Atrial fibrillation in elderly patients with severe aortic stenosis. Authors' reply

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DOI: 10.33963/v.phj.103458

Received:
October 29, 2024

Accepted:
October 29, 2024

Early publication date:
November 6, 2024

We appreciate the comments in response to our study [1] and concur with Engin and colleagues that there are several potential causes of parathyroid hormone (PTH) increase in our study population. The role of hyperparathyroidism in cardiovascular disease has already been postulated [2]. By design, our study did not aim to search for the pathophysiological background of the higher PTH concentration since we evaluated the interplay between PTH and atrial fibrillation occurrence in patients with severe calcific aortic stenosis. However, we agree that vitamin D deficiency may be associated with this phenomenon. Studies concerning the detrimental effects of vitamin D deficiency have shown its association with coronary artery atherosclerosis [3] and aortic stenosis [4]. Calcific changes in valve leaflets share common pathophysiological features with atherosclerosis. The process of valvular stenosis is complex, and numerous factors contribute to its development, including fibrosis, inflammation, oxidative stress, neovascularization, and calcification. Aging, arterial hypertension, diabetes, metabolic factors, and mineral imbalance may influence the disease progression. The relationship between vitamin D deficiency and PTH concentration is already known and undeniable, and no additional verification was required in our analysis.

Our study population presented with paroxysmal and chronic atrial fibrillation, with a duration of over three months to years. Importantly, the increased PTH concentration was demonstrated as a predictor of atrial fibrillation, independently of paroxysmal or chronic manifestation.

The relationship between PTH increase and pharmacotherapy is highly possible because elderly patients are burdened with severe longstanding co-morbidity and require complex medications. Indeed, warfarin use is associated with reduced bone density and increased risk of osteoporosis and osteoporotic fractures. Different mechanisms of this phenomenon have been proposed, including direct inhibition of gamma-carboxylation and other bone matrix proteins, and dietary changes in food rich in vitamin K. This is an important issue because patients are instructed to modify their intake of green vegetables rich in vitamin K, which in turn are an important source of folic acid. Its lower dietary intake may lead to hyperhomocysteinemia and an increased risk of osteoporotic fracture. However, we would like to underline that currently, the Polish population of patients with atrial fibrillation is sufficiently treated with non-vitamin K antagonist oral anticoagulants [5], for which interference with bone health is less probable according to recent studies [6]. Dietary changes are less restrictive with the use of novel anticoagulant therapy.

Finally, we are aware that multiple cardiovascular and non-cardiovascular diseases, as well as metabolic and biochemical imbalances, may promote atrial fibrillation development. Hypomagnesemia was mentioned in our paper as a possible factor that may endorse a PTH increase. Its role in triggering atrial fibrillation has been already established.

In the summary, Engin and colleagues highlighted the complexity of PTH concentration increases and atrial fibrillation occurrence in our study. In so doing, they placed our investigation in the context of the broad consideration on the cardiovascular pathophysiology.

Article information

Conflict of interest: None declared.

Funding: None.

REFERENCES

1. Olasińska-Wiśniewska A, Urbanowicz T, Kübler P, et al. Increased parathyroid hormone concentration as a biomarker of atrial fibrillation in severe aortic stenosis. Pol Heart J. 2024; 82(11): 1076–1082, doi: 10.33963/v.phj.102411, indexed in Pubmed: 39240914.
2. Trevisan C, Rossi A, Curreri C. Increased parathyroid hormone concentration as a biomarker of atrial fibrillation in severe aortic stenosis: Editorial comment. Pol Heart J. 2024; 82(11): 1055–1056, doi: 10.33963/v.phj.103295, indexed in Pubmed: 39743947.
3. Verdoia M, Gioscia R, Nardin M, et al. Low levels of vitamin D and coronary artery disease: Is it time for therapy? Kardiol Pol. 2022; 80(4): 409–416, doi: 10.33963/KP.a2022.0079, indexed in Pubmed: 35546270.
4. Kim IlY, Ye BM, Kim MJ, et al. 1,25-dihydroxyvitamin D deficiency is independently associated with cardiac valve calcification in patients with chronic kidney disease. Sci Rep. 2022; 12(1): 915, doi: 10.1038/s41598-022-04981-x, indexed in Pubmed: 35042976.
5. Maciorowska M, Uziębło-Życzkowska B, Gorczyca-Głowacka I, et al. Oral anticoagulation therapy in atrial fibrillation patients at high risk of bleeding: Clinical characteristics and treatment strategies based on data from the Polish multicenter register of atrial fibrillation (POL-AF). Pol Heart J. 2024; 82(1): 37–45, doi: 10.33963/v.kp.98356, indexed in Pubmed: 38230462.
6. Liu Y, Xie X, Bi S, et al. Risk of osteoporosis in patients treated with direct oral anticoagulants vs. warfarin: an analysis of observational studies. Front Endocrinol (Lausanne). 2023; 14: 1212570, doi: 10.3389/fendo.2023.1212570, indexed in Pubmed: 37842293.