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**LETTER TO THE EDITOR**

# Aortic Valve Calcification

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**To the editör,**

The basic pathology in the development of aortic valve calcification is atherosclerosis (Motawea et al., 2023). This pathology, which begins in the annulus of the aortic valve, spreads to the free edge of the valves. The thickening of the valves causes hemodynamic changes. The left ventricle responds to this pressure change by developing hypertrophy. While the mass of the heart muscle increases, the increase in fibrosis leads to irreversible heart failure. Left ventricular hypertrophy, increased myocardial oxygen consumption, and increased pressure on the coronary arteries cause anginal symptoms. The risk of syncope and sudden death increases as the degree of narrowing in the aortic valve orifice increases. When aortic valve stenosis becomes symptomatic, surgical options come to the fore. Transcatheter aortic valve replacement (TAVR) is an important option in the treatment of this pathology, which will become a significant problem in aging societies, in the patient population with high surgical mortality. Calcifications extending to the left ventricular outflow tract complicate the TAVR procedure. (Sá et al., 2022).

However, in order to use this option, the access artery must be suitable. Advanced calcification of the aortoiliac arteries is possible with the use of alternative arteries. TAVR can also be performed via graft anastomosis to the subclavian artery and sutures placed at the apex of the heart. Surgery for aortic valve calcification also has its own characteristics. The biological valve option, which is suitable for the elderly patient population, eliminates the need for lifelong use of vitamin K antagonists. In the younger population, mechanical valves are used. In recent years, valves prepared from the pericardium of the heart using the Ozaki method have begun to be used. Surgery for aortic valve calcification is also special. In cases where calcification has spread to the annulus of the valve, the septum, and the anterior valve of the mitral valve, adequate debridement of the calcifications should be performed. Paravalvular leakage may develop in patients with inadequate debridement of the valve annulus. Recently, it has been reported that paravalvular leakage has also developed after TAVR (Shehada et al., 2018).

Excessive debridement may cause destruction of the aortic root, conduction blocks and mitral valve perforation. In some cases, calcification blocks can be seen in the

aortotomy line as an extension of aortic calcifications. In cases where closure of the aortotomy is not possible, ascending aorta replacement may be required. Debris falling into the left ventricle during the decalcification process increases the risk of cerebrovascular accident in the early postoperative period (Zhang et al., 2018).

Washing the left ventricle after resection reduces this risk. Hypertrophy in the septum muscle accompanying aortic valve calcification should also be evaluated surgically during valve surgery. In cases where the valve is extremely narrow, echocardiography in the preoperative period may be insufficient to measure the subvalvular gradient. In surgical exploration, the papillary muscle not being seen when viewed from the aortic valve requires septal resection. Inadequate septal myocardial resection will cause a significant gradient to remain on the implanted valve. Another difficulty encountered during surgery for aortic valve calcification is the narrow aortic root. In these cases, valve implantation appropriate to the patient's body mass index is performed using aortic root expander techniques. The use of the Stureles valve can be used as an alternative method to root expansion (Agarwal et al., 2023).

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