CORRESPONDENCE

Epicardial adipose tissue in coronary microvascular dysfunction

International Journal of Obesity (2022) 46:1564; https://doi.org/10.1038/s41366-022-01125-z

Serkan Duyuler[1]✉ and Pınar Türker Duyuler[1]
[1]*Department of Cardiology, Bilkent City Hospital, Ankara, Turkey.*
✉*email: serkanduyuler@yahoo.com*

To the Editor:

We read the article by Mahmoud et al. entitled 'Epicardial adipose tissue differentiates in patients with and without coronary microvascular dysfunction' with interest. In this article, the authors claimed that epicardial adipose thickness may be useful in coronary microvascular dysfunction diagnosis, and interventions to decrease epicardial adipose tissue thickness may reduce coronary microvascular dysfunction [1].

Epicardial adipose tissue is a metabolically active ectopic fat accumulation regarded as a novel cardiometabolic risk factor [2]. It possesses inflammatory properties modulating local and possibly remote inflammation. Recently, we found that epicardial adipose tissue thickness is associated with the COVID-19 infection severity [3]. Evidence supports the role of inflammation in coronary microvascular dysfunction and inflammation may be the plausible link between coronary microvascular dysfunction and epicardial adiposity [4].

Coronary microvascular dysfunction can be evaluated invasively or non-invasively. However, the definition of coronary microvascular dysfunction in this article by Mahmoud et al. necessitates further clarification. In the absence of significant epicardial coronary artery stenosis, corrected TIMI frame count or TIMI flow may imply a crude status of coronary microcirculation in the advanced stages of microvascular dysfunction. In addition, the coronary slow flow may be also observed when the diffuse coronary vascular spasm or increased basal coronary vascular tone is present [5]. It is also not uncommon during radial access which was not specified in the study. Furthermore, the presence of increased left ventricular end-diastolic pressure or hypertensive heart disease may only refer to the increased likelihood of coronary microvascular dysfunction and these entities are not equivalents of coronary microvascular dysfunction. In this context, authors may clarify the definition of coronary microvascular dysfunction whether having any one of slow flow, elevated left ventricular end-diastolic pressure or hypertensive heart disease is sufficient for the diagnosis of coronary microvascular dysfunction or having all of these entities is requisite. Even though, this constellation needs to be validated with further studies.

REFERENCES

1. Mahmoud I, Dykun I, Kärner L, Hendricks S, Totzeck M, Al-Rashid F, et al. Epicardial adipose tissue differentiates in patients with and without coronary microvascular dysfunction. Int J Obes (Lond). 2021;45:2058–63.
2. Türker Duyuler P, Duyuler S, Demir M, Uçar Elalmiş Ö, Güray Ü, İleri M. Homocysteine, visceral adiposity-related novel cardiometabolic risk factors, and exaggerated blood pressure response to the exercise treadmill test. Blood Press Monit. 2017;22:333–8.
3. Turker Duyuler P, Duyuler S, Demirtaş B, Çayhan V. Epicardial and pericoronary adipose tissue in severe COVID-19 infection. Acta Cardiol. 2021;6:1–8.
4. Tona F, Serra R, Di Ascenzo L, Osto E, Scarda A, Fabris R, et al. Systemic inflammation is related to coronary microvascular dysfunction in obese patients without obstructive coronary disease. Nutr Metab Cardiovasc Dis. 2014;24:447–53.
5. Mittal SR. Diagnosis of coronary microvascular dysfunction - Present status. Indian Heart J. 2015;67:552–60.

AUTHOR CONTRIBUTIONS

Both authors (SD, PTD) conceived of the work that led to the submission. SD led the paper drafting, both authors collaboratively revised the paper. Both authors approved the final paper and agree to be accountable for all aspects of the work. SD had final responsibility for the decision to submit for publication.

COMPETING INTERESTS

The authors declare no competing interests.

ADDITIONAL INFORMATION

Correspondence and requests for materials should be addressed to Serkan Duyuler.

Reprints and permission information is available at http://www.nature.com/reprints

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Received: 13 February 2022 Revised: 10 April 2022 Accepted: 12 April 2022
Published online: 25 April 2022