minutes); may be associate with segmental biliary duct dilatation.

**PITFALLS**

Due to distorted liver architecture in cirrhosis, the percentage of false negative abnormalities or false positive as tumor is high due to regenerative/ dysplastic nodules, arterio-portal shunts, and atypical hemangiomas.

**THERAPEUTIC OPTIONS**

The therapeutical approach depend on:

- the size (>3 cm),
- number (>3 ) and
- location/ extension of the tumor in association with
- the severity of the underlying disorders (cirrhosis type/portal hypertension, age of the patient, co-morbidities).

When surgery is possible treatment involves local treatment (resection or radiofrequency ablation) or transplantation. Palliative treatment is represented by transarterial chemoembolisation (TACE) or chemotherapy, also in combined therapy.

TACE was performed, during the last three years in our department, to a total number of 144 patients (48 women and 96 men, aged between 22 and 78 years, with an average of 61.95 years). 118 patients have received only one procedure, in 25 patients we performed two procedures and in one of patients three procedures, with a total number of 171 procedures. Were treated between 1 and 3 lesions per procedure. Ultrasound guided ablation in liver tumors was performed in 75 patients.

*Follow-up imaging after TACE* is necessary between the first week and approximately two months after the procedure. It can be done by CT or by MRI. Because the
chemotherapeutic agent is mixed with lipiodol, CT scans of a tumor treated by TACE, reveal dense “opacification” associated with intratumoral necrosis (Fig. 32 on page 45). MRI is more sensitive than CT in the follow-up after TACE to depict residual tumoral tissue or tumor recurrence. In completely distroid tumors we have observed a reduction on T2 relaxation time, corresponding to the coagulative necrosis induced by TACE. Areas of high signal intensity on T2 wi and contrast enhanced T1w dynamic multiphase images, acquired 2-3 months after TACE procedure, may be considered as tumor recurrence (52).

Regarding surgical procedures performed in Clinical Institute of Fundeni, between 2001 and 2010, on patients with liver cirrhosis and hepatocarcinoma the statistics is dominate by resection, followed by surgical ablation and liver transplantation (Fig. 33 on page 44).

COURSE and PROGNOSIS

Without treatment the survival time is usually less than 1 year (. Early diagnosis and adequate therapy (43,48,49,55) improve the prognosis (Fig. 34 on page 42). In cases of "unclassified" cirrhotic nodules (less than 1 cm) imaging must be performed for the follow-up (Fig. 35 on page 43).

5 years- survival rate after transplantation is 60-70%, after resection 40-50%, after radiofrequency ablation 50%, and after transarterial chemoembolisation 5-20% (52,53).

CONCLUSIONS

- Cross-sectional imaging with CT and MRI plays an important role in the evaluation and follow-up of patients with cirrhotic liver disease and its complications.

- Interpretation must be done always in clinical context.

- MRI is the method of choice in the characterisation of liver cirrhotic nodules, by using specific cellular contrast agents, in correlation with new techniques diffusion, perfusion, spectroscopy.

- Small nodules (<1cm) must be follow up (4-5 months) by the same imaging modality.

- Multidisciplinary dialogue between the clinician (gastroenterologist, surgeon, and oncologist), radiologist, medical laboratory scientist and anatomo-pathologist allows finding the optimal solutions concerning the monitoring and correct therapeutic approach of hepatic nodules developed in the cirrhotic liver.
Acknowledgements to:

- all my colleagues from the Radiology Department of Fundeni
- Pr. Dr. Irinel Popescu- Head of the General Surgery and Liver transplantation Center of Fundeni
- Pr. Dr. Mircea Diculescu- Head of the Gastroenterology, Hepatology and Digestive tract Center of Fundeni
- Dr. Mariana Mihaila- Internal Medicine Department of Fundeni
- Dr. Vlad Herlea- Head of the Histology Department of Fundeni

Images for this section:

**Fig. 1: ETIOLOGY and INCIDENCE of LIVER CIRRHOSIS in Fundeni Clinical Institute**
**Fig. 15:** LIVER CIRRHOSIS with MULTIPLE REGENERATIVE NODULES and fibrosis, in a clinical context of Wilson disease.
Fig. 16: CT aspects in LIVER CIRRHOSIS with MULTIPLE REGENERATIVE NODULES: regenerative nodules appears discretly hyperdense on NECT, without arterial enhancement; the liver parenchyma become homogeneous in the delayed phase.
**Fig. 17:** REGENERATIVE and DYSPLASTIC LIVER CIRRHOTIC NODULES in non enhanced MRI evaluation: regenerative nodule appear iso/discretly hiperintense on T1 wi, and isointense on T2 wi; the dysplastic nodule low grade appear hyperintense on T1wi and hypointense on T2wi.
**Fig. 19:** DYSPLASTIC NODULE- ATYPICAL CT PATTERN (histology confirmation): hypervascularized hepatic nodule in arterial phase with washout in portal and delayed phase.
Fig. 18: ATYPICAL REGENERATIVE NODULE (histology confirmation): small hypoattenuating liver nodule on NECT and after iodinated contrast iv injection, in arterial and portal venous phase.
**Fig. 20**: SMALL HEPATOCARCINOMA and MULTIPLE SIDEROTIC LIVER NODULES: comparison CT/MRI: discretly hypervascularized liver nodule with wash out in portal venous phase and delayed phase; the superiority of MRI evaluation to depict siderotic liver nodules using T2*wi.
**Fig. 22:** SMALL HEPATOCARCINOMA: comparison between CT and MRI with SPIO evaluation: better delineation of the tumoral nodule (dimensions and contours) in MRI evaluation, also better evaluation of structural changes of the liver parenchyma with identification of multiple small regenerative nodules.
Fig. 21: SMALL HEPATOCARCINOMA: typical CT findings- isoattenuating lesion on NECT, important enhancement in arterial phase with rapid washout in portal phase and discreet hypoattenuation in delayed phase.
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Fig. 25: LARGE HEPATOCARCINOMA: MRI findings after contrast injection: in the periphery of the tumor on note the presence of a capsule which is effracted by the tumor; better delineation of the tumoral thrombus into the IVC.
Fig. 24: LARGE HEPATOCARCINOMA: mosaic pattern on nonenhanced MRI, located in the posterior part of the right hepatic lobe with tumoral extension into the inferior vena cave (arrow).
Fig. 26: LARGE HEPATOCARCINOMA of the left hepatic lobe with central scar and calcifications. Histology exam: poorly differentiated HCC.
Fig. 27: LARGE HEPATOCARCINOMA (yellow arrow) with SMALL SATELITE NODULES (yellow arrow head), TUMORAL RIGHT PORTAL VENOUS THROMBOSIS, non tumoral left portal venous thrombosis and hepatic perfusion disorders (blue arrow).
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**Fig. 30:** DIFFUSE RIGHT LOBE HEPATOCARCINOMA extending into the right portal vein; small ascite- MRI evaluation.
**Fig. 10:** LIVER CIRRHOSIS evaluation with SPIO in T2wi: signal drop of the liver parenchyma compared to the precontast T2 w evaluation that confirm the presence of unaltered Kupffer cells; better delination between regenerative nodules and fibrotic bands.
**Fig. 9**: LIVER CIRRHOSIS evaluation with SPIO in T1 wi: multiple regenerative nodules and fibrotic bands
Fig. 7: MRI evaluation of the liver using GD-EOB-DTPA in a cirrhotic patient (HCV) with a small nodule, hyperintense in T2wi, that enhance in arterio-portal phase, with washout in the delayed phase, deliniate in the periphery by a thinny capsule, hypointense in hepatobiliary phase (after 20 min from the contrast injection) suggestive for small hepatocarcinoma (arrow). Other small nodular enhancement is present in the dorsal part of the VII liver segment, isointense in portal and hepatobiliary phase (differential diagnosis between a true small tumoral nodule and shunt.)
**Fig. 8:** COMPLEMENTARY INFORMATIONS using GD-BOPTA: in addition to the evaluation of the liver parenchyma we can obtain informations concerning the intraabdominal vessels (particular, hepatic artery, portal vein, hepatic veins and IVC), the presence of direct and indirect signs of portal hypertension and characteristics of the thrombus (tumoral vs non tumoral).
**Fig. 6:** MRI evaluation using GD-BOPTA in a cirrhotic patient (HVC) with a large mass of the right lobe, that present typical MRI aspects in favor of a hepatocarcinoma: mosaic precontrast pattern, patchy enhancement in arterio-portal phase with wash-out in portal and delayed phase. In hepatobiliary phase (after 100 min from the contrast injection) the HCC is globally hypointense T1 relative to liver parenchyma.
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**Fig. 5:** MRI TECHNIQUE(3): importance of T2 sequence to visualise focal liver lesion and the utility of T1 pre-/ and postcontrast injection in a multiphase dynamic approach (arterial, portal and delayed phase) in combination with the MR findings obtained in the hepatobiliary phase, to characterize liver lesions.
Fig. 3: MRI TECHNIQUE (1): importance of T2* w sequences to evaluate siderotic nodules and liver hemochromatosis.
Fig. 12: Utility of the use of DOUBLE CONTRAST iv injection in liver focal lesions evaluation: Kupffer specific (SPIO) and extracellular (Gd-DTPA): diffuse right hepatic tumor extended into the right portal vein; the portal venous system evaluation is better appreciated with Gd-DTPA than with SPIO only, so using both types of contrast, we can summarize advantages of each contrast in part.
**Fig. 4:** MRI TECHNIQUE(2): importance of T1 in/out of phase sequence to delinitate the amount of liver steatosis and the lipomatous content of a liver cirrotic nodule.
Fig. 11: UTILITY of SPIO in the characterisation and detection of liver tumors compare to CT evaluation: diffuse left liver tumor associated with multiple small tumoral nodules involving the right lobe, unidentified at CT; the absence of the signal drop 10 minutes after contrast injection in the tumor compare cu the sourrounded liver is in favor of an poorly differentiated HCC.
**Fig. 14:** CURRENT ASPECTS IMAGING in LIVER CIRRHOSIS in patients of Fundeni Clinical Institute.
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Fig. 34: The Barcelona Approach: Diagnosis, Staging, and Treatment of Hepatocellular Carcinoma (48,54).
Fig. 35: Diagnostic algorithm for suspected HCC: CT, MDCT, MRI, US (48).
**Fig. 33:** SURGICAL PROCEDURES in Fundeni Clinical Institute: tumoral resection (67%), tumoral ablation (24%), liver transplantation (9%).
**Fig. 32:** Large right lobe hepatocarcinoma 6 months after TACE: CT evaluation- in site of the tumor there are necrotic areas and hyperdensities corresponding to the lipiodol particles. After contrast injection on observe on the periphery of the tumor two others tumoral nodules.
References


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