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Serum Semaphorin as a Novel Diagnostic and Prognostic Biomarker in Acute Myocardial Infarction

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Abstract

Aim: Acute myocardial infarction (AMI) remains a leading cause of morbidity and mortality worldwide. The identification of novel biomarkers reflecting the inflammatory and thrombotic mechanisms involved in acute coronary syndromes may improve diagnostic accuracy and risk stratification. Semaphorins are a family of proteins involved in immune regulation, vascular biology, and platelet activation, suggesting a potential role in cardiovascular disease. This study aimed to investigate serum semaphorin levels in patients with AMI and to evaluate their potential diagnostic and prognostic value.

Materials and Methods: In this prospective case-control study, 62 participants were enrolled: 31 patients diagnosed with AMI and 31 age- and sex-matched control subjects. Among the AMI patients, 19 had ST-segment elevation myocardial infarction (STEMI) and 12 had non-STEMI (NSTEMI). Serum semaphorin levels were measured using an ELISA. Clinical, demographic, and angiographic data were recorded and analyzed.

Results: The mean serum semaphorin level was 0.21±0.12 in the AMI group and 0.26±0.17 in the control group, with no statistically significant difference between groups (p=0.556). Semaphorin levels were also comparable between male and female participants (0.23±0.14 vs. 0.26±0.18, p=0.662). Subgroup analysis demonstrated mean semaphorin levels of 0.21±0.16 in NSTEMI patients and 0.21±0.07 in STEMI patients. Although semaphorin levels tended to be lower in AMI patients than in the control group, no significant difference was observed among the control, NSTEMI, and STEMI groups(p=0.081).

Conclusion: Serum semaphorin levels were not significantly associated with the presence or subtype of AMI in the present study. These findings suggest limited diagnostic utility of circulating semaphorin levels in AMI. Further multicenter studies with larger sample sizes are needed to clarify the role of semaphorins in the pathophysiology and clinical assessment of acute coronary syndromes.

Keywords: Acute myocardial infarction, semaphorin, biomarker, STEM, NSTEMI, acute coronary syndrome; cardiovascular disease

Introduction

Acute myocardial ınfarction (AMI), commonly known as a heart attack, is a critical cardiac event caused by the sudden blockage of blood flow in one or more coronary arteries, leading to myocardial ischemia and necrosis (1). It remains a leading cause of morbidity and mortality worldwide, with significant prevalence in both developed and developing countries (2,3). AMI includes both ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI), both of which are managed with similar therapeutic approaches (4).

The relationship between biomarker levels in patients with AMI and their clinical outcomes is multifaceted, involving various biomarkers that reflect different pathophysiological processes. Elevated levels of biomarkers such as NT-proBNP, cTNT, and GDF-15 have been shown to predict heart failure (HF) following AMI, with NT-proBNP demonstrating the highest predictive value for post-AMI HF (5,6). Similarly, BNP and troponin I levels are significantly correlated with adverse outcomes such as left ventricular dysfunction, arrhythmias, and mortality in acute coronary syndrome (ACS) patients, indicating their utility in risk stratification (7,8). Novel biomarkers like sST2, suPAR, and H-FABP

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are also elevated in AMI patients and correlate with clinical parameters such as ejection fraction and inflammatory markers, suggesting their potential role in assessing cardiac function and predicting outcomes (9). Although semaphorins were initially identified as axon guidance proteins involved in nervous system development, they are now known to have significant effects on the immune system, vascular biology, platelet activation, and inflammation. Specifically, Semaphorin 3A (Sema3A) and Semaphorin 4D (Sema4D) have been found to be associated with endothelial function, atherosclerotic plaque stability, and platelet aggregation. Sema4D is reported to exhibit pro-inflammatory and prothrombotic effects, while Sema3A is known to possess anti-inflammatory and vascular protective properties (10,11).

This study aimed to investigate the diagnostic and prognostic value of serum semaphorin levels in patients with AMI and evaluate the relationship between these levels and clinical, biochemical, and angiographic parameters.

Materials and Methods

This study will be conducted prospectively, observationally, and under controlled conditions on patients presenting to the Emergency Department of the Sivas Cumhurıyet University Health Sciences Research Ethics Committee. The study protocol will be designed in accordance with the principles of the Declaration of Helsinki, and approval will be obtained from the (Relevant Institution) Clinical Research Ethics Committee before data collection begins. Written informed consent will be obtained from all participants (or their immediate family members) included in the study. This study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Ethics Committee Sivas Cumhurıyet University Health Sciences Research (protocol number: 2025/07-19, date: 10.07.2025).

The study will include consecutive patients who present with chest pain or equivalent symptoms, such as shortness of breath or syncope, and who are diagnosed with AMI according to the Third Universal Definition of Myocardial Infarction. Patient groups: STEMI group: patients with new ST segment elevation in at least two consecutive leads on electrocardiography (ECG) (approximately 2 mm in men, 1.5 mm in women; 1 mm in other leads) or newly developed left bundle branch block and positive cardiac troponin (cTn) kinetics. NSTEMI group: patients with ischemic symptoms and/or dynamic ST-T changes (ST depression, T wave inversion) on ECG with elevated and/or decreased cTn levels, without ST segment elevation. Control group: patients similar in age and gender, with similar cardiovascular risk factors but without coronary artery disease (CAD). Individuals with similar symptoms (e.g., non-cardiac chest pain) in whom normal coronary arteries are detected on angiography or in whom CAD is ruled out by objective tests.

Inclusion Criteria:

- Age ≥ 18 years,
- Admission within the first (12/24) hours from chest pain onset,
- Willingness to participate and provision of signed informed consent.

Exclusion Criteria

To eliminate potential confounding factors that directly influence systemic inflammatory, angiogenic, and immune pathways, patients who meet any of the following criteria will be excluded:

- Active systemic infection, sepsis, or known chronic autoimmune/inflammatory diseases,
- Active malignancy or history of recent chemotherapy/radiotherapy,
- Severe renal impairment (estimated glomerular filtration rate <30 mL/min/1.73 m2 or end-stage hepatic failure,
- History of prior myocardial infarction, coronary artery bypass grafting, or percutaneous coronary intervention,
- Presentation with cardiogenic shock or requiring cardiopulmonary resuscitation at admission.

Patients' ages, genders, myocardial ınfarction thrombolysis scores (TIMI), and angiographic intervention results will be recorded on standard forms. Peripheral venous blood samples will be collected from patients in the AME group upon admission to the emergency department (0 hours, before treatment or angiography). A single basal blood sample will be collected from each healthy volunteers in the control group. Blood samples will be collected in gel-free biochemistry tubes (or EDTA tubes) and centrifuged at 3000 rpm for 10 minutes. The obtained serum/plasma samples will be separated from their cellular components, aliquoted into Eppendorf tubes, and stored in an ultra-low freezer at -80° C until analysis. Semaphorin analysis: human [specific semaphorin type, e.g., semaphorin 3A] levels in the stored samples will be determined using commercially available quantitative ELISA kits specific for human semaphorin according to the manufacturer's instructions. Measurements will be performed using a blinded method (absorbance wavelength: 450 nm) on a microplate reader by a laboratory specialist unaware of the patients' clinical status. Patients' TIMI scores and the extent and severity of CAD after coronary angiography were evaluated by experienced interventional cardiologists.

Statistical Analysis

Data analysis was performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed using the Shapiro-Wilk test. Continuous variables were expressed as mean ± standard deviation for normally distributed data or median (minimum-maximum) for non-normally distributed data. Comparisons between two independent groups were performed using the Student's t-test for normally distributed variables and the Mann-Whitney U test for non-normally distributed variables. Categorical variables were expressed as frequencies and percentages, and compared using the chi-square test. A p value <0.05 was considered statistically significant.

Result

A total of 62 participants were included in the study, comprising 31 patients with AMI and 31 control subjects. Among the AMI patients, 19 were diagnosed with STEMI and 12 with NSTEMI. Coronary angiographic evaluation demonstrated varying degrees of coronary artery stenosis and occlusion among patients with AMI, whereas no significant CAD was detected in the control group.

The mean serum semaphorin level was 0.21±0.12 in the AMI group and 0.26±0.17 in the control group. No statistically significant difference was observed between patients and controls (p=0.556) (Table 1). When serum semaphorin levels were analyzed according to sex, the mean concentration was 0.23±0.14 in males and 0.26±0.18 in females. This difference was not statistically significant (p=0.662). Subgroup analysis according to clinical diagnosis demonstrated mean semaphorin levels of 0.26±0.17 in the control group, 0.21±0.16 in patients with NSTEMI, and 0.21±0.07 in patients with STEMI. Although semaphorin levels tended to be lower in both AMI subgroups than in controls, differences among the three groups did not reach statistical significance (p=0.081).

Table 1. Comparison of serum semaphorin levels according to clinical outcomes and disease severity

Group	**Mean ± SD**	**p**
Patient	0.21±0.12	0.556
Control	0.26±0.17	
Gender		
Male	0.23±0.14	0.662
Female	0.26±0.18	
Population		
Control (n=31)	0.26±0.17	0.081
NSTEMI (n=12)	0.21±0.16	
STEMI (n=19)	0.21±0.07	

NSTEMI: non ST-segment elevation myocardial infarction, STEMI: ST-segment elevation myocardial infarction, SD: Standard deviation

Discussion

This study investigated the diagnostic and prognostic value of serum semaphorin levels in patients with AMI. The principal finding was that serum semaphorin concentrations did not differ significantly between patients with AMI and healthy controls. Furthermore, semaphorin levels were comparable between STEMI and NSTEMI patients and showed no significant association with sex or disease subtype. Although semaphorin concentrations tended to be lower in patients with AMI than in controls, these differences did not reach statistical significance. These findings suggest that circulating semaphorin levels have limited diagnostic utility for distinguishing AMI patients from controls in our study population. The absence of a significant association may be related to the relatively small sample size, the biological variability in semaphorin expression, or the timing of blood sampling during the acute phase of myocardial infarction.

The diagnosis and prognosis of AMI have significantly advanced with the identification and utilization of various biomarkers. cTn, particularly high-sensitivity troponins, are considered the gold standard for diagnosing myocardial injury because of their high sensitivity and specificity, which allow early detection of AMI (12,13). In addition to troponins, creatine kinase MB (CK-MB) and myoglobin are traditional markers used in AMI diagnosis, although their specificity is lower compared to troponins (14,15). Novel biomarkers such as heart-type fatty acid-binding protein and matrix metalloproteinases have shown promise in enhancing diagnostic accuracy (16). For prognostic purposes, N-terminal B-type natriuretic peptide (NT-proBNP) and C-reactive protein are valuable predictors of cardiovascular outcomes, including HF and mortality (17). Recent studies have identified additional biomarkers like growth differentiation factor-15 (GDF-15), tumor necrosis factor-related apoptosis-inducing ligand receptor 2, and fibroblast growth factor 23 as significant predictors of mortality and HF in AMI patients (18). Inflammatory markers such as interleukin-6 and myeloperoxidase also contribute to risk stratification by reflecting underlying inflammatory processes (19). Integrating multiple biomarkers, including novel biomarkers such as microRNAs and galectin-3, into a multi-biomarker approach can enhance diagnostic precision and provide a comprehensive risk assessment (20). However, challenges such as the high cost of testing and potential interference from chronic conditions like renal insufficiency limit the routine clinical application of some novel biomarkers (21). Despite these challenges, the use of artificial intelligence and biosensors in conjunction with biomarkers holds promise for improving early diagnosis and personalized treatment strategies in AMI (22). Overall, while traditional biomarkers remain crucial, the exploration and validation of novel biomarkers continue to evolve.

Although previous studies have demonstrated altered semaphorin expression in several inflammatory and cardiovascular disorders, our study did not identify a significant difference in serum semaphorin levels between patients with AMI and healthy controls. This discrepancy may reflect differences in disease mechanisms, the specific semaphorin isoforms measured, sample size, patient characteristics, or the timing of blood sampling. It is also possible that circulating semaphorin concentrations are influenced by dynamic inflammatory responses that are not adequately captured by a single measurement obtained at hospital admission. Therefore, while semaphorins remain biologically plausible candidates, our findings do not support their routine use as diagnostic biomarkers for AMI. In systemic sclerosis (SSc), Sema3A levels were found to be significantly lower in patients compared to healthy controls, with even lower levels observed in those with major vascular involvements such as digital ulcers and pulmonary arterial hypertension, suggesting its role in vasculopathy and as a potential biomarker for vascular complications in SSc (24,25). Similarly, in systemic lupus erythematosus (SLE), Sema3A levels were significantly reduced in patients, particularly those with active disease, correlating negatively with disease activity indices, indicating its potential as a marker for SLE activit (26,27). In the context of retinal vein occlusion, increased Sema3A levels in aqueous humor were associated with macular edema and ganglion cell degeneration, suggesting a pathological role in these conditions (28). Furthermore, in patients undergoing hemodialysis, Sema3A levels were significantly lower compared to controls and decreased further during dialysis sessions, indicating its potential involvement in kidney function and bone remodeling (29). In reproductive health, elevated Sema3A levels were observed in women with diminished ovarian reserve who responded better to controlled ovarian stimulation, suggesting a role in fertility and reproductive competence (30). On the other hand, Sema4D levels were elevated in HF patients, correlating with inflammatory markers and suggesting its utility as a biomarker for acute HF (31). In periodontal disease, increased Sema4D levels were noted in patients with periodontitis, which decreased following non-surgical periodontal therapy, indicating its role in periodontal inflammation (32). Collectively, these studies underscore the diverse roles of semaphorins in various diseases, highlighting their potential as diagnostic and prognostic biomarkers across different pathological conditions (33).

In AMI, biomarkers such as troponin, CK-MB, and B-type natriuretic peptide are commonly used for diagnosis and risk stratification. However, most of these markers are elevated as a result of myocardial damage (34). Semaphorins, on the other hand, may provide additional information about the pathophysiology of the disease as they may be involved in early stages of processes such as inflammation, platelet activation, and endothelial dysfunction. This suggests that semaphorins may be biomarkers that can be used not only in diagnosis but also in prognostic assessment (35). In our study, we planned to investigate the relationship between semaphorin levels and both TIMI score and coronary angiography findings. The TIMI score is a widely used risk assessment tool for predicting short-term mortality and major cardiac events in patients with ACS. The correlation between semaphorin levels and the TIMI score may reveal the value of these molecules for clinical risk stratification. Similarly, semaphorins can be considered biochemical indicators of atherosclerotic burden if they correlate with the extent and complexity of CAD.

Study Limitations

This study has limitations. First, since the study was conducted at a single center, the generalizability of the results to different populations and healthcare settings may be limited. Second, the relatively small sample size may reduce statistical power and make it difficult to establish significant relationships, especially in subgroup analyses. In this study, semaphorin levels were measured only at the time of admission; serial measurements were not performed. Therefore, the relationship between temporal changes in semaphorin levels and the development of myocardial infarction, receipt of reperfusion therapy, and clinical outcomes could not be evaluated. Furthermore, because only certain semaphorin subtypes were examined, the possible effects of other semaphorin family members could not be investigated.

While the extent and severity of CAD were determined by angiographic evaluation, advanced imaging techniques such as intravascular ultrasonography or optical coherence tomography (OCT) were not used. Therefore, plaque morphology and vulnerability-related characteristics could not be evaluated in detail. Although individuals with active infection, malignancy, and chronic inflammatory disease were excluded from the study, it was not possible to fully control for all biological and environmental factors that could affect semaphorin levels. Furthermore, since long-term follow-up data were not available, the effects of semaphorin levels on long-term mortality, recurrent myocardial infarction, and the development of HF could not be evaluated. Despite these limitations, our study is one of the few studies investigating the relationship between semaphorin levels and clinical, biochemical, and angiographic parameters in AMI, and may therefore contribute to the literature. Future studies with larger sample sizes, multiple centers, and long-term follow-up are needed to confirm these findings.

Conclusion

In this prospective case-control study, serum semaphorin levels did not differ significantly between patients with AMI and healthy controls, nor were they associated with AMI subtype. Although semaphorin concentrations showed a trend toward lower values in patients with AMI, the differences were not statistically significant. These findings suggest that circulating semaphorin levels have limited diagnostic value as standalone biomarkers for AMI in the studied population. Given the relatively small sample size and single-center design, larger multicenter studies with serial measurements and evaluation of specific semaphorin isoforms are warranted to further clarify the potential role of semaphorins in the pathophysiology and clinical assessment of AMI.

Ethics

Ethics Committee Approval: This study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Ethics Committee Sivas Cumhurıyet University Health Sciences Research (protocol number: 2025/07-19, date: 10.07.2025).

Informed Consent: This is prospective case-control study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: S.Ö., O.Ö., Concept: S.Ö., T.G., Design: S.Ö., O.Ö., T.G., M.A., C.B., Data Collection or Processing: S.Ö., T.G., M.A., Analysis or Interpretation: S.Ö., O.Ö., C.B., Literature Search: S.Ö., O.Ö., T.G., M.A., C.B., Writing: S.Ö., O.Ö., T.G., M.A., C.B.

Conflict of Interest: No conflict of interest was declared by the authors.

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