Literature Review of Modern Hypotheses of Seronegative Spondyloarthropathies

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Seronegative spodyloarthropathies (SpA) are a set of disorders marked by chronic inflammation of axial joints. These are portrayed by similar clinical syndromes, genetic susceptibility, and constitutional symptoms, with prominent tissue inflammation and joint destruction. Specific clinical types comprise of Ankylosing Spondylitis (AS), Reactive arthritis (RA) with Spondylitis, Psoriatic Arthritis (PA), Enteropathic Arthritis (EA) and Undifferentiated SpA. This review summarizes the proposed classification of major pathogenic mechanisms of seronegative SpA. It also considers the hypotheses of its etiology and pathogenesis in recent times. The current understanding of SpA pathogenesis is obscure. Subsequently, knowledge of early tissue, cellular, and molecular changes is incomplete. However, many researchers recognize the influence of the environment, immunologic processes, and genetics as factors of disease source and development. Majority of the focus is on HLA- B27 in predisposed individuals, immunologic processes like molecular mimicry and arthritogenic peptide hypothesis. Recent studies also highlight the pivotal roles of cytokines, tumor necrosis factor alpha (TNF-a), and angiogenesis in immune deregulation which results in disease morbidity. The most promising new direction has been the involvement of cytokines. By elucidating the central role of cytokines in etiology and pathogenesis of SpA, diagnostic and therapeutic significance is apparent.

Myocardial Bridging, From a Simple Benign Condition to Sudden Cardiac Death

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Myocardial Bridging represents an anomaly of the coronary circulation, characterized by a myocardic course of a major epicardial artery. The segment is referred to as the "tunnelled artery" as it runs beneath a layer of muscle fibbers, which varies in length and thickness. The most common site of this anomaly is the left anterior descending artery, but it can also be confined to any other coronary branches. The condition is clinically silent most of the times, being accidentally discovered during an angiographic study or at autopsies. Even though usually benign, clinical manifestations vary from ischemia to sudden cardiac death. Superficial bridges are usually of no clinical importance, while deep ones lead to different cardiac complications. The estimated frequency varies from 1.5% to 16% at coronary angiography studies, to 80% in some autopsy studies. While still debated whether just an anatomic variant or a malignant condition, different pathology studies showed morphologic alteration of the myocardium tributary to the bridged artery. Furthermore, there is evidence that the tunnelled artery is protected from atherosclerosis, while the proximal and distal segments have an increase susceptibility to atheroma plaque formation. The main physiologic effect of the myocardial bridge occurs with each systole, when the coronary artery is compressed between the overlying muscle bundle and the rest of the ventricular mass. Yet, additional research is needed to define which bridges are life-threatening and furthermore, which are the most suitable therapy options for these patients. Our goal was to review the literature regarding Myocardial Bridging and present two opposite clinical cases, one discovered by chance after the autopsy and the other of sudden cardiac.