International Journal of Cardiology 293 (2019) 192

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Letter to the Editor

Reply to the letter to the editor: "The association of B-type natriuretic peptide with left ventricular hypertrophy"

Mai Tone Lønnebakken [a,b,*], Ingeborg Eskerud [a]

[a] Department of Clinical Science, University of Bergen, Bergen, Norway
[b] Department of Heart Disease, Haukeland University Hospital, Bergen, Norway

ARTICLE INFO

Article history:
Received 22 June 2019
Accepted 3 July 2019

Keywords:
Left ventricular hypertrophy
B-type natriuretic peptide
Myocardial ischemia
Non-obstructive coronary artery disease

We thank Dr. Liu for interesting comments referring to our article on the association of left ventricular hypertrophy (LVH) and myocardial ischemia in patients with stable angina and non-obstructive coronary artery disease (CAD) [1]. We agree that myocardial ischemia in non-obstructive CAD has a multifactorial etiology. Even though we did not measure baseline B-type natriuretic peptide (BNP) levels in our study, elevated BNP levels could be expected in patients with non-obstructive CAD, particularly in concomitant LVH and myocardial ischemia. In general, BNP is secreted in response to myocardial stretch and is an unspecific biomarker, considered to be cardioprotective and has been suggested as an inhibitor of myocardial hypertrophy and ischemia induced reperfusion injury [2]. Consequently, BNP levels do not necessarily represent a confounder that needs to be adjusted for in the logistic regression model [3]. First, a causal relationship between LVH and BNP cannot be excluded. Secondly, BNP may prevent myocardial ischemia instead of being a risk factor. In our opinion, adjusting for BNP levels may introduce an overadjustment bias.

We acknowledge that subclassification of LVH into different geometric patterns have the potential to improve risk stratification [4]. However, this is a small study including 132 patients, and the prevalence of LVH in the total study population was 17%. By subclassification of LVH into concentric and eccentric geometry based on relative wall thickness, concentric LVH was significantly more common in patients with ischemia [1]. Unfortunately, our study does not have the statistical power to allow further subclassification into dilated and non-dilated LVH.

Funding/support

Financial support was obtained from the MedViz Consortium, a collaboration between the University of Bergen, Haukeland University Hospital and Christian Michelsen Research, all Bergen, Norway, and the Western Norwegian Regional Health Authorities.

Declaration of Competing Interest

The authors report no relationships that could be construed as a conflict of interest.

References

[1] I. Eskerud, E. Gerdts, T.H. Larsen, M.T. Lonnebakken, Left ventricular hypertrophy contributes to myocardial ischemia in non-obstructive coronary artery disease (the MicroCAD study), Int. J. Cardiol. 286 (2019) 1–6.
[2] T. Nishikimi, N. Maeda, H. Matsuoka, The role of natriuretic peptides in cardioprotection, Cardiovasc. Res. 69 (2) (2006) 318–328.
[3] K.J. Jager, C. Zoccali, A. Macleod, F.W. Dekker, Confounding: what it is and how to deal with it, Kidney Int. 73 (3) (2008) 256–260.
[4] C.N. Bang, E. Gerdts, G.P. Aurigemma, K. Boman, G. de Simone, B. Dahlof, et al., Four-group classification of left ventricular hypertrophy based on ventricular concentricity and dilatation identifies a low-risk subset of eccentric hypertrophy in hypertensive patients, Circulation Cardiovascular imaging 7 (3) (2014) 422–429.

* Corresponding author at: Department of Clinical Science, University of Bergen, Bergen, Norway.
E-mail address: mai.lonnebakken@uib.no (M.T. Lønnebakken).

https://doi.org/10.1016/j.ijcard.2019.07.011