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The Role of Inflammatory Biomarkers in Diabetic Mitral Annular Calcification

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Introduction

Mitral annular calcification (MAC) is a chronic and degenerative process that causes calcium and phosphate precipitation in the fibrous structure surrounding the mitral valve leaflets. It is also more than that, as MAC has been linked to lipid and mineral metabolism abnormalities, chronic kidney disease, and inflammation. Age, female sex, obesity, hypertension, left ventricular hypertrophy, dyslipidemia, diabetes mellitus, advanced chronic kidney disease, osteoporosis, and smoking are all known risk factors for MAC development. MAC, in turn, is a marker for multi-site atherosclerotic plaques in the coronary and peripheral arteries. Because MAC is more common in the elderly, a link to mitral valve disease (e.g., mitral stenosis) is also to be expected. All of these conditions have emerged as major causes of cardiovascular disease and mortality, arrhythmias (atrial fibrillation), and mitral valve surgery complications.

The most common and conclusive diagnostic method is echocardiography, which shows MAC as an irregular and echo-dense structure, the extent of which determines the severity of the calcification [1-3]. The echo density in mild and moderate cases does not exceed 180° and 180-270°, respectively, whereas calcium deposits that extend beyond 270° are considered severe. Computer tomography provides a more accurate and detailed view of the calcification within the mitral valve apparatus and aids in the differentiation of MAC from aortic valve and coronary calcium, but it is also more expensive and less widely available. MAC and aortic valve sclerosis (AVS), two risk factors for heart valve calcifications, have received increased scientific attention in recent years.

Description

Type 2 diabetes and non-alcoholic fatty liver disease (NAFLD) have both been linked to an increased risk of MAC. Furthermore, the role of underlying metabolic conditions in micro- and macro vascular complications, adverse cardiovascular events, and deaths is now well established. Heart valve calcification begins as a slow and silent process before manifesting symptomatically and becoming life threatening, so researchers are now investigating the subclinical pathogenic mechanisms that are especially active and mutually enhancing in the presence of diabetes. It's difficult to say whether insulin resistance is a cause or an effect because it appears to be a "two-way street" with consequences that go beyond the obvious glycemic imbalance associated with diabetes. Insulin resistance is commonly assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) and other similar formulae that take C-peptide levels into account [4,5].

Insulin resistance and chronic inflammation amplify each other's negative effects on many of the body's normal processes. They undermine the body's adaptive immunity by initiating the progressive reduction in mass and function of beta-cells, for example. Inflammation is measured using standardised methods based on the collection of specific proinflammatory cytokines, the most common of which is the pentraxin protein family's high-sensitivity C-reactive protein (hsCRP). HsCRP which is normally synthesised by the liver, is one of the clearest indicators of acute inflammation and, as such, is used to assess cardiovascular risk. Because smooth muscle cell lymphocytes and monocytes can produce hsCRP in atherosclerotic lesions, it is a sensitive marker for chronic inflammation as well as emerging atherosclerotic disease.

Conclusion

Interleukin 6 (IL-6) and TNF- are two hsCRP mediators that are equally useful in assessing chronic, subclinical, low-grade inflammatory status. When there is inflammation, interleukin 6 is produced, which stimulates the production of hsCRP. It also aids in the activation, growth, and differentiation of B and T cells, which are critical components of the body's immune system. Elevated IL-6 levels in the blood have been linked to diabetes and dyslipidemia, and they have been shown to have a significant predictive value for myocardial infarction and death from coronary artery disease. Furthermore, hsCRP and IL-6 have been linked to the occurrence of MAC, indicating that inflammation plays a role in the pathogenesis and progression of MAC. TNF-, on the other hand, is known for a variety of effects, including the promotion of insulin resistance and metabolic processes that release the energy required for inflammatory reactions to occur at the cellular level.

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