ISSN (P&E): 1996-7195, (O): 2957-899X DOI: https://doi.org/10.53350/pjmhs02025191.22

ORIGINAL ARTICLE

Impact of Chronic Sleep Deprivation on Blood Pressure Variability and Early Cardiovascular Dysfunction among Medical Residents: A Clinical Observational Study

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This article may be cited as:
Farooq N, Ghazanfar A, Khalid U, Afzal R, Ahmad H; Impact of Chronic Sleep Deprivation on Blood Pressure Variability and Early Cardiovascular Dysfunction among Medical Residents: A Clinical Observational Study. Pak J Med Health Sci, 2024; 19(1):4-11.

Received: 09-10-2024
Accepted:23-12-2024
Published:01-02-2025

ABSTRACT

Background: Medical residents frequently experience chronic sleep deprivation because of extended duty hours, demanding workloads, and frequent night shifts. Insufficient sleep has been associated with autonomic dysregulation, elevated blood pressure, inflammation, and subclinical cardiac dysfunction, yet limited data exist in young, otherwise healthy physicians.

Objective: To assess the impact of chronic sleep deprivation on blood pressure variability (BPV), autonomic function, inflammatory markers, and early echocardiographic indicators of cardiovascular dysfunction among medical residents.

Methods: This clinical observational study was conducted from June 2023 to March 2024 at Sheikh Zayed Hospital, Lahore, and Fatima Memorial Hospital, Lahore. A total of 110 medical residents aged 24–35 years were enrolled through consecutive sampling. Participants were divided into a sleep-deprived group (≤6 hours/night; n = 65) and an adequate-sleep group (>6 hours/night; n = 45). Sleep duration was assessed using structured questionnaires and a 7-day sleep diary. All residents underwent office blood pressure measurement, 24-hour ambulatory blood pressure monitoring (ABPM), heart rate variability (HRV) analysis, hs-CRP testing, and transthoracic echocardiography.

Results: Sleep-deprived residents exhibited significantly higher 24-hour systolic and diastolic BP, increased BPV, and a greater prevalence of non-dipping patterns. Resting heart rate was elevated, and HRV indices (SDNN and RMSSD) were significantly reduced. hs-CRP levels were higher in the sleep-deprived group, indicating low-grade inflammation. Echocardiographic evaluation showed a significantly higher E/e' ratio and a greater presence of early diastolic dysfunction.

Conclusion: Chronic sleep deprivation adversely affects cardiovascular regulation in medical residents, contributing to increased BPV, autonomic imbalance, inflammation, and early diastolic dysfunction. Structured duty schedules and sleep optimization may reduce these risks.

Keywords: Sleep deprivation, blood pressure variability, medical residents, autonomic dysfunction, heart rate variability, diastolic dysfunction, hs-CRP, ambulatory blood pressure monitoring.

INTRODUCTION

Occupational insomnia has increasingly been seen as a professional risk in clinical training settings and especially in medical residences, where many doctors are forced to work long hours, during the night, and in fast rotations[1]. The nature of residency training is rather tough, as one has to perform cognition continuously, to make clinical decisions quickly and to be physically alert. These professional demands usually come at the expense of healthy sleeping patterns with the majority of residents regularly receiving shorter than the suggested 7-9 hours of sleep per night. In the long term, sustained inadequate sleep causes physiological stress which can hasten the change in the heart even on young people who are otherwise healthy[2,3].

New data is implications short sleep duration to various poor cardiometabolic results, such as high blood pressure, autonomic dysregulation, poor endothelial functionality, insulin insensitivity, systemic inflammation and long-term cardiovascular morbidity. A reduced blood pressure variability (BPV) being the short-term variation in blood pressure in 24 hours is one of the first effects that sleep loss can produce and can be easily measured[4]. Even in case of normal mean arterial pressure, BPV is now being considered as an independent predictor of cardiovascular risks, target organ damage and mortality. The high BPV individuals have a greater vascular stiffness, lower sensitivity towards the baroreceptor, and more sympathetic dominance that indicates a pathophysiological pathway between sleep disturbance and premature cardiovascular damage[5].

Medical residents are a so-called high-risk group where sleep deprivation is no coincidence but is even built into the training model. Research has demonstrated that night shift residents often complain of disrupted sleep, circadian dysphase, heightened sympathetic response, and impaired parasympathetic rejuvenation. These physiological impairments could predispose them to increased resting blood pressure, normal nocturnal dipping absence and increased BP variability. Nevertheless, the majority of studies published have been on subjective symptomology or fatigue self-report, and limited clinical information has been available on objective cardiovascular markers in this group[6,7].

The other extremely significant factor influenced by loss of sleep on a chronic basis is the functioning of the autonomic nervous system, which is commonly measured in terms of heart rate variability (HRV). Reduced HRV indicates dysfunctions of the parasympathetic tone and increased sympathetic activity situations strongly linked with the premature cardiovascular dysfunction. In much the same manner, low-grade inflammation, which is signaled by high-sensitivity C-reactive protein (hs-CRP), is typically up-regulated in people who experience chronic sleep debt and is in addition to vascular damage and cardiac remodelling[8,9].

Notably, minor heart defects especially left ventricular diastolic dysfunction can be acquired long before one can notice any symptom[10]. The E/e 7 ratio is an early indicator of myocardial relaxation impairment which can be detected using the Echocardiographic markers. It is critical to identify these malfunctions in young medical residents at an early stage of their careers since they can develop without symptoms over time when the occupational stressors are not handled immediately.

Although the issue is becoming more relevant, no studies have measured the relationship between chronic sleep deprivation, blood pressure variation, and premature cardiovascular dysfunction among medical residents in our area. Due to the overwhelming stress loads, the long hours of duties, and sleep deprivation among most of the training hospitals, there is an urgent need of evidence which could guide institutional policies and preventive measures[11,12].

Consequently, this clinical observational study will be used to assess the effects of chronic sleep deprivation on variation of blood pressure, autonomic activity, inflammatory factors, and early cardiovascular dysfunction echocardiogram in medical residents. These associations are crucial to learn to identify the risk early, well-being of residents, and long-term preservation of cardiovascular health in this vulnerable professional group[13].

MATERIALS AND METHODS

Study Design and Setting

The research was conducted in the Departments of Medicine and Cardiology of a tertiary care teaching hospital as a clinical observation, cross-sectional study. The study was planned to last between June 2023 and March 2024, and medical residents in different medicine related specialties such as Internal Medicine, Pediatrics, Emergency Medicine, and some allied fields were contacted to take part. The major goal was to assess the impact of chronic sleep deprivation on the variability of blood pressure and the early cardiovascular dysfunction by rodent ambulatory blood pressure monitoring and echocardiography.

Study Population

The sample size was a group of postgraduate medical residents who are actively pursuing structured training programs in the hospital. The eligibility requirements included being aged between 24 and 35, and having lived not less than six months, having no previous record of hypertension, diabetes mellitus, ischemic heart disease,

structural heart disease, endocrine disorders or chronic inflammatory conditions. The inclusion criteria were that the participants should have given written informed consent voluntarily and must also have consented to receive 24-hour ambulatory blood pressure monitoring (ABPM), echocardiography, sleep assessment, and laboratory tests. The residents were not allowed to have pregnancy, antihypertensive, antiarrhythmic, corticosteroid, or psychoactive drugs, or acute febrile illness in the last two weeks. Individuals whose ABPM recording was incomplete or who did not want to fill the necessary sleep questionnaire and sleep diary also were not taken into consideration.

Sample Size and Sampling Technique.

The study used a non-probability consecutive sampling to include all the eligible residents within the ten months study period. Finally, 110 medical residents met the study inclusion criteria and underwent the study protocol. The residents were divided into two categories (sleep-deprived and adequate sleep) based on their mean duration of nightly sleep during the last three months; sleep-deprived group was considered to be those with a mean of 6 hours or less nightly sleep, whereas adequate-sleep group was regarded as those with a mean longer than 6 hours of nightly sleep. The quality of sample size used 110 was enough to identify moderate blood pressure variability difference between the two groups with an alpha of 0.05 and power of about 80, considering the potential of attrition because of the lack of complete ABPM measures.

Evaluation of Sleep Length and Quality.

The sleep measurement involved two items. First, they were all assessed using structured sleep questionnaires, which gathered data about habitual bedtime and wake time, the number of night calls per week, frequency of 24-hour shifts, self-reported sleep quality, sleep onset time and caffeine or nicotine use, especially when it was late at night. Second, the participants were having a 7-day sleep diary just before using ABPM. The records in the diary were based on the daily sleep duration, night awakenings, subjective sleep quality, and total bed time. The questionnaire and data on the duty roster were compared with the researcher to determine the accuracy of average sleep duration calculated using the diary and compared to the data on questionnaire. All these measures were taken to give an overall assessment of the quantity of habitual sleep and circadian disruption.

Clinical Evaluation

Every resident was subjected to a comprehensive clinical assessment comprising medical and occupational history, lifestyle habits as well as anthropometric measurements. Data on smoking status, physical activity level, consumption of coffee or energy drinks and family history of cardiovascular disease was taken. Measurement of height and weight was conducted using calibrated scale and calculation of body mass index (BMI) was conducted by dividing the weight (in kilograms) by the height (in meters squared). Sphygmomanometer blood pressure of the office was measured at the seat position after five minutes rest, three measurements taken with one to two minute intervals and averaged to analyze the data. Although office blood pressure was used as the initial screening test, all the cardiovascular variability assessments were conducted with the help of 24-hour ABPM.

Ambulatory Blood Pressure Monitoring and Blood Pressure Variability.

All the participants were subjected to 24-hour ABPM with a validated oscillometric measuring device attached to the non-dominant arm. The measurements were automatically noted after every 20 minutes in daytime (06:0022:00) and after every 30 minutes in the nighttime (22:0006:00). Participants were asked to carry on with regular daily life activities and not to engage in vigorous activities and to maintain a relaxed arm position with every recording. They were given a monitoring diary where they were required to record the duration of sleep, symptoms, level of activity and the time of waking up. Based on the data of the ABPM, the mean 24-hour systolic and diastolic blood pressure was calculated, as well as the mean values of daytime and nighttime. Variability of blood pressure (BPV) was measured by the standard deviation (SD) and the coefficient of variation (CV) of 24 hours systolic and diastolic pressure. Nocturnal dipping status was established by comparing the mean daytime and nighttime pressure where participants were classified into dippers, non-dippers and reverse dippers by using the already set criteria. The pre-hypertensive blood pressure profiles were established based on known norms of 24-hour ABPM in persons with no history of hypertension.

Heart Rate Variability and Resting Heart rate.

The autonomic activity was evaluated based on 5-minute resting electrocardiography. All the residents were allowed to lie in a temperature-controlled, quiet room at least 10 minutes prior to the ECG recordings. The routine lead II recording was done and time-domain analysis was used to determine the parameters of heart rate variability (HRV). The standard deviation of all normal to normal intervals (SDNN) was taken as the measure of overall HRV and the root mean square of the successive differences (RMSSD) was the measure of parasympathetic/vagal activity. Reduced SDNN and RMSSD were taken to be signs of

autonomic imbalance and sympathetic dominance. The resting heart rate was also measured with the help of the same ECG tracing.

Laboratory Investigations

Venous blood samples were gathered where possible, in the morning after an overnight fast bearing in mind the different duty shifts of the residents. The tests that were conducted in the lab were fasting blood glucose, lipid profile (total cholesterol, LDL-C, HDL-C, and triglycerides), and high-sensitivity C-reactive protein (hs-CRP) as a low-grade systemic inflammatory biomarker. All the analysis was carried out in the biochemistry laboratory of the hospital by automated means and normal in-house quality control measures.

Echocardiographic Evaluation

All participants underwent transthoracic echocardiography by an expert cardiologist who was not aware of the treatment (sleep group) given to the participants. Biplane Simpson method was used to measure left ventricular ejection fraction (LVEF). Other parameters were left ventricular mass index (LVMI), transmitral inflow velocities (E and A waves), and tissue Doppler-measured early diastolic mitral annular velocity (e +). E/e 7 ratio was measured as an approximate of left ventricular filling pressures and acted as a major pointer of early diastolic dysfunction. The presence of a high E/e 7 even under the condition of preserved LVEF was considered to be an indication of the slightest abnormalities of relaxation of the myocardium.

Operational Definitions

Chronic sleep deprivation was considered to be the average nocturnal sleep period of six hours or less per night during three or more months with continuity. More than six hours of sleep per night was referred to as adequate sleep. Greater variability of blood pressure was characterized by greater SD and CV indices of systolic and diastolic pressures compared to the group that was adequately rested.

Statistical Analysis

Statistical software was used to analyse all data. Normalcy of continuous variables was assessed and they were represented by mean standard deviation or median interquartile ranges as typed variable, and frequencies and percentages as categorical variables. The independent-samples t-test or Mann-whitney U test as a continuous variable and Chi square or Fisher exact test as a categorical variable were used to compare the sleep deprived group with the adequate sleep group. The Pearson or Spearman coefficients of correlation were conducted to examine the relationships that exist between sleep duration and cardiovascular variables such as BPV, indexes of HRV, hs-CRP, and E/e. A p-value below 0.05 was regarded as important.

Ethical Considerations

The Institutional Ethics Review Committee reviewed and approved the study protocol. All the participants signed informed consent in writing before enrollment. The confidentiality of the data collected was maintained through the use of coded identifiers to the participants and all the information collected was kept securely. The participants, who were identified to have considerably high blood pressure or abnormal BP variability or echocardiographic abnormalities were advised and used to refer to the corresponding clinical services to receive proper follow-up and management.

RESULTS

Baseline Characteristics

One hundred and ten medical residents (65 in the sleep-deprived group (1106 hours per night), and 45 in the adequate-sleep group (1106 hours per night)) took part in the research. Table 1 shows the baseline demographic and anthropometric factors. The age between the two groups did not significantly differ (28.4 ± 2.5 years vs. 27.9± 2.7 years; p = 0.32). Gender distribution was such that the sleep-deprived group consisted of 35 males (54%), and 30 females (46%), and the adequate-sleep case consisted of 23 males (51%), and 22 females (49%), which did not indicate any significant imbalance in gender. Groups had similar values of body mass index. Sleep deprived residents also reported higher weekly night calls and low subjective sleep quality (p < 0.001 each). The people on sleep deprivation were also found to have higher caffeine intake on a daily basis (p = 0.002). The summarized results presented in Table 1 indicate that both groups were similar in terms of baseline demographics with the primary differences being associated with sleep and occupational stressors.

Office and Ambulatory Blood Pressure Findings

The blood pressure measurements that were taken in the office showed that there was a big difference in the systolic and diastolic pressure of the sleep-deprived residents and those who got enough sleep. Mean systolic pressure of blood in the office setting among the sleep-deprived group was 122 ± 9 mmHg compared to 117 ± 8 mmHg in the adequate-sleep group with a p-value of 0.004, mean diastolic pressure in the sleep-deprived group was 80 ± 6 mmHg compared to 76 ± 5 mmHg in the adequate-sleep group with a p-value of 0.002. The differences were further

exaggerated by assessing them with 24-hour ambulatory blood pressure monitoring. Table 2 in summary shows that the sleep-deprived group presented a significant higher mean 24-hour systolic and diastolic pressure, daytime, and nighttime averages. In addition, nocturnal blood pressure control was also disrupted with 40 percent of sleep-deprived residents becoming non-dippers as opposed to only 18 percent of those who had sufficient sleep (p = 0.01). These results emphasize the negative cardiovascular effect that chronic inadequate sleep has on blood pressure patterns.

Table 1. Baseline Characteristics of Study Participants (including Female Gender Distribution)

Variable	Sleep-Deprived (n = 65)	Adequate Sleep (n = 45)	p-value
Age (years)	28.4 ± 2.5	27.9 ± 2.7	0.32
Male (%)	35 (54%)	23 (51%)	0.78
Female (%)	30 (46%)	22 (49%)	0.78
BMI (kg/m²)	24.6 ± 2.9	24.1 ± 3.1	0.40
Night calls/week	4.0 ± 1.3	1.8 ± 1.0	<0.001*
Poor sleep quality (%)	41 (63%)	11 (24%)	<0.001*
Caffeine intake/day (cups)	3.1 ± 1.2	2.2 ± 1.1	0.002*

*Significant at p < 0.05

Table 2. Ambulatory Blood Pressure Measurements and Dipping Patterns

Parameter	Sleep-Deprived (n = 65)	Adequate Sleep (n = 45)	p-value
24-hour SBP (mmHg)	121 ± 7	115 ± 6	<0.001*
24-hour DBP (mmHg)	79 ± 5	75 ± 5	0.002*
Daytime SBP (mmHg)	124 ± 8	118 ± 7	<0.001*
Nighttime SBP (mmHg)	116 ± 6	110 ± 6	<0.001*
Non-dippers (%)	26 (40%)	8 (18%)	0.01*
Reverse dippers (%)	6 (9%)	1 (2%)	0.12

*Significant at p < 0.05

Blood Pressure Variability

Sleep-deprived group showed a high blood pressure variability, which is one of the primary prediction factors of vascular stress and early cardiovascular dysfunction. Sleep-deprived residents had a significantly higher standard deviation of 24-hour systolic blood pressures (13.1 ± 3.4 mmHg) than the adequate-sleep group (9.7 ± 2.8 mmHg; p < 0.001) did. The same levels were recorded in the diastolic variability indices and coefficients of variation. Interestingly, 42 percent of patients in the sleep-deprived cluster exhibited a pre-hypertensive profile of BP record on ABPM but only 20 percent of patients in the well-rested group (p = 0.008). These statistics confirm the assumption that sleep deprivation is the cause of not only high average blood pressure but also erratic cardiovascular regulation.

The Variations of the Heart rate and Resting Heart rate.

There was apparent evidence of autonomic imbalance in sleep-deprived residents. The sleep-deprived group had a significantly higher resting heart rate (79 ± 7 beats/min) than the residents did (72 ± 6 beats/min; p < 0.001). Moreover, with respect to heart rate variability, there was less parasympathetic modulation. The SDNN of the sleep-deprived group was lower (123 ± 20 ms) than that of the adequate-sleep group (138 ± 23 ms; p = 0.001). There were also large changes in RMSSD (25 ± 6 ms vs. 32 ± 7 ms; p < 0.001). These results confirm sympathetic dominance and poor autonomic recovery of medical trainees when sleep deprived chronically.

Laboratory Findings

There was significant elevation of high-sensitivity C-reactive protein levels among residents who were sleep-deprived with a median of 2.0 mg/L (IQR: 1.3–2.7) as compared to residents who were sleep-adequate with a median of 1.1 mg/L (IQR: 0.717). This increase is in line with a pro-inflammatory condition, which comes with chronic sleep deprivation. There were no significant differences in fasting glucose and lipid parameters in the groups but the sleep-deprived residents tended to have higher triglyceride levels.

Echocardiographic Findings

Echocardiographic evaluation showed no significant changes in left ventricular ejection fraction in all groups, but the measurements of diastolic functions differed. Table 3 shows that sleep-deprived residents had a significantly greater mean E/e7 ratio (9.5) than their rested counterparts (8.1) (p < 0.001) and it was proved that early relaxation of the heart was impaired in sleep-deprived residents. Also, 23 percent of sleep deprived participants had echocardiographic findings of early diastolic

dysfunction as opposed to 9 percent of participants under adequate sleep (p = 0.04). Sleep-deprived residents indicated a slight, insignificant increase in left ventricular mass index, indicating potential early remodelling alterations.

The correlation analysis revealed that there were significant negative relationships between the length of average sleep and essential cardiovascular. The duration of sleep was found to have a significant negative relationship with systolic blood pressures variability (r = -0.44, p = 0.001) and resting heart rate (r = -0.36, p = 0.002). The same inverse associations were observed with the levels of hs-CRP (r = -0.28, p = 0.006) and E/e prime (r = -0.30, p = 0.004) as found that a shorter sleep period was related to increased levels of inflammation, autonomic dysfunction, and early diastolic dysfunction.

Table 3. Echocardiographic Parameters of Participants

Parameter	Sleep-Deprived (n = 65)	Adequate Sleep (n = 45)	p-value
LVEF (%)	62 ± 4	63 ± 3	0.19
LVMI (g/m²)	87 ± 12	84 ± 11	0.18
E/e' ratio	9.5 ± 1.7	8.1 ± 1.4	<0.001*
Diastolic dysfunction (%)	15 (23%)	4 (9%)	0.04*

*Significant at p < 0.05

DISCUSSION

This was a clinical observational study that was used to explore the effects of chronic sleep deprivation on blood pressure variability, autonomic balance, inflammatory markers, and early echocardiographic indices of cardiac dysfunction in medical residents working in two large tertiary care hospitals based in Lahore[14]. The results reveal a stable trend of deleterious cardiovascular changes in sleep-deprived residents even in the individuals not having clinically apparent cardiovascular disease and being very young. These findings point to the fact that chronic lack of sleep is an important work-related health risk that should be taken seriously during the residency.

Among the most notable results of this research was the increased mean 24-hours systolic and diastolic blood pressure of sleep-deprived residents. Even though the values were within the pre-hypertensive level, the regular changes recorded on ABPM suggest that the cardiovascular control changed towards anomie. These findings are consistent with the prior studies that have shown that even slight sleep deprivation can result into an enhanced sympathetic drive, amplified catecholamine release, endothelial dysfunction, and reduced baroreflex responsiveness. Notably, measured ambulatory BP is a more reliable indicator of cardiovascular morbidity than office readings, and the differences that are observed are thus probably due to physiologically significant derangements as opposed to temporary variations[15,16].

The other important abnormality among the sleep-deprived group was blood pressure variability (BPV). High BPV, expressed in much higher standard deviations and coefficients of variation of systolic and diastolic blood pressures indicate instability in autonomic and vascular regulation[17]. The previous research has demonstrated that elevated BPV correlates with endothelial damage, arterial stiffness, microvascular remodeling, and augmented risk of cardiovascular incidents over time regardless of mean blood pressure. This is only seconded by the significantly greater number of non-dipping patterns in the sleep-deprived group, as a normal nocturnal dip is representative of a parasympathetic preeminence and cardiovascular rejuvenation. The absence of this physiological dipping is a proven antecedent of left ventricular hypertrophy, cerebral vascular disease and all-cause mortality[18].

The evidence of sympathetic overactivity and parasympathetic withdrawal in this study through heart rate variability (HRV) data is also additional evidence. Reduced values of SDNN and RMSSD suggest decreased vagal tone a symptom of autonomic imbalance. These results are aligned with the research in sleep physiology that demonstrates that sleep loss causes the inability to maintain normal autonomic cycling, which results in the long-term sympathetic stimulation, heart rate acceleration, and poor capability of the cardiovascular system to respond to physiological stressors. Even at an early stage, such autonomic disturbances have been linked to increased risks of arrhythmias, hypertension, and metabolic derangements[19,20].

There seems to be an inflammatory mechanism that accompanies the cardiovascular effects of sleep deprivation. The markedly high level of hs-CRP in the sleep-deprived group indicates the condition of low-grade chronic inflammation. It has been experimentally proven that sleep deprivation triggers the amplification of such inflammatory cytokines like IL-6, TNF-alpha and CRP[7]. These mediators of inflammation facilitate the dysfunction of vascular endothelium, oxidative stress, and atherosclerosis. This autonomic stress, increased BPV and inflammation combine in a multidimensional pathophysiological state and hastens early cardiovascular injury[9,11].

The results of the echocardiographic study in this paper are very convincing that chronic sleep deprivation can be a contributory factor to premature structural and functional malformations of the heart. Although there was no difference in left ventricular ejection fraction in both groups, sleep-deprived residents showed much higher E/e' ratios, which showed myocardial relaxation is impaired and early diastolic dysfunction[8,12]. This subclinical abnormality is important particularly because the diastolic dysfunction is an early predictor of overt heart failure with preserved ejection fraction (HFpEF) that is increasingly being identified in younger adults subjected to chronic stress and poor working hours. The greater incidence of early diastolic dysfunction in the sleep-deprived group provides support to the hypothesis that the insufficient sleep can cause a sequence of functional alterations way before structural remodelling is detected[15].

The similarity of these results in various cardiovascular pathways blood pressure regulation, autonomic activity, inflammation and myocardial activity indicates that chronic sleep deprivation has a cumulative, multisystem impact on cardiovascular health[17,18]. Medical residents are a highly susceptible group because of the long working hours, working during the night, psychological stress, and circadian rhythm disruption. Other nations have come up with similar studies that have also reported an augmentation of cardiovascular strain in resident physicians, a fact that supports the fact that the problem is universal and it is not tied to particular healthcare systems[12].

There are a number of mechanisms that can describe the identified associations. The chronic loss of sleep causes the stimulation of the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system resulting in increased cortisol, catecholamines and renin-angiotensin system. These changes in hormones enhance vascular tone, retain sodium, and disrupt the production of endothelial nitric oxide. At the same time, sleep deprivation interferes with the circadian regulation of blood pressure, heart rate and inflammatory rates. All these neurohormonal and immune imbalances cause chronic cardiovascular stress, regardless of conventional risk factors, including obesity, smoking, or dyslipidemia[13,15].

There is also significance of this study in occupational health. Long-term cardiovascular effects of chronic sleep deprivation are frequently neglected in residency training programs, which consider it as an unavoidable part of medical training. Nevertheless, the evidence provided herein indicates that lack of sleep could put young physicians at the risk of premature cardiovascular dysfunction, and possibly have an adverse effect on their health in the long term, productivity, and performance in patient care. These risks could be significantly addressed by means of systemic intervention including scheduled sleep time, fewer night shifts, safeguarded resting time and sleep hygiene educations[18,20].

This study has limitations even though it is strong. Its cross-sectional design does not allow to state a clear causal connection of sleep deprivation and cardiovascular abnormalities, however, the biological plausibility and consistency of findings prove a high association[4]. The duration of sleep was measured using questionnaires and sleep diaries as opposed to objective measures of sleep, including actigraphy and polysomnography. Moreover, the two involved hospitals were located within the same city thus generalization to other groups or specialties is not possible. However, the sample size, ABPM, HRV, hs-CRP, and echocardiographic measurements are strong indicators of the findings of the study[11,17].

On the whole, the results underline the fact that sleep deprivation is not only an inconvenience in terms of lifestyle but a serious cardiovascular risk factor even in young and otherwise healthy people. Further studies, particularly longitudinal ones, are critical to the understanding of whether such abnormalities can be reversed with the help of early interventions in sleep-deprived residents or at least averted the development of overt cardiovascular disease[20].

CONCLUSION

This paper has shown that chronic sleep deprivation of medical residents is correlated with a host of undesirable cardiovascular consequences, such as heightened mean blood pressure, heightened blood pressure variability, loss of normal nocturnal dipping, autonomic imbalance, increased inflammatory compounds, and premature signs of diastolic dysfunction. Even in young and clinically healthy residents, these abnormalities raise the risk of sleep deprivation as a significant though preventable cardiovascular risk factor. The residency programs should be aware of physiological load caused by insufficient sleep and introduce the system plans to enhance sleep periods, duty hours, and cardiovascular screening. Intervention early in the development of sleep deprivation could safeguard long-term cardiovascular health of the medical trainees and promote their well-being and clinical performance.

DECLARATION

Availability of Data and Materials

The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request. Data confidentiality has been maintained in accordance with institutional policy.

Competing Interests

The authors declare that they have no competing interests.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. The study was conducted using departmental resources available at Sheikh Zayed Hospital and Fatima Memorial Hospital.

Authors' Contributions

Nouman Farooq: Study conception, data collection, ABPM coordination, manuscript writing.
Ayesha Ghazanfar: Laboratory analysis supervision, HRV data interpretation, critical manuscript revision.
Uzair Khalid: Echocardiographic data acquisition, statistical analysis, results interpretation.
Rohma Afzal: Clinical data collection, resident recruitment, methodology refinement.
Haseeb Ahmad: Data management, literature review, manuscript editing and final approval.
All authors read and approved the final manuscript.

Acknowledgements

The authors gratefully acknowledge the support of the Departments of Medicine, Cardiology, and Biochemistry at Sheikh Zayed Hospital and Fatima Memorial Hospital for facilitating this study.

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