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Author`s Reply

To the Editor,

We would like to thank the authors of the letter for their interest and criticism about our study entitled "Association of mitral annular calcification with endothelial dysfunction, carotid intima-media thickness, and serum fetuin-A: An observational study," published in the December issue of Anatolian J Cardiol 2013; 13: 752-8 (1).

We re-examined our results retrospectively. During the biochemical analysis, the diagnostic range of our spectrophotometry data was not available. For this reason, our blood samples were diluted in a higher percentage.

The differences of the results between our study and some other articles may possibly be the consequence of the dilution ratios of our spectrophotometry values.

We indicated the normal value of fetuin-A in our article. However, the aim of our study was to compare fetuin-A levels between the two groups.

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Cholesterol; is accused for the atherosclerosis proximal to myocardial bridging?

To the Editor,

We excitedly read the article about the close relationship of myocardial bridging (MB) and atherosclerosis at its proximal segment, which was suggested by Hong et al. (1), published in Anatolian J Cardiol 2014; 14: 40-7. They had suggested that the presence of MB on a coronary artery was one of the clinically independent risk factors for atherosclerosis, such as age, diabetes, and dyslipidemia. Duygu et al. (2) previously stated that MB initiated and facilitated the development and progression of atherosclerosis in the Anatolian Journal of Cardiology in 2007 (2). Systolic compression, which is a diagnostic marker for the presence of MB on the coronary angiogram, is generally supposed to account for the hemodynamic and endothelial changes that promote the atherosclerosis. In fact, the hemodynamic abnormalities are induced by MB during the diastolic period, in which the coronary artery flow and the myocardial perfusion are at their maximum (3). Since the MB behaves as an anatomic obstacle that surrounds and limits the coronary artery from its outside, a diastolic flow gradient develops at the proximal part of the bridged arterial segment. So, the diastolic gradient exerts a "seeding effect" that urges the cellular and lipid component of blood to pass into the sub-endothelial layers of the coronary artery. Phagocytic cells, cholesterol, and lipoprotein particles are the main components of an atherosclerotic plaque and also determine the vulnerability of the plaque (4). Moreover, the diastolic gradient at the proximal segment induces an increased shear stress and endothelial dysfunction, which are represented by reduced nitric oxide synthesis, antithrombotic functions, and vasodilation. These are the initial and earliest abnormalities observed in the development of atherosclerotic plaque (AP).

In the preliminary results of our study, we observed that the serum levels of total cholesterol, LDL- and VLDL-cholesterol, and triglyceride were significantly higher in patients with MB and AP (n=7) and AP (n=9) compared to patients with only MB (n=18) (unpublished data). Patients with MB who had a normal lipid profile were free from atherosclerosis, while all patients with MB and coexisting hypercholesterolemia had atherosclerotic plaque in the proximal arterial segment of the MB. It reminded us that the ancient guilty; cholesterol; was again responsible for the atherosclerosis at the proximal coronary segment of the MB. Coexistence of MB and AP in the presence of hyperlipidemia indicates that cholesterol may be a prerequisite for the development of an AP proximal to the MB. MB was surprisingly detected with a high prevalence and was found to be highly associated with atherosclerosis and sudden cardiac deaths in young and young adult subjects (5). Nevertheless, a debate about the criteria of statin therapy, target cholesterol levels, and whether it is an equivalent of atherosclerotic coronary artery disease, as well as diabetes, will develop in the management of those patients with MB. We suggest that MSCT coronary angiography, which has a great capability in the detection of MB and AP, even at the initial stages, may guide the indication of statin therapy by documenting the presence of AP in association with MB.

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Author`s Reply

The authors of this mentioned article did not send any reply for this Letter to the Editor, despite our insistent requests.

Homocysteine and masked hypertension

To the Editor,

The recent report "Homocysteine and masked hypertension" in Anatolian J Cardiol 2014; 14: 357-62 is very interesting (1). They noted that "in the individuals with no obvious health problems but with MHT, homocysteine levels may not have any significant effect upon high blood pressure levels (1)." In fact, several factors are accepted as contributing factors for "masked hypertension," including "younger age, smoking, alcohol use, contraceptive use in women, sedentary habits, and central obesity (2)". The negative finding on the role of homocysteine level in the present report should be discussed. In fact, homocysteine has been accepted as a good biomarker for identifying risk of cardiovascular disease for a long time (3). However, in addition to hypertension, other vascular pathologies are related to the change of blood homocysteine level. This fact has to be considered in the interpretation of the homocysteine level results. Another important consideration in the determination of homocysteine levels is the false positivity (4). Pre-analytical errors in specimen collection and preparation can significantly result in elevated blood homocysteine levels (4).

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Author`s Reply

The authors of this article did not send any reply to this Letter to Editor, despite our insistent requests.

Peripartum cardiomyopathy and triplet pregnancy

To the Editor,

We read with interest the article recently published by Günaydın et al. (1), entitled "Peripartum cardiomyopathy associated with triplet pregnancy," in Anatolian J Cardiol 2014; 14: 661-2. However, we have some concerns about the article. First, although the authors claimed the current patient to be the first peripartum cardiomyopathy (PPCM) patient associated with triplet pregnancy in the literature, this may not be true. Rajab et al. (2) described a 26-year-old Bahraini primigravida, at 38 weeks of gestation for elective caesarean section because of pregnancy-induced hypertension and triplets. In this article, at the 39th week, she had a cesarean section under general anesthesia but developed PPCM in the early postoperative period. Chapa et al. (3) reported follow-up data of 32 PPCM patients in 2005. They reported 4 women with multifetal gestations; 3 twins and 1 triplet. Golan et al. (4) reported a retrospective review and an analysis of 182 patients with PPCM. Twin or triplet pregnancies were reported in 15% of all patients in this study.

Our second concern is about the acute treatment of PPCM. The management of patients with PPCM is similar to that of other forms of non-ischemic dilated cardiomyopathy but must be individualized based on the patient's clinical presentation (5). In addition to the standard therapeutic options for heart failure, specific targeted agents have been advocated for the treatment of PPCM. In recent years, it has been shown that addition of bromocriptine to standard heart failure therapy in women with PPCM results in significantly greater improvements in