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Letters to the editor

Reply to: Type 2 Diabetes Complications are Associated with Liver Fibrosis Independent of Hemoglobin A1c

Dear Editor,

We thank the authors of the Letter to the Editor devised about our manuscript entitled "Type 2 diabetes complications are associated with liver fibrosis independent of hemoglobin A1c", where we demonstrate patients with complications from type 2 diabetes mellitus have 4.5 times greater odds of liver fibrosis, measured serologically by FIB-4 index, compared to those without type 2 diabetes complications. We appreciate the insightful commentary from our colleagues and provide our reply to their statements.

First off, our colleagues mention the importance of certain diabetes medications, namely metformin, and the impact they have on the development of liver fibrosis. Unfortunately, the data on metformin's impact on liver fibrosis is limited to retrospective studies and preclinical animal models. Although the studies our colleagues reference suggest a beneficial association of metformin, these data consisted mostly of nondiabetic patients, and therefore cannot be extrapolated to our study population. The fibrotic benefit of metformin in those with type 2 diabetes requires additional prospective analysis. Nonetheless, we agree studying the type of medication used to treat type 2 diabetes is important when evaluating the true association with liver fibrosis. It is therefore paramount to include them into a multivariable model when doing a retrospective analysis.

Our colleagues mention the importance of additional factors which may influence the association of type 2 diabetes complications and liver fibrosis. We agree that factors such as medication adherence and duration of type 2 diabetes may influence the development of liver fibrosis, but unfortunately were not able to capture this information reliably in our retrospective review of the record. These factors should certainly be considered when studying the impact of diabetes complications on liver fibrosis prospectively. On the other hand, our colleagues mention the importance of poor glycemic control measured by serum glucose levels over time, and its impact on liver fibrosis. We believe the hemoglobin A1c (HbA1c) to be a more reliable measure of glycemic control. Given that our findings are independent of HbA1c level, we do not think the addition of serum glucose measurements over time would affect our results. It is of no doubt that other potential confounders may exist and ultimately influence the study results, but validation of these data through coordinated prospective analyses is warranted.

Declaration of Competing Interest

None.

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