Received: 25 July 2024 | Accepted: 20 August 2024

DOI: 10.1111/jch.14899

LETTER TO THE EDITOR

WILEY

Multiple factors are related to the development of exaggerated blood pressure response to exercise

To the Editor,

I read with great interest the article titled "Usability of myocardial work parameters to demonstrate subclinical myocardial involvement in normotensive individuals with exaggerated hypertensive response in treadmill exercise testing" by Efe et al. In their detailed statistical analysis, Efe et al. reported that myocardial work parameters such as global myocardial work index (GWI) might be used to identify early signs of myocardial involvement in normotensive patients with an exaggerated blood pressure response to exercise (EBPRE).[1] Accordingly, the increase in GWI value predicts the presence of EBPRE. Myocardial work parameters are related to myocardial deformation and distortion independent from pressure and volume load which is different from previous myocardial performance parameters such as global longitudinal strain (GLS) and left ventricular ejection fraction (LVEF).[2]

In recent clinical studies, EBPRE has been found to be associated with subclinical target organ damage in normotensive individuals.[3,4] In addition, it has been suggested that EBPRE may be a predictor of future overt hypertension.[5] It is suggested that the most probable mechanism that plays a role in the development of EBPRE is the lack of enough decrement in peripheral vascular resistance in response to increased cardiac output with exercise. This inadequate decrease in peripheral vascular resistance may be related to endothelial dysfunction and subclinical vascular inflammation.[4,6] Closely related to this inadequate response in peripheral vascular resistance, various metabolic parameters (such as central adiposity, fasting blood sugar, triglyceride, total cholesterol, and impaired glucose tolerance) were also found to be associated with the development of EBPRE.[4,6,7] When deciding whether the possible role of load-independent myocardial work parameters predicts the presence of EBPRE, metabolic variables that may accompany the pathophysiology should be taken into consideration and clinicians may also interact with the manageable metabolic variables to manage the personal risk stratification.

Ali Çoner MD
Can Ramazan Öncel MD
Cemal Köseoğlu MD
Göksel Dağaşan MD

Department of Cardiology, Alanya Alaaddin Keykubat University Faculty of Medicine, Alanya, Turkey

Correspondence
Ali Çoner, MD, Department of Cardiology, Alanya Alaaddin Keykubat University Faculty of Medicine, Alanya, Turkey.
Email: conerali@hotmail.com

ORCID

Ali Çoner MD https://orcid.org/0000-0002-5711-8873

REFERENCES

1. Cagan Efe S, Buğrahan Cicek M, Unkun T, et al. Usability of myocardial work parameters to demonstrate subclinical myocardial involvement in normotensive individuals with exaggerated hypertensive response in treadmill exercise testing. *J Clin Hypertens*. 2024;26(6):687-695.
2. Ilardi F, D'Andrea A, D'Ascenzi F, et al. On behalf of the working group of echocardiography of the Italian Society of Cardiology Sic. Myocardial work by echocardiography: principles and applications in clinical practice. *J Clin Med*. 2021;10(19):4521.
3. Costa EC, Browne RAV, Câmara M, et al. Immediate post-exercise blood pressure and arterial stiffness in hypertensive and normotensive older females. *J Clin Hypertens*. 2022;24(6):704-712.
4. Çoner A, Gençtoy G, Akinci S, Altin C, Müderrisoğlu H. Assessment of vascular inflammation and subclinical nephropathy in exaggerated blood pressure response to exercise test. *Blood Press Monit*. 2019;24(3):114-119.
5. Tzemos N, Lim PO, Mackenzie IS, MacDonald TM. Exaggerated exercise blood pressure response and future cardiovascular disease. *J Clin Hypertens*. 2015;17(11):837-844.
6. Tokushige A, Ohishi M. Pathophysiology of the exaggerated blood pressure response to exercise. *Hypertens Res*. 2022;45(12):2028-2029.
7. Hamer M, Steptoe A. Vascular inflammation and blood pressure response to acute exercise. *Eur J Appl Physiol*. 2012;112(6):2375-2379.