The author suggests that the correlation between non-HDL-C levels and clinical parameters would be more informative. In our study, we also evaluated and reported correlations of non-HDL-C levels with age, weight, height, total body fat percentage, body mass index and physical activity status for both sexes. We found positive correlations with age in both sexes, and with height, weight and total body fat percentage in boys but not in girls. Our findings indicate that the unfavorable situations affecting both anthropometric values (especially related with fatness) and lipid parameters, which lead to the male predominance in cardiovascular diseases in adults begin in childhood.

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Functional status of the quadricuspid aortic valve/An uncommon coincidence of congenital quadricuspid aortic valve accompanied by hypertrophic obstructive cardiomyopathy

Kuadriküspid aort kapağın fonksiyonel durumu/Konjenital kuadriküspid aort kapak ile hipertrofik kardiyomiyopatinin nadir birlikteliği

Dear Editor,

We read with interest the recent case report by Bilge and colleagues (1) about a patient with quadricuspid aortic valve (QAV). The authors state that "the anomaly of the quadricuspid aortic valve can also cause aortic stenosis" (1). We would like to make a comment regarding this matter to avoid any misunderstanding, which could be caused by this statement. While it is true, that quadricuspid aortic valve stenosis has been described (2), it is extremely rare. In the most comprehensive review of the QAV currently available 186 cases of this anomaly were identified from the literature (3). The functional status was known in 154 cases. Of these 115 (74.7%) were regurgitant, while combined aortic valve stenosis and regurgitation was found in 13 cases (8.4%). In only one case (0.7%) the aortic valve was stenotic. A normal functioning valve was detected in 25 cases (16.2%). Dysfunction of the QAV made surgery necessary in 45.2% of the cases (3).

Further, while the association of a QAV with hypertrophic obstructive cardiomyopathy may be rare (1) it is nonetheless often associated with other congenital heart defects. In 18.3% of the cases the QAV was associated with other congenital cardiac malformations, most common being anomalies of the coronary arteries (3, 4).

Therefore, anyone who is not so familiar with this rare congenital heart defect should keep in mind that the most prevalent hemodynamic abnormality associated with a QAV is aortic regurgitation, often leading to aortic valve repair or replacement (3). Further, a QAV is often associated with other congenital heart defects.

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Author Reply

Dear Editor,

Firstly, I would like to thank my colleagues for the valuable comments they have offered. Quadricuspid aortic valve (QAV) was first defined by Balington in 1862 (1).

The mechanism of this congenital malformation is not fully enlightened. One of the leading hypotheses is an abnormal septation of embryological truncus arteriosus. Normally, after the septation of the arterial trunk, three mesenchymal swellings develop in to semilunar leaflets of the aortic and pulmonary trunk. In the setting of quadricuspid aortic valve, the fourth cusp arises during the early stage of truncal septation, resulting from either different number of primordial aortic leaflets or abnormal cusp proliferation (2).

Hurwitz and Roberts (3) described seven different types of QAV depending on the size of the cusps. In literature, the most frequent types were type B (three equal cusps and one smaller cusp) and type A (four cusps of equal size). Although the echocardiographic findings might suggest the size of the leaflets, they do not always correlate with the surgical findings. Aortic insufficiency is the predominant valvular abnormality seen in QAV as you have already described in your letter (4). It has been hypothesized that a small accessory cusp may cause an abnormal distribution of the transvalvular forces and consequently lead to aortic regurgitation. The identification of a QAV with aortic regurgitation is important because the high risk of endocarditis. Thus, endocarditis prophylaxis and echocardiographic follow-up is appropriate in managing high-risk patients. Although the QAV frequently functions abnormally, stenosis is unusual. In most of the series, aortic stenosis was demonstrated between 7-12% (5).

We think that our case is important because to our knowledge, this is the first report that demonstrates a congenital QAV associated with hypertrophic obstructive cardiomyopathy. Despite the low incidence, because QAV may also cause aortic stenosis, the reason of the transvalvular gradient must be established and a careful distinctive diagnosis in a patient with coincidental hypertrophic obstructive cardiomyopathy is required. In our patient, the aortic valve demonstrated three equally sized cusps with a one smaller extra cusp that causes mild regurgitation. The transthoracic echocardiography was helpful to diagnose QAV and transvalvular pressure gradient. Transesophageal echocardiography was used to evaluate further the aortic structure. Additionally, aortography and left ventriculography were performed to support the hypertrophic obstructive cardiomyopathy diagnosis.

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Evaluation of hemodynamic changes in patients with mitral valve replacement using dobutamine stress echocardiography

Mitral kapak replasmanı yapılan hastalarda dobutamin stres ekokardiyografi ile hemodinamik değişikliklerin değerlendirilmesi

Dear Prof. Dr. Bilgin Timuralp
Editor-in-Chief
The Anatolian Journal of Cardiology

The article by Tulga Ulus A. et al. (1) published in the recent issue of The Anatolian Journal of Cardiology investigated the hemodynamic response to Dobutamine stress (DSE) of bicarbon mechanical mitral valve of different size and evaluated the relationship between prosthetic size and cardiac recovery-remodeling process, 4.0±2.2 years after the operation.

The study demonstrated that, at follow-up, only the patients who had undergone mitral valve replacement (MVR) with small-sized valve prosthesis (≤29mm) achieved a decrease in cardiac mass index and a preserved left ventricular (LV) systolic function. On the contrary, the patients with large-sized mitral valve prosthesis achieved a decrease in systolic pulmonary artery pressure (PAP) and a non significant decrease in cardiac mass index, but had worse LV systolic and diastolic function (with higher volumes and diameters).

During DSE, PAP and diastolic mitral valve gradients increased in all patients, especially in patients with valve prosthetic size ≤29mm. However, in patients with prosthetic size >29mm, LV systolic and diastolic parameters worsened (LV end-diastolic dimension and LV end-systolic dimension resulted to be higher and isovolumic relaxation time to be more prolonged as compared with patients with smaller size valve).

The authors conclude that "prosthetic mitral valve size has an effect on cardiac remodeling in the late postoperative period…further investigations should be carried out to put forward more definite results" (1).

The conclusion seems to be in contrast with the detailed discussion regarding the relationship between the prosthetic valve size and cardiac remodeling and in which the role of mitral valve pathology, the hemodynamic consequences and surgical technique are analysed.

The patients with mitral insufficiency or with a combined type of lesions (stenosis and insufficiency) are more prone to have larger ventricles, an altered ventricular geometry, with more spherical morphology, and anatomic and functional damage of myocardial fibers. In these patients a large-sized prosthetic valve are frequently implanted because of mitral annulus dilatation; the MVR with chordal transection, without posterior leaflet preservation, leads to more spherical ventricular geometry, reducing the possibility of favourable reverse remodeling. Therefore the patients implanted with >29mm mitral prosthesis showed a worse recovery of LV size and function and a less decrease of LV mass index as a result of baseline valve pathology, type of hemodynamic consequences (LV enlargement, mitral regurgitation) and surgical technique. During DSE ventricular systolic and diastolic parameters worsened to, as a consequence of impaired LV contractility response.

The patients implanted with ≤29mm mitral prosthesis (valve replacement with chordal transection, without posterior leaflet preservation), had more frequently lone mitral stenosis or combined mitral and aortic disease (consequently underwent double valve replacement), smaller ventricles and higher EF and FS. In these patients, after operation, ventricular size significantly and faster decreased, ventricular systolic function increased and hemodynamic response to DSE resulted normal as consequences of preserved contractility response.

Therefore this interesting study outlines the needs of accurately studying all patients with mitral valve disease before and after MVR, for the evaluation of surgical, anatomical and functional results, and the utility of performing physical or pharmacological stress for the evaluation of prosthetic valve hemodynamic performance.

Cardiac remodeling seems to be related with preoperative valvular pathology, hemodynamics and ventricular size and performance; therefore, the optimal timing for MVR is warranted.

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