Editorial

Aortic Stenosis and Hypertension: Is There Any Relationship?

Constadina Aggeli, Konstantinos Lampropoulos, Christodoulos Stefanadis

1st Department of Cardiology, University of Athens Medical School, Hippokration Hospital, Athens, Greece

Key words:
Aortic valve stenosis, hypertension.

Address:
Constadina Aggeli

47 Georgiou Papandreou St.
15773 Zografou
Athens, Greece
e-mail:
caggeli@otenet.gr

Aortic stenosis (AS) is the third most common cardiac disease in developed countries after hypertension and coronary artery disease (CAD), and the most common native lesion. AS is also the most common reason for aortic valve replacement, accounting yearly for about 40,000 valve operations in Europe and 95,000 in the United States. Many studies have shown that AS is a worldwide problem in industrialised countries and constitutes a significant health problem, especially in the elderly.[1]

Epidemiological, histopathological, and experimental evidence indicates that calcification of the aortic valve is an active rather than a passive biological process within the valve leaflet, causing regulated bone formation. Furthermore, there are histopathological data suggesting that calcifying AS is in some ways similar to atherosclerosis.

Several studies have addressed the prevalence of various cardiovascular risk factors in patients with AS. Arterial hypertension has been shown to be more frequent in patients with AS compared to those without. It seems that the frequency of this combination has been underestimated. Antonini-Canterin and colleagues[2] reported a prevalence of 32% for systolic hypertension in a series of 193 patients with severe symptomatic AS. In a more recent study, Briand et al[3] showed that the prevalence of systemic hypertension in patients with AS can reach as high as 50%. On the other hand, the impact of hypertension on the assessment of AS has not been clarified. Recently, interest in this issue has been renewed and several experimental and clinical studies have been carried out. There has been discussion of how arterial hypertension might influence the aortic valve area and the transvalvular pressure gradient. Some investigators believe that there is a direct influence of blood pressure on the indices of AS severity; others could not prove an independent effect.

There is controversy as to whether blood pressure does directly affect common indices of AS severity. It is well known that indices of AS severity are flow dependent. Thus, acute changes in blood pressure can significantly alter these indices as a consequence of concomitant changes in transvalvular flow. Systemic arterial hypertension may result in an increase in systolodiastolic hypertension, a decrease in aortic compliance, or both.[3] On the other hand it is not well known whether the presence of hypertension plays an additional role in left ventricular remodelling and symptom development in patients with AS. Arterial hypertension and AS are the two main pathological models of left ventricular systolic overload. Furthermore, blood pressure is an important determinant of global left ventricular afterload, which seems to be associated with mortality in patients with AS.

The left ventricle of patients with AS and concomitant systemic hypertension is

subjected to two additive loads: a valvular load and an arterial load. On the other hand, it is well known that the assessment of patients with AS includes the measurement of transvalvular pressure gradient and aortic valve area, as well as assessment of left ventricular geometry and function. According to a study by Kadem et al,[4] when assessing the severity of aortic stenosis it is important to consider the status of arterial haemodynamic function. Those investigators postulated that the presence of hypertension affects the assessment of AS, since increasing systemic pressure causes a decrease in pressure gradients and an increase in valve area. This conclusion was drawn from a study of pigs with supravalvular stenosis induced by banding the ascending aorta. This banding of the aorta may have caused an increase in the anatomical and effective orifice area. The most recent experimental study was published by Mascherbauer et al.[5] They did not find any influence of blood pressure on aortic valve area or pressure gradients.

In contrast with experimental studies, in clinical studies the interpretation of the results poses many difficulties. Razzolini's group[6] demonstrated a small but linear increase of pressure gradients across a porcine bioprosthesis with rising systemic pressure. But these results are not consistent with fluid dynamic theory. In another interesting study, Little et al[7] used handgrip exercise and phenylephrine infusion to increase blood pressure in patients with AS. In this patient population, blood pressure and systemic vascular resistance increased at peak intervention, whereas the transvalvular flow rate decreased. Moreover, the aortic valve area decreased while the mean transvalvular gradient did not change. The only independent predictor of the change in aortic valve area was the change in cardiac output.[8] Khot and colleagues[9] found that nitroprusside improves cardiac function in patients with left ventricular systolic dysfunction and AS. Furthermore, Chockalingam et al[10] demonstrated that angiotensin-converting enzyme inhibitors caused an increase of ejection fraction while pressure gradients and valve areas remained unchanged.

This issue remains open, since there are insufficient data so far to substantiate one view or the other. AS severity may be assessed after blood pressure optimisation. An acute increase in blood pressure or chronic arterial hypertension may have different impacts on the assessment of AS. Therefore, more research data are needed before we can adequately evaluate the relationship between blood pressure and the assessment of AS severity.

References

1. Spargias K, Manginas A, Pavlides G, et al. Transcatheter aortic valve implantation. The first Greek experience. Hellenic J Cardiol. 2008; 49: 397-407.
2. Antonini-Canterin F, Guoquian H, Cervesato E, et al. Symptomatic aortic stenosis: Does systemic hypertension play an additional role? Hypertension. 2003; 41: 1268-1272.
3. Briand M, Dumesnil JG, Kadem L, et al. Reduced systemic arterial compliance impacts significantly on left ventricular afterload and function in aortic stenosis: implications for diagnosis and treatment. J Am Coll Cardiol. 2005; 46: 291-298.
4. Kadem L, Dumensil J, Rieu R, Durand LG, Garcia D, Pibarot P. Impact of systemic hypertension on the assessment of aortic stenosis. Heart. 2005; 91: 354-361.
5. Mascherbauer J, Fuchs C, Stoiber M, et al. Systemic pressure does not directly affect pressure gradient and valve area estimates in aortic stenosis in vitro. Eur Heart J. 2008; 29: 2049-2057.
6. Razzolini R, Gerosa G, Leoni L, Casarotto D, Chioin R, Dalla-Volta S. Transaortic gradient is pressure-dependent in a pulsatile model of the circulation. J Heart Valve Dis. 1999; 8: 279-283.
7. Little SH, Chan KL, Burwash IG. Impact of blood pressure on the Doppler echocardiographic assessment of severity of aortic stenosis. Heart. 2007; 93: 848-855.
8. Pibarot P, Dumesnil JG. Assessment of aortic stenosis severity: check the valve but don't forget the arteries! Heart. 2007; 92: 780-782.
9. Khot UN, Novaro GM, Popovic ZB, et al. Nitroprusside in critically ill patients with left ventricular dysfunction and aortic stenosis. N Engl J Med. 2003; 348: 1756-1763.
10. Chockalingam A, Venkatesam S, Subramaniam T, et al. Safety and efficacy of angiotensin-converting enzyme inhibitors in symptomatic severe aortic stenosis: symptomatic cardiac obstruction-pilot study of enalapril in aortic stenosis (SCOPE-AS). Am Heart J. 2004; 147: 848-855.