

Letter to the editor

Independently from mitral regurgitation, Barlow disease may cause left ventricular enlargement and thereby anticipation of surgery

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Submitted December 3, 2014; accepted February 10, 2015

Key words

Mitral valve prolapse, left ventricle enlargement, cardiac surgery.

Sir:

We read the article 'Anatomic characteristics of bileaflet mitral valve prolapse – Barlow disease – in patients undergoing mitral valve repair' by Rostagno et al. (2014) with great interest. In this article, they shared their surgical observations about bileaflet mitral valve prolapse (MVP) in terms of mitral valve repair.

We appreciated their experiences on mitral valve repair in patients having MVP. However, we have some doubts about the timing of surgery. They reported that almost half of the patients were in New York Heart Association functional class (NYHA) I or II and that the left ventricular (LV) end-diastolic and end-systolic diameters of the patients were 58.02 ± 6.14 and 34.81 ± 6.88 mm, respectively. Moreover, LV ejection fraction of the patients were $62.32 \pm 7.11\%$. All of the above mentioned findings suggest the possibility that surgical intervention was premature in some patients according to guidelines (Vahanian et al., 2012). This may result from the good experience of the surgical team in the repair procedures for MVP. They may desire that mitral regurgitation (MR) disappears before impairment in systolic function of LV. This may have logical reasons. Instead of replacement, they repair mitral valves, thereby i) patients take not so much surgical risk, and ii) they are protected the patients from the negative effects of long-term mechanical valve. It is well known that capabilities may sometimes influence decision-making process. However, there is no randomized clinical trial investigating the optimal timing for surgical intervention in patients with MR who are candidate for mitral valve repair. In this context, new studies investigating the optimal timing for surgery are needed for patients with MR having high probability to undergo mitral valve repair.

Furthermore, we previously conducted a research on patients with classic MVP (Yiginer et al., 2012) and demonstrated that patients with classic bileaflet MVP have

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enlarged LV even in the absence of moderate or severe MR. LV end-diastolic diameters of 50% patients with MVP were found above the normal limits (>57 mm) in spite of the absence of significant MR. Additionally, systolic and diastolic diameters and volumes of LV in the MVP group were found to be significantly higher than those in the control groups. Ejection fraction of the LV in patients with MVP was not different from the healthy controls. MVP, which sometimes accompanies Marfan syndrome and Ehler-Danlos syndrome, is a connective tissue disorder of the heart (Delling and Vasan, 2014). Mitral valve apparatus is a component of the cardiac fibrous skeleton which supports the myocardial muscle as a connective tissue. Coexistences of MVP with tricuspid valve prolapse and enlargement of the ascending aorta in some patients supports the idea of 'MVP is a connective tissue disorder of the heart'. In this context, Barlow disease is related to pathological and/or geometrical changes of the fibrous skeletal structure of the heart and this may manifest in clinical practice as LV enlargement. LV internal diastolic and systolic diameters are essential parameters for timing surgical intervention in MVP complicated with MR. Additionally, in grading chronic MR, increased LV diameters support the presence of severe MR. Increased LV diameters in classic MVP may lead to overestimate of MR and to premature timing for surgical intervention. Therefore, being aware of this phenomenon may affect the decision about the timing of surgery in the patients with MVP complicated with MR in clinical practice.

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