Role of MRI in characterization of hypervascular hepatic focal lesions in cirrhotic patients

Thesis

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دور الرنين المغناطيسي في وصف البؤر الكبديه غزيرة الأوعية الدمويه في مرضى التليف الكبدي

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Sarmad Sami Rasheed

Contents

List of Abbreviations

3D	**:**	Three dimensional.
ADC	**:**	Apparent diffusion coefficient.
APA	**:**	Arterio-portal anastomoses.
BH	**:**	Breath hold.
CT	**:**	Computed tomography.
CV	**:**	Central venule.
DW MRI	**:**	Diffusion weighted magnetic resonance Imaging.
DWI	**:**	Diffusion weighted imaging.
EPI	**:**	Echo planner imaging.
FFE	**:**	Fast field echo.
Fig	**:**	Figure.
FLL	**:**	Focal liver lesions.
FNH	**:**	focal nodular hyperplasia.
FS	**:**	Fast spin.
FSE	**:**	Fast spin echo.
GB	**:**	Gall bladder.
Gd DTPA	**:**	Gadolinium diethylenetriamine pentaacetic acid (hepatocyte-specific contrast agent taken by hepatocytes and excreted into biliary system).
GRAPPA	**:**	Generalized auto-calibrating partially parallel acquisition.
GRE	**:**	Gradient recalled echo.
HA	**:**	Hepatic artery.
HCC	**:**	Hepatocellular carcinoma.
HCV	**:**	Hepatitis c virus.
HMS	**:**	Hepatic microvascular subunits.
IQR		Interquartile range
IVC	**:**	Inferior vena cava.
Min	**:**	Minute.
MRI	**:**	Magnetic resonance imaging.
msec	**:**	Millisecond.
NEX	**:**	Number of excitations.
PSC	**:**	Primary sclerosing cholangitis.
PV	**:**	Portal vein.
RT	**:**	Respiratory triggered.
SE	**:**	Spin echo.
Sec	**:**	Second.
SGE	**:**	Spoiled gradient echo
SI	**:**	Signal intensity.
SNR	**:**	Signal to noise ratio.

SOR	**:**	Standard of reference.
SPAIR	**:**	Spectral attenuated inversion recovery (fat suppression mri technique).
T	**:**	Tesla.
TE	**:**	Echo time.
THRIVE	**:**	High resolution isotropic volume examination.
TR	**:**	Repetition time.
TSE	**:**	Turbo spin echo.
US	**:**	Ultrasonography.
VIBE	**:**	Volumetric interpolated breath hold examination.
WIs	**:**	Weighted images.

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Abstract

Liver cirrhosis is a common problem in Egypt with most of the cause are viral induced liver cirrhosis **(Robert et al., 2008).**

Distinguishing between HCC and benign hypervascular lesions in liver cirrhosis remains a major challenge in management of patients at risk for developing hepatocellular carcinoma. The differential diagnosis of a hypervascular liver lesion in cirrhotic liver can be narrowed to a few entities, including pseudolesions (for very small lesions),regenerative nodules, dysplastic nodules, and HCCs. Occasionally; a cirrhotic liver may have preexisting flash-filling hemangiomas that may mimic malignant lesions. Small, arterially-enhancing lesions detected with MRI have a low likelihood of representing HCC, and MRI follow-up of such lesions is a reasonable approach. Lesions that increase in size, convert to hypointense on subsequent T1W images, convert to hyperintense in T2W images, or develop rim enhancement on follow-up MRI images are concerning and should prompt consideration of intervention**(Parente et al., 2012).**

Magnetic resonance imaging (MRI) represents an extremely useful method in detecting of early HCC and in follow up post locoregional therapy**.**

Keywords: Hypervascular focal lesions**,** Liver cirrhosis, HCC, MRI, dynamic study, DWI, ADC maps, T1 WIs, T2 WIs.

Introduction

Cirrhotic livers are characterized by irreversible remodeling of the hepatic architecture, including bridging fibrosis and a spectrum of hepatocellular nodules ***(Forner et al., 2008)***

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 non-triadal arteries. High-grade dysplastic nodules and early HCCs usually are hypovascular, but they may enhance in the arterial phase, whereas those that are larger and more advanced usually appear as hypervascular nodules.

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.

In several studies, including a meta-analysis, the specificities of MR imaging and CT were found to be comparable for depicting HCC in the cirrhotic liver,