10% of BAV stenosis patients in a cohort of 96 congenitally malformed aortic valve patients who underwent simultaneous AVR and proximal aortic surgery [17]. Lower prevalence of aortic media EFL in both study subgroups compared with our findings may have resulted from inclusion of the patients with smaller diameters of the proximal aorta in their study (i.e. ≥45 mm). In accordance with our findings, presence of BAV insufficiency was associated with a much higher prevalence of significant aortic media EFL compared with BAV stenosis (i.e. OR 8.8; 95% CI 2.9–28.1) in the above mentioned study [17]. Another recent study, which included a significant proportion of BAV insufficiency patients (i.e. 60% of the total study population), showed a high prevalence of moderate/severe histological alterations in the aortic media even in the absence of clinically relevant proximal aortic dilatation [18]. In their multiple regression analysis, these authors demonstrated a significant association between moderate/severe aortic media EFL and diameter of the aortic annulus, which is in turn an indicator of aortic root disease (i.e. root phenotype of BAV disease) [18]. Similarly, Cotrufo and co-workers were able to show significant differences in the expression and spatial distribution of extracellular matrix proteins in the proximal aorta between patients with BAV stenosis vs BAV insufficiency in a series of biomolecular investigations [8].

Study limitations

There are some important limitations of our study. The retrospective design is a clear limitation, which may be overcome only by a randomized controlled trial. The limited number of included patients (i.e. 15% of consecutive BAV patients who underwent AVR during the study period) may be explained by conservative approach to the proximal aorta in our institution during the study period. The third limitation is that we have no data on the serial measurements of the downstream aorta for the whole study population. The available echocardiographic data are of screening value and only sufficient to exclude a clinically relevant progression of distal aortic disease. Therefore, clinically silent progression of downstream aortic disease may not be excluded.

CONCLUSIONS

The current study demonstrates that BAV patients with aortic valve insufficiency and a proximal aorta of ≥50 mm have a significantly higher rate of moderate/severe EFL compared with their counterparts with BAV stenosis.

Conflict of interest: none declared.

REFERENCES


eComment. Evaluating aortic media changes in ascending aorta aneurysm with bicuspid aortic valve disease

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We read with great interest the paper by Girdauskas and colleagues who found that patients with bicuspid aortic valve (BAV) insufficiency and a proximal aorta of ≥50 mm had a significantly higher rate of moderate/severe proximal aortic media elastic fibre loss as compared to their counterparts with BAV stenosis [1]...
expression in mildly dilated ascending aortas in BAV stenosis and tricuspid aortic valve stenosis groups and also a decreased TGFβR1 expression only in BAV [2]. The existence of difference or not of the expression of extra domain-A splicing variant of fibronectin in BAV stenosis versus BAV insufficiency groups is an interesting issue as it was shown that its expression was activated in the media of mildly dilated BAV and tricuspid aortic valve (TAV) [2].

Another topic is the evaluation of smooth muscle cell (SMCs) phenotype and orientation in ascending aorta samples (both aortic curvatures) in BAV stenosis versus BAV insufficiency groups as different flow derangements and shear stress anomalies might be observed in these patients. Is there a different pattern of ascending aorta medial remodelling in these groups? Smooth muscle cells switched their phenotype in mildly dilated BAV and tricuspid aortic valve (TAV) [2]. Smoothelin and myocardin mRNAs decreased in all samples from BAV and TAV, with the exception of BAV convexity. There was also a change in orientation of smoothelin-positive SMCs and an increase of α-smooth muscle actin mRNA [2]. The expression of genes that are involved in extracellular matrix remodelling is another field which we could search for differences of expression of matrix metalloproteinase-2 (MMP-2), tissue inhibitor of metalloproteinases-2, MMP-9 and other MMPs in BAV stenotic versus BAV insufficiency groups. The MMP-2 mRNA was increased in the convexity of mildly dilated ascending aortas of BAV and TAV groups [2].

Differences in the biosynthesis of collagen and cross-linking in ascending aorta aneurysm of patients with BAV stenosis versus BAV insufficiency is another field of investigation. Are there any differences in collagen turnover in BAV stenosis versus BAV insufficiency groups? It was found that similar collagen turnover rates were observed in dilated and non-dilated aortas of BAV patients [3]. Is the nature of proximal aortic aneurysm (collagen biosynthesis and cross-linking) similar in BAV stenotic and BAV insufficiency groups? Is the collagen network similar in both groups? What about the ratio of hydroxylysyl pyridinoline to lysyl pyridinoline, two distinct forms of collagen cross-linking? Is there any defect in post-translational collagen modification? In dilated aortas from patients with BAV a deficiency was found at the level of lysyl hydroxylase (PLOD1) which showed reduced PLOD1 expression [3]. Could microRNA (miR)-29 expression be involved in the ascending thoracic aorta with aneurysm progression in BAV patients? It was demonstrated that decreased levels of miR-29b in the aortic wall could attenuate aortic aneurysm progression in two different mouse models of abdominal aortic aneurysms [4]. BAV-related ascending aorta dilation represent a potential field of investigation for future research.

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References