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Assessment of stress level, sleep quality and heart rate variability among the individuals with symptoms of depression

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ABSTRACT

Aims: Long-term stress is a contributing factor to the development of major depressive disorder. Persistent stress can lead to sleep disturbances, which in turn, sustain sympathetic nervous system activation and elevate cortisol levels. Stress, poor sleep quality, and depression adversely affect mental and physical health, leading to increased morbidity and mortality. This study aims to assess stress levels, sleep quality, and heart rate variability (HRV) among individuals with depressive symptoms.

Methods: This cross-sectional study enrolled 80 participants (mean age of 23.4±5.57 years) comprising 40 individiuals diagnosed with the symptoms of depression by a psychiatrist and 40 healthy controls. Stress levels, sleep quality, depression levels, and cardiac autonomic function were measured using the Perceived Stress Scale (PSS), the Pittsburgh Quality of Sleep Index (PSQI), the Patient Health Questionnaire-9 (PHQ-9) and the Hamilton Depression Rating Scale (HAM-D) for depression, and HRV analysis for cardiac autonomic function.

Results: Participants with depressive symptoms demonstrated significantly poorer HRV parameters compared to healthy controls ($p<0.05$), as indicated by lower root mean square of successive differences (31.74±18.74 vs. 44.57±23.79), higher low frequency (LF) power in normalized units (LF nu: 70.57±12.45 vs. 42.69±11.96), and lower high frequency (HF) power in normalized units (HF nu: 29.48±11.57 vs. 55.39±11.45). Depression severity was significantly higher in patients with depression compared to healthy participants including PHQ-9 scores (9.90±3.84 vs. 1.98±1.33) and HAM-D scores (12.05±2.83 vs. 4.10±1.68) ($p<0.001$). Additionally, patients with depression exhibited significantly elevated PSS scores (27.42±3.21 vs. 13.35±2.68) and PSQI scores (8.62±2.12 vs. 3.67±1.67), indicating higher stress levels and poorer sleep quality ($p<0.001$).

Conclusions: Individuals with depressive symptoms exhibited significantly lower HRV parameters, higher perceived stress levels, and poorer sleep quality compared to healthy controls. The findings suggest that depression is associated with altered autonomic function and increased stress perception. These results highlight the importance of monitoring HRV, stress, and sleep quality as potential biomarkers for depression severity and treatment response.

Introduction

Depression represents a paramount global health challenge, emerging as a leading cause of disability worldwide. According to the Global Burden of Disease study, the prevalence of depressive episodes exhibits a marked gender disparity, affecting 1.9% of men and 3.2% of women (1). The impact of major depressive disorder transcends socioeconomic boundaries, establishing itself as the primary cause of years lived with disability in 56 countries, the secondary cause in another 56, and the tertiary cause in 34 nations (2). Regional studies from South India have revealed particularly concerning statistics, with depression prevalence reaching 15.1% (3). Projections indicate that depression's burden on global health systems may soon surpass that of any other disease (1).

Contemporary societal transformations, characterized by rapid changes in work patterns and lifestyle dynamics, have precipitated an escalation in stress levels among the general population (15.1%) (3). These changes have heightened vulnerability to psychosomatic disorders (4). The manifestations of stress extend beyond psychological domains, potentially triggering a cascade of physical symptoms including headaches, gastritis, dermatological conditions, and insomnia, while also increasing risk for serious conditions such as ulcers, hypertension, cardiovascular disease, and cerebrovascular events (5). The chronic stress response involves complex metabolic alterations, potentially manifesting as autonomic dysfunction and disruptions in hormonal homeostasis, cytokine profiles, and cellular growth factor expression (6).

The relationship between depression and cardiovascular health has garnered significant attention, with studies consistently demonstrating reduced heart rate variability (HRV) in depressed individuals. The increased incidence of cardiovascular conditions among patients with depression suggests autonomic dysfunction as a potential pathophysiological mechanism (7). Regulation of heart rate and rhythm occurs through the intricate interplay between components of the sympathetic and parasympathetic nervous systems. While sympathetic activation accelerates heart rate, parasympathetic influence, primarily mediated through vagal pathways, provides counterbalancing deceleration. HRV, defined as the temporal variation between consecutive heartbeats measured via standard electrocardiogram, serves as a non-invasive tool for assessing cardiac autonomic status.

Research utilizing HRV as an objective measure of psychological stress has consistently revealed stress-induced alterations in HRV parameters, predominantly characterized by attenuated parasympathetic activity. Neuroimaging studies have strengthened this association by demonstrating connections between HRV and cortical regions involved in stress processing (8). This evidence base supports the validity of HRV as an objective tool for assessing psychological stress and overall health status.

The triad of mental stress, poor sleep quality, and depression presents significant risks to health and well-being. While these conditions can be effectively managed through medical and complementary therapeutic approaches, their early identification and monitoring remain crucial for preventing adverse outcomes. Despite the clear importance of objective stress assessment in depression, studies using physiological measures such as HRV in this population remain limited (9,10). This cross-sectional comparative study addresses this research gap by examining stress levels, sleep quality, and HRV in patients with depressive symptoms and in healthy controls.

Methods

Study Design and Participants

This cross-sectional study was conducted at Sri Ramachandra Medical College and Research Institute in Chennai from 2021 to 2023. The study enrolled 80 participants aged 18-35 years, aligning with the required sample size to detect a medium effect size (f^2=0.15) in regression analysis using 2-3 predictors with 80% power at α=0.05, and minimizing age-related physiological variations that could affect HRV measurements. Participants were divided into two groups: 40 individuals with depressive symptoms and 40 healthy controls.

The depression group comprised recently diagnosed patients (within the last month) who scored above 5 on the Patient Health Questionnaire-9 (PHQ-9) (11). These participants had not initiated antidepressant treatment and had no history of depression treatment. The control group consisted of apparently healthy individuals with PHQ-9 scores below 5. Exclusion criteria included signs of other organic mental disorders, the current use of medications known to affect HRV, and a history of cardiovascular disease or other conditions affecting HRV. All participants remained medication-free for at least two weeks prior to the study to minimize potential impacts on HRV measurements. Written informed consent was obtained from the patient prior to participation in the study. The study received ethical approval from the Sri Ramachandra Institute of Higher Education and Research Institutional Ethics Committee (approval no: CSP/22/MAR/108/248, date: 14.07.2022).

Outcome measurements

Perceived Stress Scale (PSS)

The PSS, developed by Cohen et al. (12), was used to assess stress levels. This 10-item instrument measures global perceived stress over the past 30 days on a 5-point scale ranging from 0 (never) to 4 (very often). Total scores range from 0-40, with higher scores indicating greater perceived stress. Score interpretation: 0-13: Low stress; 14-26: Moderate stress; 27-40: High perceived stress. The scale demonstrates robust internal consistency, with Cronbach's alpha ranging from 0.78 to 0.91 across studies (13).

Pittsburgh Quality of Sleep Index (PSQI)

Sleep quality was assessed using the PSQI (14), which comprises 19 self-rated questions and 5 bed partner/roommate-rated questions (only self-rated items contribute to scoring). Seven component scores are derived, each ranging from 0 (no difficulty) to 3 (severe difficulty). These components sum to a global score (range 0-21), with higher scores indicating poorer sleep quality. The PSQI has demonstrated good internal consistency, with a Cronbach's alpha of approximately 0.83.

HRV

HRV, which represents cardiac beat-to-beat variation (15), was measured using the AD instrument-Equivital device. Each participant underwent a standardized protocol that began with a 10-minute seated rest period, followed by the placement of Equivital device leads and the securing of the measurement belt. HRV recording was then conducted for 15 minutes. The analysis incorporated both time-domain and frequency-domain parameters. Time-domain analysis included the measurement of the average R-R interval (RR) and the standard deviation of normal-to-normal intervals intervals, which provide insights into overall HRV. For frequency-domain analysis, the fast Fourier transform was employed to calculate the power spectral density, with a specific focus on low frequency (LF) and high frequency (HF) power components, measured both in milliseconds and in normalized units. The LF/HF ratio was calculated to assess autonomic nervous system balance (16).

Depression assessment

Depression severity was evaluated through two complementary instruments. The PHQ-9 served as a self-administered assessment tool, consisting of 9 items based on DSM-IV criteria for major depressive disorder (17). This instrument provides scores ranging from 0 to 27, with higher scores indicating greater depression severity. Additionally, the Hamilton Depression Rating Scale (HAM-D) was administered by clinicians to provide a comprehensive assessment of depression symptoms (18,19). This scale consists of 17 to 21 items, depending on the version used, with scores ranging from 0 to 52 on the 17-item version. Higher scores on the HAM-D indicate greater depression severity, and the scale is particularly valued for its sensitivity to change over time. The PHQ-9 has demonstrated good internal consistency, with a Cronbach's alpha typically ranging from 0.86 to 0.89.

Study procedure

The study followed a systematic assessment sequence for all participants. Initially, both groups underwent depression screening using the PHQ-9 and HAM-D scales, administered by qualified psychiatrists. Following this, participants completed the PSS to assess their stress levels and the PSQI to evaluate sleep quality. The final component of the assessment involved HRV measurement, which was consistently performed between 9 and 11 A.M. for all participants to minimize diurnal variation in autonomic function.

Statistical Analysis

Results are expressed as mean ± standard deviation. Data analysis was performed using R statistical software version 3.1.1. The Kolmogorov-Smirnov test assessed data normality. Between-group comparisons utilized Student's t-test for parametric data. Statistical significance was set at $p<0.05$.

Results

The study included 40 healthy participants who served as controls, and 40 patients with depressive symptoms. The demographic comparison (Table 1) revealed no significant differences in age (mean age: 23.4±5.57 years; p=0.14) or gender distribution (49% female, 51% male; p=0.3) between the groups, indicating balanced baseline characteristics for age and sex. Table 2 presents a comparison of HRV parameters between the control and depression groups. Notably, root mean square of successive differences (RMSSD) values were significantly higher in healthy controls (44.57±23.79 ms.) compared to the depression group (31.74±18.43 ms.) (p=0.012), indicating enhanced parasympathetic activity in controls. LF power (n.u.) was significantly lower in healthy controls (42.69±11.96 n.u.) compared to the depression group (70.57±12.45 n.u.) (p<0.001), signifying imbalanced autonomic activity. HF power (n.u.) was significantly higher in healthy controls (55.39±11.45 n.u.) compared to the depression group (29.48±11.57 n.u.) (p<0.001), indicative of altered autonomic balance. The LF/HF ratio was significantly lower in healthy controls (0.85±0.39) compared to the depression group (2.99±1.88) (p<0.001), reflecting an imbalanced sympathetic-vagal ratio in depression. The depression group demonstrated markedly higher scores on PHQ-9 (9.90±3.84 vs. 1.98±1.33, p<0.001) and HAM-D (12.05±2.83 vs. 4.10±1.68, p<0.001), indicating greater depression severity (Table 3). Additionally, the depression group reported significantly higher perceived stress levels as measured by the PSS scale (27.42±3.21 vs. 13.35±2.68, p<0.001) and poorer sleep quality on the PSQI (8.62±2.12 vs. 3.67±1.67, p<0.001).

Table 1. Description of the study participants

Variable	Overall (n=80)	Healthy controls (n=40)	Patients with depression (n=40)	p-value
	Mean ± SD	Mean ± SD	Mean ± SD	
Age (years)	23.4±5.57	22.4±4.32	24.8±6.42	0.14
Gender	n (%)	n (%)	n (%)	
Female	39 (49)	17 (42)	22 (55)	0.3
Males	41 (51)	23 (57)	18 (45)	

SD: Standard deviation

Table 2. Comparison of short term HRV parameters between healthy control group and depression group

Characteristic	Overall (n=80)	Control (n=40)	Depression (n=40)	t-value/p-value
Mean R-R interval (ms)	753.21±90.84	765.52±92.18	740.91±8.92)	1.24/0.3
Average HR (bpm)	81.14±9.51	79.82±9.18	82.45±9.77	-1.24/0.3
RMSSD (ms)	38.15±22.11	44.57±23.79	31.74±18.43	**2.57/0.012**
Total power (ms^2)	3,280.61±3,637.95	3,686.86±4,148.27	2,874.35±3,043.36	1.02/0.3
LF power (ms^2)	1,143.22±1,327.13	1,061.27±1,385.21	1,225.18±1,278.71	-0.57/0.7
LF power (n.u)	56.63±18.55	42.69±11.96	70.57±12.45	**-10.46/<0.001**
HF power (ms^2)	1,160.71±1,982.54	1,589.15±2,192.62	732.28±1,666.62	**1.96/<0.001**
HF power (n.u)	42.44±17.34	55.39±11.45	29.48±11.57	**10.06/<0.001**
LF/HF ratio	1.92±1.73	0.85±0.39	2.99±1.88	**-7.07/<0.001**

R-R, R R interval, bpm: Beats per minute, RMSSD: Root mean square of successive differences, LF: Low frequency, HF: High frequency, ms: Milliseconds, nu: Normalized units

Table 3. Comparison the PHQ-9 scores, HAM- D scores, PSS scores and PSQI scores between healthy control group and depression group

Characteristic	Overall (n=80)	Control (n=40)	Depression (n=40)	t-value/p-value
PHQ 9	5.94±4.91	1.98±1.33	9.90±3.84	**12.40/<0.001**
HAM D	8.07±4.62	4.10±1.68	12.05±2.83	**15.36/<0.001**
PSS	20.39±7.67	13.35±2.68	27.42±3.21	**21.45/<0.001**
PSQI	6.15±3.13	3.67±1.67	8.62±2.12	**11.83/<0.001**

Data is represented as mean ± SD. Independent t test used
PHQ-9: Patient health questionnaire-9, HAM-D: Hamilton Depression Rating Scale, PSS: Perceived stress scale, PSQI: Pittsburgh Sleep Quality Index

Discussion

This study demonstrates significant autonomic dysfunction in depression, characterized by sympathetic predominance and reduced parasympathetic activity, as evidenced by increased LF power and LF/HF ratio alongside reduced RMSSD and HF power compared to healthy controls. These findings align with previous research by Wang et al. (20) and Kumar et al. (21), who reported similar autonomic imbalances in depression.

The observed autonomic dysfunction correlates with clinical severity, as evidenced by significantly higher scores on validated depression scales (PHQ-9 and HAM-D) in the affected group. The concurrent elevation in perceived stress levels and deterioration in sleep quality suggest a complex interplay between mood, stress, and autonomic regulation. These findings support the growing body of evidence linking depression with dysregulation of the autonomic nervous system.

Our study reveals a robust association between depression and elevated stress levels, with the depression group showing significantly higher PSS scores. This finding aligns with a previous study demonstrating that each unit increase in perceived stress corresponds to a 1.40-fold increase in the odds of depression (95% confidence interval: 1.35-1.44). The bidirectional nature of this relationship suggests that stress and depression may create a self-reinforcing cycle that exacerbates autonomic dysfunction (22).

Sleep disturbances emerged as another significant factor, with patients with depression showing markedly elevated PSQI scores. This finding corresponds with regional research indicating that individuals with depression face a 4.3-fold higher risk of poor sleep quality (23). The concurrent presence of sleep disruption, autonomic dysfunction, and depression likely reflects shared underlying neurobiological mechanisms, including alterations in neurotransmitter systems (serotonin, norepinephrine, and dopamine), hypothalamic-pituitary-adrenal axis dysregulation, and elevated inflammatory markers (24). This biological overlap may explain the observed clustering of symptoms and physiological changes.

Our results have important clinical implications. The distinct HRV profile associated with depression could serve as a biomarker for the early detection and monitoring of the condition. The marked reduction in parasympathetic activity (as indicated by decreased RMSSD and HF power) suggests that interventions aimed at enhancing vagal tone, such as HRV biofeedback or vagal nerve stimulation, may be beneficial. Furthermore, the strong associations between depression, stress, and sleep disturbances indicate that comprehensive treatment approaches addressing all these domains may be more effective than targeting depression alone.

Study Limitations

Several limitations should be considered when interpreting these findings. First, the cross-sectional design precludes determination of causality between autonomic dysfunction and depression. Second, the moderate sample size may limit the generalizability of the findings. Third, while we controlled for major confounders, other factors affecting HRV, such as physical activity levels and dietary habits, were not assessed. Fourth, the complex interactions between stress, sleep, and autonomic function make it challenging to isolate the primary drivers of the observed changes.

Conclusion

This study provides compelling evidence of the profound impact of depression on both physiological and psychological parameters. The observed alterations in cardiac autonomic function, characterized by sympathetic predominance and parasympathetic withdrawal, suggest a potential mechanism linking depression to increased cardiovascular risk. The significant associations with stress levels and sleep quality underscore the multifaceted nature of depression and highlight the importance of comprehensive treatment approaches. These findings contribute to our understanding of depression's biological underpinnings and may inform the development of more effective therapeutic strategies.

Ethics

Ethics Committee Approval: The study received ethical approval from the Sri Ramachandra Institute of Higher Education and Research Institutional Ethics Committee (approval no: CSP/22/MAR/108/248, date: 14.07.2022).

Informed Consent: Informed consent was obtained from the participants after explaining the detailed procedure of the study.

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Footnotes

Authorship Contributions

Concept: S.S, P.R., Design: S.J., P.R., Data Collection or Processing: M.R., D.K.S., Analysis or Interpretation: M.R., M.K., S.J., Literature Search: M.R., D.K.S., S.J., S.S., Writing: M.R., D.K.S., M.K., S.S., P.R.

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