Left Ventricular Dysfunction in Severe Aortic Valve Disease

Jae Yeong Cho, MD, PhD
Department of Cardiovascular Medicine, Chonnam National University Hospital, Gwangju, Korea

Introduction

Aortic valve disease is a common disorder that affects about 2% of the whole adult general population. Aortic stenosis (AS) is mostly due to lesions of the valve leaflets while aortic regurgitation (AR) is a disease of leaflet or aortic root disease. Those two disease entity appears to be different from each other, however, they both affect left ventricle (LV) in similar ways.

Mechanism of Left Ventricular dysfunction in Severe Aortic Stenosis and Regurgitation

The natural history of aortic disease is strongly associated with LV dysfunction. Progressive obstruction of LV outflow by AS leads to compensatory changes in ventricular morphology from hypertrophy by pressure overload. Those with severe AR shows LV remodeling from a combination of both pressure and volume overload. If untreated, LV cavity dilatation and following dysfunction may occur and develop symptoms of heart failure that leads to hospitalization and eventually cardiac death.

In the beginning, pressure hypertrophy in AS normalizes wall stress through preservation of LV ejection fraction and stroke volume. Myocardial ischemia can occur and myocardial apoptosis and fibrosis develops. Therefore, pathologic mechanisms of LV dysfunction comprise of abnormal contraction of microtubules within the myocyte cytoskeleton, intermittent ischemia, neurohormonal activation, and abnormalities in calcium handling. As AS become more advanced, the compensation of the LV become inadequate as the high afterload and these accompanying pathologic mechanisms result in deterioration of systolic function and cavity dilation.

The regurgitation and excess ventricular afterload in chronic AR stimulate cardiomyocyte hypertrophy. This facilitates chamber dilatation that compensate the increase in regurgitant volume, normalize wall stress, and also maintain a normal cardiac output. Ventricular adaptation to the volume and pressure overload with increased chamber compliance leads to tolerance of AR for many years. However, once the preload reserve is decompensated, the high afterload leads to deterioration in LV performance and heart failure.

Challenges in Patients with Aortic Valve Disease and Left ventricular dysfunction

It is very difficult to establish the severity of the lesion, risk assessment for patient prognosis, and to determine appropriate surgical intervention for those patients with critical valvular disease. For patients with AS and ventricular dysfunction, differentiating truly severe AS and a concomitant AS and cardiomyopathy would be challenging and crucial at the same time.

Conclusion

Severe aortic valve disease has significantly associated with LV dysfunction in terms of pressure and volume overload. Severe aortic valve disease affects LV function and vice versa. In daily clinical practice, it is sometimes hard to distinguish truly severe aortic valve disease from pseudo-severe disease in the setting of severe LV dysfunction. LV dysfunction mechanism in severe aortic valve disease provide us better understating of pattern of adaptation and decompensation of LV against pressure and volume overload, which in turn may help us with searching for better treatment strategy for systolic heart failure.