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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