Unexpected Worsening of Mitral Regurgitation After Surgical Closure of Atrial Septal Defect

Masumi Iwai-Takano, MD, Hiroyuki Yaoita, MD, Masayoshi Oikawa, MD, Hironori Kaneko, MD, Akiko Nakamura-Shichishima, MD, Hiroko Tajima, MD, Kazuko Akutsu, MD, Akiami Yoshihisa, MD, Osamu Yamaguchi, MD and Yukio Maruyama, MD
First Department of Internal Medicine, Fukushima Medical University, Fukushima, Japan

Abstract
A 16-year-old male with a history of surgically-closed atrial septal defect (ASD) 2 years before was admitted to our hospital for lung congestion caused by severe mitral regurgitation (MR). Before the closure of the ASD, his echocardiographic findings showed mild prolapse of the anterior mitral leaflet (AML) with the hypoplastic posterior mitral leaflet (PML), but only mild MR. On admission, severe MR was observed by echocardiography in association with an increase in mitral anural size and the progression of AML prolapse. It was assumed that the worsening of MR was due to increased blood volume in the left-side of the heart after closing the ASD combined with cardiac growth. After mitral valvuloplasty, MR and heart failure disappeared.

Key words: atrial septal defect, mitral regurgitation, mitral valve prolapse

Case

A 16 year old high school student was admitted to our hospital with a complaint of shortness of breath on effort in 2004. He had a history of surgical closure of the atrial septal defect (ASD) in 2002. On admission, a mitral regurgitant murmur (Levine III/VI) was audible at the apex. X ray showed lung congestion and pleural effusion. Junctional rhythm similar to that before ASD closure was observed on electrocardiogram. The transthoracic Doppler echocardiographic findings on admission were compared to those before surgical closure of the ASD. Before the closure, echocardiography detected ASD with high pulmonary-systemic flow ratio (2.75) and resultant distortion of the left ventricle. Despite mild prolapse of the anterior mitral leaflet (AML) with the hypoplastic posterior mitral leaflet (PML) and short chordae, mitral regurgitation (MR) was trivial (Figure 1). In 2004, although there was neither residual shunt flow nor distortion of left ventricle, the center of the AML prolapsed without myxomatous change and/or calcification. The left atrium was

Fig. 1. Transthoracic echocardiographic findings on long-axial view before atrial septal defect (ASD) closure. Note the long anterior mitral leaflet and hypoplastic posterior mitral leaflet.
enlarged, and severe MR jet blew from the AML to the posterior wall of the left atrium (Figure 2).

During these 2 years, he gained about 10 cm in height, and the left ventricular end-diastolic and end-systolic diameters increased [LVDd, mm (index, mm/m²): 24 (24) to 34 (28), LVDs, mm (index, mm/m²): 35 (35) to 46 (38)]. The mitral annular diameter also increased from 22 mm (index, 22 mm/m²) to 38 mm (31 mm/m²). We assumed that both an increased blood volume in the left side of the heart due to ASD closure and the growth of his heart itself resulted in the increased mitral annular diameter, and augmented MR in conjunction with the hypoplastic PML. Mitral valvuloplasty was then successfully performed. Intraoperative findings included enlarged AML but with no myxomatous change, and hypoplastic PML with shortened chordae. After mitral valvuloplasty, MR and heart failure were improved.

Discussion

By echocardiographic findings, 50-95% [1-2] of ASD patients have MR due to prolapse of the AML and/or PML. The mechanism of such MR was considered to be deformation of the left ventricular cavity, which causes dislocation of the mitral valve [3]. The distance between the posteromedial papillary muscle and the mitral annulus is shorter in ASD patients than in normal subjects, so that the medial half of the AML prolapses [4]. Closure of ASD leads to normalized left ventricular geometry and a lower degree of prolapse and MR [2]. Our patient developed congestive heart failure in order to diffuse severe MR after ASD closure. Augmentation of MR after ASD closure is rare [1], and no mechanism have been reported. We considered that the increase in left ventricular volumes after ASD closure and patient growth caused the worsening of MR on the basis of hypoplasty of the mitral valve, i.e., enlarged AML and hypoplastic PML. We should keep in mind whether it is necessary to add mitral valvuloplasty to ASD closure even if preoperative MR is small in such cases.

References