The significance of epicardial adipose tissue in individuals with diabetes mellitus type 2 without clinical heart failure

Ana Đuzel Čokljat[1]*, Petra Grubić Rotkvić[2], Damir Čokljat[1], Zdravko Babić[3]

[1]General Hospital Dubrovnik, Dubrovnik, Croatia
[2]University Hospital Centre Zagreb, Zagreb, Croatia
[3]University Hospital Centre "Sestre milosrdnice" Zagreb, Croatia

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***ADDRESS FOR CORRESPONDENCE:** Ana Đuzel Čokljat, Opća bolnica Dubrovnik, Ulica Roka Mišetića 2, HR-20000 Dubrovnik, Croatia. / Phone: +385-99-2995-290 / E-mail: ana29.djuzel@gmail.com

ORCID: Ana Đuzel Čokljat, https://orcid.org/0000-0003-0095-2987 • Petra Grubić Rotkvić, https://orcid.org/0000-0002-2587-1932 Damir Čokljat, https://orcid.org/0009-0008-3221-7534 • Zdravko Babić, https://orcid.org/0000-0002-7060-8375

Aim. To explore the effect of epicardial adipose tissue (EAT) on myocardial function in stage A and stage B (pre-heart failure) patients with diabetes mellitus type 2 (T2DM).

Materials and Methods. We have conducted a review of a relevant literature comprising an area of interest as stated.

Results. Patients with T2DM have a larger amount of EAT compared to non-diabetic individuals.[1] Since all of them, if asymptomatic, are at risk of developing HF (stage A), and if additionally proven structural/functional cardiac abnormalities and/or elevated natriuretic peptides, then diagnosed with pre-HF (stage B),[2] we have searched for a potential mechanism of EAT impact. Firstly, EAT might infiltrate into adjacent myocardium, which disturbs myocardial architecture, stimulates myocardial thickening, and promotes diastolic dysfunction.[3] Secondly, a mechanical effect of thickened EAT resembles that of a constrictive pericarditis: if a pericardial dilatation is non-congruent with EAT expansion (i.e. pericardial restraint), adjacent myocardium is not able to further dilate in case of demanding hemodynamic state because of limited pericardial pliability.[4] Since cardiovascular outcome trials have shown that glucagon-like peptide 1 receptor agonists (GLP1R) agonists reduce the incidence of major cardiovascular events, with effect sizes beyond regulation of glycemia, and EAT expresses GLP1R, it is possible that delta EAT plays an important role in cardiovascular improvement.[5]

Conclusion: EAT could serve as a novel tool for risk estimation of progression from asymptomatic to clinical (symptomatic) HF in individuals with T2DM.[1-5] In addition, EAT may serve as a target for existing and novel therapies.

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LITERATURE

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