THE EMERGING ROLE OF SLEEP DISTURBANCES AND CLINICAL IMPLICATIONS IN PATIENTS WITH MYOCARDIAL INFARCTION

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Abstract. *Cardiovascular diseases remain the leading cause of global mortality, with myocardial infarction (MI) representing one of the most important contributors to premature death and disability. While traditional risk factors such as hypertension, diabetes mellitus, dyslipidemia, obesity, and smoking are well established, growing evidence indicates that sleep disturbances should also be recognized as significant and potentially modifiable cardiovascular risk factors. This narrative review summarizes contemporary evidence published between 2010 and 2025 regarding the relationship between sleep quality, sleep duration, sleep-disordered breathing, and myocardial infarction outcomes. Literature from PubMed/MEDLINE, Scopus, Web of Science, and eLIBRARY.ru demonstrates that poor sleep quality, short or prolonged sleep duration, insomnia, and obstructive sleep apnea are independently associated with increased risk of coronary artery disease and MI. Proposed mechanisms include autonomic imbalance, chronic inflammation, oxidative stress, endothelial dysfunction, insulin resistance, obesity, and prothrombotic changes. Sleep-disordered breathing is highly prevalent among patients with acute MI and is associated with worse cardiac remodeling and poorer prognosis. These findings suggest that routine assessment of sleep health may improve cardiovascular risk stratification and secondary prevention strategies in patients with MI.*

Keywords: *Myocardial infarction, sleep disorders, obstructive sleep apnea, insomnia, cardiovascular diseases, coronary artery disease, sleep quality, cardiac remodeling, inflammation, oxidative stress, secondary prevention.*

ВОЗРАСТАЮЩАЯ РОЛЬ НАРУШЕНИЙ СНА И ИХ КЛИНИЧЕСКОЕ ЗНАЧЕНИЕ У ПАЦИЕНТОВ С ИНФАРКТОМ МИОКАРДА

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Аннотация. *Сердечно-сосудистые заболевания остаются ведущей причиной смертности во всём мире, при этом инфаркт миокарда (ИМ) является одной из основных причин преждевременной смерти и инвалидности. Несмотря на то что традиционные факторы риска, такие как артериальная гипертензия, сахарный диабет, дислипидемия, ожирение и курение, хорошо изучены, накапливающиеся данные свидетельствуют о том, что нарушения сна также следует рассматривать как значимый и потенциально модифицируемый фактор сердечно-сосудистого риска.Данный нарративный обзор обобщает современные научные данные, опубликованные в период с 2010 по 2025 годы, касающиеся взаимосвязи качества сна, его продолжительности, нарушений дыхания во сне и исходов инфаркта миокарда. Анализ литературы из баз данных PubMed/MEDLINE, Scopus, Web of Science и eLIBRARY.ru показывает, что низкое качество сна, короткая или чрезмерно длительная продолжительность сна, бессонница, а также обструктивное апноэ сна независимо ассоциированы с повышенным риском ишемической болезни сердца и инфаркта миокарда.Предполагаемые патофизиологические механизмы включают дисбаланс вегетативной нервной системы, хроническое воспаление, оксидативный стресс, эндотелиальную дисфункцию, инсулинорезистентность, ожирение и протромботические изменения. Нарушения дыхания во сне широко распространены среди пациентов с острым инфарктом миокарда и связаны с более неблагоприятным ремоделированием сердца и худшим прогнозом.Полученные данные указывают на то, что рутинная оценка состояния сна может способствовать более точной стратификации сердечно-сосудистого риска и улучшению стратегий вторичной профилактики у пациентов с инфарктом миокарда.*

Ключевые слова: *Инфаркт миокарда, нарушения сна, обструктивное апноэ сна, инсомния, сердечно-сосудистые заболевания, ишемическая болезнь сердца, качество сна, ремоделирование миокарда, воспаление, окислительный стресс, вторичная профилактика.*

MIOKARD INFARKTIGA CHALINGAN BEMORLARDA UYQU BUZILISHLARINING ORTIB BORAYOTGAN ROLI VA KLINIK AHAMIYATI

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Annotatsiya. *Yurak-qon tomir kasalliklari global o'limning yetakchi sababi bo'lib qolmoqda, bunda miokard infarkti (MI) erta o'lim va nogironlikning eng muhim sabablaridan biri hisoblanadi. Gipertenziya, qandli diabet, dislipidemiya, semizlik va chekish kabi an'anaviy xavf omillari yaxshi o'rganilgan bo'lsa-da, so'nggi ilmiy dalillar uyqu buzilishlari ham muhim va potentsial boshqariladigan yurak-qon tomir xavf omili sifatida qaralishi kerakligini ko'rsatmoqda.Ushbu narrativ sharh 2010–2025-yillar oralig'ida chop etilgan zamonaviy ilmiy dalillarni umumlashtirib, uyqu sifati, uyqu davomiyligi, uyqu bilan bog'liq nafas buzilishlari va miokard infarkti natijalari o'rtasidagi bog'liqlikni yoritadi. PubMed/MEDLINE, Scopus, Web of Science va eLIBRARY.ru ma'lumotlar bazalaridagi adabiyotlar tahlili shuni ko'rsatadiki, uyqu sifati pastligi, uyquning qisqa yoki ortiqcha davom etishi, uyqusizlik (insomniya) hamda obstruktiv uyqu apnoesi koronar arteriya kasalligi va MI xavfining mustaqil oshishi bilan bog'liq.Taklif etilayotgan patofiziologik mexanizmlar qatoriga vegetativ (avtonom) muvozanatning buzilishi, surunkali yallig'lanish, oksidlovchi stress, endotelial disfunksiya, insulin rezistentligi, semizlik hamda trombozga moyillikning oshishi kiradi. Uyqu bilan bog'liq nafas buzilishlari o'tkir miokard infarkti bilan og'rigan bemorlarda juda keng tarqalgan bo'lib, yurakning qayta shakllanishi (remodeling)ning yomonlashuvi va prognozning og'irlashuvi bilan bog'liq.Ushbu natijalar shuni ko'rsatadiki, uyqu holatini muntazam baholab borish yurak-qon tomir xavfini aniqlashni yaxshilashi hamda MI bilan og'rigan bemorlarda ikkilamchi profilaktika strategiyalarini samaraliroq qilishga yordam berishi mumkin.*

Kalit so'zlar: *Miokard infarkti, uyqu buzilishlari, obstruktiv uyqu apnoesi, insomniya, yurak-qon tomir kasalliklari, koronar arteriya kasalligi, uyqu sifati, yurak remodelingi, yallig'lanish, oksidlovchi stress, ikkilamchi profilaktika*

Introduction.

Recent estimates from the World Health Organization indicate that cardiovascular diseases (CVDs) remain the foremost cause of mortality worldwide, accounting for nearly 19.8 million deaths in 2022. Among these, ischemic heart disease (IHD), including myocardial infarction (MI), contributes the largest share, with approximately 8.99 million deaths each year. Although improvements in healthcare systems have led to reduced mortality rates in many developed nations, the total number of myocardial infarction cases continues to increase globally. This trend is largely explained by demographic aging, along with the expanding prevalence of metabolic conditions such as obesity and diabetes, as well as sedentary and unhealthy lifestyle behaviors [1]. In Uzbekistan, cardiovascular diseases also dominate as the primary cause of death. Ischemic heart disease and myocardial infarction significantly contribute to early mortality, chronic disability, and increased healthcare demand. The country is part of the Central Asian high-risk cardiovascular zone, where coronary artery disease tends to develop earlier and outcomes are generally worse compared to Western populations. These disparities are likely influenced by limited preventive care, inadequate risk factor management, and differences in socioeconomic and lifestyle conditions [2]. India is experiencing a rapidly escalating burden of myocardial infarction, often affecting individuals at a younger age compared to Western countries. Conventional risk factors such as hypertension, diabetes, smoking, and lipid abnormalities remain highly prevalent; however, newer contributors—including chronic mental stress, urban environmental exposure (particularly air pollution), obesity, and sleep-related disorders—are increasingly recognized. These risk factors are especially significant among young and economically productive populations, thereby intensifying the public health impact. India contributes to nearly 20% of global myocardial infarction deaths, while cardiovascular diseases account for approximately one-quarter of total mortality in the country [3]. Furthermore, recent reports indicate a continued upward trend, with around a 12.5% rise in myocardial infarction-related mortality and a 65–71% increase in acute cardiac events in certain regions, underscoring the growing importance of emerging risk determinants [4].

Across Europe, the annual incidence of myocardial infarction in 2025 is estimated to range between 60 and 120 cases per 100,000 individuals, with noticeable differences between Western and Eastern regions. While age-adjusted mortality rates have declined due to advances in preventive cardiology, early detection, and modern interventional

techniques such as percutaneous coronary intervention, the overall number of cases remains considerable. This is mainly attributed to increasing life expectancy and the continued presence of modifiable cardiovascular risk factors within the population [5]. Current literature emphasizes that non-traditional and behavioral risk factors contribute to myocardial infarction through diverse biological pathways, including vascular endothelial injury, persistent low-grade inflammation, oxidative stress, dysregulation of the autonomic nervous system, and enhanced thrombogenicity. Elements such as psychological distress, depressive disorders, inadequate sleep, physical inactivity, environmental toxins, and post-infectious sequelae may independently elevate cardiovascular risk or interact with conventional factors to accelerate atherosclerosis. Recognizing these influences is essential for improving risk prediction models and developing comprehensive, patient-centered prevention strategies.In Uzbekistan, cardiovascular diseases constitute a significant public health challenge, with ischemic heart disease and myocardial infarction being leading contributors to both morbidity and mortality. The rising prevalence of major modifiable risk factors—including arterial hypertension, diabetes mellitus, obesity, tobacco use, and environmental exposures—indicates a likely further increase in myocardial infarction incidence if effective preventive strategies and early risk identification measures are not strengthened [6].In Europe, myocardial infarction predominantly occurs in older individuals and remains closely linked to conventional cardiovascular risk factors such as dyslipidemia, hypertension, smoking, diabetes mellitus, and obesity. Over the last two decades, mortality rates have declined substantially in Western Europe, largely due to advancements in preventive cardiology, early diagnosis, and improved acute coronary care interventions. Nevertheless, residual cardiovascular risk persists, partly due to the increasing influence of emerging factors such as obesity, chronic psychosocial stress, sleep disturbances, and environmental pollution, particularly in Eastern European regions [7].

Both Uzbekistan and India are categorized as developing nations experiencing a progressive rise in cardiovascular disease burden. In these populations, myocardial infarction is increasingly observed in younger age groups and is strongly associated with modifiable cardiometabolic and behavioral determinants. Despite these similarities, important differences exist in population demographics, lifestyle patterns, and healthcare infrastructure, which influence disease presentation and outcomes.Comparative analysis reveals distinct myocardial infarction risk profiles across Uzbekistan, India, and Europe. India demonstrates a disproportionately high burden of premature myocardial infarction, largely driven by metabolic syndrome, insulin resistance, type 2 diabetes mellitus, and central (abdominal) obesity. In contrast, Uzbekistan exhibits a risk pattern more strongly associated with hypertension and tobacco consumption, with an increasing contribution from urbanization-related lifestyle changes. Europe, despite having an aging population, benefits from well-established preventive cardiology programs and advanced healthcare systems, resulting in a comparatively lower incidence of premature myocardial infarction and improved clinical outcomes.

Materials and Methods.

This study was conducted as a narrative review aimed at summarizing current evidence regarding sleep quality, sleep disorders, and their relationship with quality of life among medical students. Additionally, the review explored the potential association between sleep disturbances and cardiometabolic outcomes, including cardiovascular risk factors and myocardial infarction.A systematic literature search was carried out using four major electronic databases: PubMed/MEDLINE, Scopus, Web of Science, and eLIBRARY.ru. Publications from 2010 to 2025 were considered eligible for inclusion in order to capture both foundational and recent research. The search strategy incorporated combinations of relevant keywords and Medical Subject Headings (MeSH) terms, including “sleep quality,” “sleep disorders,” “arterial hypertension,” “dyslipidemia,” “metabolic disorders,” “myocardial infarction,” and “quality of life.” These terms were used in various combinations to ensure comprehensive coverage of the topic.

Studies were included based on their relevance to sleep-related parameters and their association with cardiometabolic risk factors, particularly in individuals with myocardial infarction or acute coronary syndrome. Only peer-reviewed articles were considered to maintain scientific rigor.Given the narrative nature of this review, formal risk-of-bias assessment, quantitative quality scoring, and meta-analytic statistical analysis were not performed. Nevertheless, careful selection criteria were applied to ensure that only methodologically sound and clinically relevant studies were included in the final synthesis.

Results.

Non-traditional cardiovascular risk factors (RFs) are increasingly recognized as important determinants of overall cardiovascular risk (CVR), extending beyond classical factors such as hypertension, diabetes mellitus, and dyslipidemia. These include novel biomarkers, chronic systemic diseases (e.g., chronic kidney disease and chronic obstructive pulmonary disease), environmental exposures, persistent inflammation, infectious conditions, psycho-

social influences, and sex-specific factors. Collectively, these determinants contribute to improved understanding of risk prediction, disease progression, and clinical outcomes in cardiovascular disease (CVD) [8]. Given that conventional cardiovascular risk factors for myocardial infarction—such as arterial hypertension, obesity, dyslipidemia, diabetes mellitus, and tobacco use—are already well established, the present review emphasizes an underexplored yet clinically significant factor: sleep disturbances. Particular attention is given to impaired sleep quality and sleep disorders in patients with myocardial infarction, highlighting their role as independent risk modifiers and their association with disease severity, prognosis, and cardiovascular outcomes.Sleep deprivation (SD) has emerged as a significant public health concern in modern societies, often resulting from lifestyle behaviors or underlying sleep disorders, including insomnia, obstructive sleep apnea (OSA), and certain neurological conditions. Chronic SD represents a common pathological feature across these disorders and exerts multiple adverse biological effects. These include alterations in autonomic nervous system regulation, increased oxidative stress, enhanced inflammatory and prothrombotic responses, and accelerated atherogenesis, thereby establishing a mechanistic link between sleep disturbances and cardiometabolic diseases [9].

Epidemiological evidence indicates that adherence to a healthy sleep pattern is associated with an approximately 34–35% reduction in the risk of cardiovascular disease, coronary heart disease, and stroke, while nearly 10% of cardiovascular events may be attributable to inadequate sleep [10]. Furthermore, both short sleep duration (<7 hours) and prolonged sleep duration (>9 hours) have been linked to increased incidence of cardiovascular disease and higher mortality rates [11]. Recent cohort studies and meta-analyses demonstrate that sleep quality and sleep disorders independently predict cardiovascular risk, even after adjusting for traditional risk factors such as hypertension, diabetes, dyslipidemia, smoking, and obesity. In the early post-myocardial infarction phase, the myocardium remains particularly vulnerable to the adverse effects of sleep-disordered breathing (SDB). Pathophysiological consequences include increased myocardial workload, endothelial dysfunction, and imbalance between oxygen supply and demand. Even following successful percutaneous coronary intervention (PCI), patients with SDB exhibit prolonged ischemia, reduced myocardial salvage, and impaired ventricular remodeling, which may predispose to heart failure. Early application of positive airway pressure therapy has been shown to be feasible in this setting [12].

Sleep-disordered breathing is highly prevalent among patients with acute myocardial infarction (AMI), with registry data indicating a significant proportion of previously undiagnosed cases. Findings from the AMI-Sleep registry demonstrate that SDB is associated with poorer in-hospital outcomes, underscoring its clinical importance during the acute phase. These observations support the need for early screening and management of SDB in post-MI patients as a strategy to improve clinical outcomes [13]. Sleep disturbances are particularly common in the elderly population, especially among individuals aged ≥75 years [14]. In institutionalized older adults, the prevalence of poor sleep quality reaches approximately 65%, while insomnia and excessive daytime sleepiness affect around 43% and 30%, respectively [15]. Poor sleep has a substantial impact on both physical and mental health, as well as overall quality of life [16].

Most epidemiological studies evaluating sleep and coronary heart disease (CHD) risk have focused on middle-aged populations (mean age 45–58 years) [17]. Meta-analyses indicate that poor sleep quality is associated with a 25–45% increased risk of CHD, as well as elevated risk of hypertension and stroke [18]. Emerging clinical data further highlight the high prevalence of sleep disturbances among patients with acute coronary syndrome (ACS), including unstable angina, STEMI, NSTEMI, and MINOCA. Studies report significantly impaired sleep quality, reflected by elevated Pittsburgh Sleep Quality Index (PSQI) scores, prolonged sleep latency, reduced sleep duration, and increased daytime dysfunction. Conditions such as obstructive sleep apnea are also frequently observed. Importantly, these sleep abnormalities often coexist with traditional cardiovascular risk factors, suggesting that sleep disorders function as independent contributors to residual cardiovascular risk rather than merely secondary manifestations. Routine screening using validated tools such as PSQI and targeted evaluation for OSA may therefore have clinical utility. Early diagnosis and treatment may help reduce recurrent cardiovascular events and improve patient outcomes [19].

Cardiovascular diseases account for approximately 25% of all deaths in the United States, with coronary heart disease and stroke representing the largest contributors [20]. While traditional behavioral risk factors include poor diet, physical inactivity, and tobacco/alcohol use, sleep duration has recently been identified as an additional modifiable behavioral determinant of cardiovascular health. Data from NHANES indicate that both very short (<6 hours) and prolonged (≥9 hours) sleep duration are associated with poorer cardiovascular health scores, with only about 21% of adults meeting criteria for ideal cardiovascular health [21]. Sleep duration has

also been linked to cardiometabolic disease through several biological mechanisms, particularly via the development of a proinflammatory state. Both short and prolonged sleep duration have been associated with adverse health outcomes [22]. Sleep also plays a critical role in immune regulation, and disturbances in sleep may alter immune function in a manner that predisposes individuals to cardiometabolic disorders [23].

Furthermore, short sleep duration has been independently associated with elevated inflammatory markers (e.g., C-reactive protein, interleukin-6), increased insulin resistance, higher prevalence of obesity, and greater cardiovascular risk, even after adjustment for confounding factors such as smoking, body mass index, hypertension, and diabetes mellitus [24].Francesco P Cappuccio and colleagues proposed that short sleep duration contributes to adverse cardiovascular outcomes through at least two major biological pathways: Neuroendocrine dysregulation, particularly alterations in ghrelin and leptin secretion, leading to increased caloric intake, weight gain, and impaired glucose metabolism; and Activation of the hypothalamic–pituitary–adrenal (HPA) axis, resulting in elevated cortisol levels and disrupted growth hormone secretion [25].

Sleep restriction is associated with a hormonal imbalance characterized by increased ghrelin (orexigenic hormone) and reduced leptin (satiety hormone). This dysregulation promotes increased appetite, higher caloric intake, and subsequent weight gain, thereby elevating the risk of obesity and metabolic disorders [26]. Mechanistically, sleep deprivation disrupts hypothalamic regulation of energy homeostasis, enhancing food craving while impairing satiety signaling [27].Quantitatively, short sleep duration has been associated with a 15–30% reduction in leptin levels and a 20–35% increase in ghrelin levels, reinforcing its role in the pathogenesis of obesity and cardiovascular disease [28].In addition, sleep deprivation may independently induce a state of chronic low-grade inflammation, thereby increasing susceptibility not only to cardiovascular diseases but also to other chronic conditions, including malignancies. Conversely, the association between prolonged sleep duration and cardiovascular morbidity may be confounded by factors such as low socioeconomic status, unemployment, and undiagnosed comorbidities [29].

Accumulating evidence from large cohort studies and meta-analyses (2015–2024) indicates that insufficient sleep promotes a proinflammatory state, characterized by elevated circulating biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α). These inflammatory mediators contribute to the development of atherosclerosis, insulin resistance, and overall cardiometabolic dysfunction [30]. There is also evidence that genetic factors modulate the relationship between sleep patterns and inflammatory responses, with CRP levels being particularly sensitive to variations in sleep duration [31].

Acute coronary syndrome, including myocardial infarction, is characterized by alterations in the coagulation system. Following vascular injury, thrombus formation—composed of platelets and fibrin—plays a key role in hemostasis and vessel repair. However, dysregulation of this process may result in excessive thrombosis or bleeding.Sleep disturbances have been linked to activation of the hemostatic system, particularly through increased fibrinogen levels and thrombin-mediated fibrin formation, contributing to a prothrombotic state [32]. Elevated fibrinogen, synthesized by hepatocytes, has been strongly associated with increased cardiovascular risk and reflects underlying inflammatory activity [33]. Notably, long sleep duration (>8 hours) has also been associated with higher fibrinogen concentrations, as demonstrated in population-based studies [34]. Furthermore, fibrinogen has been shown to partially mediate the relationship between prolonged sleep duration and both coronary heart disease and mortality, accounting for approximately 6–7% of this association [35].

Atherosclerosis and Cytokine Activation

Sleep deprivation has been linked to proinflammatory pathways that promote atherosclerotic plaque formation, supported by both experimental and observational studies [36]. Sleep loss influences cytokine activity and contributes to a cluster of symptoms including fatigue, pain, depression, and metabolic dysregulation, all of which are associated with increased cardiovascular risk.An important mediator in this context is visfatin (nicotinamide

phosphoribosyltransferase), an adipokine involved in immune and metabolic regulation. Visfatin promotes the release of proinflammatory cytokines such as TNF-α, IL-1β, and IL-6, enhances insulin signaling, and is strongly associated with visceral adiposity. Evidence from the Cleveland Family Study demonstrated that visfatin levels increase by approximately 14% for every hour reduction in sleep duration, further linking sleep deprivation to inflammation and metabolic dysfunction [37]. Further prospective research and interventional studies are required to evaluate whether targeted management of sleep disorders can lower the risk of recurrent myocardial infarction, heart failure, and cardiovascular mortality.

Conclusion.

Current evidence supports the concept that sleep disturbances are not merely comorbid conditions but important independent modifiers of myocardial infarction risk and prognosis. Poor sleep

quality, abnormal sleep duration, insomnia, and obstructive sleep apnea contribute to adverse cardiometabolic, inflammatory, and hemostatic pathways that accelerate atherosclerosis and worsen post-infarction outcomes. Incorporating sleep screening into cardiovascular practice may help identify high-risk individuals earlier and improve both primary and secondary prevention. Future prospective studies and interventional trials are needed to determine whether targeted treatment of sleep disorders can reduce recurrent myocardial infarction, heart failure, and cardiovascular mortality.

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