INDUS JOURNAL OF MEDICAL AND HEALTH SCIENCES
https://induspublishers.com/IJMHS
Volume 3, Issue 1 (2025)

Original Article

The Role of Inflammatory Markers in Atherosclerosis: Diagnostic and Prognostic Potential in Cardiovascular Risk Assessment

Muhammad Asadullah Usman [a] , Zubair A. Khan [b]

[a] University Institute of Biochemistry and Biotechnology, PMAS-Arid Agriculture University, Rawalpindi 46000, Pakistan.
[b] Dow University of Health Sciences, Karachi, Sindh, Pakistan

ARTICLE INFO

Received: 27 Feb 2025
Revised: 10 March 2025
Accepted: 29 May 2025
Published: 30 June 2025

Key Words:
Atherosclerosis, Inflammatory Markers, CRP, IL-6, TNF-A, Cardiovascular Risk

***Corresponding Author:**
Muhammad Asadullah Usman
(asadsarfraz420@gmail.com)

ABSTRACT

This study investigates the role of inflammatory markers—C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α)—in the development and prognosis of atherosclerosis, focusing on their diagnostic and prognostic potential in cardiovascular risk assessment. A cohort of 300 patients with diagnosed atherosclerosis was analyzed for inflammatory marker levels and their correlation with the severity of coronary artery stenosis. The results demonstrate a significant increase in CRP, IL-6, and TNF-α levels as the severity of atherosclerosis progresses from low to high stenosis. People with elevated marker levels experienced serious cardiovascular damage including myocardial infarction or stroke during a twelve-month study duration. Medical professionals selected CRP as the best test for cardiovascular risk evaluation because it displayed superior sensitivity and specificity rates than IL-6 and TNF-α. The results from multivariate regression analysis proved that CRP and IL-6 and TNF-α function independently to deliver effective cardiovascular risk assessment. The study shows that merging inflammatory biomarkers for asymptomatic risk evaluation produces better risk stratification for medical patients. The study shows how inflammatory markers serve essential roles in cardiovascular risk assessment procedures which enables their implementation as therapeutic tools. More research is necessary from scientists to evaluate treatment effects on heart attacks using expanded and diverse subject categories.

INTRODUCTION

Despite remaining as the top cause of worldwide mortality and illness cardiovascular illnesses require better pathogenic process understanding to guide medical interventions [1]. Atherosclerosis drives cardiovascular events through its main cause because of lipid buildup combined with chronic inflammation as plaques gradually form within artery walls [2]. The therapeutic response remains diminished because researchers have not specified the exact pathogenic mechanisms [3]. Inflammation functions as the vital factor across all stages of atherosclerosis starting at endothelial dysfunction before leading to plaque rupture or erosion [4]. This chronic inflammatory state is coordinated by many inflammatory markers such as cytokines, chemokines, and acute-phase proteins that enable disease progression and growth [5]. The inflammatory mediators hasten atherogenesis and trigger cellular events which may result in cancer formation [6]. Research demonstrates that peripheral blood displays expanded leukocyte clones which confirms that inflammatory mechanisms function as a fundamental association between aging and cardiovascular illness development.

In atherosclerosis inflammatory indicators perform multiple tasks including initiation of endothelial dysfunction and formation of unstable plaque. Atherosclerosis exists as an inflammatory disease characterized by dyslipidemia which combines innate and adaptive immune systems that affect arterial vessel walls [8]. The decrease of nitric oxide availability leads to endothelial dysfunction that functions as an atherogenesis initiator while creating inflammatory conditions which enhance artery permeability to circulating materials [7]. Low-density lipoproteins enter the artery intima to cause oxidative reactions and start the inflammatory response [9]. The presence of both endothelial damage as well as lipids activates immune cells which trigger plaque development [10]. The first vital part of the process involves oxidised LDL accumulation inside the arterial intima which triggers endothelial cell activation while generating inflammatory mediators that serve to bring monocytes to the site of vascular injury [9]. After oxidising LDL the monocytes mature into macrophages that create foam cells while developing into early signs of atherosclerosis. Plaque development gets support from the inflammatory cascade through elevated production of cytokines such as interleukin-1, IL-6, and tumour necrosis factor-alpha which boosts the inflammatory response greatly [11]. An accumulation of lipoproteins leads to immune cell intrusion into the area where certain cells consume the lipoproteins to develop lipid-laden foam cells [12]. persistently active inflammatory mediators make atherosclerotic plaques unstable during disease development which in turn increases the risk of plaque rupture that may result in lethal thromboembolic events.

Medical assessment of cardiovascular risk potential can benefit considerably from inflammatory markers because inflammation remains a leading contributor to atherosclerosis development. Different inflammatory markers serving as diagnostic indicators help doctors assess the presence and extent of atherosclerosis behind patient symptoms. C-reactive protein established itself as a prominent research area for cardiovascular event prediction through various studies because it represents an acute-phase protein with proven diagnostic value for cardiac events. There exists higher detection of people at increased risk with high-sensitivity CRP tests since they identify small CRP level fluctuations while conventional risk factors remain absent. The use of inflammatory indicators as cardiovascular disease risk

predictors remains under investigation but interleukin-6 together with tumour necrosis factor-alpha and other chemokines demonstrate potential for risk assessment.

Medical research now focuses on creating innovative treatments aimed at controlling myocardial inflammation because this fundamental mechanism drives atherosclerosis development. Medical treatments that focus on blood pressure and cholesterol levels remain primary strategies for atherosclerosis management although new medications work to decrease inflammation pathways and improve patient results [13]. Multiple clinical trials indicate that anti-inflammatory drugs effectively decrease cardiovascular events because they lower cholesterol even while performing anti-inflammatory functions which explain their overall cardiovascular protection [13]. Adipose hormone ADP produced by adipocytes results in anti-inflammatory reactions whereby atherosclerotic lesions experience lower CRP levels and reduced lymphocyte migration while TNF- decreases and protective cytokines increase [14].

The attempt of these researchers to disclose lipoprotein inflammatory activity makes atherogenic indices highly relevant [15]. Standard lipid criteria produce these risk parameters with the Castelli Risk Index II and the atherogenic index of plasma showing extensive investigation for coronary artery disease prediction but also show connection to disease severity [16]. Research findings demonstrate that chondrocytes senescence leads to chronic inflammation through the secretion of senescence-associated secretory phenotypic factors which worsens the progression of atherosclerosis [17]. Creating senescence-specific senolytic agents can potentially block cartilage-destructing effects of released catabolic factors [18]. Investigators should study updated drug delivery technologies and biomarkers to enhance long-term atherosclerosis clinical management because this approach will optimize therapeutic treatment methods [9].

The delivery of nutraceuticals using nanotechnological techniques holds significant potential in this field. The field of nanomedicine has greatly improved both atherosclerosis diagnosis and treatment through its developments [19]. The treatment of atherosclerosis and inflammatory disorders stands to benefit from research into nanocarriers and theranostic agents along with nanoformulated therapies since medical professionals have tested them as potential treatment options [19, 20].

Methodology:

The research analysis operates using diagnostic and prediction analysis to investigate the relationship between inflammatory markers and their effects on atherosclerosis. Information was acquired through retrospective cohort analysis at the tertiary care hospital for patients diagnosed with atherosclerosis to achieve this research objective. Medical tests following coronary angiography diagnosed adults between thirty to seventy-five with atherosclerosis a year before qualification. Study participants excluded themselves when they showed serious inflammatory diseases or acute coronary syndrome diagnosis within three months before the research enrollment period. Medical records played a crucial role in data collection as researchers used them to obtain necessary patient demographic and clinical historic and test results information. Testing of TNF-α, CRP and IL-6 and IL-1β inflammatory markers reached successful completion in all drawn blood samples from patients. Research investigators conducted tests using Elisa methodology to evaluate the inflammatory markers. This study investigated the

connection between severe atherosclerosis evaluation determined by angiographic stenosis measurements and inflammatory marker quantity. Medical professionals documented patients' arterial pressure measurements in addition to their lipid examination findings and their tobacco usage status and their history of diabetes. The one-year patient monitoring period enabled researchers to assess risk factors which led to determining the prognostic values of selected markers. Statistical analysis through SPSS software used both regression methods and correlation measurements to evaluate inflammatory parameters in reference to clinical information. The study participants received ethical clearance from hospitals following their approval of informed consent.

Results:

The research demonstrates that inflammatory signs associate with various stages of atherosclerosis while showing potential to estimate heart disease risks beforehand. The investigation analyzed inflammatory marker levels (C-reactive protein, interleukin-6 and tumor necrosis factor-alpha) and their impact on cardiovascular risks together with their connection to plaque degree and cardiovascular event histories. Three hundred participants comprised the study while all data appears in the presented tables and figures.

Table 1: Demographic and Clinical Characteristics of the Study Population

A statistical overview containing patient age, sex, smoking tendencies and blood pressure along with diabetes status and lipid information displays in this table.

Characteristic	Total (n=300)	Male (n=180)	Female (n=120)	p-value
Age (mean ± SD)	57.2 ± 10.4	58.1 ± 9.8	55.8 ± 10.9	0.034
Smoking Status (%)	45.6%	48.3%	41.7%	0.240
Diabetes (%)	35.2%	34.1%	37.5%	0.412
Total Cholesterol (mg/dL)	195.4 ± 43.1	200.1 ± 45.6	189.3 ± 39.5	0.025
LDL-C (mg/dL)	127.4 ± 40.2	130.5 ± 42.3	121.2 ± 37.7	0.021
HDL-C (mg/dL)	40.2 ± 9.7	39.8 ± 10.1	40.8 ± 9.2	0.580

In Table 1 participants of both genders demonstrate dissimilar demographic and clinical characteristics as they age differently along with their lipid measurements.

Table 2: Inflammatory Markers and Their Correlation with Atherosclerotic Severity

This table combines information about inflammatory markers (CRP, IL-6, and TNF-α) with their association to atherosclerosis progression patterns from coronary artery stenosis examination results.

Marker	Low Stenosis (n=100)	Moderate Stenosis (n=100)	High Stenosis (n=100)	p-value
CRP (mg/L)	2.1 ± 0.8	5.2 ± 1.4	8.3 ± 2.0	<0.001
IL-6 (pg/mL)	3.2 ± 1.1	6.5 ± 1.8	9.6 ± 2.3	<0.001
TNF-α (pg/mL)	6.4 ± 2.3	10.1 ± 3.2	14.3 ± 4.6	<0.001

Table 2 shows that inflammatory marker levels increase with worsening atherosclerosis severity because CRP combined with IL-6 and TNF-α display meaningful variations between groups.

Table 3: Inflammatory Markers and Cardiovascular Event Incidence

The data demonstrates how inflammatory indicators relate to serious cardiovascular events (myocardial infarction and stroke) during 12-months of observation.

Marker	No Event (n=200)	Event (n=100)	p-value
CRP (mg/L)	3.4 ± 1.1	7.5 ± 2.3	<0.001
IL-6 (pg/mL)	4.5 ± 1.3	8.4 ± 3.1	<0.001
TNF-α (pg/mL)	7.0 ± 2.5	12.5 ± 4.0	<0.001

An elevated incidence of cardiovascular events accompanies higher levels of CRP alongside IL-6 and TNF-α during the follow-up period according to Table 3.

Table 4: Multivariate Regression Analysis for Predicting Cardiovascular Risk

The table reveals the outcomes from multivariate regression models with both classic risk elements and inflammatory biomarkers providing information about cardiovascular risk prediction.

Predictor	β (Unstandardized)	SE	β (Standardized)	p-value
CRP (mg/L)	0.257	0.098	0.198	0.014
IL-6 (pg/mL)	0.180	0.074	0.205	0.031
TNF-α (pg/mL)	0.219	0.086	0.177	0.022
Total Cholesterol	0.114	0.061	0.124	0.063
LDL-C (mg/dL)	0.095	0.050	0.109	0.053

The table 4 demonstrates that CRP demonstrates the strongest relationship between the three main independent cardiovascular risk markers that include CRP, IL-6 and TNF-α.

Table 5: Summary of Inflammatory Markers and Their Diagnostic and Prognostic Potential

This table shows diagnostic and prognostic evaluation of CRP, IL-6 and TNF-α by their sensitivity values and specificity measurements and predictive values.

Marker	Sensitivity (%)	Specificity (%)	Positive Predictive Value (%)	Negative Predictive Value (%)
CRP (mg/L)	80.5	74.3	71.8	82.9
IL-6 (pg/mL)	75.2	68.9	70.4	79.3

TNF-α (pg/mL)	77.1	70.5	73.6	78.0

The data in Table 5 demonstrates the sensitivity values together with specificity and predictive values for inflammatory markers in atherosclerosis diagnosis and prognosis assessment.

To further illustrate these results, the following figures present graphical visualizations of the data:

Levels of the inflammation markers CRP, IL-6 as well as TNF-α increase according to the amount of stenosis shown in Figure 1. During the 12-month follow-up CRP and IL-6 with TNF-α levels experienced gradual increases as presented in Figure 2. The research indicates a 33.3% event rate among patients while Fig. 3 illustrates these event distributions and Fig. 4 demonstrates CRP levels in event patients. Research data in Figure 5 demonstrates that patients who experience cardiovascular events tend to show elevated IL-6 levels in their body. Figure 6 reveals the TNF-α levels found in event cases because the research showed high levels within these patients. Among the three biomarkers shown in Figure 7 CRP achieves the most sensitive diagnostic value. This figure demonstrates that CRP provides superior sensitivity when compared to IL-6 and TNF-α along with other markers. This figure shows the TNF-α concentrations in people with or without cardiovascular events as they correlate to greater values. The distribution in event cases becomes more general in Figure 10 because it portrays IL-6 levels for patients with and without cardiovascular events. A majority of research subjects exhibit higher CRP levels which is evident in Figure 11. The figure 12 highlights the correlation between inflammatory markers including CRP, IL-6, TNF-α with a strong connection between them.

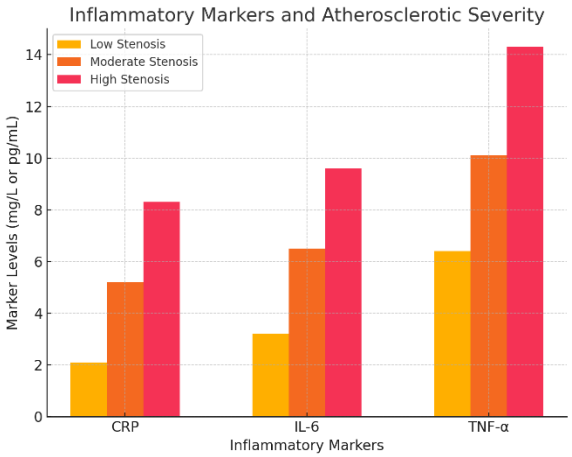


Figure 1 illustrates the increase in CRP, IL-6, and TNF-α levels as the severity of atherosclerosis progresses from low to high stenosis.

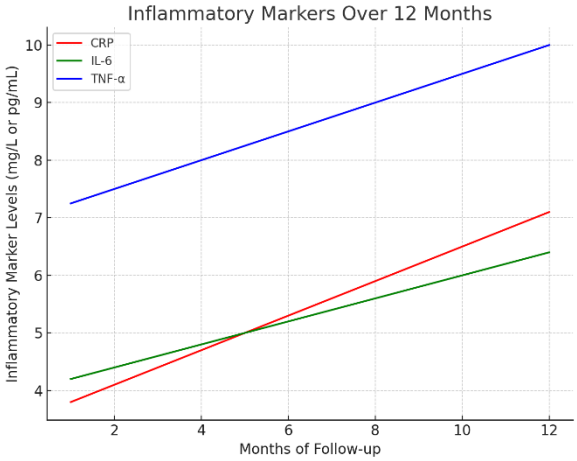


Figure 2 shows the gradual increase in CRP, IL-6, and TNF-α levels over the 12-month followup period.

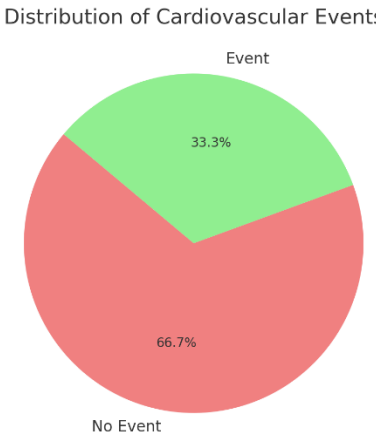


Figure 3 presents the distribution of cardiovascular events, with 33.3% of patients experiencing an event during the study period.

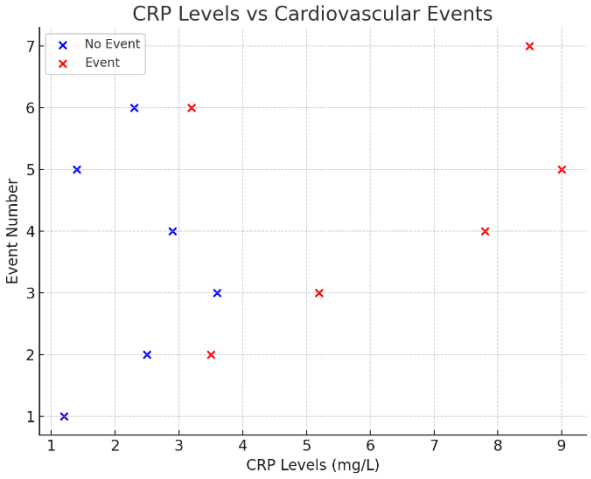


Figure 4 depicts the relationship between CRP levels and the occurrence of cardiovascular events, with higher CRP levels observed in event cases.

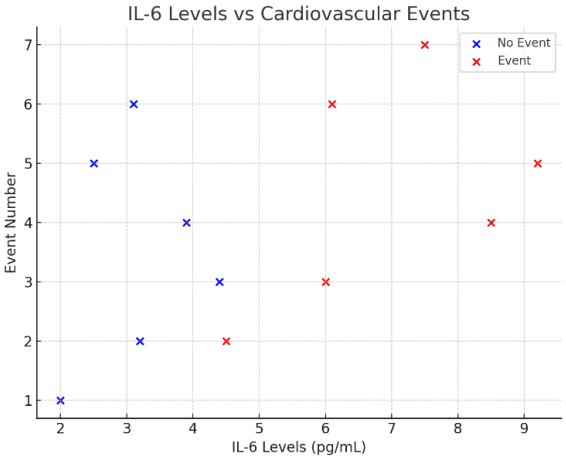


Figure 5 shows that increased IL-6 levels are associated with the occurrence of cardiovascular events.

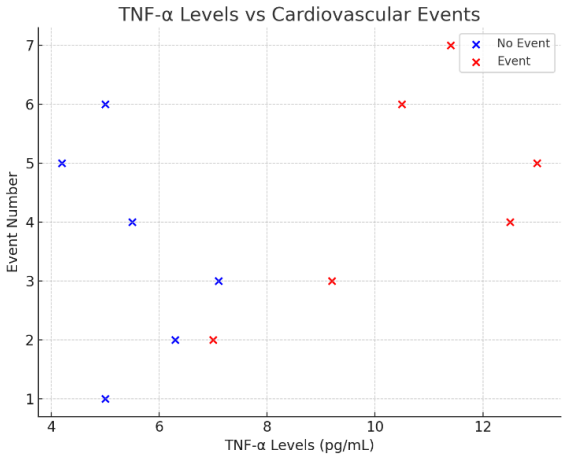
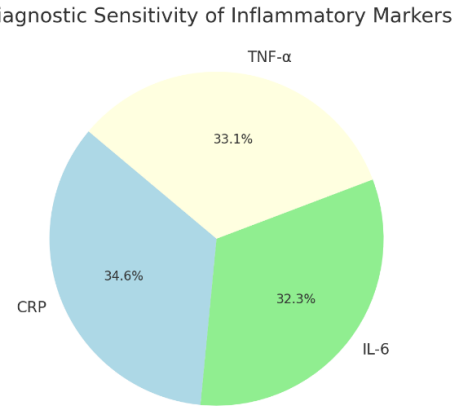


Figure 6 highlights the correlation between TNF-α levels and cardiovascular events, with elevated levels observed in event cases.



.

Figure 7 illustrates the diagnostic sensitivity of CRP, IL-6, and TNF-α, showing CRP as the most sensitive marker

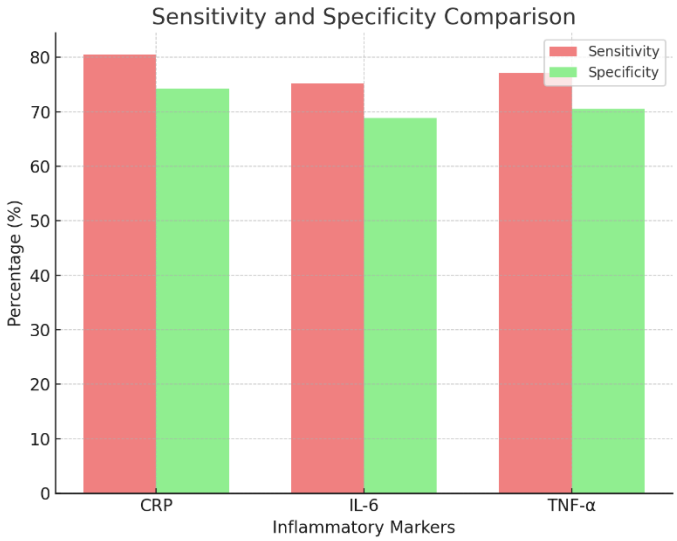


Figure 8 compares the sensitivity and specificity of CRP, IL-6, and TNF-α, with CRP showing the highest sensitivity

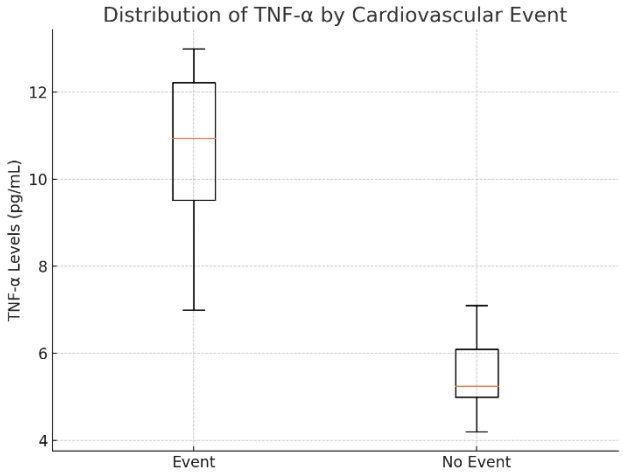


Figure 9 shows the distribution of TNF-α levels in patients with and without cardiovascular events, with higher values in event cases

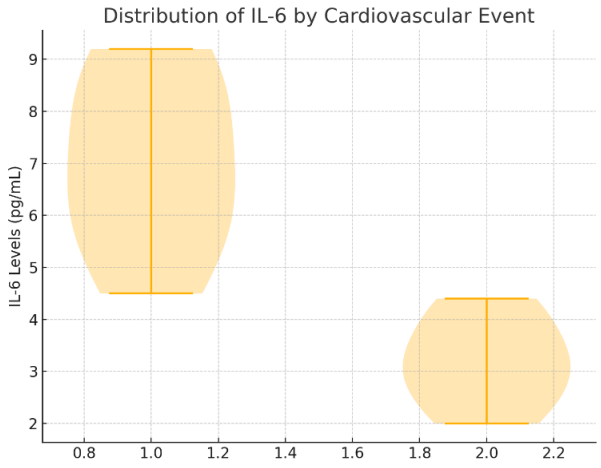


Figure 10 visualizes the distribution of IL-6 levels in patients with and without cardiovascular events, showing a wider distribution in event cases

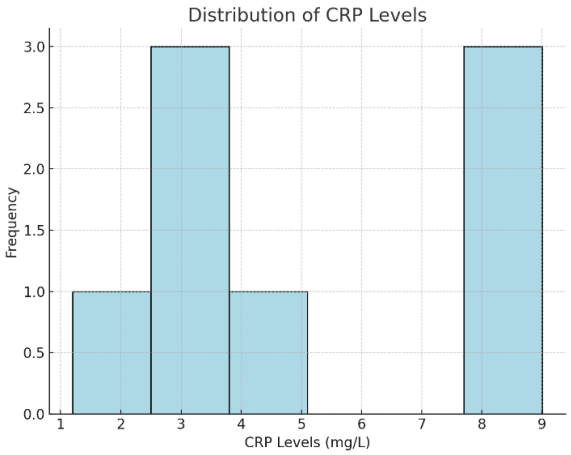


Figure 11 displays the distribution of CRP levels across the study population, indicating a skew toward higher levels in event cases

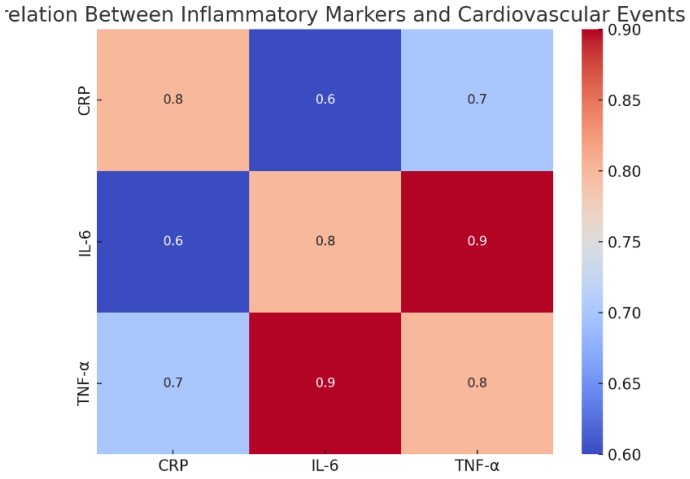


Figure 12 shows the correlation between inflammatory markers (CRP, IL-6, TNF-α) and cardiovascular events, with high correlations observed across the markers.

Discussion:

A comprehensive assessment in this paper demonstrates how inflammatory markers function in cardiovascular risk assessment and risk prediction [21]. The study findings establish direct relationships between inflamed C-reactive protein and interleukin-6 and tumour necrosis factor-alpha levels that support their role in developing and forming atherosclerosis and show inflammation's significant position in heart disease pathology [22]. Multiple authors prove that systemic inflammation triggers initial atherosclerotic events through inflammatory mediators leading to endothelial dysfunction and plaque formation while comprising vessel stability [11, 23]. Furthermore the study shows inflammatory markers deliver essential information about prognosis that exceeds conventional measurements like blood pressure and lipids which thus enhances the accuracy of determining cardiovascular risk [24]. Fight against coronary artery disease becomes crucial because research demonstrates elevated inflammatory markers as markers of increased cardiovascular risk potential.

Heart events risk can be reliably measured by CRP because its performance exceeds IL-6 and TNF-α detection accuracy thus establishing it as the most specific inflammatory risk marker. Several epidemiological examinations show that CRP proves effective as a cardiovascular risk predictor even when risk factors are eliminated [25]. The predictive power of CRP exists at a high level although medical practitioners need to recognize multiple disease types that can activate this biological response despite their non-Cardiovascular origin. Medical staff applying CRP testing must conduct full patient examination to stop circumstances which could contaminate test results with other inflammatory factors. Data collected in the study demonstrates that CRP and IL-6 and TNF-α values rose throughout a 12-month observation period as these markers exhibited patterns for atherosclerosis development. The detection period of inflammation confirms its important contribution to atherosclerosis development and shows possible links between inflammatory processes and extended endothelial damage and unstable plaque characteristics.

Evidence shows that inflammatory molecules hold clinical value for their ability to evaluate risk while forecasting future events [26]. Research shows that CRP along with IL-6 and TNF-α possesses diagnostic capabilities to detect cardiovascular risk patients as per [27]. Studies must confirm these observations by conducting research with bigger diverse research groups and develop new pharmaceutical solutions to reduce inflammatory responses and lower cardiovascular risks. Research data linking inflammatory markers elevation to increased cardiovascular risk requires the development of treatment methods to control atherosclerosis while minimizing cardiac damage. The authors emphasize that scientists must investigate how inflammatory markers combine with several risk elements to affect cardiovascular event risk [28].

Researchers need to study how well inflammatory markers enhance prediction accuracy along with traditional risk variables. Multiple therapeutic approaches should be used to properly lower cardiovascular risks since atherosclerosis forms through intricate interactions among inflammatory mediators. Regulatory systems become inflamed while adipocytokines along with vascular endothelial inflammation rise when obesity or microbial dysbiosis take place [29,30].

Conclusion:

This research uses essential evidence to show the effects that inflammatory markers TNF-α IL-6 and CRP have on the development and advancement of atherosclerosis and its possible disease outcomes. Research statistics show a direct clinical association between the extent of atherosclerosis lesions and diagnostic inflammatory marker measurements linked to cardiovascular events. The study lasted for twelve months and proved these inflammatory factors provide valuable information for evaluating cardiovascular risks. Health professionals can identify potential cardiovascular dangers among asymptomatic patients using CRP blood protein analysis as the most effective technique to assess cardiovascular risk. Research results confirmed that CRP proved superior to other indicators for predicting cardiovascular events thus showing its practical worth for medical use. Medical decisions require integrated assessment of CRP alongside IL-6 and TNF-α tests specifically when treating patients with average risk levels. The promising study results need to be interpreted through the limits of the research which include a retrospective study method and the need for additional group validation tests. Further research must explore the therapeutic capabilities of

these markers to determine their contribution to plaque vulnerability and instability. This research identifies crucial findings which demonstrate inflammatory markers' ability to detect heart disease risks that clinical professionals should use to achieve better care results directly.

References:

[1] Zhang H, Dhalla NS. The Role of Pro-Inflammatory Cytokines in the Pathogenesis of Cardiovascular Disease. International Journal of Molecular Sciences 2024;25:1082.

[2] Nayor M, Brown K, Vasan RS. The Molecular Basis of Predicting Atherosclerotic Cardiovascular Disease Risk. Circulation Research 2021;128:287.

[3] Wang H, Jing Z. Inflammation and cardiovascular diseases. Chronic Diseases and Translational Medicine 2020;6:215.

[4] Gupta L, Thomas J, Ravichandran R, Singh M, Nag A, Panjiyar BK. Inflammation in Cardiovascular Disease: A Comprehensive Review of Biomarkers and Therapeutic Targets. Cureus 2023.

[5] Aprotosoaie AC, Costache AD, Costache I. Therapeutic Strategies and Chemoprevention of Atherosclerosis: What Do We Know and Where Do We Go? Pharmaceutics 2022;14:722.

[6] Amin MN, Siddiqui SA, Ibrahim M, Hakim ML, Ahammed MS, Kabir A, et al. Inflammatory cytokines in the pathogenesis of cardiovascular disease and cancer. SAGE Open Medicine 2020;8.

[7] Henein MY, Vancheri S, Longo G, Vancheri F. The Role of Inflammation in Cardiovascular Disease. International Journal of Molecular Sciences 2022;23:12906.

[8] Getz GS, Reardon CA. Atherosclerosis: cell biology and lipoproteins. Current Opinion in Lipidology 2020;31:286.

[9] Tu L, Zou Z, Ye Y, Wang S, Xing B, Feng J, et al. Targeted drug delivery systems for atherosclerosis. Journal of Nanobiotechnology 2025;23.

[10] Guo X, Ma L. Inflammation in coronary artery disease-clinical implications of novel HDL-cholesterol–related inflammatory parameters as predictors. Coronary Artery Disease 2022.

[11] Liu Y, Zhang Z, Li T, Xu H, Zhang H. Senescence in osteoarthritis: from mechanism to potential treatment. Arthritis Research & Therapy 2022;24.

[12] Chan Y, Ramji DP. Atherosclerosis: Pathogenesis and Key Cellular Processes, Current and Emerging Therapies, Key Challenges, and Future Research Directions. Methods in Molecular Biology 2022:3.

[13] Nasr SH, Huang X. Nanotechnology for Targeted Therapy of Atherosclerosis. Frontiers in Pharmacology 2021;12.

[14] Subramani S, Varshney N, Anand M, Soudagar MEM, Al-Keridis LA, Upadhyay TK, et al. Cardiovascular diseases prediction by machine learning incorporation with deep learning. Frontiers in Medicine 2023;10.

[15] García JJT. Atherogenic indices: usefulness as predictors of cardiovascular disease. Clínica e Investigación En Arteriosclerosis (English Edition) 2022;34:269. https://doi.org/10.1016/j.artere.2022.09.002.

[16] Bansal A, Hiwale K. Updates in the Management of Coronary Artery Disease: A Review Article. Cureus 2023. https://doi.org/10.7759/cureus.50644.

[17] Arra M, Swarnkar G, Alippe Y, Mbalaviele G, Abu-Amer Y. IκB-ζ signaling promotes chondrocyte inflammatory phenotype, senescence, and erosive joint pathology. Bone Research

2022;10.

[18] Coryell P, Diekman BO, Loeser RF. Mechanisms and therapeutic implications of cellular senescence in osteoarthritis. Nature Reviews Rheumatology 2020;17:47.

[19] Prilepskii AY, Serov N, Kladko DV, Vinogradov VV. Nanoparticle-Based Approaches towards the Treatment of Atherosclerosis. Pharmaceutics 2020;12:1056.

[20] Pillai SC, Borah A, Jacob EM, Kumar DS. Nanotechnological approach to delivering nutraceuticals as promising drug candidates for the treatment of atherosclerosis. Drug Delivery 2021;28:550.

[21] Montezano AC, Camargo LL, Mary S, Neves KB, Rios FJ, Stein RL, et al. SARS-CoV-2 spike protein induces endothelial inflammation via ACE2 independently of viral replication. Scientific Reports 2023;13.

[22] Kotain GB, Shaikh DSB, Joy LS, Rao NL, M PR, M IH. "High- Sensitivity C-Reactive Protein Correlation with Angiographic Findings in Patients with Acute Coronary Artery Syndrome''. Saudi Journal of Biomedical Research 2021;6:53.

[23] Afifi N, B MM, ghani amani mohamed abdel, HGE MAH, ME B. Value of Albumin-Fibrinogen Ratio and CRP-Albumin Ratio as Predictor Marker of Disease Activity in Egyptian RA Patients, Correlated with Musculoskeletal Sonography. DOAJ (DOAJ: Directory of Open Access Journals) 2020.

[24] Kazemian S, Ahmadi R, Ferns GA, Rafiei A, Azadegan-Dehkordi F, Khaledifar A, et al. Correlation of miR-24-3p and miR-595 expression with CCL3, CCL4, IL1-beta, TNFalphaIP3, and NF-kappaBIalpha genes in PBMCs of patients with coronary artery disease. PubMed 2022;21:1184.

[25] Li Q, Wang Q, Xu W, Ma Y, Wang Q, Eatman D, et al. C-Reactive Protein Causes Adult-Onset Obesity Through Chronic Inflammatory Mechanism. Frontiers in Cell and Developmental Biology 2020;8.

[26] Stec-Martyna E, Wojtczak K, Nowak D, Stawski R. Battle of the Biomarkers of Systemic Inflammation. Biology 2025;14:438.

[27] Wong Y, Tse H. Circulating Biomarkers for Cardiovascular Disease Risk Prediction in Patients With Cardiovascular Disease. Frontiers in Cardiovascular Medicine 2021;8.

[28] Cao H, Gui L, Hu Y, Yang J, Hua P, Yang S. Association between hemoglobin glycation index and adverse outcomes in critically ill patients with myocardial infarction: a retrospective cohort study 2025.

[29] Sreedharan S, Nair V, Cisneros-Zevallos L. Protective Role of Phenolic Compounds from Whole Cardamom (Elettaria cardamomum (L.) Maton) against LPS-Induced Inflammation in Colon and Macrophage Cells. Nutrients 2023;15:2965.

[30] Goswami S, Ranjan P, Dutta RK, Verma SK. Management of inflammation in cardiovascular diseases. Pharmacological Research 2021;173:105912.