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CLINICAL—LIVER

Magnetic Resonance Imaging More Accurately Classifies Steatosis and Fibrosis in Patients With Nonalcoholic Fatty Liver Disease Than Transient Elastography

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See Covering the Cover synopsis on page 540; see editorial on page 553.

BACKGROUND & AIMS: Noninvasive methods have been evaluated for the assessment of liver fibrosis and steatosis in patients with nonalcoholic fatty liver disease (NAFLD). We compared the ability of transient elastography (TE) with the M-probe, and magnetic resonance elastography (MRE) to assess liver fibrosis. Findings from magnetic resonance imaging (MRI)–based proton density fat fraction (PDFF) measurements were compared with those from TE-based controlled attenuation parameter (CAP) measurements to assess steatosis. **METHODS:** We performed a cross-sectional study of 142 patients with NAFLD (identified by liver biopsy; mean body mass index, 28.1 kg/m^2) in Japan from July 2013 through April 2015. Our study also included 10 comparable subjects without NAFLD (controls). All study subjects were evaluated by TE (including CAP measurements), MRI using the MRE and PDFF techniques. **RESULTS:** TE identified patients with fibrosis stage $\geq$2 with an area under the receiver operating characteristic (AUROC) curve value of 0.82 (95% confidence interval [CI]: 0.74–0.89), whereas MRE identified these patients with an AUROC curve value of 0.91 (95% CI: 0.86–0.96; $P = .001$). TE-based CAP measurements identified patients with hepatic steatosis grade $\geq$2 with an AUROC curve value of 0.73 (95% CI: 0.64–0.81) and PDFF methods identified them with an AUROC curve value of 0.90 (95% CI: 0.82–0.97; $P < .001$). Measurement of serum keratin 18 fragments or alanine aminotransferase did not add value to TE or MRI for identifying nonalcoholic steatohepatitis. **CONCLUSIONS:** MRE and PDFF methods have higher diagnostic performance in noninvasive detection of liver fibrosis and steatosis in patients with NAFLD than TE and CAP methods. MRI-based noninvasive assessment of liver fibrosis and steatosis is a potential alternative to liver biopsy in clinical practice. UMIN Clinical Trials Registry No. UMIN000012757.

Keywords: Diagnosis; Classification; Alanine Transaminase; Overweight.

Nonalcoholic fatty liver disease (NAFLD) is an important cause of chronic liver injury in many countries.[1,2] A recent study has shown that the risk of developing NAFLD is 4–11 times higher in patients with metabolic syndrome as compared with healthy individuals.[3] NAFLD ranges from benign nonalcoholic fatty liver (NAFL) to nonalcoholic steatohepatitis (NASH). This latter condition includes progressive fibrosis[4] and hepatocellular carcinoma.[5,6] Liver biopsy is recommended as the gold standard method for the diagnosis and grading of steatosis, hepatic inflammation, and hepatocellular ballooning, and the staging of liver fibrosis in patients with NASH.[7] However, because of increased cost, possible risk, and health care resource use, an invasive liver biopsy is a poorly suited diagnostic test for such a prevalent condition.[8] In addition, the histologic lesions of NASH are unevenly distributed throughout the liver parenchyma, therefore, liver biopsy sampling error can result in substantial stratification and staging inaccuracies.[9]

Assessment of the severity of liver fibrosis and steatosis is important in the evaluation of the stage of NAFLD. Transient elastography (TE; Fibroscan, EchoSens, Paris, France) is a useful technique that allows rapid and noninvasive measurement of mean tissue stiffness.[10] We have reported that liver stiffness measurement (LSM) obtained using TE was useful for estimation of severity of liver fibrosis in NAFLD.[11,12] Meta-analysis of TE has also shown that LSM accurately reflected liver fibrosis.[13] In addition,

Abbreviations used in this paper: **AUROC, area under the receiver operating characteristic; BMI, body mass index; CAP, controlled attenuation parameter; CI, confidence interval; LSM, liver stiffness measurement; MRE, magnetic resonance elastography; MRI, magnetic resonance imaging; NAFL, nonalcoholic fatty liver; NAFLD, nonalcoholic fatty liver disease; NAS, Nonalcoholic Fatty Liver Disease Activity Score; NASH, nonalcoholic steatohepatitis; PDFF, proton density fat fraction; ROI, region of interest; TE, transient elastography.**

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recent interest has shifted toward the controlled attenuation parameter (CAP), which is based on the properties of ultrasonic signals acquired using TE.[14] Recent clinical studies using the TE system have demonstrated an increase in the CAP with severe fat accumulation and liver stiffness with advanced fibrosis in NAFLD patients.[15–17] Body mass index (BMI) >28 kg/m^2 has been identified as an independent risk factor for failure to measure liver elastography and fat accumulation.[18] Therefore, TE has the limitation that sometimes LSM and CAP cannot be measured in morbidly obese patients with NAFLD.

Magnetic resonance elastography (MRE) is a magnetic resonance imaging (MRI)-based method for quantitatively imaging tissue stiffness, and is available from several manufacturers of MRI scanners as an option that includes special hardware and software. Quantitative stiffness images (elastograms) of the liver can be obtained rapidly during breath-hold acquisitions, and can therefore be readily included in conventional liver MRI protocols.[19] Multiple studies have also shown that MRE-based LSM provides an accurate biomarker for detecting the presence of fibrosis in patients with chronic liver dysfunction.[20–24] Indeed, MRE has been reported to be a useful method for the diagnosis of liver fibrosis in patients with NAFLD, even in the early stages.[25–27] Proton density fat fraction (PDFF) measurement is an MRI-based method for quantitatively assessing hepatic steatosis and is available from several manufacturers of MRI scanners as an option. MRI-determined PDFF correlates with histologically determined steatosis grade in patients with NAFLD.[28,29] Although MRI can be performed even in morbidly obese patients with NAFLD, where it is sometimes difficult to perform TE, there are no reports that have directly compared the diagnostic accuracy of MRI and TE for assessing both fibrosis and steatosis in patients with NAFLD.

Table 1. Clinical, Serologic, and Histologic Characteristics of Control Subjects and Patients With NAFLD

Characteristic	Control	NAFLD	*P* value
n	10	142	
Age, y, mean ± SD	52.1 ± 15.1	57.5 ± 14.6	.362
Sex, male/female	6/4	81/61	.321
BMI, *kg/m^2*, mean ± SD	21.9 ± 0.69	28.1 ± 4.63	<.001
Platelets, */10^4 μL*, mean ± SD	22.8 ± 4.31	20.9 ± 7.69	.442
AST, *IU/L*, mean ± SD	23.4 ± 8.12	44.5 ± 26.3	<.001
ALT, *IU/L*, mean ± SD	24.3 ± 7.31	56.2 ± 42.6	<.001
γ-GTP, *IU/L*, mean ± SD	39.0 ± 6.73	80.0 ± 87.7	.297
C-reactive protein, *mg/L*, mean ± SD	0.06 ± 0.03	0.17 ± 0.09	.003
Creatinine, *mg/dL*, mean ± SD	0.63 ± 0.32	0.77 ± 0.42	.672
Fasting blood glucose, *mg/dL*, mean ± SD	92.1 ± 16.3	110.3 ± 28.2	.001
Fasting insulin, *μU/mL*, mean ± SD	7.47 ± 2.98	19.2 ± 20.8	.001
HbA1c, mean ± SD	5.62 ± 0.61	6.43 ± 1.10	.002
Diabetes mellitus, %	0	71 (50.0)	
Hypertension, %	0	45 (31.7)	
Dyslipidemia, %	0	94 (66.2)	
Length of specimens, *mm*, mean ± SD		21.3 ± 1.94	
Number of portal areas, mean ± SD		14.3 ± 4.42	
Steatosis grade, n			
5%–33%		59	
33%–66%		59	
>66%		24	
Lobular inflammation (n)			
None		6	
<2 foci per 200× field		78	
2–4 foci per 200× field		52	
>4 foci per 200× field		6	
Liver cell ballooning (n)			
None		32	
Few balloon cells		96	
Many balloon cells		14	
NAFL/NASH, n		34/108	
NAS, n			
1/2/3/4/5/6/7		6/15/32/51/30/5/3	
Fibrosis stage, n			
None		14	
Perisinusoidal or periportal		51	
Perisinusoidal and portal/periportal		32	
Bridging fibrosis		34	
Cirrhosis		11	

ALT, alanine transaminase; AST, aspartate aminotransferase; γ-GTP, gamma-glutamyl transferase.

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