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Letter and Reply

Pulmonary hypertension in chronic kidney disease: what could change the fate?

To the Editor:

Recently, the interest is increasing in studying the association between pulmonary hypertension (PH) and chronic kidney disease (CKD). PH is rare in general but may be present in 30–40% (on the basis of echocardiographic studies) of the patients with end-stage renal disease, being associated with high morbidity and mortality [1]. The reason why PH is frequently associated with CKD or end-stage renal disease is still unclear and presumed to be multifactorial [2]. In the results from the Chronic Renal Insufficiency Cohort study group, the likelihood of prevalent PH was increased with older age, presence of anemia, left ventricular hypertrophy, and lower levels of left ventricular ejection fraction [1].

In the past issue of this *Journal*, Kim et al [3] investigated associations between PH, peripheral vascular calcifications (VCs), and major cardiovascular events in dialysis patients. In this retrospective study, echocardiography and plain radiographs were used to estimate pulmonary artery systolic pressure and simple VC score of the hands and pelvis, respectively. The authors concluded that severe VCs were independently associated with PH and that PH was predictive of major cardiovascular events. Accordingly, they suggested that careful attention should be paid to the presence of VCs and PH in dialysis patients because of the occurrence of major cardiovascular events.

Then, how can we block the bad scenario beginning from VCs? The authors stated that to prevent VC, early initiation of hyperphosphatemia management or use of non–calcium-based phosphate binders is necessary in CKD patients. I doubt if either early control of hyperphosphatemia or use of non–calcium-based phosphate binders has clear evidence to support their application in clinical practice. Actually, VCs are most often detected incidentally on imaging studies for other purposes. Most nephrologists do not screen or attempt to quantify VC in all CKD patients because no specific therapy is available beyond careful attention to calcium and phosphate balance.

Finally, the timing of echocardiography may be important in dialysis patients. The authors were also aware of the possibility of inconstancy in timing of echocardiography according to hemodialysis (HD) session, but they found that the prevalence of PH was not statistically affected by the timing of echocardiography (before or after HD, data not shown). However, reproducibility of their data may need to be tested. Previous studies have reported that pulmonary arterial pressure values tend to regress after each HD session [4,5]. Appropriate dialytic therapy would be the most feasible tool to control PH because volume status may affect diastolic function of the heart.

Conflicts of interest

The author has no conflicts of interest to declare.

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In reply:

We appreciate your interest in our recent article entitled "Relationship between pulmonary hypertension, peripheral vascular calcification, and major cardiovascular events in dialysis patients" [1]. Chronic kidney disease with a milieu of accelerated extraosseous calcification may result in vascular calcification (VC) not only in peripheral artery but also in the coronary or pulmonary arteries [2,3]. Theoretically, VC could involve pulmonary vascular beds and subsequently increase the risk of pulmonary hypertension. Our group previously demonstrated that low intact PTH (parathyroid hormone) and diabetes were independent risk factors for predicting VC and also that simple VC score ≥ 1 was a significant variable for predicting severe coronary artery calcification in patients with peritoneal dialysis [2].

As you pointed out in the letter, VC is still most often detected incidentally on imaging studies obtained for other purposes. However, we need to pay attention to central VCs including pulmonary vascular beds and coronary arteries that can substantially affect cardiovascular outcomes. Relatively easy and inexpensive procedures can detect them during our routine clinical practices in chronic kidney disease patients. In addition, although definitive treatments or preventive measures are not currently possible, we need to carefully monitor these patients with VCs for the presence of ischemic heart disease or pulmonary hypertension. Specific treatment strategies against VC may be realized in the near future.

With regard to timing of echocardiography in measuring pulmonary artery pressure, we completely agree with your opinion. The inconsistency in timing of echocardiography in our study might have affected the results, and we recognized it as a limitation of our study. To verify this, future larger prospective studies will be required.

Conflicts of interest

The author has no conflicts of interest to declare.

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