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The Role of Cardiovascular Biomarkers in Early Detection of Myocardial Fibrosis

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Abstract

Myocardial fibrosis is a pathological condition characterized by the excessive accumulation of extracellular matrix components particularly collagen within cardiac tissues. This structural remodeling contributes to the increased myocardial stiffness, impaired relaxation and the progression to heart failure. Early and accurate detection of myocardial fibrosis remains challenging due to the limitations of the conventional diagnostic methods such as imaging biopsy. However, advancements in cardiovascular biomarkers offer a promising avenue for early diagnosis and disease monitoring. This review comprehensively discusses the established biomarkers including galectin-3, soluble suppression of tumorigenicity-2 (sST2) and B-type natriuretic peptide (BNP)/N-Terminal proBNP (NT-proBNP) which have demonstrated significant clinical utility in detecting and prognosticating myocardial fibrosis. Moreover, Galectin-3 is a key mediator of fibrotic processes and an independent predictor of adverse cardiac remodeling. The sST2 reflects myocardial stress and provides rapid insights into acute changes in cardia function whereas, BNP and NT-proBNP correlate with ventricular strain and offer prognostic information on disease severity and therapeutic response. Additionally, emerging biomarkers such as microRNAs (miR-21 and miR-29) and collagen-derived peptides (PICP and PIIINP) have shown promising results in enhancing diagnostic accuracy and understanding fibrotic dynamics. The integration of these biomarkers into clinical practice may facilitate early diagnosis followed by personalized treatment strategies and improved patient outcomes in myocardial fibrosis.

Keywords: Acute coronary syndrome, HIV infection, coronary intervention, etc

Introduction

Myocardial fibrosis is a pathological condition characterized by the excessive accumulation of extracellular matrix components particularly collagen within cardiac tissues. This structural remodeling contributes to the increased myocardial stiffness, impaired relaxation and the progression to heart failure. Early and accurate detection of myocardial fibrosis remains challenging due to the limitations of the conventional diagnostic methods such as imaging biopsy. However, advancements in cardiovascular biomarkers offer a promising avenue for early diagnosis and disease monitoring. This review comprehensively discusses the established biomarkers including galectin-3, soluble suppression of tumorigenicity-2 (sST2) and B-type

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Pathophysiology of Myocardial Fibrosis

Activation of cardiac tissue's fibroblasts leads to myocardial fibrosis by excessive accumulation of extracellular matrix proteins, especially collagen types I and III [1]. The process occurs due to chronic inflammation, oxidative stress and neuro-hormonal activation mainly by the renin-angiotensin-aldosterone system (RAAS) and sympathetic nervous system [2]. Over time, myocardial fibrosis results in increased stiffness of the myocardium, altered contractile function followed by ventricular remodeling that predispose to heart failure [3]. Two primary forms of myocardial fibrosis exist; which include interstitial and replacement fibrosis. In interstitial fibrosis, collagen is laid between the fibers of the myocardium, while in replacement fibrosis, the space resulting after the death of the myocytes is occupied by the tissue of fibrosis [4]. It is difficult to establish an early stage of fibrosis using conventional diagnostic means. Cardiovascular biomarkers, however, might serve the purpose of an early marker of such changes [5].

Methodology

A systematic search strategy was conducted to identify relevant studies for the review. The preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines were followed to ensure transparency and rigor in the selection process. The search was performed across multiple electronic databases including Google Scholar (n=65), PubMed (n=35) and Science Direct n=10). However, no records were obtained from research registers. A total of 105 records were initially retrieved from these sources. After removing 55 duplicate and irrelevant records, 50 studies proceeded to the screening phase. During this stage, titles and abstracts were carefully examined resulting in the exclusion of 10 studies due to their irrelevance to the research objectives. The remaining 40 studies were then sought for full-text retrieval and all were successfully obtained without

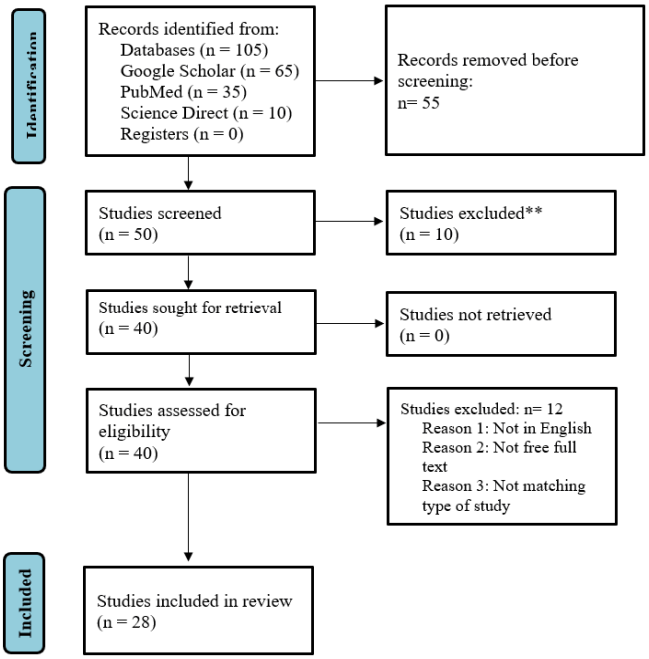

Figure: PRISMA flow diagram illustrating the search strategy used for the article selection.

any losses. In the eligibility assessment phase, the full texts of 40 studies were thoroughly reviewed based on pre-defined inclusion and exclusion criteria. Moreover, 12 studies were excluded for the following reasons: language barriers (n=1), no full-text access (n=2) and non-confirmatory with the study type required for the review (n=9). Ultimately, 28 studies met the eligibility criteria and were included in the final analysis. This rigorous selection process ensured that only-high quality and relevant studies were considered therefore enhancing the validity and reliability of the review findings.

Cardiovascular Biomarkers for Myocardial Fibrosis Detection

Galectin-3

Galectin-3 is a beta-galactoside-binding lectin has a role in inflammatory and fibrotic processes and thus presents a valuable biomarker of myocardial fibrosis [6]. It is secreted from macrophages due to inflammation and cellular stress by stimulating the activation of fibroblasts and deposition of extracellular matrix components into myocardial tissue [7]. The elevation of galectin-3 has been associated with more severe fibrosis, the progression of heart failure and poorer cardiovascular outcomes [8]. A 2021 meta-analysis found that galectin-3 is significantly predictive of future cardiac events, and that a higher concentration of galectin-3 portends a greater degree of fibrosis. Because galectin-3 is sensitive to early fibrosis, its role as an independent predictor of adverse cardiac remodeling in patients with heart failure or chronic hypertension is increasingly being acknowledged [9]. Besides being considered a marker of cardiac health, galectin-3 is now explored for its potential therapeutic value [10].

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Scientists are investigating ways to interfere with the action of galectin-3 so that fibrotic processes in the heart would be slowed down or even reversed [11]. Moreover, the associations between galectin-3 concentrations and other markers of fibrosis including BNP and sST2 will be analyzed to establish whether this set of markers would effectively enhance sensitivity and the prediction of outcomes. Apart from that, it also can be a biomarker and give hints regarding the possible risk for readmission into the hospital among the patients suffering from heart failure, thereby gaining additional importance in managing long-term outcomes in patients [12]. Moreover, studies have shown that galectin-3 can serve as a biomarker that differentiates between the various categories of heart failure; namely between heart failure with preserved ejection fraction (HFpEF) and heart failure with reduced ejection fraction (HFrEF). This is a difference of great significance since the pathophysiology and treatment approach differ between these two forms of heart failure. Targeting galectin-3 along with other markers may thus offer a possible avenue for clinicians to offer individualized treatments and achieve better outcomes in the patients diagnosed with fibrotic heart diseases [13].

Soluble Suppression of Tumorigenicity-2 (sST2)

Specifically, this is a soluble member of the interleukin-1 receptor family participating in fibrosis and inflammation [14]. sST2 is released upon cardiac stress, a reflection of myocardial stretch and injury, and very sensitive to acute changes in myocardial structure [15]. The association of high levels of sST2 with worse prognosis and severe fibrosis among patients with heart failure, so it is viewed as an important prognostic marker [16]. What separates sST2 from others is the ability to depict acute shifts in cardiac function, which means providing immediate insight into the state of fibrotic myocardial tissue [17]. This biomarker also adds information regarding active cardiac remodeling to other fibrosis markers with a high value for adverse cardiac events [18].An acute change can be detected by sST2 and therefore, these are particularly useful in emergency settings, where knowing what one could expect from cardiac function rapidly could be very critical. Additionally, this biomarker may provide insights into short term risk stratification guidance for clinicians to make rapid decisions on treatment. Other studies are now underway with an endeavor to know whether sST2 will indeed monitor the response to heart failure treatments, especially those focused at a reduction in myocardial stress and fibrosis [19]. Recent studies have also suggested that the evaluation of sST2 will be useful in identifying the individuals at risk for arrhythmia secondary to fibrosis changes in the myocardial tissue. This information is critically important because arrhythmias may lead to sudden death and are a major cause of death in patients with heart failure. Therefore, incorporating measurement of sST2 with assessment of cardiac biomarkers may optimize the patient's ability to predict and prevent adverse events and can be extremely useful as an additive to overall cardiovascular risk appraisal [20].

B-type Natriuretic Peptide (BNP) and N-terminal proBNP (NT-proBNP)

B-type natriuretic peptide (BNP) and its precursor, NT-proBNP, are peptides released in response to ventricular stretch and increased wall tension. Traditionally, used as marker of heart failure, they are also valuable in detecting myocardial fibrosis as elevated levels correlate with fibrosis severity and ventricular remodeling [21]. BNP is directly related to the severity of fibrosis in heart failure patients and can identify at high risk who might benefit from early therapeutic interventions. NT-proBNP is also widely used since it is a more stable form of BNP that has a longer half-life and is reliably correlated with ventricular strain and fibrosis [22].

These peptides not only assist in the diagnosis of heart failure but are also helpful in assessing the effectiveness of treatments [23]. Thus, alteration in the levels of BNP or NT-pro-BNP may better indicate whether the patient is responding to therapies intended to reduce cardiac strain and fibrosis. Other than that, BNP has been considered as a marker for inclusion into multi-marker panels to provide a better and more complete picture of the state of the heart, by combining it with specific fibrosis markers like galectin-3 and sST2 [24]. The prognostic values of BNP and NT-proBNP have been well established, but further research will be in improving specificity for the detection of fibrosis through further investigations of their interactions with other biomarkers [25]. This article concludes that clinicians can track the progression of fibrotic changes and possible cardiac events by following changes in BNP and NT-proBNP concentrations so that they may guide interventions and possible improvement in patients with myocardial fibrosis.

Emerging Biomarkers in Myocardial Fibrosis Detection

MicroRNAs

MicroRNAs like miR-21 and miR-29 are the non-coding RNAs that modulate the gene expression. These are highly involved in the pathways of proliferation, differentiation, and fibrotic processes in cells. Scientific reports of research have shown that the expression of miR-21 is increased in the process of myocardial fibrosis; this activates more fibroblasts along with an increase in the synthesis of collagen. miR-29 has been reported as an inhibitor of fibrosis, whose overexpression is inversely proportional to enhanced collagen deposition, both these microRNAs can complement the already established markers such as galectin-3 and BNP that provide more insight into changes occurring within fibrosis.

Collagen-Derived Peptides

Collagen synthesis and degradation release specific

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peptides including the C-terminal pro-peptide of type 1 collagen (PICP) and the N-terminal pro-peptide of type III collagen (PIIINP). Elevated levels of these peptides indicate the active collagen deposition which is a hallmark of myocardial fibrosis. Biomarkers associated with collagen provide a direct measure of fibrotic activity. Due to this correlation, they have been investigated as potential tools for monitoring the disease severity among patients with cardiac conditions.

Table 1: Emerging Biomarkers in Myocardial Fibrosis Detection

Biomarkers	Source	Role	Clinical Relevance
miR-21	Non-coding RNA	Increases fibroblast activity and collagen synthesis	Indicator of fibrosis progression
miR-29	Non-coding RNA	Inhibits fibrosis and collagen deposition	Potential therapeutic target for reversing fibrosis
Collagen-Derived Peptides	Collagen synthesis and degradation	Measures active fibrotic activity	Direct indicator of fibrosis severity

Conclusion and Future Directions

Myocardial fibrosis significantly contributes to cardiac dysfunction and the eventual progression to heart failure highlighting the need for early and accurate detection. Traditional diagnostic methods, while informative often identify fibrosis at advanced stages. This review focuses on the growing significance of cardiovascular biomarkers as a non-invasive and reliable indicators for detecting and monitoring myocardial fibrosis. Established biomarkers such as galectin-3, sSt-2 and BNP/NT-proBNP provide crucial insights into fibrotic activity, cardiac remodeling and patient prognosis. Furthermore, emerging biomarkers including specific microRNAs and collagen-derived peptides hold promise for refining diagnostic precision and advancing our understanding of fibrotic mechanisms. Future research should focus on validating these biomarkers in larger cohorts exploring their interactions and developing multi-marker panels to improve sensitivity and specificity. Utilizing these advanced biomarker profiles, clinicians can better predict disease progression, modified therapeutic interventions leading to improve clinical outcomes for patients with myocardial fibrosis.

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