

In conclusion, despite advances in diagnosis and treatment, infective endocarditis is still associated with mortality rates. These interrelations need to be clarified with further studies in Turkey.

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Elevated mean pulmonary artery pressure in patients with mild-to-moderate mitral stenosis: a useful predictor of worsening renal functions?

To the Editor,

We read the article entitled "Elevated mean pulmonary artery pressure in patients with mild-to-moderate mitral stenosis: a useful predictor of worsening renal functions?" by Zorkun et al. (1) published in August issue of *The Anatolian Journal of Cardiology* 2013; 13: 457-64 with interest. They concluded that elevated mean pulmonary artery pressure at the time of initial evaluation, in patients with the mild -to-moderate mitral stenosis, might help to predict worsening renal function. Frankly, we appreciate the authors for their informative and original study. However, we have some criticism about this study.

As mentioned by authors, Heywood et al. (2) found no association between renal dysfunction and left ventricular systolic dysfunction in patients with acute heart failure. They also concluded that renal failure might be more closely associated with diastolic dysfunction. Because it was preserved, Zorkun et al.(1) overlooked the right ventricular systolic function but there were no significant differences in diastolic function between the two groups.

In a study, Bilen et al. (3) demonstrated that patients with mitral stenosis had lower left ventricle functions using 2D strain imaging, and this was independent of the hemodynamic severity of mitral stenosis.

The authors cite experimental studies of renal venous congestion to consolidate for their conclusion, but the directionality of their results appears to be discordant with the cited models. Furthermore, relief of

venous congestion leads to a prompt and reproducible improvement in renal function. The authors did not explain how they measured or established venous congestion. In the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial, Nohria et al. (4) found a lack of association between worsening renal function and baseline, or changes in hemodynamic parameters including right atrial pressure.

Central venous pressure (CVP) is an important and easy to use cardiac parameter. There is good evidence from experimental data that, apart from decreased renal blood flow, an increase in CVP in the context of significant right ventricular dysfunction or tricuspid regurgitation may lead to decreased renal perfusion by elevation of renal venous pressure. In the isolated perfused rat kidney, an increase in CVP has been shown to be followed by a significant reduction in glomerular filtration rate, sodium excretion, and fractional excretion of sodium, which resolved after restoration of normal CVP levels (5).

In the Zorkun et al.'s (1) study, there are insufficient patients with comorbidities that contribute to intrinsic renal disease in the patients with worsening renal function on follow up group. So, we think that this is a selection bias.

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DOI:10.5152/akd.2014.5085



Author's Reply

To the Editor,

We thank to authors for their valuable comments, appreciate their interest to our study titled "Elevated mean pulmonary artery pressure in patients with mild-to moderate mitral stenosis: a useful predictor of worsening renal functions?" published in August issue of *The Anatolian Journal of Cardiology* 2013; 13: 457-64 (1).

In their letter to the editor, the authors highlighted several points to clarify. We would like to reply to all issues raised in their letter.

1. We have indicated all current and possible limitations of our study with clear and definite expressions in our manuscript as; "Although a lack of invasive measurements was the major limitation of our study, we did not consider invasive assessment, since it might cause ethical problems if performed in cases of mild-to-moderate MS. Central venous pressure and inferior vena cava diameters, which remain other important study limitations, were also not recorded in our study. Because right ventricular systolic function was preserved, this issue was overlooked. Male gender was also found to be a predictor of WRF (worsening renal function); however, it is better not to generalize about this, since there were relatively few male patients in the cohort, which is another limitation of this study. The number of patients enrolled in this study was another limitation; therefore, our findings should not be generalized. These findings should be supported by further studies conducted with a sufficient number of patients".
 2. We were in hope that, our published data would support future research aimed at elucidating the pathophysiology leading to worsening renal function in mitral stenosis, therefore a better understanding of the mechanisms of the cardiorenal interaction.
 3. On the contrary of the authors' expression, there was no any attempt to consolidate our results in related manuscript. The reason for citing an experimental study was the lack of any clinical study on this topic. As our study results represent very first findings in this subject, the mentioned citation was aimed to explain the problem and targeted to point possible underlying mechanism.
 4. As we did not evaluate "venous congestion" in our study, we kindly suggest reading related references in our manuscript to get more in detail.
 5. We have published to share a small group of patients' results. Therefore, a statement as "a selection bias" is unmeritorious. Using such expression requires a previous experience or performing a larger study.
- In conclusion; the manuscript itself may give all related answers of possible questions. Mankind always sets itself only such tasks as it can solve; since, looking at the matter more closely.

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Aggregation of lipoprotein(a) to apolipoprotein A-I and coronary artery problem

To the Editor,

Sir, the recent report on aggregation of lipoprotein(a) to apolipoprotein A-I and coronary risk factor is very interesting published in September issue of The Anatolian Journal of Cardiology 2013; 13: 543-51 (1). Onat et al. (1) concluded that "Lp (a) may aggregate in a pro-inflammatory milieu to apoA-I, rendering apoA-I atherogenic." The mechanism underlying the atherogenic is an issue for discussion. Aggregation might lead to a bigger complex molecule but this cannot be sufficient for explanation for triggering the atherogenic. There should be some vascular insult that will be the starting point of atherogenic. A possible mechanism to be mentioned is the energy fluctuation during formation of intravascular lipoprotein complex. In fact, many previous reports confirming the formation of complex can result in energy insult to the vessel and lead to vascular disorder [the good example is the formation of hemoglobin A1C (2)].

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Available Online Date: 19.03.2014

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DOI:10.5152/akd.2014.5365



Author's Reply

To the Editor,

We appreciated to learn the comment by Wiwanitkit to our prospective population-based study published in September issue of The Anatolian Journal of Cardiology 2013; 13: 543-51 (1) indicating that aggregation of lipoprotein (Lp) (a) in a proinflammatory setting to apolipoprotein (apo) A-I, the major protein constituent of HDL particles, may lead to impairment of the antioxidant and atheroprotective functions of apoA-I, which may ultimately become diabetogenic or atherogenic, a process representing HDL dysfunction and autoimmune activation. The author points out that the aggregation process per se may be inadequate to induce atherogenicity which may require the mediation of energy fluctuation in the course of intra-