Indirect insulin resistance markers in relation to nonalcoholic fatty liver disease among patients with type 1 diabetes mellitus

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Authors' reply We would like to thank Chen et al[1] for their interest in and valuable comments on our recently published work on the insulin resistance markers and their association with nonalcoholic fatty liver disease (NAFLD) in individuals with type 1 diabetes mellitus (T1DM).[2] The authors pointed out some probable limitations of our study, which we address below.

One of the mentioned downsides was that we had based our analysis on anthropometric measurements, blood pressure values, and laboratory test results rather than on diagnoses of comorbidities. In fact, according to the adopted estimated glucose distribution rate (eGDR) formula, we took into consideration the diagnosis of arterial hypertension; whereas in the visceral adiposity index and triglyceride to high-density lipoprotein cholesterol ratio calculation formulae, we used anthropometric measurements and lipid profile components, regardless of concomitant (treated or untreated) dyslipidemia. We believe that anthropometric measurements and laboratory test results accurately described the metabolic state of the analyzed population, even if they were a result of implemented treatment. Moreover, as stated in the manuscript, we assume that T1DM does not typically coexist with comorbidities typical for metabolic syndrome,[3] which develop rather as a result of reduced insulin sensitivity. We also believe that in T1DM, the phenomenon of insulin resistance is secondary to the glycemic control, insulin administration regimen, as well as lifestyle and dietary habits, unlike in type 2 diabetes, where insulin resistance is the essence of the disease. Thus, we cannot agree with the authors of the letter that taking into account the diagnoses of comorbidities, instead of anthropometric measurements and laboratory results, also in the statistical analyses, would have eliminated the confounding effect.

The other mentioned downside was that we had not considered the duration of comorbidities, such as hypertension or hyperlipidemia, or other conditions (ie, the intensity of tobacco smoking and alcohol consumption). We were not able to present these data as they were not available. We analyzed the duration of T1DM, as we acknowledged the fact of elevated risk for NAFLD with increasing level of glycated hemoglobin.[4] We presented data on the percentage of smokers (18%) in the study group; however, the information on pack-years smoked was not available, and thus not reported. Regarding alcohol consumption, in the *Patients and methods* section we stated that patients with overconsumption of alcohol had been excluded from the study.

We agree with the comment that certain medications / supplements or dietary components (such as vitamin E, ω-3 fatty acids, caffeine, polyphenols, and the Mediterranean diet) can affect the risk of NAFLD. Unfortunately, data on dietary habits in the study group were not available and could not be included in the analysis.

Indisputably, taking into consideration the issues mentioned by Chen et al[1] can allow the readers to acknowledge probable limitations of our study and understand it despite the related bias. Still, we believe that our analysis has taken us a step closer to elucidating the complex phenomenon of NAFLD in T1DM.

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Published online: August 30, 2023.
Pol Arch Intern Med. 2023
doi:10.20452/pamw.16553

ARTICLE INFORMATION

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CONFLICT OF INTEREST None declared.

HOW TO CITE Grzelka-Woźniak A, Uruska A, Szymańska-Garbacz E, et al. Indirect insulin resistance markers in relation to nonalcoholic fatty liver disease among patients with type 1 diabetes mellitus. Authors' reply. Pol Arch Intern Med. 2023; 133: 16553. doi:10.20452/pamw.16553

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