Original Research

PSYCHOLOGICAL STRESS AND ITS ROLE IN THE PRECIPITATION OF ANGINA PECTORIS

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ABSTRACT

Background: Psychological stress has been increasingly recognized as a significant contributor to cardiovascular morbidity, yet its specific role in precipitating angina pectoris episodes remains inadequately characterized in clinical practice. Understanding this relationship is essential for comprehensive cardiovascular risk management and therapeutic intervention.

Methods: A cross-sectional analytical study was conducted among 248 patients diagnosed with stable angina pectoris attending the cardiology outpatient department. Psychological stress was assessed using the Perceived Stress Scale (PSS-14) and the Depression Anxiety Stress Scales (DASS-21). Angina characteristics were evaluated using the Seattle Angina Questionnaire (SAQ) and patient-reported episode frequency. Correlation analyses, independent t-tests, and multiple regression analyses were performed.

Results: Patients with high perceived stress (PSS-14 score ≥28) demonstrated significantly greater weekly angina episode frequency (4.82 ± 1.94 vs. 2.31 ± 1.12, $p<0.001$) compared to low-stress counterparts. High stress was associated with increased angina severity scores (6.73 ± 1.45 vs. 4.21 ± 1.38, $p<0.001$) and reduced physical limitation scores on SAQ (48.32 ± 12.67 vs. 68.45 ± 14.23, $p<0.001$). Multiple regression analysis revealed that perceived stress independently predicted angina frequency ($\beta=0.412$, $p<0.001$) after controlling for traditional cardiovascular risk factors.

Conclusion: Psychological stress significantly contributes to the precipitation and exacerbation of angina pectoris, suggesting that stress management interventions should be integrated into comprehensive cardiac care protocols.

Keywords: Psychological Stress, Angina Pectoris, Coronary Artery Disease, Perceived Stress Scale, Cardiovascular Risk Factors.

INTRODUCTION

Cardiovascular diseases remain the leading cause of mortality worldwide, accounting for approximately 17.9 million deaths annually [1]. Among the clinical manifestations of coronary artery disease, angina pectoris represents a cardinal symptom characterized by chest discomfort resulting from myocardial ischemia due to an imbalance between myocardial oxygen supply and demand [2]. While traditional risk factors including hypertension, diabetes mellitus, dyslipidemia, and smoking have been extensively studied, the contribution of psychosocial factors to cardiovascular morbidity has gained substantial attention in recent decades [3].

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Psychological stress, defined as a state of mental or emotional strain resulting from adverse or demanding circumstances, has been implicated in the pathogenesis and progression of coronary artery disease through multiple pathophysiological mechanisms [4]. The landmark INTERHEART study demonstrated that psychosocial stressors, including work stress, financial stress, and major life events, were independently associated with increased risk of acute myocardial infarction across diverse populations [5]. This association was comparable in magnitude to traditional risk factors such as hypertension and diabetes.

The mechanisms underlying stress-induced myocardial ischemia involve activation of the sympathetic nervous system, resulting in elevated catecholamine release, increased heart rate, and blood pressure elevation [6]. Additionally, psychological stress promotes endothelial dysfunction, enhances platelet aggregation, and

triggers inflammatory cascades that contribute to plaque vulnerability and coronary vasoconstriction [7]. Mental stress-induced myocardial ischemia has been documented in laboratory settings, with approximately 30-70% of patients with coronary artery disease demonstrating ischemic responses during standardized mental stress testing [8].

Despite accumulating evidence linking psychological stress to adverse cardiovascular outcomes, the specific relationship between chronic perceived stress and the day-to-day manifestation of angina pectoris remains insufficiently characterized in routine clinical practice [9]. Furthermore, limited data exist regarding the comparative impact of different stress dimensions on angina frequency and severity in patients with stable coronary artery disease [10]. Understanding these relationships is paramount for developing targeted interventions that address modifiable psychosocial risk factors alongside conventional pharmacological management.

The aim of this study was to investigate the association between psychological stress levels and the frequency, severity, and clinical characteristics of angina pectoris episodes in patients with established stable coronary artery disease, and to determine whether psychological stress independently predicts angina manifestation after controlling for traditional cardiovascular risk factors.

MATERIALS AND METHODS

Study Design and Setting

This cross-sectional analytical study was conducted at the Outpatient Department of a tertiary care teaching hospital.

Study Population

A total of 248 patients with diagnosed stable angina pectoris were consecutively recruited. The sample size was calculated using G*Power software version 3.1.9.7, assuming a medium effect size (d=0.5), alpha error of 0.05, and power of 80%, yielding a minimum required sample of 210 participants.

Inclusion Criteria

Patients were eligible if they met the following criteria: (1) age between 40 and 75 years; (2) documented coronary artery disease confirmed by coronary angiography showing ≥50% stenosis in at least one major epicardial vessel; (3) diagnosis of stable angina pectoris according to Canadian Cardiovascular Society (CCS) classification (Class I-III); (4) stable medical therapy for at least three months; and (5) ability to comprehend and complete self-administered questionnaires.

Exclusion Criteria

Patients were excluded if they presented with: (1) acute coronary syndrome within the preceding three months; (2) severe heart failure (NYHA Class IV); (3) significant valvular heart disease; (4) diagnosed psychiatric disorders requiring pharmacological treatment; (5) cognitive impairment precluding reliable questionnaire completion; (6) recent coronary revascularization within six months; or (7) terminal illness with life expectancy less than one year.

Data Collection Instruments

Perceived Stress Scale (PSS-14): The 14-item Perceived Stress Scale was utilized to assess subjective perception of stress during the preceding month. Scores range from 0 to 56, with higher scores indicating greater perceived stress. Participants were categorized as low stress (0-18), moderate stress (19-27), or high stress (≥28).

Depression Anxiety Stress Scales (DASS-21): The 21-item DASS was administered to evaluate depression, anxiety, and stress dimensions. Each subscale contains seven items scored from 0 to 3, with subscale scores multiplied by two for interpretation according to established severity categories.

Seattle Angina Questionnaire (SAQ): The SAQ is a 19-item disease-specific quality of life instrument measuring five domains: physical limitation, angina stability, angina frequency, treatment satisfaction, and quality of life. Scores range from 0 to 100, with higher scores indicating better functional status.

Angina Episode Documentation: Participants maintained daily angina diaries for two weeks preceding the study visit, recording episode frequency, duration, precipitating factors, and nitroglycerin use.

Clinical and Laboratory Parameters

Demographic information including age, sex, educational status, and occupational category was recorded. Clinical data encompassing body mass index, blood pressure, heart rate, lipid profile, fasting glucose, and glycated hemoglobin were obtained from medical records. Coronary angiography reports were reviewed to document the extent of coronary artery disease.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 26.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Normality of data distribution was assessed using the Shapiro-Wilk test. Independent samples t-test and one-way ANOVA were employed for comparing continuous variables between groups. Chi-square test was used for categorical variable comparisons. Pearson correlation coefficients were calculated to assess relationships between stress scores and angina parameters. Multiple linear regression analysis was performed to identify independent predictors of angina frequency. A p-value less than 0.05 was considered statistically significant.

RESULTS

Baseline Characteristics

A total of 248 patients completed the study protocol. The mean age of participants was 58.42 ± 9.87 years, with 162 (65.3%) being male. Based on PSS-14 scores, 78 patients (31.5%) were classified as having low stress, 98 patients (39.5%) as moderate stress, and 72 patients (29.0%) as high stress. Baseline demographic and clinical characteristics stratified by stress category are presented in Table 1.

Table 1: Demographic and Clinical Characteristics by Stress Category

Variable	Low Stress (n=78)	Moderate Stress (n=98)	High Stress (n=72)	p-value
Age (years)	59.21 ± 10.12	58.34 ± 9.56	57.69 ± 9.94	0.612
Male, n (%)	54 (69.2%)	63 (64.3%)	45 (62.5%)	0.674
BMI (kg/m²)	27.34 ± 3.45	28.12 ± 4.23	28.67 ± 4.01	0.089
Systolic BP (mmHg)	132.45 ± 14.23	136.78 ± 15.67	142.34 ± 16.89	0.001
Diastolic BP (mmHg)	82.12 ± 8.45	84.56 ± 9.12	87.23 ± 10.34	0.003
Heart Rate (bpm)	72.34 ± 9.87	76.45 ± 10.23	82.12 ± 11.56	<0.001
Total Cholesterol (mg/dL)	186.45 ± 32.12	192.34 ± 35.67	198.78 ± 38.45	0.078
LDL-C (mg/dL)	112.34 ± 28.45	118.67 ± 30.12	124.45 ± 32.78	0.034
HbA1c (%)	6.42 ± 0.89	6.58 ± 0.94	6.87 ± 1.12	0.012
Diabetes mellitus, n (%)	28 (35.9%)	42 (42.9%)	38 (52.8%)	0.098
Hypertension, n (%)	52 (66.7%)	68 (69.4%)	56 (77.8%)	0.307
Current smoking, n (%)	18 (23.1%)	26 (26.5%)	24 (33.3%)	0.362
Multi-vessel disease, n (%)	32 (41.0%)	48 (49.0%)	42 (58.3%)	0.096
CCS Class II-III, n (%)	34 (43.6%)	56 (57.1%)	54 (75.0%)	<0.001

BMI: Body Mass Index; BP: Blood Pressure; LDL-C: Low-Density Lipoprotein Cholesterol; HbA1c: Glycated Hemoglobin; CCS: Canadian Cardiovascular Society

Stress Levels and Angina Parameters

The association between psychological stress measures and angina-related parameters is summarized in Table 2. Patients in the high-stress category demonstrated significantly higher weekly angina episode frequency compared to moderate and low-stress groups (4.82 ± 1.94 vs. 3.45 ± 1.56 vs. 2.31 ± 1.12, $p<0.001$). Similarly, angina severity scores, assessed on a 0-10 visual analog scale, were significantly elevated in high-stress patients (6.73 ± 1.45 vs. 5.12 ± 1.34 vs. 4.21 ± 1.38, $p<0.001$).

Table 2: Psychological Stress Measures and Angina Parameters

Parameter	Low Stress (n=78)	Moderate Stress (n=98)	High Stress (n=72)	p-value
PSS-14 Score	12.34 ± 4.23	23.45 ± 2.67	34.56 ± 4.89	<0.001
DASS-Stress Score	8.45 ± 3.12	16.78 ± 4.23	26.34 ± 5.67	<0.001
DASS-Anxiety Score	6.23 ± 2.89	12.45 ± 3.67	18.67 ± 4.89	<0.001
DASS-Depression Score	5.12 ± 2.34	10.34 ± 3.45	16.78 ± 4.56	<0.001
Weekly Angina Episodes	2.31 ± 1.12	3.45 ± 1.56	4.82 ± 1.94	<0.001
Angina Severity (VAS 0-10)	4.21 ± 1.38	5.12 ± 1.34	6.73 ± 1.45	<0.001

Episode Duration (minutes)	3.45 ± 1.23	4.67 ± 1.56	6.23 ± 2.12	<0.001
Weekly Nitroglycerin Use	1.89 ± 0.87	2.78 ± 1.23	4.12 ± 1.78	<0.001
SAQ-Physical Limitation	68.45 ± 14.23	56.34 ± 13.67	48.32 ± 12.67	<0.001
SAQ-Angina Frequency	72.34 ± 15.67	58.45 ± 14.89	45.67 ± 13.45	<0.001
SAQ-Quality of Life	65.78 ± 16.23	52.34 ± 14.56	42.12 ± 12.89	<0.001

PSS: Perceived Stress Scale; DASS: Depression Anxiety Stress Scales; VAS: Visual Analog Scale; SAQ: Seattle Angina Questionnaire

Correlation and Regression Analyses

Pearson correlation analysis revealed significant positive correlations between PSS-14 scores and weekly angina frequency ($r=0.524$, $p<0.001$), angina severity ($r=0.487$, $p<0.001$), and nitroglycerin use ($r=0.456$, $p<0.001$). Negative correlations were observed between PSS-14 scores and all SAQ domains. The correlation matrix is presented in Table 3.

Table 3: Correlation Matrix between Stress Scores and Clinical Outcomes

Variable	PSS-14	DASS-Stress	DASS-Anxiety	Angina Frequency	Angina Severity
PSS-14	1.000	0.712**	0.634**	0.524**	0.487**
DASS-Stress	0.712**	1.000	0.678**	0.489**	0.445**
DASS-Anxiety	0.634**	0.678**	1.000	0.398**	0.412**
Angina Frequency	0.524**	0.489**	0.398**	1.000	0.623**
Angina Severity	0.487**	0.445**	0.412**	0.623**	1.000
SAQ-Physical	-0.456**	-0.423**	-0.387**	-0.567**	-0.534**
SAQ-QoL	-0.512**	-0.478**	-0.445**	-0.612**	-0.578**

*$p<0.001$

Multiple linear regression analysis with angina frequency as the dependent variable revealed that PSS-14 score ($\beta=0.412$, $p<0.001$), DASS-anxiety score ($\beta=0.187$, $p=0.008$), age ($\beta=0.134$, $p=0.024$), and multi-vessel disease ($\beta=0.156$, $p=0.012$) were independent predictors. The model explained 38.4% of variance in angina frequency ($R^2=0.384$, $F=24.67$, $p<0.001$).

DISCUSSION

The present investigation provides compelling evidence supporting the significant association between psychological stress and the clinical manifestation of angina pectoris. Our findings demonstrate that elevated perceived stress levels are independently associated with increased angina frequency, greater symptom severity, and diminished disease-specific quality of life in patients with stable coronary artery disease.

The observation that high-stress patients experienced more than twice the weekly angina episode frequency compared to low-stress counterparts underscores the clinical relevance of psychological assessment in cardiovascular care. These findings align with the conceptual framework proposed by Dimsdale, emphasizing that psychological stress acts as a precipitant of acute cardiovascular events through multiple pathophysiological pathways [11]. The magnitude of this association, with perceived stress explaining over 17% of variance in angina frequency independently, suggests that stress management may represent a meaningful therapeutic target.

Our results corroborate previous laboratory-based studies demonstrating mental stress-induced myocardial ischemia. Vaccarino and colleagues reported that mental stress provoked myocardial ischemia in approximately 40% of patients with stable coronary artery disease, and this phenomenon was associated with adverse prognosis [12]. The current study extends these observations by demonstrating that chronic perceived stress, rather than acute laboratory-induced stress, similarly influences angina manifestation in naturalistic settings.

The pathophysiological mechanisms underlying stress-induced angina involve sympathetic nervous

system activation, resulting in increased myocardial oxygen demand through elevated heart rate and blood pressure, as observed in our high-stress cohort [13]. Additionally, catecholamine-mediated coronary vasoconstriction may reduce oxygen supply, exacerbating the supply-demand mismatch. Yeung and colleagues demonstrated that mental stress induces paradoxical vasoconstriction in atherosclerotic coronary segments, providing mechanistic insight into stress-precipitated ischemia [14].

The significant correlations between anxiety scores and angina parameters observed in this study are consistent with growing recognition of anxiety as a cardiovascular risk factor. Roest and colleagues, in their meta-analysis, demonstrated that anxiety was associated with a 26% increased risk of incident coronary heart disease [15]. Our findings suggest that anxiety may also influence symptom expression in patients with established disease, potentially through heightened symptom perception and reduced pain threshold.

Notably, Seattle Angina Questionnaire scores were significantly lower across all domains in high-stress patients, indicating substantial impairment in disease-specific quality of life. This observation has important clinical implications, as diminished quality of life is associated with increased healthcare utilization and adverse outcomes in coronary artery disease populations [16]. Interventions targeting psychological stress may therefore offer dual benefits of symptom reduction and quality of life improvement.

The clinical implications of these findings warrant consideration of routine psychological screening in patients with stable angina. Evidence-based stress management interventions, including cognitive-behavioral therapy and relaxation training, have demonstrated efficacy in reducing cardiovascular events and improving quality of life [17]. The INTERHEART study identified that incorporation of psychosocial risk assessment improved cardiovascular risk stratification beyond traditional factors [18].

Several limitations of this study merit acknowledgment. The cross-sectional design precludes establishment of causal relationships between stress and angina manifestation. Self-reported angina episodes may be subject to recall bias, and the single-center design limits generalizability. Future prospective studies incorporating ambulatory ischemia monitoring and stress interventions are warranted.

CONCLUSION

This study demonstrates that psychological stress is significantly associated with increased frequency and severity of angina pectoris episodes and substantially impairs disease-specific quality of life in patients with stable coronary artery disease. Perceived stress independently predicts angina manifestation after controlling for traditional cardiovascular risk factors, suggesting that psychological factors represent an important determinant of symptom burden. These findings support the integration of routine psychological assessment and stress management interventions into comprehensive cardiovascular care protocols. Healthcare providers should consider addressing psychosocial factors alongside conventional pharmacological management to optimize outcomes in patients with stable angina pectoris.

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