Pakistan Journal of Medical & Health Sciences Vol.20 (01), 2026 www.pjmhsonline.com

ISSN (P&E): 1996-7195, (O): 2957-899X DOI: https://doi.org/10.53350/pjmhs02026201.1

EDITORIAL

Sleep Disorders as an Emerging Cardiometabolic Risk Factor: Why Clinicians Must Take It Seriously

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This Editorial may be cited as: Shuja N; Sleep Disorders as an Emerging Cardiometabolic Risk Factor: Why Clinicians Must Take it Seriously. Pak J Med Health Sci, 2025;20(01):1-2.

Received: 02-08-2025
Accepted: 15-01-2026
Published: 30-01-2026

INTRODUCTION

Sleep has traditionally been viewed as a passive physiological state; however, contemporary research now positions it as a central component of cardiometabolic health[1]. Sleep disorders which include obstructive sleep apnea, insomnia, restless legs syndrome, circadian rhythm disturbances, and chronic sleep deprivation are increasingly recognized as independent risk factors contributing to metabolic syndrome, type 2 diabetes mellitus, hypertension, obesity, and cardiovascular morbidity[2]. Despite this growing body of evidence, sleep is one of the most neglected aspects of routine clinical evaluation, particularly in cardiology, endocrinology, and primary care settings[3]. As cardiometabolic diseases continue to rise in South Asia and worldwide, sleep must be considered a vital variable in both prevention and disease modification[4].

Sleep Disorders as the Missing Component in Cardiometabolic Assessment

Clinicians routinely focus on traditional risk determinants such as hypertension, dyslipidemia, smoking, sedentary lifestyle, hyperglycemia, and obesity[5]. However, sleep disturbances exert equally powerful effects on cardiometabolic physiology[6]. Many patients with poorly controlled hypertension, unexplained weight gain, or rapidly worsening metabolic syndrome often harbor unrecognized sleep disorders[7]. In South Asian clinical settings, sleep problems are under-screened primarily due to time constraints, lack of awareness, and insufficient integration into routine medical history-taking[8]. Yet, sleep disorders frequently precede or exacerbate metabolic dysfunction long before overt disease appears[9].

Biological Pathways Linking Sleep and Cardiometabolic Disease

Multiple interrelated mechanisms explain how disrupted sleep contributes to cardiovascular and metabolic pathology[10]. Sleep fragmentation, intermittent hypoxia, and circadian misalignment elevate sympathetic nervous system activity, producing sustained increases in blood pressure and heart rate even during daytime[11]. Poor sleep alters metabolic hormone balance by reducing insulin sensitivity, increasing glucose intolerance, and disrupting appetite-regulating hormones such as ghrelin and leptin[12]. These changes promote visceral adiposity, weight gain, and insulin resistance, driving the development of metabolic syndrome[13]. Furthermore, sleep disorders elevate inflammatory cytokines including IL-6, TNF-α, and CRP, creating a persistent low-grade inflammatory state that accelerates endothelial injury and atherogenesis[14]. Intermittent hypoxia associated with obstructive sleep apnea increases oxidative stress and impairs nitric oxide production, resulting in endothelial dysfunction[15]. These changes collectively disturb cardiac autonomic regulation, reducing heart rate variability and predisposing patients to arrhythmias, sudden cardiac events, and overall heightened cardiovascular risk[12].

Why Sleep Disorders Cannot Be Ignored in Clinical Practice

Ignoring sleep disorders leads to significant diagnostic and therapeutic gaps[15]. Many patients with resistant hypertension fail to respond to multiple antihypertensive agents simply because underlying obstructive sleep apnea has not been recognized[11]. Similarly, patients with diabetes may struggle with glycemic control despite optimal pharmacotherapy because untreated sleep problems worsen insulin resistance[13]. Obesity becomes more difficult to manage when hormonal disturbances caused by poor sleep perpetuate increased appetite and reduced energy

expenditure[5]. Sleep disorders also increase the rate of myocardial infarction, stroke, and heart failure, making early recognition essential[8]. Clinically, identifying sleep disturbances is far easier and more cost-effective than managing long-term complications of cardiometabolic disease[9], yet sleep remains an overlooked dimension[10].

Integrating Sleep Into Routine Clinical Evaluation

There is a pressing need to embed sleep assessment as a core component of clinical evaluation[14]. A few focused questions regarding snoring, breathing pauses during sleep, excessive daytime sleepiness, morning headaches, insomnia symptoms, and sleep patterns can significantly improve early detection[12]. Diagnostic pathways such as polysomnography, overnight oximetry, and actigraphy should be integrated into care protocols for high-risk patients, particularly those presenting with resistant hypertension, obesity, or metabolic syndrome[15]. Screening instruments such as the STOP-BANG questionnaire or the Epworth Sleepiness Scale provide rapid and clinically meaningful insights[6]. Incorporating sleep considerations into cardiometabolic risk stratification tools will improve diagnostic accuracy and allow clinicians to initiate earlier interventions[13].

Therapeutic Importance of Addressing Sleep Disorders

Treating sleep disorders can yield profound metabolic and cardiovascular benefits[11]. Continuous positive airway pressure (CPAP) therapy in obstructive sleep apnea reduces blood pressure, improves insulin sensitivity, enhances endothelial function, and lowers cardiovascular risk[15]. Non-pharmacological methods such as sleep hygiene training, cognitive behavioral therapy for insomnia, structured weight reduction programs, and circadian alignment strategies create long-term health improvements[12]. Addressing sleep problems in shift-workers, who represent a growing population in urban South Asia, can dramatically improve metabolic parameters and reduce cardiovascular strain[10]. Integrating sleep into multidisciplinary cardiometabolic management alongside exercise, diet, and pharmacotherapy provides a more complete and biologically aligned therapeutic approach[14].

CONCLUSION

Sleep disorders represent a rapidly emerging and clinically significant cardiometabolic risk factor[13]. The evidence linking sleep with cardiovascular and metabolic disease has become unequivocal, yet clinical practice remains slow to adapt[15]. A paradigm shift is urgently needed[9]. Clinicians must treat sleep as a measurable and modifiable vital sign, deserving the same attention as blood pressure, glucose levels, and lipid panels[11]. Early recognition, structured screening, and targeted therapy for sleep disorders can substantially reduce the burden of cardiometabolic diseases and improve patient outcomes[14]. The challenge now is not a lack of evidence, but the need for clinicians to translate this evidence into everyday practice[15]. Only by taking sleep seriously can we meaningfully advance cardiometabolic health and long-term disease prevention[1].

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