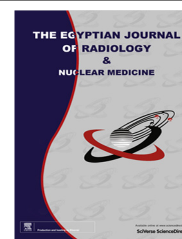
The Egyptian Journal of Radiology and Nuclear Medicine (2014) 45, 619–626

Egyptian Society of Radiology and Nuclear Medicine

The Egyptian Journal of Radiology and Nuclear Medicine

www.elsevier.com/locate/ejrnm
www.sciencedirect.com



REVIEW

MRI evaluation of fatty liver in day to day practice: Quantitative and qualitative methods

Kiran Gangadhar *, **Kedar N. Chintapalli** [1], **Gilbert Cortez** [1], **Sandhya Vinu Nair** [1]

Department of Body Imaging and Intervention, UTHSCSA, San Antonio, TX, USA

Received 2 March 2014; accepted 16 May 2014
Available online 10 June 2014

KEYWORDS

Hepatic steatosis;
Non-alcoholic fatty liver disease;
MRI liver;
MRI methods;
Chemical shift imaging

Abstract Intracellular fat accumulation is a common feature of liver disease. Steatosis is the histological hallmark of non-alcoholic fatty liver disease (NAFLD) but also may occur with alcohol abuse, viral hepatitis, HIV and genetic lipodystrophies, and chemotherapy. This condition is common in the Western population and is typically associated with obesity and the metabolic syndrome. Early diagnosis and early treatment of NAFLD are important to prevent the development of end-stage liver disease and cancer. In addition, liver fat is a risk factor for postoperative complications after liver resection and transplantation. MRI has become a primary modality to assess hepatic steatosis, both qualitatively and quantitatively. In this article we discuss various MRI methods for evaluation of hepatic steatosis.
© 2014 Production and hosting by Elsevier B.V. on behalf of Egyptian Society of Radiology and Nuclear Medicine. Open access under CC BY-NC-ND license.

Contents

1. Introduction . . . 620
2. Chemical shift imaging (dual echo) . . . 620
3. Multi-echo Dixon sequences . . . 621
4. MR spectroscopy . . . 624
5. Conclusion . . . 625

Abbreviations: BMI, body mass index; NAFLD, nonalcoholic fatty liver disease; NASH, nonalcoholic steatohepatitis; PDFF, proton density fat fraction; MRI, magnetic resonance imaging; MRS, magnetic resonance spectroscopy; GRE, gradient echo; TE, echo time; TR, repetition time; USG, ultrasonography; LFT, liver function tests.

* Corresponding author. Address: 4900 Medical Drive, #811, San Antonio, TX, USA. Tel.: +1 314 262 3552.
E-mail addresses: kirang.585@googlemail.com, gangadhar@uthscsa.edu (K. Gangadhar), chintapalli@uthscsa.edu (K.N. Chintapalli), cortezG3@uthscsa.edu (G. Cortez), VinuNair@uthscsa.edu (S.V. Nair).
[1] 7703 Floyd Curl Dr San Antonio, TX 78229, USA.

Peer review under responsibility of Egyptian Society of Radiology and Nuclear Medicine.

0378-603X © 2014 Production and hosting by Elsevier B.V. on behalf of Egyptian Society of Radiology and Nuclear Medicine.
Open access under CC BY-NC-ND license. http://dx.doi.org/10.1016/j.ejrnm.2014.05.015

Authors' contributions . . . 625
Conflict of interest . . . 625
Disclosures . . . 625
Consent . . . 625
Human and animal rights . . . 626
Acknowledgements . . . 626
References . . . 626

1. Introduction

Fatty liver disease refers to a spectrum of conditions characterized by accumulation of increasing amounts of triglycerides within the hepatocyte. There is a wide variation in incidence rates for NAFLD (1). An estimated prevalence of 25–35% is seen in the general population of the United States (2). Whereas, a study from Greece revealed evidence of steatosis in 31% and NASH in 40% of autopsied cases of ischemic heart disease or traffic accident death after exclusion of hepatitis B seropositivity or known liver disease (3). The prevalence of fatty liver disease is higher among those who consume large quantities (> 60 g per day) of alcohol (45%), those with hyperlipidemia (50%) or obesity (body mass index, > 30 kg/m^2) (75%), and those with both obesity and high alcohol consumption (95%) (4).

The exact pathogenesis of NAFLD remains poorly understood. The current hypothesis by experts in the field is that several insults are involved in causing progressive liver injury (5). With the initial hit, macrovesicular steatosis results. Insulin resistance (6) most likely plays a central role in the net retention of lipids, particularly triglycerides, within the hepatocytes. Although the mechanisms have not been completely elucidated, this is thought to result from decreased disposal of fatty acids due to impaired mitochondrial β-oxidation (7). The second insult is generally due to oxidative stress, which causes peroxidation of lipids in the hepatocyte membrane, cytokine production, and Fas ligand induction (8) and is in large part responsible for the progression from steatosis to NASH to cirrhosis. Bacterial toxins (9), overproduction of cytokines (especially tumor necrosis factor-α) (10), and alteration of hepatocyte ATP stores and cytochrome P450 Cyp2E1/Cyp4A enzyme activity (11) are also considered as potential triggers for disease progression and fibrogenesis.

Furthermore, hepatic steatosis has been reported to affect the progression of various chronic liver diseases. For example, hepatic steatosis has been found to adversely affect the progression of hepatic fibrosis and the response to antiviral treatment in patients with hepatitis C (12–14). Moreover, hepatic steatosis has been found to be an important cofactor in liver injury observed in patients with hemochromatosis and alcoholic liver disease. Therefore, the assessment of hepatic steatosis may have important clinical implications in the management of patients with chronic liver disease (15). In order to grade steatosis, pathologists visually estimate the fraction of hepatocytes that contain fat droplets. Typically, a five-point ordinal scale is used (0%, 1–5%, 6–33%, 34–66%, or > 67%). The size of fat droplets is not considered (16). However, most agree that a very mild degree of steatosis involving less than 5% of hepatocytes may not actually represent a true pathologic abnormality (2).

MRI is an attractive modality to assess hepatic steatosis. In and out of phase MRI, calibrated with robust liver/fat standards, have been found to be superior in quantifying hepatic steatosis, when compared with non-invasive methods (17). Advanced MRI techniques currently under development have demonstrated high potential for accurate detection and quantification of hepatic steatosis using proton density fat-fraction (18). MR imaging (chemical shift imaging, multi-echo Dixon method) and MR spectroscopy for quantifying liver fat will be discussed in later sections.

2. Chemical shift imaging (dual echo)

On MRI, fatty liver has high signal intensity on T1-weighted images. In addition, several MRI sequences, including fat-suppression sequences and chemical shift imaging with OP gradient recalled echo sequences facilitate the detection of fat (19). The magnitude-based approach is probably the most commonly used MR approach for liver fat assessment in current practice. Typically, two gradient echoes are acquired, one employing a TE in which the water peak (4.7 ppm) and the dominant fat peak (1.3 ppm) are "out of phase" and hence subtractive, and the other using a TE in which the two peaks are "in phase" and therefore additive. Because two echoes are acquired, this is often called "dual-phase" or "dual echo" imaging. The TE corresponding to in-phase and out-of-phase (IOP) depend on the magnet field strength. At 1.5 T, the chemical shift between water and the dominant fat peak (3.4 ppm) corresponds to a resonance frequency difference of 217 Hz (i.e. the main fat peak resonates 217 Hz slower than the water peak). Therefore, at 1.5 T, signals from water and the main fat peak oscillate with a period of 4.6 ms (1000 ms/217 Hz). At 3 T, the chemical shift corresponds to a frequency difference of 434 Hz (double that at 1.5 T). Therefore, the oscillation period at 3 T (2.3 ms) is half that at 1.5 T (4.6 ms) and the corresponding IP and OP echo times at 3 T are halved: water and the main fat peak are in phase every 2.3 ms (i.e., 2.3, 4.6, and 6.9 ms) and out of phase at 1.15 ms and every subsequent multiple of 2.3 ms (i.e., 1.15, 3.45, and 5.75 ms) (20).

To estimate the signal fat-fraction, we assume (1) the signal intensity from fat is less than the signal intensity from water (i.e. $S_{Fat} \leqslant SI_{Water}$); (2) the signal intensity from OP images represents the difference in water and fat signals (i.e., $S_{OP} = S_{Water} - S_{Fat}$); and (3) the signal intensity from IP images represents the sum of water and fat signals (i.e., $S_{IP} = [S_{Water} + S_{Fat}]$) (21). Fat Signal Percentage is calculated as $[S_{IP} - S_{OP}]/[2 \times SIP] \times 100$. The dynamic range of magnitude-based chemical shift techniques has typically a 0–50% signal fat-fraction (Figs. 1–3) (18).

The use of MR imaging with the chemical shift imaging for the detection and quantification of fatty liver provides the

Download English Version:

https://daneshyari.com/en/article/4224113

Download Persian Version:

https://daneshyari.com/article/4224113

Daneshyari.com