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CASE REPORT

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Early detection of cardiac remodeling using biomolecular markers and imaging analysis in a young hypertensive patient: A case report

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ABSTRACT

Herbal background: Cardiac remodeling is a progressive structural and functional alteration of the heart often resulting from chronic hypertension or myocardial stress. Early identification of this process remains a clinical challenge, as conventional methods may not detect subtle molecular or anatomical changes in asymptomatic individuals.

Case summary: We report the case of a 29-year-old male patient with a recent diagnosis of mild hypertension who presented with nonspecific symptoms such as fatigue and occasional palpitations. While initial electrocardiography and basic echocardiographic assessments appeared unremarkable, advanced investigations revealed elevated levels of circulating biomarkers including NT-proBNP and Galectin-3. Further molecular profiling showed upregulation of extracellular matrix-related genes. Cardiac MRI identified early signs of left ventricular hypertrophy, confirming the onset of cardiac remodeling. Prompt initiation of pharmacological therapy and lifestyle modifications led to stabilization of biomarker levels and clinical improvement over a three-month follow-up period.

Conclusion: This case highlights the critical role of biomolecular diagnostics and advanced imaging in the early detection of cardiac remodeling, particularly in young patients with minimal symptoms. Incorporating biotechnological tools into routine cardiovascular screening may enable timely intervention and improved long-term outcomes.

KEYWORDS

Cardiac remodeling; Biomolecular markers; Hypertension; Cardiac MRI; NT-proBNP; Galectin-3

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Introduction

Cardiac remodeling is a compensatory response of the heart to chronic pressure or volume overload, resulting in structural and functional alterations such as ventricular hypertrophy, fibrosis, and changes in chamber geometry. These changes, although initially adaptive, can ultimately impair cardiac function and contribute to the development of heart failure [1].

One of the primary contributors to cardiac remodeling is hypertension, which induces mechanical stress on the myocardium. Over time, this stress can activate a cascade of molecular and cellular processes that lead to myocardial stiffening, reduced compliance, and eventual decline in systolic and diastolic function. Similarly, myocardial injury, such as that caused by ischemia, can also initiate remodeling by triggering inflammatory and fibrotic pathways [2].

Despite its clinical significance, a major challenge in managing cardiac remodeling is that most cases are diagnosed only after substantial damage has occurred, often when symptoms become pronounced or when traditional imaging tools reveal overt structural abnormalities. This delay in detection limits the effectiveness of early intervention strategies. With the emergence of biotechnology-driven diagnostics, particularly biomolecular markers such as NT-proBNP, Galectin-3, and gene expression profiling, it is now possible to detect early-stage remodeling at the molecular level well before anatomical changes are visible [3]. These tools offer a new frontier in preventive cardiology.

This case report aims to demonstrate the utility of combining molecular biomarkers with advanced imaging to detect early cardiac remodeling in a young hypertensive patient who presented with minimal clinical symptoms. The case highlights the potential for integrating biotechnological advancements into routine cardiovascular assessment to improve early diagnosis and treatment outcomes.

Case Presentation

The patient profile

A 29-year-old male presented to the cardiology outpatient clinic with no significant past medical history. He had a family history of hypertension and led a sedentary lifestyle, often experiencing high levels of occupational stress [4]. The patient had not been previously diagnosed with any cardiovascular conditions and was not on any medications.

Clinical symptoms

He reported intermittent fatigue and occasional palpitations over the previous month. There was no history of chest pain, dyspnea, syncope, or reduced exercise tolerance.

Initial workup

On examination, the patient's blood pressure was elevated at 148/92 mmHg, confirmed on two separate readings. Heart rate and other vitals were within normal limits. A 12-lead ECG showed normal sinus rhythm without evidence of hypertrophy

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or ischemia. A transthoracic echocardiogram revealed preserved left ventricular ejection fraction (LVEF 60%) and normal chamber dimensions. However, a mild increase in septal wall thickness (1.2 cm) was noted, considered borderline and non-diagnostic [5].

Advanced diagnostics

To explore possible early cardiac remodeling, the patient underwent a series of advanced diagnostic tests:

Blood biomarkers

- NT-proBNP: Mildly elevated at 176 pg/mL (normal <125 pg/mL).
- Galectin-3: Slightly above normal at 18 ng/mL (normal <17.8 ng/mL).

Gene expression profiling

Peripheral blood mononuclear cells were analyzed using quantitative real-time PCR (qRT-PCR). Results indicated upregulation of genes associated with early myocardial fibrosis and remodeling, notably COL1A1 and TGF-β1 [6].

Cardiac imaging

A cardiac MRI revealed early concentric left ventricular hypertrophy, with an increase in myocardial mass index. There was no late gadolinium enhancement, ruling out fibrosis or infarction [7]. The MRI findings supported subtle structural changes consistent with the early stages of remodeling.

Intervention

The patient was started on low-dose metoprolol succinate (25 mg once daily) to control blood pressure and reduce myocardial stress. He was advised to adopt lifestyle modifications, including a low-sodium diet, regular aerobic exercise, and stress management techniques such as mindfulness and yoga.

Outcome

At a three-month follow-up, the patient reported reduced fatigue and no further palpitations. Blood pressure had improved to an average of 124/80 mmHg, and repeat biomarker levels showed decreased NT-proBNP and Galectin-3 concentrations. A follow-up cardiac MRI showed no further structural progression. The intervention successfully stabilized the early remodeling process.

Investigative Focus: Biotechnology Angle

In this case, a combination of advanced biotechnological techniques was employed to detect early-stage cardiac remodeling that might have been missed using conventional diagnostics alone. The goal was to identify subclinical myocardial changes in a young hypertensive patient before irreversible structural damage could occur.

Techniques used

1. **Enzyme-linked immunosorbent assay (ELISA):** Blood samples were analyzed using ELISA to quantify protein biomarkers associated with cardiac stress and remodeling [8]. The patient's levels of NT-proBNP (176 pg/mL) and Galectin-3 (18 ng/mL) were found to be mildly elevated, suggesting increased myocardial strain and fibrotic activity despite a lack of clinical symptoms.
2. **Quantitative real-time PCR (qRT-PCR):** Peripheral blood mononuclear cells (PBMCs) were isolated and subjected to qRT-PCR analysis. This revealed upregulation of genes such as COL1A1 and TGF-β1, both of which are key regulators of extracellular matrix remodeling and fibrosis. These gene-level alterations provide a molecular signature of early cardiac remodeling [9].
3. **Advanced imaging analysis:** A cardiac MRI was conducted to obtain high-resolution images of myocardial structure and tissue composition. The imaging revealed subtle concentric left ventricular hypertrophy and an increased myocardial mass index, despite normal ejection fraction and no evident fibrosis on echocardiography [10]. While no AI-assisted imaging tools were used in this case, integration of such platforms could enhance image interpretation in future diagnostics.

Justification

Standard cardiovascular tests such as ECG and basic echocardiography often fail to detect early remodeling, especially in young and asymptomatic individuals [11]. By using biomolecular and genetic profiling, this case demonstrates the utility of biotechnology in identifying pre-clinical myocardial alterations, enabling timely therapeutic intervention.

Outcome interpretation

Thanks to this multi-layered biotechnology-based assessment, cardiac remodeling was detected before irreversible structural changes occurred. The patient was placed on an appropriate treatment plan, including lifestyle modifications and pharmacologic therapy, which led to clinical improvement and stabilization of biomarkers at the three-month follow-up [12]. The case exemplifies the power of early molecular diagnostics in preventive cardiology.

Discussion

Cardiac remodeling is a progressive, compensatory process that involves structural and functional changes in the myocardium in response to stress, such as chronic hypertension or myocardial injury. If left unrecognized in its early stages, remodeling can advance to irreversible ventricular dysfunction and ultimately heart failure [13]. The uniqueness of this case lies in the early detection of remodeling in a young, minimally symptomatic hypertensive patient, using a biotechnology-driven approach.

In many clinical scenarios, especially among younger individuals with no overt symptoms, standard diagnostics such as ECG and echocardiography may fail to detect early pathophysiological changes. In this case, echocardiography was largely unremarkable, showing preserved ejection fraction and borderline septal wall thickness. However, molecular biomarkers such as NT-proBNP and Galectin-3, which are sensitive indicators of myocardial stress and fibrosis, were mildly elevated. Their interpretation required careful correlation with imaging and genetic markers to support a preclinical diagnosis [14].

The application of quantitative real-time PCR (qRT-PCR) added further depth to the diagnostic process by detecting

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upregulation of COL1A1 and TGF-β1, genes associated with extracellular matrix remodeling [15]. These findings underscore the potential of gene expression profiling as an emerging tool in the early identification of cardiac stress, especially before irreversible fibrosis or chamber dilation occurs.

Furthermore, the use of cardiac MRI proved instrumental in confirming structural changes not visible on echocardiography. The identification of subtle left ventricular hypertrophy validated the biochemical and molecular findings, forming a complete and conclusive diagnostic picture [16].
This case also reflects the importance of early therapeutic intervention. By initiating beta-blocker therapy and implementing lifestyle changes, the patient's blood pressure was brought under control, and biomarker levels decreased significantly within three months [17]. Early diagnosis enabled targeted management, potentially preventing long-term adverse cardiac outcomes.

In comparison with existing literature, most reports of cardiac remodeling focus on advanced stages or post-myocardial infarction scenarios. This case highlights a critical diagnostic gap the absence of early identification strategies in young hypertensive patients, who may silently progress to structural heart disease. The integration of biotechnology-based diagnostics with imaging tools could help address this gap by enabling personalized and preventive cardiology [18].

Conclusions

This case underscores that early-stage cardiac remodeling can be effectively detected through the integration of molecular diagnostics and advanced imaging techniques, even in young, asymptomatic hypertensive patients. Standard clinical assessments alone may not reveal early myocardial changes, but the use of biomarkers such as NT-proBNP and Galectin-3, coupled with gene expression profiling and cardiac MRI, can uncover subtle yet clinically significant alterations.

The outcome in this patient demonstrates that early identification enables timely intervention, preventing disease progression and reducing long-term cardiovascular risk. Most importantly, this case illustrates the growing role of biotechnology in enhancing diagnostic precision, supporting a shift toward personalized and preventive strategies in cardiovascular care.

The successful integration of molecular and imaging-based tools into routine clinical practice offers a promising pathway to improve outcomes in at-risk patients—particularly those who may otherwise remain undiagnosed until irreversible damage occurs.

Patient Consent

Written informed consent was obtained from the patient for the publication of this case report, including all associated clinical information, diagnostic findings, and imaging data. The patient was made aware that identifying details would remain confidential and that all data would be used strictly for academic and scientific purposes.

Disclosure statement

The authors declare that they have no competing interests.

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