Evaluation of Cirrhosis and Chronic Hepatitis with Diffusion Weighted Imaging and ADC Measurements

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Introduction

The natural history of chronic hepatitis involves progression of hepatic fibrosis to cirrhosis. Assessment of hepatic fibrosis is very important in the evaluation of these patients for the purpose of staging, because fibrosis is the main determinant of clinical outcomes. The diagnosis of early stages of fibrosis permits treatment of the cause of the disease and has been shown to prevent progression to cirrhosis. Conversely, if chronic liver disease is detected at the cirrhotic stage, it is irreversible and often revealed by its complications which can be life threatening. Liver histology is considered the gold standard for establishing the severity of hepatic fibrosis. However, percutaneous liver biopsy is associated with morbidity (and rarely, mortality), patient discomfort, and expense. Additionally, interobserver variability and sampling error may lead to erroneous staging. Magnetic resonance imaging (MRI) has been widely used to evaluate the cirrhotic liver disease. Several morphological imaging characteristics of cirrhosis have been described in the literature. However, these changes occur at the cirrhotic stage, and no imaging test provides reliable detection of early fibrosis. Imaging therefore cannot obviate the need for liver biopsy. MR measurement of the apparent diffusion coefficient (ADC) allows quantification of microscopic molecular motion of water. Liver ADC measurements were obtained in patients with cirrhosis, chronic hepatitis, and normal livers, with the goal of eventual utilization of the ADC for staging of hepatic fibrosis.

Materials and Methods

34 patients were imaged on a GE 1.5T Excite Spectrometer using a torso-phased-array coil. Following the acquisition of our routine pre contrast sequences, diffusion-weighted imaging (DWI) was performed during suspended respiration using single shot echoplanar imaging with isotropic diffusion weighting (TR/TE 6,000/62.5 msec, 40x40 cm FOV, 128x128 matrix size, 10mm slice thickness, b values of 0 and 500 s/mm^2, 24 sec breath-hold). ADC maps were generated using vendor-provided software (FuncTool 2, GEMS). Cirrhosis was determined by histology and clinical/imaging features. Chronic hepatitis was determined by viral titer, liver function test and/or liver biopsy.

Results

19 patients had cirrhosis, 4 patients had chronic hepatitis, and 11 patients had no known liver disease (normal liver). The mean ADC and standard deviation for patients with cirrhosis was (1.15 +/- 0.16)x10^{-3}mm^2/s, for patients with chronic hepatitis was (1.38 +/- 0.17)x10^{-3}mm^2/s, and for normal liver was (1.58 +/- 0.15)x10^{-3}mm^2/s. The liver ADC values were significantly lower in patients with cirrhosis compared with normal livers ($p<0.0001$). Liver ADC values in patients with chronic hepatitis and early fibrosis without cirrhosis were higher than in cirrhosis but lower than in normal livers (Figure 1).

Discussion

Taouli et al (1) were the first to show that the ADC of cirrhotic liver is lower than that of the normal liver, possibly reflecting increased cellularity/fibrosis, leading to decreased free water motion. Aube et al (2) reported similar findings and correlated the ADC values with the Child-Pugh scores of cirrhosis. Both studies used an upper b-value of 500 sec/mm^2, which is believed to allow most accurate quantification of ADC. However, both studies used relative small patient populations: 9 cases of cirrhosis in Taouli et al (1) and 13 cases of cirrhosis in Aube et al (2). Neither study included patients with chronic liver disease without cirrhosis. Koinuma et al (3) measured liver ADC in patients with cirrhosis and chronic hepatitis. However, the authors used a b-value of 195 sec/mm^2. The use of small b values (<300 s/mm^2) likely overestimates ADCs (3) and higher b values (>1000 s/mm^2) likely underestimates ADCs (1-2). In this study, we therefore measured ADC values in cirrhotic and chronic hepatitis patients, along with normal livers, using this b-value of 500 s/mm^2 (and 0 s/mm^2 as the lower b-factor). Although preliminary, our data suggest that ADC values drop with the progression of fibrotic liver disease, and that liver ADC below 1.25-1.30 X 10^{-3} may signal almost certain cirrhosis. The study is, however, limited due the small patient population enrolled thus far. We plan to extend the study to confirm our initial findings in a larger patient population. ADC trends may allow monitoring of progression of fibrotic liver disease and obviate the need for serial liver biopsy in the future.

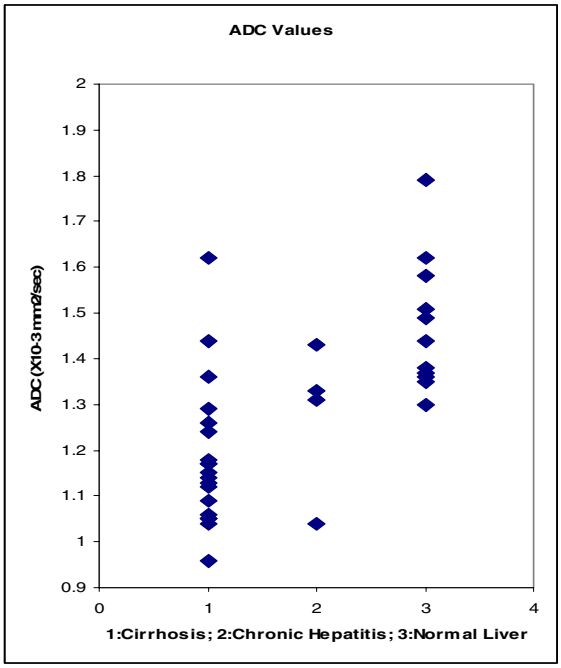

Fig. 1: Scatterplot of liver ADC values in patients with cirrhosis (n=19), chronic hepatitis (n=4), and no known liver disease (normal liver) (n=11). Mean ADC and standard deviation is (1.15 +/- 0.16)x10^{-3}mm^2/s for cirrhosis, (1.38 +/- 0.17)x10^{-3}mm^2/s for chronic hepatitis and (1.58 +/- 0.15)x10^{-3}mm^2/s for normal liver. P value 4.7 X10^{-5} between cirrhosis and normal liver.

References

1, Taouli B. *et al.* Radiol 2003;226:71-78.
2. Aube C. *et al*, J Radiol 2004;85:301-306.
3. Koinuma M. *et al.* JMRI 2005;22:80-85.