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Aortic aneurysm from physiopathology to risk assessment: A comprehensive review

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Introduction: Abdominal aortic aneurysm (AAA) is present in 9 % of men older than 65 years old and is among top 10 leading cause of death in the US. AAA measurement has become the standard approach to assess the risk of AAA rupture. Other methods to predict AAA growth and rupture prematurely would lead to better outcomes. Therefore, we performed a comprehensive review on the physiopathology of AAA assessment.

Methods: We aimed to evaluate biological changes within the aortic wall as well as inflammatory processes, biomechanical factors and other non-invasive methods that might predict earlier aneurysm growth and rupture.

Discussion: The aortic wall is structured by 3 distinct adventitia, aortic matrix and endothelium. The adventitia is affected by inflammatory mechanism of the aneurysm formation; the aortic matrix allocates the collagen and elastin that are degraded by proteolytic enzymes, whereas in the endothelium there is a decrease in nitric oxide release causing abnormal shear stress of the wall, release of oxidative stress, and intra-luminal thrombus formation. The protein kinase Jun N-terminal (JNK) promotes activation of inflammatory cytokines in vascular smooth muscle cells. eNOS deficiency is associated with increased atherosclerosis and spontaneous aortic aneurysm and dissection. Angiotensin receptors activation leading NADPH oxidase release and subsequently oxidative stress byproducts will cause vascular inflammation and wall remodeling. Atherosclerotic plaques host macrophages that express proteolytic enzymes responsible for collagen and elastin breakdown; also lead to wall hypoxia with consequent neovascularization and wall weakness. Biomechanical parameters are wall strength and the forces applied to the AAA wall calculated by multiaxial analysis and physical laws were proven to predict AAA growth.

Conclusion: Better understanding of those parameters will improve AAA management and therapy.

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