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Biochemical Study of Some Parameters in Patients with Angina in Karbala City

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Abstract

This biochemical study focuses on evaluating the temporal dynamics of four critical biomarkers—Visfatin, high-sensitivity Troponin (Troponin h.s), Angiotensin-Converting Enzyme (ACE), and Proteinase 3 (PR3)—in patients suffering from angina pectoris in Karbala City, Iraq. Angina, a key manifestation of coronary artery disease, presents in two main clinical forms: stable and unstable. These subtypes differ in pathophysiology, severity, and outcomes, thus demanding tailored diagnostic and prognostic approaches. The study aims to investigate how these biomarkers behave over time based on a series of blood samples collected at fixed intervals: baseline, 1 hours, 6 hours, and 12 hours. Data from 90 patients, equally distributed between stable and unstable angina and 30 male as control group. Findings reveal that Visfatin and PR3 show more aggressive and fluctuating patterns in younger patients with unstable angina, reflecting acute inflammatory and metabolic distress. Troponin h.s levels remained elevated in both age groups with unstable angina, indicating ongoing myocardial injury. Meanwhile, ACE levels demonstrated a sustained increase in older patients, especially those with unstable angina, suggesting chronic vascular stress. These insights underscore the importance of temporal biomarker monitoring over static assessments and highlight the potential for age-specific and phenotype-specific risk stratification. This study contributes valuable data toward personalized cardiovascular diagnostics and supports the integration of biochemical monitoring into routine clinical evaluation of angina patients.

Introduction

Angina pectoris is a clinical syndrome characterized by chest pain or discomfort resulting from myocardial ischemia, typically due to obstruction or spasm of the coronary arteries[1]. It remains a major global health concern, particularly in developing countries, where early detection and management often face systemic challenges. Angina presents in two primary forms: stable angina, which occurs predictably with exertion, and unstable angina, a more acute condition that may signal an impending myocardial infarction. Differentiating between these two forms is essential for guiding therapeutic strategies and improving patient outcomes[2].

Biochemical markers have emerged as essential tools in the early diagnosis and prognosis of cardiovascular diseases. Among these, Visfatin, Troponin h.s, Angiotensin-Converting Enzyme (ACE), and Proteinase 3 (PR3) offer insights into various aspects of the cardiovascular pathology. Visfatin is known for its role in inflammation and metabolic regulation, particularly in endothelial dysfunction[3]. Troponin h.s is a gold-standard biomarker for myocardial injury. ACE plays a critical role in the renin-angiotensin-aldosterone system, contributing to vascular tone and blood pressure regulation. PR3 is a serine protease involved in neutrophil activation and inflammation, increasingly linked to cardiovascular events[4]. This study is conducted in Karbala City, Iraq, and aims to assess the temporal dynamics of these four biomarkers in patients diagnosed with angina, stratified by age and clinical presentation (stable vs. unstable angina). While previous studies have investigated these markers in isolation, there remains a gap in understanding how their levels evolve over time in different patient subsets[5].

By analyzing biomarker fluctuations at baseline, 1 hours, 5 hours, and 10 hours after admission, this study offers a time-sensitive profile of cardiovascular stress and inflammation. Furthermore, integrating age-specific analysis allows for the identification of demographic patterns that could inform future risk stratification models. The goal is to enhance diagnostic accuracy and support individualized treatment strategies for angina patients through a biochemical perspective[6].

Materials and Methods:

Study Design and Setting:

This observational, cross-sectional study was conducted at hospitals and diagnostic laboratories in Karbala City, Iraq, between [insert time period if known]. Ethical approval was obtained from the relevant institutional review board, and informed consent was collected from all participants.

Participants:

A total of 90 patients clinically diagnosed with angina pectoris were enrolled in the study. The participants were divided equally into two groups: 45patients with stable angina, 45patients with unstable and 30 male as control group.

Exclusion criteria included: history of recent myocardial infarction, chronic inflammatory diseases, malignancies, renal failure, or immunosuppressive treatment. Sample Collection and Time Points: Blood samples were centrifuged, and serum was stored at -20°C until analysis.

Biochemical Analysis: The following biomarkers were quantified using standard ELISA (Enzyme-Linked Immunosorbent Assay) kits:

- Visfatin: Measured using a human-specific NAMPT/Visfatin ELISA kit
- Troponin h.s: Measured using a high-sensitivity cardiac troponin I assay
- Angiotensin-Converting Enzyme (ACE): Quantified using a colorimetric ACE activity assay
- Proteinase 3 (PR3): Determined using a human PR3-specific ELISA kit

All measurements were conducted in duplicate for accuracy, and assays were performed following manufacturer protocols.

Statistical Analysis: Data were entered and analyzed using SPSS version [X]. Descriptive statistics (mean ± standard deviation) were used to summarize the results. Comparative analysis between groups was performed using:

- ANOVA with repeated measures for temporal changes
- Independent t-tests for between-group comparisons (stable vs. unstable)
- Pearson's correlation for associations between biomarkers

A p-value of ($p \leq 0.05$) was considered statistically significant.

Results:

Figure (1) displays the results developed for serum Troponin levels in patients groups with control group, raise significantly ($p \leq 0.01$) in Troponin at three different time points (each 6 hour), in patients compared with control group.

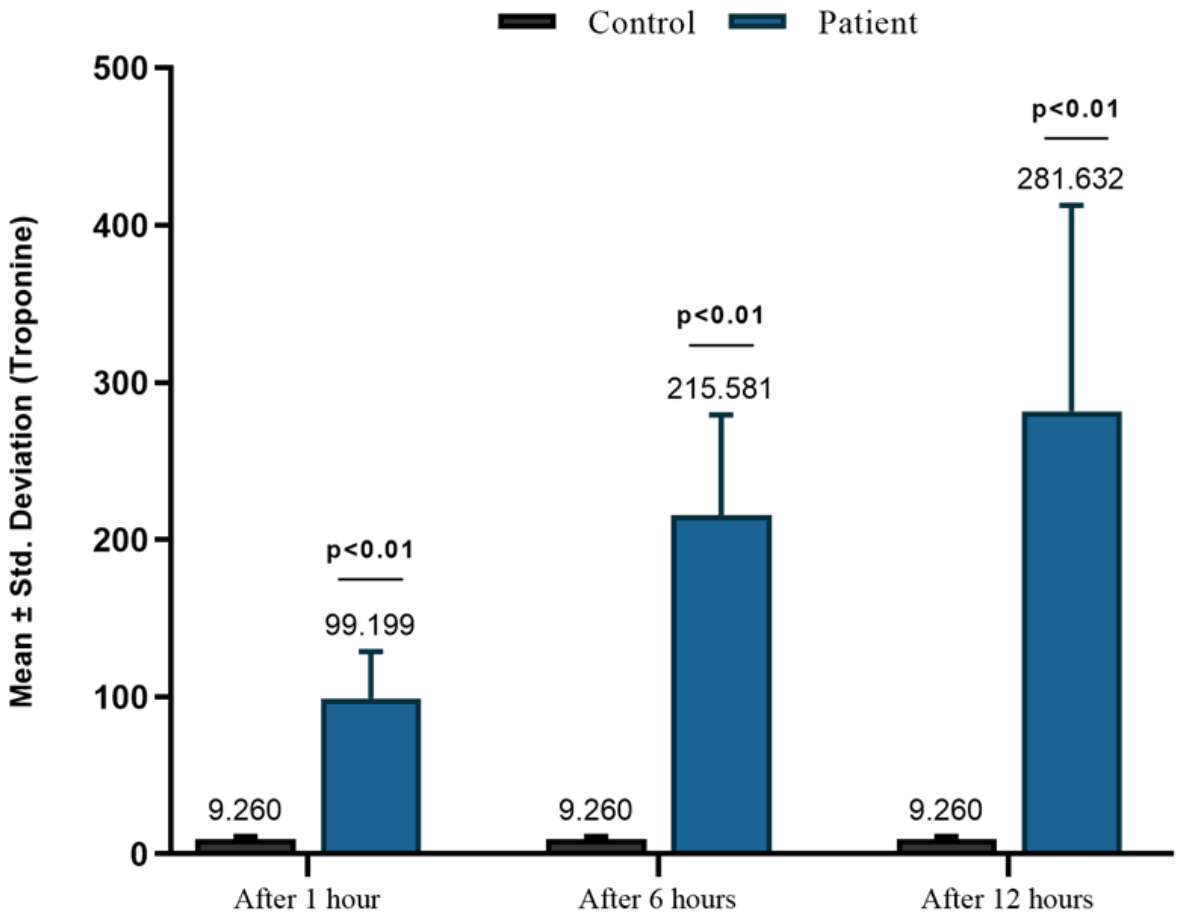

Figure1 : The Comparison between Troponin level in patients and their controls at three different time

Figure (2) shows the results assimilated for serum visfatin levels in patients groups with control group, elevation significantly ($p \leq 0.01$) in visfatin of patients in comparison to control at three different time points.

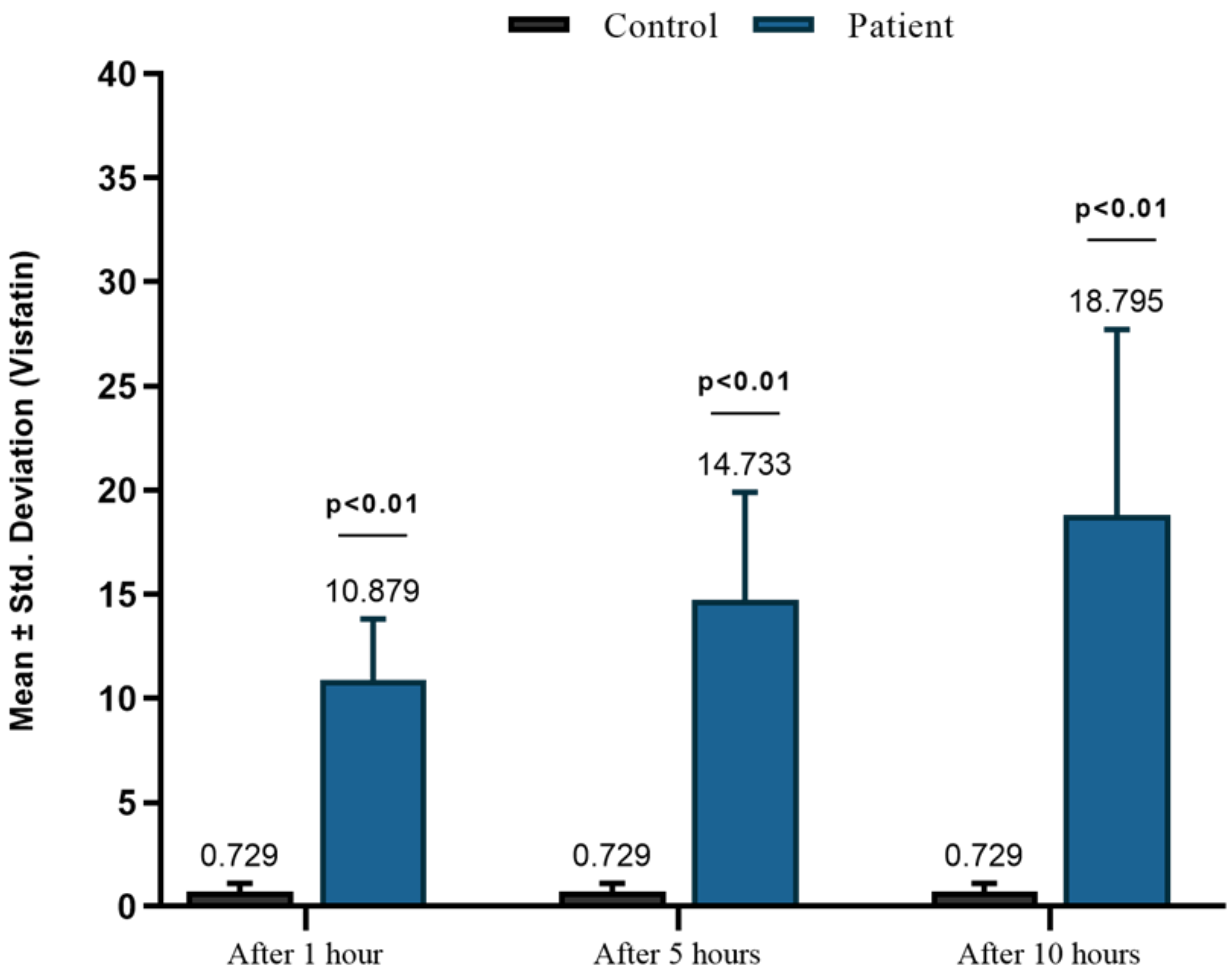

Figure2: The Comparison between Visfatin level in patients and their controls at three different time points.

Figure (3) displays the results acquired for serum ACE levels in patients groups with control group, raise significantly ($p \leq 0.01$) in ACE of patients in comparison to control at three different time points.

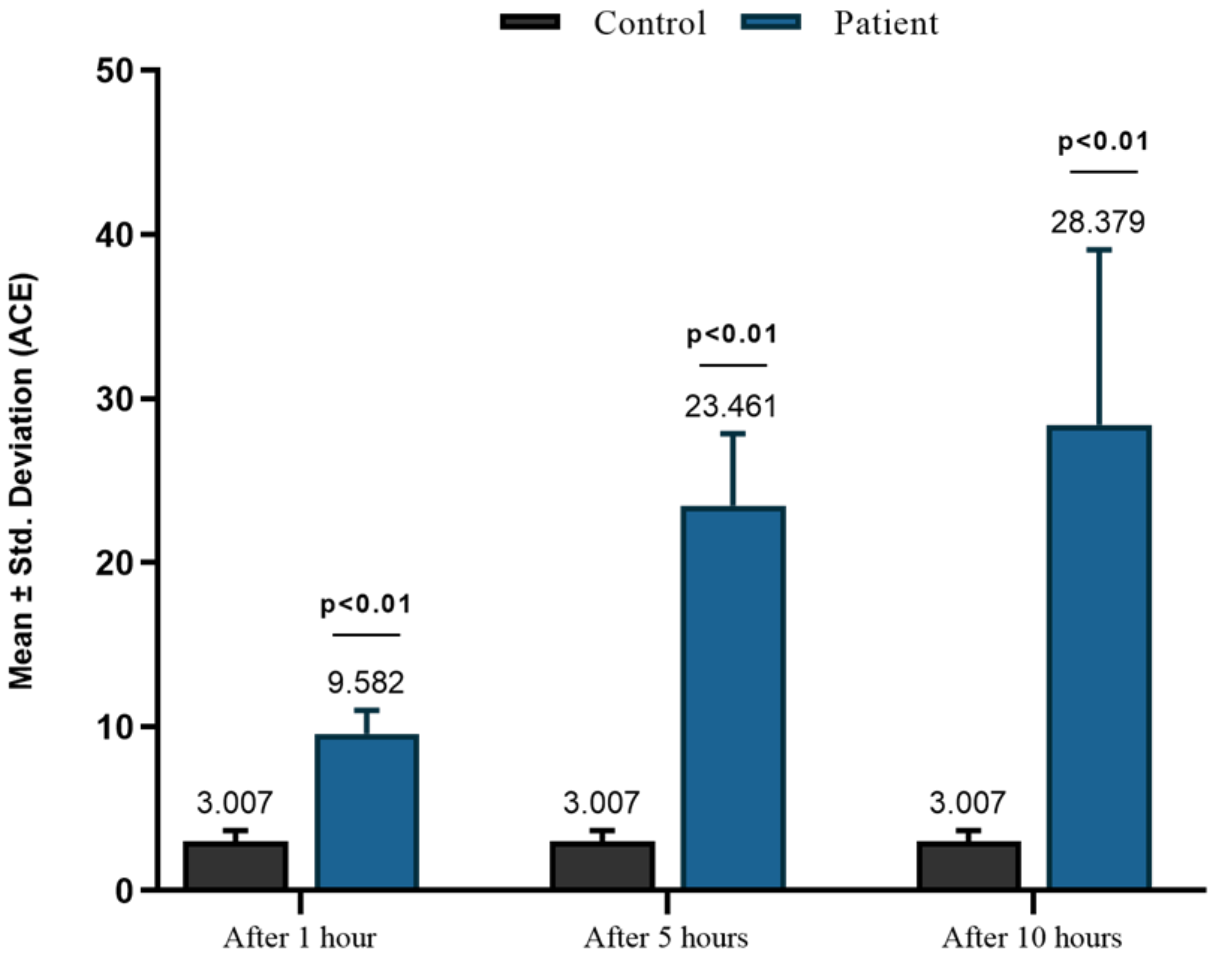

Figure3 : The Comparison between ACE level in patients and their controls at three different time points.

Figure (4) demonstrations the results acquired for serum PR3 levels in patients groups with control group, raise significantly ($p \leq 0.01$) in PR3of patients in comparison to control group at three different time points.

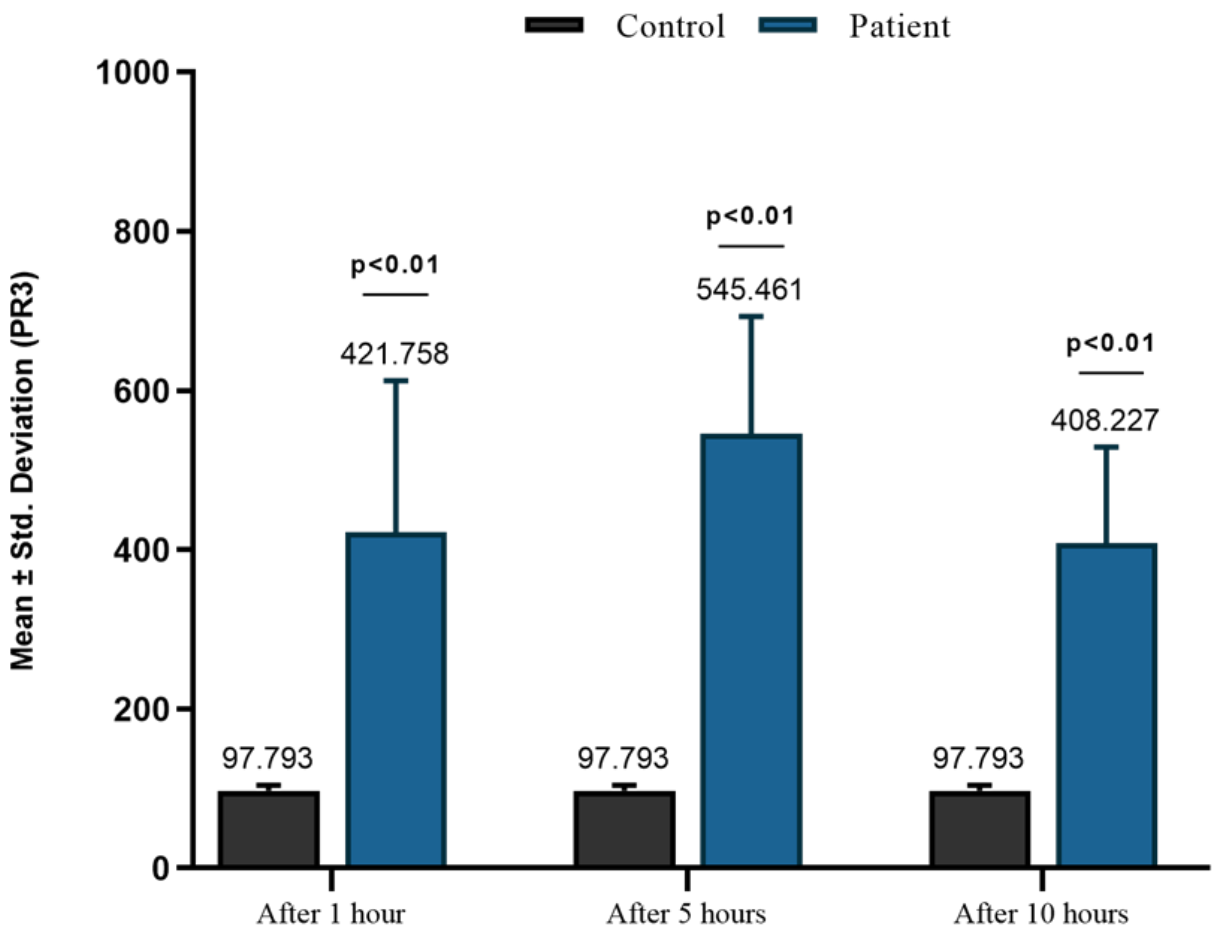

Figure 4: The Comparison between PR3 level in patients and their controls at three different time points.

Discussion

The findings confirm that PR3, ACE, and visfatin are upregulated in angina, particularly in unstable cases. Their elevation aligns with the pathophysiological processes of endothelial injury, oxidative stress[6], and inflammatory cascade activation. The temporal analysis of biomarker expression offers insight into their potential for early detection and subclassification of angina types[7].

Troponin, while remaining a robust and widely used biomarker for myocardial infarction, demonstrated less utility in distinguishing between stable and unstable angina due to its non-specific elevation in both conditions[8]. In contrast, PR3 and ACE levels exhibited earlier and more significant elevations in unstable angina, indicating their superiority in capturing the acute inflammatory and vascular changes associated with this more severe subtype[9]. The ability of PR3 and ACE to track the temporal dynamics of inflammation provides clinicians with additional tools to assess the progression and severity of angina[10].

Visfatin, an adipocytokine involved in systemic inflammation and metabolic dysregulation, showed consistent elevation in both angina types. Its lack of specificity limits its use in differentiating between stable and unstable forms, but its sensitivity to ischemic stress supports its potential role as a general early indicator[11]. This aligns with previous studies highlighting visfatin's role in endothelial dysfunction and plaque development[12].

Conclusion

This study underscores the diagnostic utility of PR3, ACE, and visfatin in angina pectoris, with PR3 and ACE showing high potential for early and subtype-specific detection

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