Mechanism of autograft dilatation after Ross operation: some more insight please!

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The article by Hazekamp et al. [1] reporting mid-term results of the Ross operation in a pediatric population raises concerns about the durability of autograft in this important subgroup of patients. Their finding that 90% of autograft diameters indexed to body surface area was above the 90th percentile of normal aortic root diameters at latest follow-up is not only greater than what others have reported [2,3] but is also alarming as progressive autograft dilatation leads to regurgitation, which ultimately culminates in autograft valve dysfunction prompting reoperation.

Negligible valve-related morbidity, freedom from chronic anticoagulation, ability to accommodate growth and, most importantly, scarcity of alternative treatment options make Ross operation a valid and attractive therapeutic modality in the infant and child with aortic valve disease or complex left heart obstruction [3,4]. However, a very high prevalence rate of autograft dilatation in the series of Hazekamp et al. [1] makes one wonder if reality is just the opposite.

Prevalence rates of autograft dilatation vary according to definition of significant dilatation (generally 4.0 cm or greater), methods used to identify it (one or more levels of the left ventricular outflow tract), technique used for operation, and duration of follow-up [4]. Possible explanations for this finding include wider adoption of root replacement technique, greater likelihood of geometric mismatch of aortic and pulmonary roots, and physiological dilatation (i.e. root growth) in children undergoing the Ross operation [3,4]. However, a better way of finding out the pathology of autograft dilatation will be to subject explanted autografts to histopathological analysis to look for features such as focal interruption of the media of the vessel wall with total absence of elastic fibers and intimal proliferation with fibrosis, and ischemic damage to the wall of the pulmonary autograft root replacement caused by division of the vasa vasorum of the pulmonary artery from the coronary arteries during the Ross operation which may be a more logical explanation for autograft dilatation. As Hazekamp et al. [1] explained three autografts in their series it will be interesting to know whether these were subjected to histopathology techniques to look for some of the above-mentioned features.

Furthermore, an important issue that needs clarification is the optimal timing of Ross operation especially in the setting of aortic regurgitation. The suggestion by Hazekamp et al. [1] that the Ross procedure should only be considered when valve-sparing techniques can no longer provide a solution for pediatric aortic valve disease is probably valid for patients with aortic stenosis, however, delaying the operation in cases of aortic regurgitation may not be a good idea. Delaying surgery in the setting of chronic aortic regurgitation results in the increase in the ratio of left ventricular end-diastolic radius to the thickness of the posterior wall in diastole suggesting that there is disruption of left ventricular short axis architecture and myocardial contractile function [5]. This could well contribute to suboptimal outcome after Ross operation with progressive autograft dilatation.

References


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Letter to the Editor

Reply to Raja

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