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Letters to the editor

Reply to: The presence of diabetes impacts liver fibrosis and steatosis by transient elastography in a primary care population

Dear Editor,

We thank the authors of the Letter to the Editor devised about our manuscript entitled "The presence of diabetes impacts liver fibrosis and steatosis by transient elastography in a primary care population", where we demonstrate a strong association between type 2 diabetes mellitus (T2DM) and increased hepatic steatosis and fibrosis on noninvasive measurement with transient elastography (TE). We appreciate the insightful commentary, but respectfully disagree with some of their conjecture.

First off, we agree the duration of T2DM and poor glycemic control influence the progression of liver disease. The primary endpoint of our study, however, was to evaluate whether the presence of T2DM influences steatosis and fibrosis on TE, compared to those *without* T2DM irrespective of disease duration. Data on duration of disease is not reliable and therefore not included in our results. However, the primary care-based population of this cohort represents the disease phenotype of the community and enhances the generalizability of our results regardless of duration of disease. Secondly, the profound statistically significant results we demonstrate are likely to remain significant even if duration of T2DM were accounted for. We also agree that poor glycemic control likely influences liver disease progression. However, the studies quoted in the Letter to the Editor describe cohorts with hepatitis C virus, which is a vastly different population we evaluated in this study. Regardless, we adjust for hemoglobin A1c, a measure of glycemic control, in our regression model which reduces the impact it may have on our results.

The authors in the Letter to the Editor also point out differences in the baseline demographics between the two study groups. This is an inherent characteristic of observational studies that can only be avoided by randomization. Additionally, mentioned factors such as EZH1, EZH2 and OAP, are not routinely measured in the clinical scenario we are evaluating and are not relevant in this study. The authors of the Letter also mention there was no explanation for our adjustment strategy, however, this is false. I would like to refer them to the Statistical analysis section, which explains the *stepwise* method employed for our regression analysis, a commonly used technique for multivariable adjustment. While we agree that longitudinal, prospective trials are required in this area of study, we are confident our results are hypothesis generating and reproduce similar findings demonstrated in study populations outside of the United States.

Conflict of interest

There are no conflicts of interest or funding to report for the creation of this manuscript.

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