O MUNDO DA SAÚDE

ARTICLE ORIGINAL

DOI: 10.15343/0104-7809.202650e18082025I
ISSN: 1980-3990

The Potential of Serum Tenascin-C as a Biomarker of Severity and Progression in Coronary Artery Disease: a Narrative Review

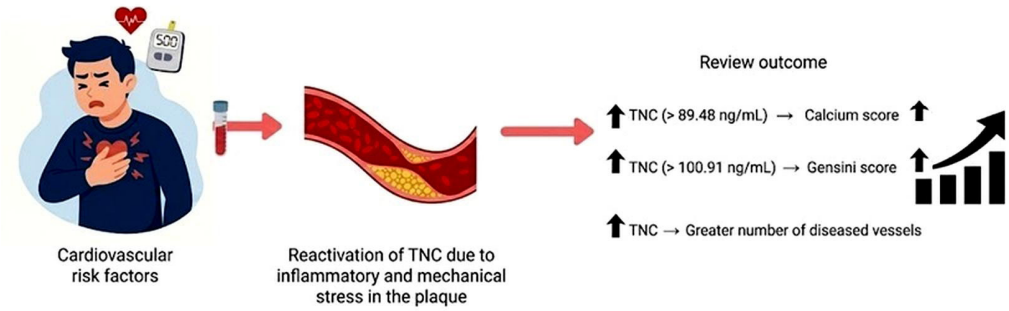
Vanessa Ellen Silva Carmo[1] **Hideki Zimermann Kamitani**[1] **Ívine Santos Carvalho**[1] **Williames Matheus Malaquias da Silva**[1] **João Pedro Lopes Azevedo**[1] **Vanessa Souza Silva Medrado**[1] **Rodrigo Mendes**[2] **Pedro Pereira Tenório**[1]

[1]Universidade Federal do Vale do São Francisco – UNIVASF. Paulo Afonso/BA, Brasil.
[2]Irmandade Santa Casa de Misericórdia de São Paulo. São Paulo/SP, Brasil.
E-mail: pedro.tenorio@univasf.edu.br

Highlights

- *Coronary Artery Disease is a condition that is difficult to diagnose and is often only identified in advanced stages and after complications have occurred.*
- *Elevated serum TNC was observed in patients with greater coronary involvement.*
- *Studies demonstrate a correlation between TNC levels and CAD severity scores.*
- *TNC levels are increased even in stable angina.*
- *TNC is associated with other inflammatory markers in cardiovascular contexts.*

Graphical Abstract



Abstract

Tenascin-C (TNC) is an extracellular matrix glycoprotein whose expression in adults is associated with inflammatory and vascular remodeling processes, such as atherosclerosis. This exploratory narrative review resulted in the inclusion of studies that evaluated the association between serum TNC levels and the severity of Coronary Artery Disease (CAD). The findings indicate that TNC levels above 89.48 ng/mL exhibit a sensitivity of 86.4% and a specificity of 77.6% for identifying the presence of CAD, whereas values above 100.91 ng/mL are predictors of complex and severe coronary lesions (Gensini Score). It is concluded that serum TNC is a promising marker for risk stratification and monitoring of CAD progression, although its standardization in clinical practice requires validation in larger cohort studies.

Keywords: Tenascin-C. Biomarker. Coronary Artery Disease.

Associate Editor: Edison Barbieri
Mundo Saúde. 2026,50:e18082025
O Mundo da Saúde, São Paulo, SP, Brasil.
https://revistamundodasaude.emnuvens.com.br

Received: 10 september 2025.
Accepted: 30 march 2026.
Published: 23 april 2026.

INTRODUCTION

Cardiovascular diseases (CVD) are the leading cause of mortality worldwide, accounting for approximately 17.9 million deaths annually, according to the World Health Organization (WHO). Within the spectrum of CVD, Coronary Artery Disease (CAD) is the most prevalent heart disease globally. Although multifactorial, its primary etiology is atherosclerosis of the coronary arteries, which reduces vascular blood flow. This reduction in coronary reserve leads to a state of chronic myocardial hypoxemia, which may progress to acute coronary syndrome (ACS) or even acute myocardial infarction (AMI), with consequent tissue necrosis[1,2]. The formation of atherosclerotic plaques results from a chronic inflammatory process initiated by the influx of lipids, particularly LDL-c, and inflammatory cells, such as foam cells, into the subendothelial tissue. In addition, there is hyperplasia of smooth muscle cells in the tunica intima, further exacerbating vascular stenosis[3,4].

Currently, the methods used for CAD detection are still considered complex and costly, such as genetic testing; insufficiently sensitive for early lesions, such as imaging examinations; or highly nonspecific, such as lipid profile measurement, which delays and hinders diagnosis[2]. In this context, although there is still a limited number of studies correlating variations in serum TNC levels with atherosclerosis, some studies have identified increased levels of this glycoprotein in CAD. Given the lack of accessible methods for detecting CAD at early stages, as well as the limited number of studies in this field, this review aims to discuss the use of serum TNC measurement in individuals with CAD and to encourage the development of new clinical studies evaluating the relationship between serum TNC and the monitoring of diseases of atherosclerotic etiology.

TENASCIN-C

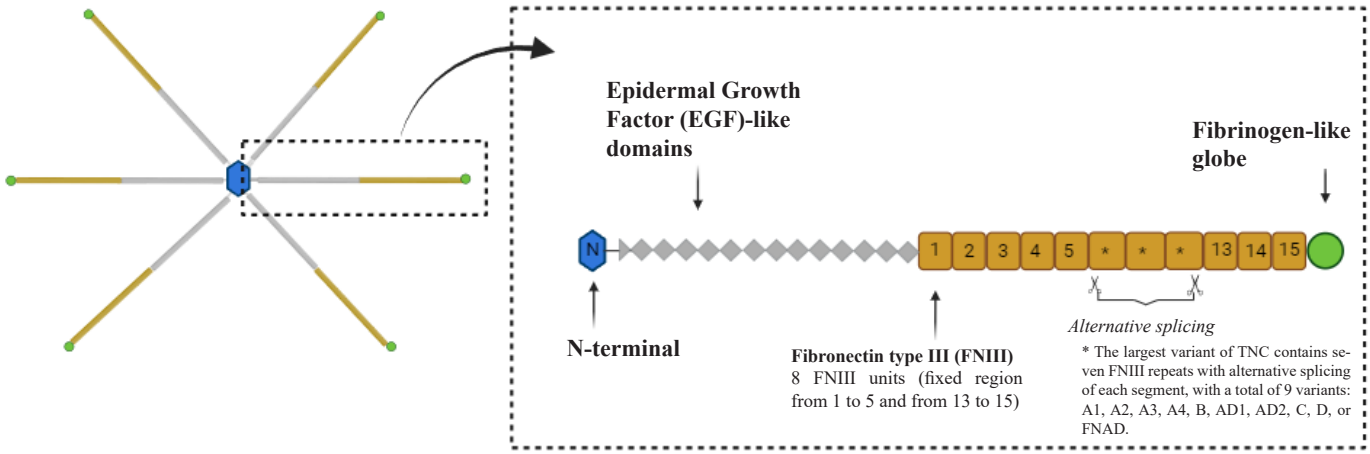


Figure 1 - Structural representation of Tenascin-C: a hexameric extracellular matrix (ECM) glycoprotein. Each subunit is composed of four domains: the N-terminal or Assembly Domain, Epidermal Growth Factor (EGF)-like domains, Fibronectin type III (FNIII) repeats, and a Fibrinogen-like globe. The FNIII domain contains a constant region, represented by repeats 1–5 and 13–15, and a variable region, represented by asterisks within the FNIII repeats, responsible for the existence of different isoforms generated by alternative splicing.

Tenascin-C (TNC) is a glycoprotein present in the extracellular matrix (ECM) that plays, during the embryonic period, an important role related to cellular differentiation, adhesion, and effects on the cell cycle, such as growth and apoptosis[5]. In adulthood, TNC concentration remains low; however, in certain pathological conditions, such as neoplasms, trauma, and inflammatory diseases, its serum levels are substantially elevated.

TNC exhibits a hexameric structure, and each subunit contains four domains: the N-terminal domain, epidermal growth factor (EGF)-like domains, fibronectin type III (FNIII) repeats, and a fibrinogen-like globe (Figure 1). Each TNC domain is capa-

Mundo Saúde. 2026,50:e18082025
DOI: 10.15343/0104-7809.202650e18082025I

ble of interacting with other ECM molecules through different types of receptors. These interactions may result in structural alterations, modifying the stiffness of the cellular environment or regulating the action of ECM components on cells. In addition, TNC isoforms, arising from alternative splicing of genes encoding part of the FNIII domain, are also associated with increased TNC diversity and its ability to interact with other compounds. Due to its structure and interaction with different receptors, TNC is capable of inducing a series of processes such as cell migration, adhesion, dissemination, as well as the synthesis of proteases and pro-inflammatory cytokines[6,7].

TNC expression is tightly regulated across different stages in the human organism. In the embryo, TNC contributes to the morphogenesis of essential tissues, such as limb formation, skeletal organization, as well as ligaments and tendons. It accumulates around the epithelium of the lungs, kidneys, developing teeth, and around certain cells of the developing central nervous system (CNS)[8].

Its presence in healthy adult individuals is more localized and at relatively low levels, being found in some connective tissues resistant to tensile stress, beneath epithelial tissues, and in specific stem cell niches. In addition to its role in physiological conditions, TNC is also rapidly produced in response to stress conditions that require extensive cellular turnover, plasticity, and tissue remodeling. Its presence is observed in cellular, mechanical, or chemical injuries, following viral and bacterial infections, within the tumor stroma, and in chronic inflammatory conditions such as atherosclerosis[6,7,8,9,10].

Alterations in TNC levels under pathological conditions demonstrate the involvement of this glycoprotein in inflammation, fibrosis, and consequently in tissue remodeling[7,11]. In this context, although reference values for normal TNC concentrations vary among the analyzed studies, Gao *et al.* (2019)[12] define a serum level ≤ 89.48 ng/mL as a reference value in healthy adults. Furthermore, current evidence indicates that TNC participates in mediating stress responses in organs of the cardiovascular system, particularly in CAD[12].

CORONARY ARTERY DISEASE

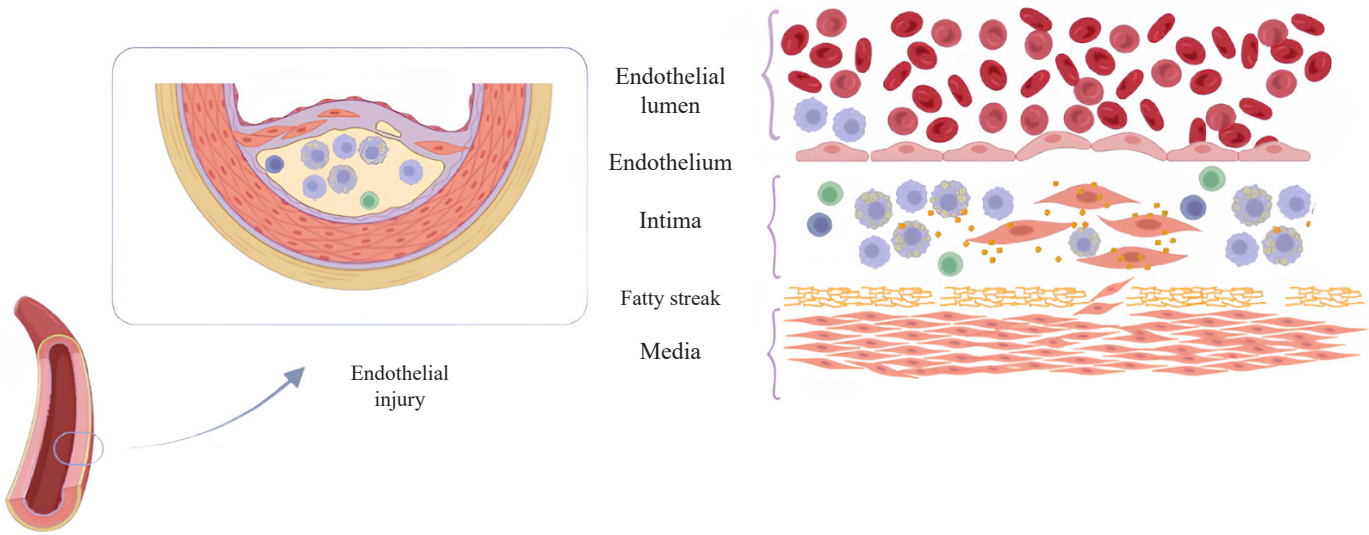


Figure 2 - Schematic representation of atherosclerosis: Initially, the formation of the atherosclerotic plaque begins with endothelial injury and dysfunction at sites of disturbed blood flow, followed by the accumulation of LDL (c). Subsequently, LDL (c) deposits in the tunica intima and undergoes modification by reactive oxygen species, which favors its phagocytosis by macrophages and the formation of foam cells. Finally, the accumulation of oxidized LDL (oxLDL), followed by activation of T cells and macrophages, increases the production of pro-inflammatory cytokines in this region, thereby establishing a chronic inflammatory state.

Coronary Artery Disease (CAD) is a condition with high morbidity and mortality, significant prevalence, and substantial impact on the economy and healthcare systems of multiple countries[13]. CAD is characterized by a reduction in blood flow through the coronary arteries, either due to partial narrowing or complete obstruction of the lumen, with atherosclerosis being its primary cause. The formation of atherosclerotic plaques in the coronary arteries develops similarly to that in other arteries, resulting from abnormal deposition of cholesterol, blood cells, and lipoproteins — particularly low-density lipoprotein (LDL) — in the tunica intima of the arteries[14] (Figure 2). Triggering factors include genetic predisposition and individual lifestyle habits that create a pro-inflammatory and pro-thrombotic environment within the myocardial vascular supply[15].

In addition to these factors, plaque formation and its associated inflammatory cascade promote a reduction in coronary reserve, impairing blood supply to the myocardium. Subsequently, plaques may undergo erosion and rupture, potentially progressing from thrombosis to embolism, which can lead to ischemia at various sites, posing harmful or even fatal consequences for the individual[16]. Therefore, CAD is commonly associated with angina pectoris and acute myocardial infarction (AMI)[15].

Although various methods are available for detecting atherosclerosis, they present limitations, particularly in the coronary arteries, including high cost, invasive procedures, low sensitivity, restricted ability to assess atheroma composition, low resolution, and exposure to ionizing radiation[17]. The limitations of available diagnostic tests, combined with the potential severity of cardiovascular diseases, underscore the need to identify biomarkers with high specificity and sensitivity at an accessible cost to assist in CAD detection.

In light of the above, this exploratory narrative review aims to evaluate the evidence regarding the association between serum Tenascin-C levels and the severity or progression of Coronary Artery Disease (CAD), discussing its potential as a complementary tool in cardiovascular risk stratification.

METHODOLOGY

This study consisted of a narrative literature review that included original articles retrieved from databases of the United States National Library of Medicine service for free access to MEDLINE (PubMed), Scientific Electronic Library Online (SciELO), the Virtual Health Library (VHL), and the Brazilian Digital Library of Theses and Dissertations (BDTD). The search strategy involved identifying Health Sciences Descriptors (DeCS/BIREME) and Medical Subject Headings (MeSH/PubMed), which generated the following general descriptors: ("Tenascin-C") AND (atherosclerosis OR atheroscleroses).

The inclusion criteria were: (1) availability of full text; (2) publication in English, Portuguese, or Spanish; (3) studies addressing the relationship between CAD and TNC. The exclusion criteria were: (1) review articles; (2) editorials (Figure 3).

The exploratory analysis of the data focused on identifying cutoff values for serum TNC and its ability to predict atherosclerotic burden. Source selection was based on academic relevance and the recency of evidence, aiming to construct a rationale linking glycoprotein expression to the severity and progression of vascular disease.

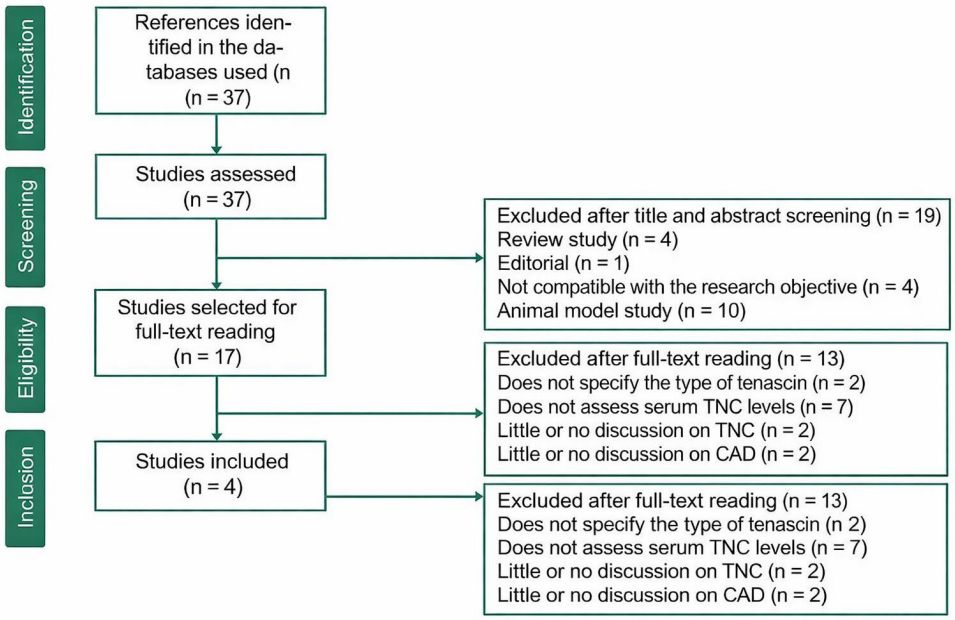


Figure 3 - Flowchart of included and excluded articles, according to the established inclusion and exclusion criteria.

Mundo Saúde. 2026,50:e18082025
DOI: 10.15343/0104-7809.202650e18082025I

RESULTS

The selection of articles was based on those aligned with the study objective, which consists of evaluating the relationship between CAD and TNC. Initially, the aim was to investigate the relationship between the serum presence of TNC in cardiovascular diseases and atherosclerosis; however, the identified studies directed the focus toward assessing the association between elevated serum TNC and CAD (Table 1).

In the study by Mehri *et al.* (2021)[15], 80 serum samples were analyzed, including 20 from the control group and 60 from patients with CAD, aiming to evaluate differences in serum levels of distinct compounds, including TNC, between these groups. The serum analysis of TNC in both groups demonstrated a statistically significant difference ($p = 0.01$), with a mean TNC level of 1049 ± 355 (ng/mL) in the CAD group and 683 ± 145 (ng/mL) in the control group. The CAD group was subdivided into three groups based on the number of occluded vessels, as assessed by angiography: 20 patients with one occluded vessel, 20 with two occluded vessels, and 20 with three occluded vessels. The analysis of these three subgroups also indicated a significant difference compared to the control group ($p < 0.05$)[15]. The identified studies thus directed the focus toward evaluating the association between elevated serum TNC and CAD.

Table 1 - Synthesis of the included studies evaluating Tenascin-C (TNC) in Coronary Artery Disease (CAD).

Title	Authors	Year	Methodology	Summary	Level of Evidence
Evaluation of the serum levels of Mannose binding lectin-2, Tenascin-C, and total antioxidant capacity in patients with coronary artery disease	Mehri *et al.*[15]	2021	Analysis of 60 serum samples from patients with CAD and 20 healthy serum samples. Assessed using the ELISA method.	Investigates serum TNC levels in patients with CAD and the relationship between biochemical parameters and CAD progression. Serum TNC levels were significantly increased in the CAD group compared to the control group. The study suggests that CAD may be diagnosed at early stages based on the evaluation of these serum parameters. Further research is needed to establish its diagnostic and therapeutic value.	3B
Tenascin-C: A Potential Biomarker for Predicting the Severity of Coronary Atherosclerosis	Gao *et al.*[12]	2019	Selective coronary angiography performed in 157 patients with chest pain suspected of atherosclerosis. Patients were divided into CAD and non-CAD groups according to symptoms and angiographic findings.	Evaluates the value of TNC in relation to the severity of atherosclerosis by comparison with the Gensini score. Higher serum TNC levels were detected in the CAD group, with a significant positive correlation observed with the Gensini score. TNC levels may be considered in CAD risk assessment prior to angiography.	3B
Relation between coronary artery calcium score and serum Tenascin-C level in patients without known coronary artery disease	Yildiz *et al.*[18]	2015	Selection of 90 patients with chest pain, without known CAD, and grouping according to their coronary artery calcium scores (CACS). Patients were assessed for risk factors and underwent laboratory analyses for biochemical parameters, including TNC.	Investigates the relationship between coronary artery calcium score (CAC) and serum TNC levels. Mean serum TNC levels were significantly higher in patients with higher CACS compared to the low-CACS group and the control group. A significant relationship between CAC and serum TNC levels was demonstrated, suggesting that TNC measurement may be used to identify elevated CAC and, consequently, CAD risk.	3B
Clinical implications of Tenascin-C and OX40 ligand in patients with acute coronary syndrome	Yang; Ren[19]	2014	Serum measurement of TNC and OX40L in 50 healthy individuals and 170 patients with stable angina, unstable angina, and acute myocardial infarction.	Evaluates serum levels of TNC and OX40L in acute coronary syndrome (ACS). TNC levels in serum samples from ACS patients were significantly higher compared to those from control and stable angina groups, and both TNC and OX40L levels increased with clinical severity progression from stable angina to ACS.	3B

In the study conducted by Gao *et al.* (2019)[12], 157 patients presenting with angina/chest pain and suspected coronary atherosclerosis underwent coronary angiography. According to angiographic findings and clinical presentation, they were divided into two groups: CAD (81 patients) and non-CAD (76 patients). Serum TNC levels were measured in these groups to assess their relationship with atherosclerosis severity, using the Gensini score, which evaluates disease severity based on lesion location and degree of luminal narrowing. The study demonstrated significantly higher mean serum TNC levels in the CAD group ($p < 0.001$). ROC curve analysis established a serum TNC cutoff value of 89.48 ng/mL, yielding a sensitivity of 86.4% and a specificity of 77.6% for identifying CAD. Furthermore, values above 100.91 ng/mL showed a sensitivity of 82.7% and specificity of 79% for predicting a high Gensini score. A positive correlation between serum TNC levels and the Gensini score was also observed ($p < 0.01$) across all patients, indicating TNC as a predictor of higher Gensini scores. These findings suggest that serum TNC levels may indicate the severity of atherosclerosis even prior to angiography. However, serum TNC levels also exhibited a positive correlation with other biomarkers and biochemical indices[12].

The study by Yildiz *et al.* (2015)[18] evaluated 90 patients presenting with chest pain without a prior diagnosis of CAD. A 64-slice computed tomography scan was performed to determine the coronary artery calcium score (CACS), allowing classification into the following groups: CACS = 0 (control group; 30 patients), CACS between 0 and 400 (low-CACS group; 30 patients), and CACS ≥ 400 (high-CACS group; 30 patients). Serum TNC levels in the high-CACS group were 84 (2–456) ng/mL, compared to 28 (0.7–212) ng/mL in the low-CACS group and 4.7 (0.03–30.7) ng/mL in the control group. These findings indicate that mean TNC levels were substantially higher in both high- and low-CACS groups compared to the control group, demonstrating a correlation between CACS and TNC, as well as their association with CAD. ROC curve analysis identified a serum TNC cutoff value of 8.09 ng/mL, with a sensitivity of 72% and specificity of 82% for predicting CACS > 100, while a value of 1.19 ng/mL was established as the cutoff for CACS = 0. Additionally, the study demonstrated a correlation between TNC levels and triglyceride levels[18].

In Yang and Ren (2014)[19], a total of 220 individuals were analyzed, including 170 patients with angiographically confirmed CAD and 50 healthy individuals. The CAD group was subdivided into 120 patients with acute coronary syndrome (ACS) — including 70 with unstable angina and 50 with recent AMI — and 50 patients with stable angina. Serum TNC levels were markedly higher in CAD patients (39.39 ± 19.80 ng/mL) compared to both the control group and the stable angina group (28.65 ± 12.32 ng/mL, $p < 0.01$; and 31.22 ± 18.92 ng/mL, $p < 0.05$, respectively). The study also evaluated OX40, a member of the tumor necrosis factor (TNF) receptor superfamily expressed in activated T cells, which was elevated in the CAD group and showed a positive correlation with TNC serum levels[19].

DISCUSSION

The elevation of TNC in CAD may be explained by its expression by vascular smooth muscle cells and activated macrophages within the atheroma, where it promotes inflammation and extracellular matrix remodeling. TNC interacts with multiple cellular receptors, resulting in structural alterations and amplification of protease and pro-inflammatory cytokine synthesis. This sustained inflammatory microenvironment directly contributes to intimal hyperplasia, lipoprotein retention, and increased vulnerability of the atherosclerotic plaque, facilitating its histopathological progression and eventual evolution toward acute coronary syndromes.

The analysis of the four selected studies indicates that Tenascin-C (TNC) transcends its role as a mere structural protein, acting as a dynamic biomarker of vascular integrity. Unlike traditional markers of myocardial necrosis, TNC expression in adults is virtually absent under physiological conditions and is specifically reactivated in response to inflammatory stress and extracellular matrix (ECM) remodeling characteristic of atherosclerosis[6,7,8].

CAD is a silent disease and difficult to diagnose in its early stages. At this stage, current approaches rely on clinical history combined with basic biochemical tests, such as lipid profile evaluation and markers for comorbid conditions (e.g., diabetes), to stratify risk. However, these assessments are nonspecific for CAD detection. Genetic testing has emerged as a screening tool prior to more invasive procedures; nevertheless, it remains costly and limited in accessibility, as only a few institutions provide such services[20]. Regarding TNC expression in patients with early-stage vascular involvement, available studies have not clearly established how early serum TNC elevation may occur, which may

limit its clinical applicability in early detection. Nonetheless, it may serve as a complementary test to current methods. For instance, Yang and Ren (2014)[19] observed increased serum TNC and other biomarkers in patients with stable angina compared to controls, even before progression to ACS, suggesting that TNC, combined with clinical history in individuals with stable angina and atherosclerotic risk factors, may be considered[21].

Moreover, physical testing and imaging methods, particularly coronary computed tomography angiography, are important tools for CAD detection in clinical practice[22]. However, access to these examinations is often limited, and they have constraints in early detection. In addition, they are invasive techniques involving exposure to ionizing radiation, catheter insertion, and contrast agents. In this context, TNC measurement may be advantageous, as demonstrated by Gao *et al.* (2019)[12], where serum TNC levels were able to predict severe CAD even prior to the Gensini index.

Nevertheless, the use of serum TNC measurement in CAD diagnosis still requires further advancement for clinical application. This is due to the limited number of available studies and the relatively small sample sizes in the analyzed works[12,15,18,19]. Furthermore, it is important to note that TNC is also elevated in other diseases and conditions associated with increased levels of additional biomarkers. In the analyzed studies, positive correlations were observed with NT-proBNP, HbA1c, and OX40 levels — the latter demonstrating superior diagnostic accuracy in predicting adverse events in patients with ACS[19]. While TNC reflects structural matrix damage, OX40L signals immune activation, making their combined assessment a powerful indicator of atherosclerotic plaque instability. Additionally, renal function may influence TNC levels, complicating interpretation. Therefore, the analysis of serum TNC levels and their correlation with other biomarkers requires further investigation[12]. Despite promising results, the real clinical feasibility of TNC measurement as a routine biomarker still faces substantial challenges. Currently, detection methods — predominantly based on enzyme-linked immunosorbent assays (ELISA) — are largely restricted to research settings, with higher costs and lower levels of automation compared to established and low-cost cardiovascular biomarkers, such as high-sensitivity troponin and C-reactive protein (CRP). Furthermore, pre-analytical and analytical issues, including biological sample stability over time and inter-assay reproducibility, still lack rigorous standardization protocols. For TNC to be incorporated into routine clinical practice in a cost-effective manner, the development of automated and commercially scalable detection platforms will be essential.

Current evidence, although limited and derived from cross-sectional studies, strongly suggests that serum Tenascin-C is a promising biomarker for stratifying disease severity and monitoring CAD progression, showing correlation with angiographic and calcium scores. To establish its role in early diagnosis or screening, long-term prospective cohort studies with larger populations and standardized measurement methods are imperative.

CONCLUSION

Current evidence, although limited and derived from cross-sectional studies, strongly suggests that serum Tenascin-C is a promising biomarker for stratifying disease severity and monitoring CAD progression, correlating with angiographic and calcium scores. To establish its role in early diagnosis or screening, long-term prospective cohort studies with larger populations and standardized measurement methods are imperative.

CRediT author statement

Conceptualization: Carmo, VES; Kamitani, HZ; Carvalho, IS; Silva, WMM; Mendes, R; Tenório, PP; Azevedo, JPL; Medrado, VSS. Methodology: Carmo, VES; Kamitani, HZ; Carvalho, IS; Silva, WMM; Mendes, R; Tenório, PP; Azevedo, JPL; Medrado, VSS. Validation: Carmo, VES; Kamitani, HZ; Carvalho, IS; Silva, WMM; Mendes, R; Azevedo, JPL; Medrado, VSS; Tenório, PP. Statistical analysis: Carmo, VES; Kamitani, HZ; Carvalho, IS; Silva, WMM. Formal analysis: Carmo, VES; Kamitani, HZ; Carvalho, IS; Silva, WMM. Investigation: Carmo, VES; Kamitani, HZ; Carvalho, IS; Silva, WMM. Resources: Carmo, VES; Kamitani, HZ; Carvalho, IS; Silva, WMM; Mendes, R; Azevedo, JPL; Medrado, VSS; Tenório, PP. Writing – original draft: Carmo, VES; Kamitani, HZ; Carvalho, IS; Silva, WMM. Writing – review & editing: Mendes, R; Tenório, PP. Visualization: Mendes, R; Tenório, PP. Supervision: Mendes, R; Tenório, PP. Project administration: Mendes, R; Tenório, PP.

All authors have read and agreed to the published version of the manuscript.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the workreported in this paper.

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How to cite this article: Carmo, V.E.S., Kamitani, H.Z., Carvalho, I.S., Silva, W.M.M., Azevedo, J.P.L., Medrado, V.S.S., Mendes, R., Tenório, P.P. (2026). The Potential of Serum Tenascin-C as a Biomarker of Severity and Progression in Coronary Artery Disease: a Narrative Review. O Mundo Da Saúde, 50. https://doi.org/10.15343/0104-7809.202650e18082025I. Mundo Saúde. 2026,50:e18082025.