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In Response to Çağrı Yayla, et al.

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We thank the authors[1] for their interest in our article[2] and their valuable comments. These authors[1] could not understand if the patients had a decreased left ventricular ejection fraction (LVEF) after primary percutaneous coronary intervention (pPCI) and they asked if there was a difference in the success rate of pPCI between the LVEF <50% and LVEF ≥50% groups. They also wondered about the use of spironolactone.

Different causes of cardiac remodeling share common molecular, biochemical, and mechanical pathways. The process begins with myocyte damage and then the extracellular matrix, fibroblasts, and inflammatory cells are also involved and then the process is shaped by the effect of hemodynamic load, neurohormonal activation, and other factors.[3] Some previous studies reported that glypican-6 (GPC6) regulates cardiomyocyte growth via extracellular-signal-regulated kinase signaling.[4]

In our study,[2] we evaluated the predictive value of GPC6 for cardiac remodeling after myocardial infarction. Patients with a previous history of heart failure with reduced ejection fraction were excluded. Patients without a history of heart failure with reduced ejection fraction (low or normal ejection fraction before pPCI) were included in the study and compared with their sixth month follow-up. Coronary revascularization of the patients was achieved. Those with unsuccessful revascularization were not included in the study.

We thought of spironolactone like you. Therefore, we evaluated the effect of spironolactone in the regression analysis, but we found that it was not an independent predictor. For this reason, we did not think that the results we obtained in our article were related to spironolactone.

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