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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

functional capacity and left ventricle function than with standard therapy alone. We have added bromocriptine in the acute phase of PPCM to standard heart failure therapy in our clinical practice since 2010.

Our last concern is about the duration of therapy. In the current study, the patient had normal left ventricle ejection fraction in the 6th month, but the authors did not report whether they continued the heart failure therapy or not after 6 months. Currently, there is no clear consensus on the appropriate duration of heart failure drug therapy. It is also unknown when to discontinue heart failure medications in recovered PPCM patients or whether there is any deterioration in left ventricular function after an initial recovery in these patients. Recently, we published the results of 42 prospectively followed PPCM patients (5). Four patients showed delayed deterioration (12, 24, 26, and 34 months after diagnosis) during the study period. The findings of late deterioration indicate the need for close follow-up with periodic determination of cardiac function in women in whom medications are discontinued after complete recovery. Due to the probability of either delayed recovery or deterioration of left ventricular function in PPCM, long-term follow-up may be needed not only in non-recovered patients but also in patients with complete recovery. After clinical and echocardiographic evidence of full recovery, it may be acceptable to gradually taper the drug doses over a period of 12 to 24 months; however, we suggest that ACE inhibitors and beta-blockers be continued for at least 2 years after complete recovery.

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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 'Letter to the Editor,' published November 2014 issue in The Anatolian Journal of Cardiology (1) for entitled "Peripartum cardiomyopathy associated with triplet pregnancy." Triplet pregnancy and peripartum cardiomyopathy (PPCM) are uncommon separately in clinical practice; thus, their coexistence is even less common. We performed a search in Pubmed/Medline, Scopus, and Türkiye Atıf Dizini using combinations of the following keywords: "triplet pregnancy," "peripartum cardiomyopathy," and "multiple gestations." We could not find any topic about them. One case (2) that was mentioned by the authors was published in Bahrain Medical Bulletin. This journal is not indexed in the databases mentioned above. Therefore, we were unable to reach this information. We thank the authors for their notice and reminder. There may be some cases that were missed due to undetected mother deaths or undiagnosed patients on World-wide. Today, in vitro fertilization is gradually becoming more common. It shows that we can encounter such situations more frequently in the future due to multiple gestations. This association has been presented for the first time in this topic as a case report. In this respect, our publication is valuable. The authors stated that their study (3) is the largest series of patients with PPCM in Turkey. In that study, while there were twin pregnancies, triplet pregnancies were not detected.

There are no standard, universally accepted guidelines for the management of PPCM. The treatment of patients with PPCM is similar to other forms of non-ischemic dilated cardiomyopathy. However, it must be individualized based on the patient's clinical presentation (3). Prolactin may have adverse effects on the heart muscle by restricting its blood supply and causing cell death (4). Although early studies suggest that bromocriptine may be beneficial in the treatment of PPCM, large double-blind randomized trials are essential to confirm the results of smaller studies (5). More research is needed to determine its safety and efficacy. Also, there is no information about bromocriptine in the 2013 ACCF/AHA Guidelines for the Management of Heart Failure and 2012 ESC Guidelines for the Diagnosis and Treatment of Acute and Chronic Heart Failure. If larger studies substantiate and it takes part in universally accepted guidelines with strong recommendations, bromocriptine can be administered safely. Our patient improved gradually with conventional treatment of heart failure. Therefore, no additional treatment was considered, like bromocriptine.

Today, there is no clear consensus on the appropriate duration of peripartum cardiomyopathy drug therapy. Biteker et al. (3) published the results of 42 prospectively followed PPCM patients. Four patients showed delayed deterioration during the study period. The findings of late deterioration indicate the need for close follow-up, with periodic determination of cardiac function in women in whom medications are discontinued after complete recovery. The patient is still in our follow-up. It was about 1.5 years after diagnosis, and deterioration was not observed. Metoprolol, spironolactone, furosemide, and ramipril, all with oral use, were administered in the daily treatment of the patient in the first 6 months, as mentioned in our letter. Spironolactone and furosemide were stopped, and only 50 mg of metoprolol and 5 mg of ramipril were given to the patient after 6 months up to 1 year. We stopped all medications at 12 months after the diagnosis. Because we thought this cardiomyopathy was associated with pregnancy, the effects of pregnancy were loss completely, and the patient had a complete recovery. The authors suggest that ACE inhibitors and beta-blockers should be continued for at least 2 years after complete recovery. We take into consideration the authors' recommendations about the duration of therapy after complete recovery.

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Heart rate recovery and methodological issues

To the Editor,

We read with great interest the article, entitled "Heart rate recovery may predict the presence of coronary artery disease" by Akyüz et al. (1) published in Anatolian J Cardiol 2014; 14: 351-6.

They observed in a retrospective analysis that abnormal heart rate recovery at 1 min (HRR1) was associated with the presence of angiographically proven coronary artery disease. This study strengthens previous research that the heart rate information gleaned from a standard exercise test can be used to supplement prognostic and diagnostic data. There are some methodological issues that need to be clarified in order to understand how these data were obtained. The authors' statement that "post-exercise HRR was measured in the sitting position during the cool-down period after the cessation of peak exercise" might lead to misunderstandings and is inappropriate with regard to terminology. Exercise testing can be terminated (cessation of exercise) abruptly with the patient in the standing or sitting positon (no 'cool-down' period), or the patient keeps walking in a predetermined speed and incline (cool-down period), which can be a 2-minute cool-down at 1.5 mph on a 2.5° grade or a 1-minute cool-down at 1 mph at 0% incline (2, 3). In protocols using cool-down, heart rate recovery at 1 minute is calculated by taking the difference between the heart rate at peak exercise and heart rate 1 minute later, which is 1 minute after the beginning of the cool-down period (2). Similarly, in exercise tests that stop abruptly, heart rate recovery at 1 minute is calculated by taking the difference between the heart rate at peak exercise and heart rate 1 minute later, at which time the patient is at complete rest in the supine or sitting positon. Abnormal HRR1 is usually defined as heart rate that declines ≤12 beats/min in the first minute after exercise for protocols that use a post-exercise cool-down or ≤18 beats/min in the first minute postexercise for protocols that stop exercise abruptly (2, 4). Since the authors defined abnormal HRR1 as ≤21 beats, we assume that there was no cool-down period in their study. Although the authors mentioned heart rate reserve in the results section and tables, they did not define it in the methods. It is not clear whether heart rate reserve is in beats per minute or in percentages. Heart rate reserve in beats per minute is calculated as [(220-age in years) - resting heart rate in beats per min], while heart rate reserve in percentages is calculated as (peak heart rate- resting heart rate in beats per min)/[(220-age in years) - resting heart rate in beats per min] multiplied by 100 (2). Heart rate reserve in percentages is also an indicator of chronotropic response. Heart rate reserve below 80% is considered to be evidence of an impaired chronotropic response, which is a powerful indicator of mortality (5). We believe that caregivers should be familiar with these parameters and consider for routine incorporation into exercise test interpretation.

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Author`s Reply

To the Editor,

We would like to thank the authors for their comments and criticism of our original investigation (1), entitled "Heart rate recovery may predict the presence of coronary artery disease," published in Anatolian J Cardiol 2014; 14: 351-6. We wrote in the methodology section that "post-exercise HRR was measured in the sitting position during the cool-down period after the cessation of peak exercise." "Cooling down" commonly refers to easy exercise following strenuous exercise. In contrast, the "cool-down period" refers to the length of the warming-down time. In the