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Letter to the Editor

Visceral Fat and Liver Fat as Risk Factors of Metabolic Syndrome

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We have read with great interest the article "Comparison of Visceral Fat and Liver Fat as Risk Factors of Metabolic Syndrome" in a recent issue of the Journal of Korean Medical Science by Kang et al. (1). Although Dr. Kang and colleagues reported liver fat may be a more important risk factor than visceral fat in this study, we would like to add a few comments on this study to avoid misinterpretation of results.

Our first concern is the some methodological problems regarding the measurement of intra-hepatic fat infiltration. The authors mentioned they applied a liver-to-spleen attenuation ratio ≤ 1.1 at the T12 level to evaluate fatty liver. To assess the prevalence of steatosis by CT, several diagnostic criteria can be applied: liver attenuation ≤ 40 HU, liver to spleen attenuation difference and liver-to-spleen attenuation ratio ≤ 1.1. Among these criteria, a liver attenuation value ≤ 40 HU is known to represent the most accurate method in assessing hepatic fat (2, 3). Considering the importance of attenuation value itself, the authors should have used not only liver-to-spleen attenuation ratio but also the attenuation value and L-S difference of CT. Furthermore, attenuation value of MS group was relatively high in this study (55.7 ± 10.1). Thus, it is difficult to quantify the accumulation of liver fat by liver-to-spleen ratio only.

We are also concerned about the large (200 mm^2) region of interest (ROI) used by authors. When analyzing the body composition, careful choice of measurement location with adequate ROI size is crucial. Most previous studies measured liver attenuation with relatively small ROI size (100-150 mm^2), because the ROI for the liver should be placed manually to avoid major vessels and bile duct. Kang et al. might have difficulty in placing ROI without large vascular structure at T 12 level. In addition, the authors described that they obtained the mean liver attenuation from an average of 4 selected areas including the right anterior lobe, right posterior lobe, and left-interior lobe of the liver. However, figure 1 of article only shows that ROI was drawn inside right posterior, right anterior and left medial lobe. We wonder where left-interior lobe mentioned by authors is located.

Another question concerning technical aspect is the attenuation range of adipose tissue. Although the authors mentioned visceral fat area was measured using an attenuation range of 30 to -190 Hounsfield units (HU), this range include a skeletal muscle tissue. The adipose regions should be obtained using the range below -30 HU for pixels. This is the most important technical consideration in measuring the visceral fat area that Kang et al. have overlooked.

Final comment on method of study is that the authors presented they made a diagnosis of metabolic syndrome by NCEP-ATP III criteria. However, it seems that diagnosis of metabolic syndrome in this study was based on the International Diabetes Federation (IDF) definition; Central obesity with any two of the following risk factors (increased TG levels, decreased HDL levels, elevated blood pressure or the use of antihypertensive medication, increased FBS or the use of anti-diabetics).

Our final concern is related to the conclusions of the article. Kang et al. concluded fatty liver was found to be significantly associated with metabolic syndrome, but visceral obesity was not a risk factor of metabolic syndrome. However, this associations found by the authors may be confounded by statistical error. The small sample size and influence of extreme value of this study might produce an extraordinary high odds ratio and very wide confidence interval (odds ratio 71.3; 95% CI 13.04-389.53). This width of the confidence interval just gives us some idea about uncertainty of this study. A possible way for better estimation would be to collect more data before any definite association can be said. Taken together, the results of Kang et al. (1) should be interpreted with caution.

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