

Comment on “Coronary artery calcium in type 2 diabetes: a nested case-control study”

To the Editor The coronary artery calcium score (CACS) emerges as a useful marker of coronary artery atherosclerosis and cardiovascular risk.^{1,2} Coronary calcifications are related to coronary artery plaque burden but not to plaque instability and cannot be used to exclude coronary atherosclerosis. The measurement of the CACS may play an important role in the reclassification of subjects into high and low risk of cardiovascular death.

Due to an increasing burden of diabetes in the Polish population, the correct assessment of atherosclerotic burden and risk stratification in these subjects is crucial.³ To date, there have been several studies aiming to determine the role of CACS measurement in patients with diabetes. Sosnowski et al.⁴ conducted an interesting nested case-control study comparing the CACS between subjects with and without type 2 diabetes. In their report, symptomatic subjects with diabetes demonstrated a higher CACS, showing a link between diabetes and coronary calcifications independent of other risk factors. Their findings support the presence of higher coronary artery plaque burden and cardiovascular risk in diabetic subjects. Interestingly, despite the exclusion of individuals with high pretest probability of coronary artery disease, the high proportion of patients with diabetes had a CACS higher than 400, which is considered as a surrogate for high cardiovascular risk. It is widely accepted that the CACS, plaque morphology, and plaque burden assessed by multidetector computed tomography provide an incremental prognostic value in subjects with suspected coronary artery disease.⁵ However, it is still unclear whether and how this increased risk should be managed. Nevertheless, the reclassification of cardiovascular risk based on the CACS and plaque morphology seems to be an attractive concept, and more data are needed to modify the preventive strategy based on imaging data. The issue is particularly important in diabetic subjects with a high cardiovascular risk per se.

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Authors' reply We read the commentary of Miszałski-Jamka with great attention. Our single-center study was conducted on a sample of patients with type 2 diabetes and angina-like symptoms but without a high risk of coronary artery disease (CAD). Thus, the conclusions drawn from studies on coronary artery calcium in asymptomatic populations might not necessarily be applicable (especially regarding risk stratification). On the other hand, as diabetic neuropathy in type 2 diabetes of unknown duration (the onset cannot be determined) might have already developed, a substantial proportion of such patients would never experience angina-like chest pain or discomfort. Thus, the recommendations for coronary artery calcium (CAC) scoring in asymptomatic subjects might be useful in such patients (not examined in our study).

In our center, CAC scoring is scheduled for symptomatic patients (with or without diabetes) without objective indications for invasive coronary angiography (presence of ischemia). If any

coronary calcifications are detected, a decision for invasive study seems more justified (a positive CAC score denotes the presence of coronary atherosclerotic plaques, not necessarily significant). In the absence of calcified plaques, noninvasive computed tomography (CT) coronary angiography is performed, allowing for the fact that, as stated by Miszalski-Jamka, the absence of coronary calcifications does not exclude the presence of significant noncalcified lesions. As our CAC scoring approach cannot be considered as being in line with the guidelines (CAC scoring for asymptomatic and CT coronary angiography for symptomatic patients), we were unable to find any comparators for our findings. In summary, our diagnostic CT steps were aimed to exclude CAD rather than confirm its presence.

We are aware that in our study, due to a retrospective design, several important clinical data are missing, especially in the population of patients with diabetes. In a prospectively designed study, one should also consider glucose balance control (glycated hemoglobin), renal functional status (albuminuria, creatinine clearance), inflammatory markers (C-reactive protein, fibrinogen, and others), left ventricular hypertrophy, and concomitant diseases (for example, tobacco-related or inflammatory diseases). We found that the classification of patients based on the absence or presence of traditional risk factors cannot explain the presence of more advanced coronary artery calcified plaques in patients with type 2 diabetes.

Currently, there is no need for the reclassification of symptomatic patients with diabetes on the basis of CAC scoring. Rather, a more detailed, individual examination for the presence of other factors might be necessary not only to explain the absence or presence of coronary calcifications, but also to ensure that other factors are controlled, or at least considered.

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