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Author`s Reply

To the Editor,

We thank you for your comments on our study published in the September 2014 issue of The Anatolian Journal of Cardiology entitled "Assessment of serum hepcidin levels in patients with non-ST elevation myocardial infarction (NSTEMI)." (1). They have raised some questions. Hepcidin is produced mainly in the liver and increases in response to inflammation, and its expression is regulated by anemia, hypoxia, and inflammation (2). In this single-center study, we evaluated whether the level of hepcidin increased in the acute phase in NSTEMI, known as acute inflammatory aggravation of a chronic atherosclerotic process.

There are conflicting results for hepcidin in coronary artery disease patients (3, 4). The first remark was about blood sampling time and symptom onset. We did not investigate hepcidin kinetics in this study; our aim was fundamentally to use hepcidin as a new cardiac marker instead of troponin. Another remark was about the time interval between the onset of the symptoms and blood sampling. According to our study design, we aimed to compare hepcidin levels with troponin levels in the diagnosis of NSTEMI. It is important that the hepcidin levels did not increase; meanwhile, the levels of troponin were increased in NSTEMI patients in the acute phase. The observed differences in these parameters, performed simultaneously from the same patients, forced us to think that there was no need to take the time interval between the onset of symptoms and blood sampling. The other remark was about the study of Suzuki et al. (4). The patient population and the design of the two studies were different, as the authors (4) studied ST elevation myocardial infarction patients, but we did not. Also, the sample of their study was extremely low, and their aim was also different. As stated in the criticism, if we performed a correlation analysis between CRP and hepcidin levels, it should have corroborated our results, showing hepcidin as a surrogate marker of inflammation.

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Transcatheter closure of antegrade pulmonary blood flow with Amplatzer septal occluder after Fontan operation

To the Editor,

We read the article of Karagöz et al. (1), entitled "Transcatheter closure of antegrade pulmonary blood flow with Amplatzer muscular VSD occluder after Fontan operation," published in The Anatolian Journal of Cardiology 2014; 14: 565, with great interest. Recently, in our clinic, we closed residual antegrade pulmonary blood flow with an Amplatzer septal occluder device after Fontan operation.

Our patient's initial diagnosis was unbalanced complete atrioventricular septal defect and double outlet right ventricle with D-transposed great arteries. His first surgery was a pulmonary banding operation when he was 2.5 months old. When he was 6.5 years old, a bi-directional Glenn operation was performed (with antegrade flow). He underwent an extracardiac Fontan operation at the age of 11 years in our clinic. During his hospital stay, 10 days after the Fontan procedure, massive pleural effusion, edema, and ascites were detected. Echocardiography revealed significant antegrade flow to the pulmonary artery. The patient underwent cardiac catheterization to close the antegrade flow. Mean pulmonary artery pressure was 33 mm Hg. The right ventriculogram and main pulmonary artery angiogram showed normally branched pulmonary arteries, with a narrowing in the main pulmonary artery owing to his first operation-pulmonary banding. The narrow part of the pulmonary artery was 9 mm, and the proximal and distal sides of this narrow part were 24.3 mm and 21.5 mm, respectively. An 11-mm Amplatzer septal occluder (AGA Medical, MN, USA) device was deployed at the narrow region. After deployment of the device, the mean pulmonary artery pressure decreased to 26 mm Hg, which was also high but at least lower than the pre-intervention pressure.

Residual forward flow from the ventricle to the pulmonary artery, via either a native pulmonary outflow tract or a previously banded or ligated main pulmonary artery, leads to ineffective even hazardous pulmonary blood flow and unnecessary ventricular volume overload in Fontan patients. This in turn can lead to persistent pleural effusions or ventricular failure, especially in patients with transposed great arteries, in whom surgical dissection of the main pulmonary artery during the Fontan procedure would be difficult or hazardous. At least 5 of 8 patients from the Desai et al. (2) series had transposed great arteries. Similarly, our case had transposed great arteries. It may be difficult to locate and close pulmonary antegrade flow due to the anatomy of the

great arteries in patients with transposed great arteries who had a sternotomy redone in the Fontan operation.

If it is complicated to close the pulmonary antegrade flow during the Fontan procedure due to transposition of the great arteries, transcatheter intervention can be performed safely and effectively after the surgery.

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Author`s Reply

To the Editor,

We would like to thank the authors of the letter for their interest about our paper entitled "Transcatheter closure of antegrade pulmonary blood flow with Amplatzer muscular VSD occluder after Fontan operation.", published in the September issue of The Anatolian Journal of Cardiology 2014; 14: 565 (1). In 1971, Fontan and Baudet described a surgical procedure for repair of tricuspid atresia that built on experimental and clinical research from the 1940s. Today, the Fontan procedure is the most commonly performed staged palliative surgical procedure in patients with single ventricle physiology to ultimately create a circulatory system driven by a single ventricle without passing the right ventricle (2). It has been performed to treat several complex congenital heart diseases, including tricuspid atresia, hypoplastic left heart syndrome, pulmonary atresia with intact ventricular septum, and double-inlet ventricle.

At the time of the Fontan procedure, it is necessary to remove all origins of supplemental pulmonary blood flow to avoid volume loading of the heart. However, this can result in acute reduction in ventricular preload and diastolic dysfunction in the early postoperative period (3). In addition, some studies reported that non-pulsatile pulmonary blood flow decreased capillary flow and increased vascular resistance (2). On the other hand, there is a risk of persistent pleural effusions or progressive ventricular failure in patients having forward flow from the ventricle to the pulmonary arteries after Fontan procedure (3). As a result, it is controversial as to whether additional sources of systemic to pulmonary artery flow are beneficial or not.

Transcatheter closure of accessory antegrade pulmonary blood flow is an alternative to surgery, because it is less invasive, easy to perform, reliable, and more comfortable (4, 5). Numerous kinds of devices are now commercially available for the closure. Petko et al. (4) showed that the off-label use of Amplatzer Septal or Ductal Occluders or an Amplatzer Vascular Plug for the closure was effective for the reduction of ventricular volume load and resolution of the pleural effusions, which can occur as a complication after cavopulmonary shunt or Fontan procedure. Desai et al. (3) also reported that the use of a Raskind Umbrella Occluder or Amplatzer Septal or ductal occluder for the closure was a safe and effective technique after cavopulmonary shunt or Fontan procedure.

In my opinion, an issue that is worthy of discussion may be the thrombotic problems in the author's case. Devices can be placed to the pulmonary artery band or pulmonary valve tissue or above the pulmonary valve (4). The place and approach for occlusion can be modified by patient anatomy and technical ease. By the way, if there is room between the pulmonary valve and device, the stasis of blood in the room can lead to formation of a thrombus. The thrombus is also possible for patients who have undergone surgical ligation of main pulmonary artery distally to the pulmonary valve, creating a pulmonary artery stump (6). In conclusion, we think that patients with a risk of thrombosis over time should be followed up more often in clinical practice, and anticoagulation may be considered in these cases.

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Basilic vein transposition should be the first option

To the Editor,

We have read with great interest the article, entitled 'Long-term patency of autogenous saphenous veins vs. polytetrafluoroethylene