



Identification of potential early biomarkers of aortopathy

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Abstract:

Bicuspid aortic valve is the most common congenital heart disease and in approximately 50% of cases it is associated with aneurysms of any or all segments of the aorta. Ascending aortic aneurysm is responsible for 1%-2% of all deaths in industrialized countries and poses a challenge on the growing and aging society. Although aortic aneurysms are generally benign, a progressive increase in their diameter can lead to catastrophic and fatal event of acute aortic dissection and aortic rupture. Comprehensive genetic, molecular and proteomic analyses have increased our understanding of the complex cellular processes and signaling involved in the pathophysiology of ascending aortic aneurysms. To support the two known hypotheses proposed to explain the causality in aneurysm formation, intrinsic factors related to vascular remodeling were further investigated and analyzed in patients with congenital BAV and Marfan Syndrome (MFS). In MFS, mutations are present in the gene encoding for the extracellular matrix protein fibrillin-1, which causes the dysregulation of transforming growth factor-beta signaling. The second hypothesis

proposes a weakening of the aortic wall by steady laminar shear stress caused by the malformed BAV. This theory is supported by the asymmetrical extracellular matrix protein expression patterns and vascular smooth muscle cell apoptosis within the proximal BAV aorta, indicating shear stress-induced changes at certain sites. The interactions between mechanical forces and biological functions are intimately coupled. The medial degeneration in thoracic aortic aneurysms in MFS and tricuspid aortic valve patients shows a progressive increase as the patient's age. The BAV and ascending aortic aneurysm appears to share multiple etiologies. In BAV-associated aortopathy, the flow conditions and oxidative stress play a decisive role in the dilation of the ascending aorta. Summary of some of the recent developments in the field of cellular and molecular analysis, targeting and highlighting the molecular aspects which could help in elucidating the etiology of aortopathy and help in the development of biomarkers in the clinical setting will also be provided.

Biography:

Salah A Mohamed has completed his PhD from University of Bremen and Postdoctoral studies from Institute of Legal Medicine and Department of Cardiac Surgery, University of Luebeck. Currently he is an Associate Professor of Experimental Cardiac Surgery at University of Luebeck. He has published more than 40 papers in reputed journals and has been serving as an Editorial Board Member of repute.