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Letter to the Editor

Hepatic steatosis, visceral adiposity, insulin resistance, adiponectin, and inflammation

To the Editor:

We read with great interest the article by Wasada et al [1] on the association of liver steatosis with insulin sensitivity in obese patients. They report that, when compared with a demographically matched healthy group, decreased insulin sensitivity and altered adipokine metabolism in hepatic steatosis are not associated with the presence of increased adiposity in people with fatty liver (FL). The results are interesting and likely to contribute to our understanding of the pathophysiology of nonalcoholic fatty liver disease (nonalcoholic steatohepatitis); however, we have some concerns about the data presented by the authors.

Although it was stated in the text that patients with serum glucose of at least 6.11 mmol/L were not included in the study, Table 1 of the article by Wasada et al shows that some of the patients with either mild or moderate to severe FL as well as body mass index–matched non-FL controls have overt glucose dysregulation or diabetes mellitus. We know from previous studies that hypoadiponectinemia occurs not only in diabetes but also in people with impaired glucose tolerance [2], which is a condition with insulin resistance as well. An oral glucose tolerance test for those individuals with increased fasting serum glucose before enrollment is mandatory in such instances. Moreover, in Table 1, some of the patients with moderate to severe FL seem hypertensive, although it was also noted that no patients with systolic blood pressure greater than 130 mm Hg were included in the study. As in the case of hyperglycemia, increased blood pressure has been shown to be closely associated with low blood adiponectin concentration [3,4], which can be an explanation of a greater decrease in the blood level of this peptide in moderate to severe FL group compared with patients with mild FL.

We conclude that, before making certain interpretations, presence of major confounders for hypoadiponectinemia and insulin resistance raises some questions about the data presented. In such a case, statistical correlations may also be misleading. It would be appreciated if the authors could present some more data (adiponectin, insulin, and homeostasis model assessment score) adjusted for major confounders. This could provide the readers of the journal clearer information in relation to the link between adiposity, adipokines, insulin sensitivity, and nonalcoholic steatohepatitis. The authors should also clarify the medications used by the patients. If some patients were on treatment for diabetes or hypertension, their medications are also likely to interfere with the results [5,6].

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