Letter to Editor

Noninvasive Methods to Diagnose NAFLD

Sir,

We read with great interest the article on noninvasive methods of diagnosing hepatic steatosis by AlShaalan et al. published in this Journal.^[1] We wish to supplement additional noninvasive radiological markers available for the diagnosis of NAFLD.

Qualitative grading of steatosis has been proposed on ultrasound. Grade I is increased liver echogenicity and clear depiction of hepatic and portal vein walls, Grade II is increased liver echogenicity obscuring the hepatic and portal vein walls, and Grade III is increased liver echogenicity and significant posterior shadowing that impairs evaluation of the deep liver parenchyma and diaphragm.^[2] Acoustic radiation force impulse (ARFI) elastography measures liver stiffness as a function of the extent of hepatic infiltration in the form of shearing velocity. Normal velocity of liver is 1 m/s. This velocity decreases once there is fatty infiltration.

Dual-energy computed tomography (CT) is a relatively new technique for the assessment of hepatic steatosis. An increase in fat content leads to a decrease in the CT numbers at low energy; as the energy level increases, the fat attenuation increases. Raptopoulos et al. found that an attenuation change of more than 10 HU with a tube potential change from 140 and 80 kVp was indicative of fatty infiltration of more than 25%.^[3]

MR elastography can be used to measure liver stiffness. MRE is a three-step technique: (1) Generating mechanical waves in tissue; (2) imaging the waves with a special MRI sequence, and (3) processing the wave information to generate elastogram images that quantitatively depict tissue stiffness. These quantitatative images typically depict shear stiffness in units of kilopascals (kPa), and may be displayed in a gray scale or with a color scale. Normal liver parenchyma has shear stiffness values less than 3 kPa. In patients with NAFLD, steatosis alone does not appear to have a significant effect on hepatic stiffness. However, it has been shown that if the disease progresses to nonalcoholic steatohepatitis, hepatic stiffness does increase, even before the onset of fibrosis.^[4]

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