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**The role of MRI in differentiation between Hepatocellular
Carcinoma and benign hyper vascular lesions in cirrhotic
patients.**

Essay

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Aim of the work

This study aims to describe the role of MRI in early detection and characterization of HCC and differentiating it from other non-malignant focal lesions. Using unenhanced, contrast-enhanced and diffusion-weighted MR images, for a better patient management plan.

List of Abbreviations

2D	Two dimensional
3D	Three dimensional
3D-GRE	Three Dimensional Gradient Recalled Echo
ADC	Apparent diffusion coefficient
AFP	Alpha-fetoprotein
CCA	Cholangiocarcinoma
CNR	contrast to noise ratio
CHA	Common hepatic artery
DWI	Diffusion-weighted imaging
ECF	Extracellular fluid
ETL	Echo train length
FLC	Fibrolamellar carcinoma
FNH	Focal nodular hyperplasia
FOV	Field of vision
FRFSE	Fast recovery FSE
FSE	Fast spin echo

Gd	Gadolinium
Gd-BOPTA	Gadobenate dimeglumine
GRE	Gradient Recalled Echo
HBV	Hepatitis B virus
HCA	Hepatocellular adenoma
HCC	Hepatocellular carcinoma
HCV	Hepatitis C virus
HASTE	Half-Fourier acquisition single-shot turbo spin-echo
Hz	Hertz
IP	In-phase
IVC	Inferior vena cava
MIP	Maximum intensity projection
MDCT	Multi detector CT
NEX	number of excitations
NRH	Nodular regenerative hyperplasia
OP	Out-of-phase
PI	Parallel imaging
PV	Portal vein
RES	Reticuloendothelial system

RF	Radio-frequency
ROI	Region of interest
RT-FSE	Respiratory triggered FSE
SGE	Spoiled gradient echo
SMA	Superior mesenteric artery
SNR	Signal to-Noise Ratio
SPIO	Super Paramagnetic Iron Oxide
SSFSE	Single-shot fast spin-echo
T	Tesla
TE	Time of Echo
THED	Transient hepatic enhancement difference
TR	Time of Repetition
US	Ultrasound

List of cases

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Introduction

Liver cirrhosis is a progressive diffuse fibrosis with architectural distortion and nodular regeneration. It is characterized by a spectrum of hepatocellular nodules that mark the progression from regenerative nodules to low_ and high_ grade dysplastic nodules, followed by small and large hepatocellular carcinomas (HCC). Vascularity patterns change gradually as the nodules evolve, with an increasing shift from predominantly venous to predominantly arterial perfusion. (**Parente et al, 2012**).

All types of cirrhosis can lead to hepatocellular carcinoma. Cirrhosis is present in up to 90% of patients with hepatocellular carcinoma (HCC).

(**Mittal S et al, 2013. and, Hussain et al, 2009**).

Hepatocellular carcinoma (HCC) is the most common histologic type of primary liver cancer, accounting for between 85% and 90% of these tumors. The overall prognosis of patients with liver cancer is poor, and an understanding of this disease and its risk factors is crucial for screening at-risk individuals, early recognition, and timely diagnosis. Most HCCs arise in the background of chronic liver disease caused by hepatitis B virus, hepatitis C virus, and chronic excessive alcohol intake. These underlying causes are characterized by marked variations in geography, gender, and other well-documented risk factors, some of which are potentially preventable. (**Pellicelli A et al, 2012. and, McGlynn K et al, 2011**)

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Distinguishing between HCC and benign hypervascular lesions in these cases remains a major challenge in management of patients at risk for developing hepatocellular carcinoma (**Choi JY et al, 2014**)

Biopsy of arterially enhancing lesions detected in cirrhotic livers is neither feasible nor advisable. Therefore, such lesions are often followed with periodic imaging to assess interval change. (**Korpraphong et al, 2009**).

Various types of hypervascular lesions are common among patients with cirrhosis. The ability to differentiate between malignant and benign nodules is limited; nodules are primarily characterized on the basis of differences in vascularity. Regenerative and low-grade dysplastic nodules have predominantly portal venous blood supplies and demonstrate as much enhancement as the liver parenchyma. High-grade dysplastic nodules and HCCs demonstrate a loss of portal vascularization and have more arterial supply. The transition from regenerative and dysplastic nodules to HCC is not characterized by discrete steps; rather, it is marked by a continuum of vascular pattern changes. Many of the intermediate stages are atypical, making their characterization difficult. (**Bruix et al 2011**).

Common hypervascular liver lesions include hemangioma, focal nodular hyperplasia (FNH), hepatocellular adenoma (HCA), hepatocellular carcinoma (HCC), fibrolamellar carcinoma (FLC), and others as metastases from primary tumors such as islet cell tumor, carcinoid, renal cell carcinoma, melanoma, and thyroid carcinoma. Other conditions that could mimic hypervascular liver

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lesions include hepatic artery pseudoaneurysm, transient hyperintensity disorder, and passive hepatic congestion. **(Lee YJ et al, 2015).**

If cirrhosis is present, the differential diagnosis of a hypervascular liver lesion can be narrowed to a few entities, including arterioportal shunts or pseudo lesions (for very small lesions), dysplastic nodules, and HCCs. Occasionally, a cirrhotic liver may have preexisting flash-filling hemangiomas that may mimic malignant lesions. **(Morana G et al, 2011)**

Early detection of HCC is necessary for the most effective triage of patients and in planning management strategies like resection, transplantation, tumor ablation (using radiofrequency, cryotherapy or percutaneous ethanol injection) and chemo-embolization **(Bondioni MP et al, 2011) .**

Magnetic resonance imaging (MRI) is extremely useful in the detection and characterization of regenerating and dysplastic nodules and HCC. Several studies have demonstrated the superiority of MRI in both lesion detection and characterization of focal hepatic lesions when compared to CT **(Kozaka K et al, 2014)**

As MRI techniques become more sensitive for the detection of HCC, the rate of detection of lesions other than HCC has also increased. Small arterially enhancing lesions are particularly common in patients with the chronic liver disease who are also at risk for HCC, although only a minority of these lesions are subsequently shown to represent HCC **(Korpraphong et al, 2009).**

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With a combination of basic T1 and T2 weighted sequences, diffusion weighted imaging DWI, and use of gadolinium-enhanced MRI, most liver lesions can be adequately diagnosed (**Haradome H et al, 2012**)

Even in very complicated cases, an analysis of signal intensity data and dynamic enhancement patterns after intravenous contrast administration and the status of the remainder of the liver allows for accurate differential diagnosis of hypervascular focal lesions. (**Ronot M et al, 2014**)

Characterizing hypervascular lesions in patients with cirrhosis is challenging as differentiating HCC from other hypervascular lesions is a key step in treating patients with cirrhosis, and is the responsibility of the radiologist.

(**Hanna RF et al, 2008**).

DW-MRI has already been extensively researched for hepatic imaging and gains sufficient accuracy in the differentiation of benign and malignant liver disease. DW-MRI relies heavily on the ADC-calculation (**Lewis S, Kamath A et al, 2015**).