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Perspective

Galectin-3's Role in the Diagnosis of Patients with Coronary Artery Disorders

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DESCRIPTION

Cardiovascular disease (CVD) continues to be the leading cause of death in the globe and presents significant treatment challenges. A continuing inflammatory response, which is essential for the development of this prevalent ailment, characterizes atherosclerosis, a degenerative vascular disease that affects all organ systems. Aorta and peripheral arteries are not exempt from this pathologic condition; however epicardial or extra- and intracranial arteries are typically the focus of atherosclerosis and associated problems. Standard assessments of cardiovascular risk factors, such as smoking, arterial hypertension, dyslipidemia, and diabetes mellitus alone, cannot identify the presence of atherosclerotic disease. Gal-3 belongs to the broad galectin family, which is involved in a variety of physiological and pathological processes, including inflammation and the development of fibrous tissue. Gal-3, a monocyte and macrophage active metabolite is important for cell growth, proliferation, apoptosis, differentiation, adhesion, and tissue regeneration. It is also involved in many physiological and pathological processes. Its predictive relevance for heart failure has long been established, as was already indicated, but it is also acknowledged as a potential marker for the assessment of atherosclerotic disease.

Gal-3 has been shown to be elevated in the myocardium and blood of people with Heart Failure (HF), and its level is related to the development and severity of HF. According to certain theories, Gal-3 may actively contribute to the onset of HF through myocardial fibrosis, and its suppression may be a new method of treating the condition. But as of now, there is no proof linking Gal-3 to the histologically confirmed myocardial fibrosis in HF patients. Additionally, a recent research conducted in endurance athletes found no correlation between plasma Gal-3 and myocardial fibrosis identified by cardiac magnetic resonance imaging. In end myocardial biopsies from individuals with HF of hypertensive origin, Gal-3 is connected to the production and deposition of fibrillar collagen types I and III. Additionally, connections between Gal-3 and circulating peptides that have been put up as indicators of the extracellular synthesis of mature procollagen type I, which forms fibrils, and collagen type I are known to occur. Procollagen Type I (PICP) and mature, fibril-forming collagen type III (from its precursor, procollagen type III) both have C-terminal propeptides. Investigations were also conducted on the Procollagen Type IIII's N-Terminal Propeptide (PIIINP). In order to achieve these goals, an invasive investigation was conducted to quantify the levels of Gal-3 in the myocardium and plasma, as well as its correlation with histological and molecular indicators of cardiac fibrosis.

No matter the patient's sex, age, LDL cholesterol level, or history of myocardial infarction, high levels of Gal-3 are linked to the existence of large carotid plaques. In individuals with a history of myocardial infarction, Gal-3 and carotid intima-media thickness are independent predictors of higher mortality. Similar to this, coronary angiography patients with low blood Gal-3 levels experienced fewer Cardiovascular (CV) events during short-term follow-up. The lack of carotid plaques was a stronger predictor of a decreased risk of CV events than low serum Gal-3 levels [16]. Greater Gal-3 levels were linked to higher CV mortality during long-term follow-up. Serum Gal-3 levels were considerably greater in patients with Acute Coronary Syndrome (ACS) compared to patients without ACS, as well as in patients with angiographically confirmed Coronary Artery Disease (CAD) compared to patients without CAD. Gal-3 was an independent predictor of CAD and was linked to Syntax score complexity. In the one-year follow-up MACE, patients with increased Gal-3 had a considerably higher MACE rate.

Despite receiving the best possible medication therapy and non-pharmacological treatments, patients with chronic coronary syndrome frequently have acute coronary syndrome as well as other adverse events (such as atrial fibrillation or heart failure). In order to provide the best possible pharmacological therapy, it is crucial to identify the patients in this category who most are at risk for MACE and who need further intervention. 17.8 ng/mL, 17.8 ng/mL-23.9 ng/mL, and > 23.9 ng/mL were designated as low, moderate, and high risk, respectively, for MACE in a research that included patients with heart failure. In individuals with suspected CAD, Gal-3 may be a helpful biomarker in diagnosing and evaluating the degree of coronary heart disease.

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Greater severity of coronary heart disease (three-vessel disease) might be anticipated in the group of people with confirmed CVD and increased Gal-3 serum levels. Gal-3 serum levels may also be a useful tool for identifying high-risk individuals with stable coronary artery disease, particularly those who would benefit most from early revascularization, whether by CABG or PCI, along with optimization of drug therapy to halt disease progression toward fibrosis, heart failure, or the emergence of MACE.