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Dilatation of the ascending aorta associated with bicuspid aortic valve

Salah A Mohamed
University of Lubeck, Germany

The ascending aorta and the semilunar valves share common embryological origins, in which the contributions of various cell populations (e.g. cardiac neural crest cells) are involved. If during valvulogenesis, the original semilunar valve fails to separate and remains fused at the valve commissures, it results in the development of a Bicuspid Aortic Valve (BAV). BAV is the most common type of congenital cardiac malformations with an estimated incidence of 1-2% in the general population. This anomaly leads to an increased risk for severe cardiovascular events, which are not only due to valvular dysfunction itself but are further caused by concomitant dilatation of any or all of the segments of the proximal aorta occurring in roughly 40-60% of BAV patients, thus representing a significant risk factor for catastrophic clinical events involving high mortality and morbidity. With respect to operative criteria that are always seriously and controversially debated, surgical treatments are primarily decided in case of most serious causality. From the molecular biological respects, disturbed remodeling of the extracellular matrix in the aortic wall and an increased incidence of vascular smooth muscle cell loss play an essential role in the pathogenesis of thoracic aortic aneurysms associated with BAV. Here we report the extrinsic factors involved in hemodynamic alterations associated with increased wall shear stress due to modified flow profile and discuss the intrinsic factors of congenital aortic fragility, which is responsible for medial degeneration in the vessel wall. We discuss the genetic basis and basic pathology underlying BAV and ascending thoracic aortic aneurysms and compare these with known mechanisms underlying other aortic pathologies.

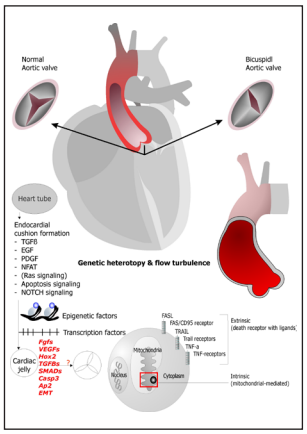

Figure-1: The genetic basis and pathogenesis of the bicuspid aortic valve (BAV) and BAV-associated aortopathy appears to be multifactorial. At the onset of valvulogenesis, a number of mechanisms (e.g. genes, epigenetic factors, fluid forces) may be involved, either alone or in combination, in the pathogenesis.

Recent Publications

1. Mohamed S A (2017) Genetic basis and hemodynamic aortopathy of the ascending aorta and dissection. *Cardiac surgery*. Avid Science: 2-39.
2. Mohamed S A (2017) Heart, aorta and aortic valve development and cardio-vascular malformations. *Human Genetics & Embryology*; 7: 139.

Biography:

Salah A Mohamed is an Associate Professor of Experimental Cardiac Surgery. He has published in many reputed scientific journals on the topics of aortic and aortic valve disease, genetics and biomarker discovery. His laboratory also focuses on understanding the causes of atrial fibrillation. He is an Editorial Board Member of many scientific and medical journals.

salah.mohamed@uksh.de