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Insomnia as a Contributor to Hypertension and Somatic Distress: A Case Report

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ABSTRACT: Insomnia is increasingly recognized as a systemic disorder involving persistent hyperarousal with broad psychiatric and cardiovascular consequences. This case report describes a 49-year-old woman who developed functional dyspepsia after taking an analgesic, which led to chronic insomnia and, subsequently, essential hypertension (blood pressure reaching 173/93 mmHg) accompanied by generalized somatic distress. Her sleep pattern became non-restorative, with reduced sleep drive and dependence on hypnotics. Psychiatric evaluation revealed Nonorganic Insomnia, a Moderate Depressive Episode, and Somatization Disorder, with an Insomnia Severity Index score of 16. Her clinical course illustrates a psychophysiological cascade. Gastrointestinal distress disrupted sleep architecture, sustained sympathetic nervous system activation, and dysregulated the hypothalamic–pituitary–adrenal axis, thereby contributing to hypertension and somatic amplification. This case highlights the need to view insomnia as a systemic condition rather than an isolated symptom. Early recognition and non-pharmacological interventions—particularly Cognitive Behavioral Therapy for Insomnia (CBT-I)—may prevent long-term cardiovascular and psychiatric complications.

Keywords: Insomnia, Hypertension, Somatic Distress, Gut-Brain Axis, Cognitive Behavioral Therapy for Insomnia.

INTRODUCTION

Insomnia is increasingly recognized as a complex systemic disorder rather than a simple complaint of reduced sleep. In modern sleep medicine, it is defined as a 24-hour condition of sustained hyperarousal that affects physiological, cognitive, and emotional processes throughout the day. Patients with insomnia show elevated metabolic activity, higher cortisol secretion, increased heart rate, and heightened alertness even during the daytime, indicating that the disorder persists beyond nighttime sleep disturbance (St-Onge et al., 2016). Over the past decade, neurophysiological and

neuroimaging studies have consistently demonstrated that individuals with chronic insomnia exhibit increased activation in brain regions responsible for threat detection, autonomic regulation, and emotional processing, suggesting that the nervous system remains in a constant "fight-or-flight" state (Riemann et al., 2020). This state of persistent arousal disrupts sleep continuity, shortens restorative sleep stages, and generates a cycle in which the body becomes less able to initiate or maintain sleep despite physical fatigue(Hapsari & Kurniawan, 2019; Wuestenberghs et al., 2022).

Epidemiologically, insomnia affects an estimated 10–15% of the adult population worldwide as a clinical disorder and up to 35% when including chronic insufficient sleep or subthreshold symptoms (Palagini et al., 2021). The prevalence is higher among women, older adults, individuals exposed to chronic stress, and patients with chronic pain or functional gastrointestinal disorders(Zhu et al., 2021). Its economic and public health impact is substantial: insomnia is associated with increased disability, workplace absenteeism, medication dependence, and healthcare utilization. More importantly, it is strongly linked to long-term morbidity, including cardiovascular disease, psychopathology, and metabolic syndrome (Hirotsu et al., 2015).

Cardiovascular consequences are among the most serious systemic outcomes of insomnia. Multiple meta-analyses have shown that individuals with chronic insomnia face a 1.3–1.5 times higher risk of developing hypertension compared to good sleepers (M. Li et al., 2020; Q. Wang et al., 2015). Insomnia interferes with the normal nocturnal decline in blood pressure (known as the dipping pattern), increases sympathetic tone, and disrupts neuroendocrine function. Elevated catecholamines and chronic activation of the hypothalamic–pituitary–adrenal (HPA) axis lead to sustained cortisol secretion, vascular stiffness, and endothelial dysfunction (Okamoto & Uchida, 2022; Shiromani & Saper, 2019). Patients with insomnia who do not experience daytime sleepiness—paradoxically feeling "wired but tired"—demonstrate the most severe form of hyperarousal, and this subgroup is at the greatest cardiovascular risk (Javaheri & Redline, 2017).

In addition to cardiovascular consequences, insomnia has extensive links with gastrointestinal physiology through the gut–brain axis. The gut–brain axis represents a bidirectional communication system involving autonomic pathways, inflammatory signaling, neurotransmitters, and microbiota activity (Gheorghe et al., 2021). Functional gastrointestinal disorders such as reflux disease, irritable bowel syndrome, and dyspepsia frequently co-occur with insomnia(Sateia et al., 2021; Walker et al., 2022). Gastric discomfort, epigastric pain, nausea, or bloating may disrupt sleep initiation and cause repeated night awakenings (Fass & Dickman, 2005; Korsnes & Talley, 2022b). Conversely, inadequate or fragmented sleep increases visceral sensitivity, alters gastric motility, and lowers the threshold for pain perception. Sleep deprivation also disturbs microbiome composition, increasing inflammatory cytokines and reducing short-chain fatty acid production, which worsens gut discomfort (Sen & Smith, 2023).

These bidirectional mechanisms create a self-reinforcing loop: abdominal discomfort disrupts sleep; poor sleep increases gastrointestinal sensitivity; the heightened sensitivity strengthens discomfort; and discomfort intensifies insomnia. Over time, the system shifts into chronic dysregulation, where sleep and gut function deteriorate simultaneously. When this loop persists,

patients develop systemic symptoms including fatigue, palpitations, headache, muscle tension, and mood disturbances.

Psychologically, insomnia is associated with anxiety, depression, irritability, and cognitive decline, but in many Asian contexts—including Indonesia—emotional distress is often expressed through physical complaints rather than psychological vocabulary. Somatic idioms of distress are deeply embedded in cultural communication styles, where symptoms such as chest tightness, tremor, body heaviness, and gastric pressure carry emotional meanings (Fernandez-Mendoza & Vgontzas, 2013). As a result, insomnia may remain undiagnosed for months or years, because patients seek treatment for physical symptoms and not for sleep disturbance. This cultural pattern contributes to underdiagnosis, late treatment, and prolonged suffering.

The present case demonstrates how a seemingly local gastrointestinal disturbance can evolve into systemic illness mediated by insomnia(Chellappa & Aeschbach, 2021; Lombardi & Parati, 2005). A 49-year-old woman experienced functional dyspepsia after taking an analgesic. The visceral discomfort repeatedly disrupted her sleep until insomnia became chronic. Persistent hyperarousal contributed to the development of essential hypertension and pervasive somatic distress. Even after medical investigations showed no structural abnormalities, she experienced palpitations, chest tightness, gastric pressure, and muscle pain, illustrating the process of somatic amplification. The case highlights that insomnia should not be dismissed as a minor symptom but treated as a systemic condition with cardiovascular and psychosomatic implications(Espie et al., 2020; Korsnes & Talley, 2022a).

Early identification of insomnia is crucial for preventing chronic medical and psychiatric deterioration. Although hypnotic medications may provide short-term symptom relief, international guidelines recommend Cognitive Behavioral Therapy for Insomnia (CBT-I) as the first-line treatment because it corrects hyperarousal and maladaptive sleep behaviors (Trauer et al., 2015). Without behavioral treatment, insomnia becomes self-perpetuating, leading to medication dependence, emotional dysregulation, and physiological deterioration. This case underscores the need for holistic intervention that integrates gastroenterological, psychiatric, and behavioral approaches(Ali et al., 2013).

Theoretical Framework and Hypothesis

The multifaceted presentation of insomnia can be understood through a psychophysiological cascade framework, which proposes that an initial somatic disturbance triggers a chain of physiological and psychological dysregulations, ultimately resulting in multi-system illness. Unlike a simple comorbidity model that views concurrent disorders as separate entities, this framework emphasizes a sequential and reciprocal relationship among gastrointestinal, sleep, cardiovascular, and psychiatric systems.

In this context, functional gastrointestinal disorders—such as dyspepsia—may serve as the precipitating event. Visceral distress disrupts sleep-promoting neural pathways and activates the gut–brain axis, producing a state of conditioned hyperarousal (Fass & Dickman, 2005; Gheorghe

et al., 2021; Li, Zhang, & Chen, 2024). Persistent insomnia then contributes to sympathetic nervous system overactivity and hypothalamic–pituitary–adrenal (HPA) axis dysregulation, maintaining cortisol hypersecretion and increasing cardiovascular risk (Buckley & Schatzberg, 2005). This physiological overactivation enhances bodily vigilance, lowering the threshold for somatic perception and potentially masking underlying emotional distress—a phenomenon consistent with masked depression(Vgontzas & Chrousos, 2002).

The current case exemplifies this cascade. The patient's analgesic-induced gastric disturbance initiated persistent visceral discomfort, which disrupted sleep continuity. As her insomnia became chronic, sustained hyperarousal contributed to the onset of essential hypertension and amplification of somatic complaints. Over time, prolonged physical discomfort and unrefreshing sleep facilitated the emergence of depressive symptoms. This bidirectional feedback loop demonstrates how untreated insomnia can serve as both a mediator and amplifier of systemic illness.

METHOD

1. Case Identification

The case was obtained from the outpatient psychiatric clinic at RSI Jemursari, Surabaya, Indonesia. The patient provided written informed consent for the anonymized use of her clinical data for educational and publication purposes. Identifying information was omitted or altered to maintain confidentiality, in accordance with institutional and ethical standards.

2. Diagnostic Procedure

A comprehensive psychiatric evaluation was performed by a licensed psychiatrist using the Pedoman Penggolongan dan Diagnosis Gangguan Jiwa di Indonesia III (PPDGJ-III), harmonized with the International Classification of Diseases, 10th Revision (ICD-10). Medical comorbidities and sleep-related symptoms were cross-verified with hospital medical records.

3. Data Collection

Clinical information was collected through direct patient interviews, medical documentation, and psychiatric assessments. The severity of insomnia was measured using the Insomnia Severity Index (ISI) during her consultation in June 2025 (Clinical Records, Case Based Discussion, 2025; Clinical Records, Student Scientific Paper, 2025). Blood pressure and physical symptoms were documented across multiple visits to track clinical progression.

4. Case Documentation

The patient's narrative was documented verbatim where relevant. Culturally specific expressions—such as ampeg (gastric fullness), nderedeg (palpitations or trembling), and boyoken

(musculoskeletal ache)—were retained with contextual explanations to preserve the authenticity of her subjective experience while ensuring cross-cultural comprehension.

5. Data Analysis

A descriptive and interpretive approach was applied, guided by the psychophysiological cascade framework. Clinical observations were analyzed in relation to current evidence on insomnia, hypertension, and somatic distress to explore potential causal pathways and systemic interconnections.

RESULT AND DISCUSSION

The clinical presentation in this case demonstrates a systemic cascade of pathophysiological and psychological events rather than isolated symptoms. Although the patient initially sought treatment for gastric discomfort, the longitudinal trajectory—from functional dyspepsia to chronic insomnia, essential hypertension, and somatic distress—shows that insomnia served as a central mediator linking multiple organ systems. Understanding this pattern requires exploring the gut–brain axis, autonomic dysregulation, conditioned hyperarousal, and cognitive–emotional mechanisms that shape somatic perception.

The Gut–Brain Axis as the Primary Mechanism

The first disturbance occurred after analgesic-induced functional dyspepsia. Functional dyspepsia commonly triggers visceral hypersensitivity through vagal activation, mucosal irritation, serotonergic imbalance, and disrupted gastric motility (Korsnes & Talley, 2022). In this patient, nocturnal discomfort caused repeated awakenings. Each episode of gastric tension activated autonomic arousal, making it increasingly difficult to return to sleep. Over time, the brain learned to associate bedtime with discomfort and vigilance, forming the basis of conditioned insomnia.

The relationship is bidirectional. Poor sleep worsens gastrointestinal regulation by increasing gastric acid secretion, slowing motility, and intensifying visceral perception. Sleep deprivation also alters microbiome composition, increases inflammatory cytokines, and reduces short-chain fatty acids important for enteric regulation (Shi et al., 2025). Thus, dyspepsia was not merely a trigger—it became part of a self-reinforcing gut–sleep dysregulation loop (Fass & Dickman, 2005; Gheorghe et al., 2021).

Importantly, normal endoscopy or laboratory findings do not eliminate symptoms. In functional dyspepsia, discomfort is driven by visceral hypersensitivity and autonomic amplification rather than structural pathology. Therefore, the patient's recurrent abdominal tightness was physiologically real, even without anatomical abnormalities.

Insomnia as Sustained Hyperarousal

A hallmark of this case was the absence of daytime sleepiness despite prolonged sleep loss. Instead of feeling drowsy, the patient experienced persistent tension, restlessness, and heightened alertness—classic signs of hyperarousal. Chronic insomnia is characterized not by insufficient opportunity to sleep, but by continuous internal activation. Biologically, insomnia is linked to elevated sympathetic tone, increased cortisol, accelerated heart rate, increased metabolic activity, and persistent beta-wave EEG activity during attempted sleep (Basta et al., 2007).

This explains why patients often describe feeling "physically exhausted but unable to sleep." Their nervous system remains physiologically primed for threat detection, and sleep becomes inhibited even when the body is fatigued. Once established, this hyperarousal state becomes self-sustaining.

Somatization and Masked Depression

As the disorder evolved, the patient's presentation shifted from a purely sleep-related complaint to a combination of somatic and affective symptoms. She initially denied depressive mood but consistently emphasized physical sensations such as gastric fullness (ampeg), palpitations (nderedeg), and musculoskeletal pain (boyoken). These culturally rooted terms describe visceral tightness, autonomic arousal, and body heaviness—symptoms often associated with masked depression in non-Western contexts (Fernandez-Mendoza & Vgontzas, 2013; Wichniak et al., 2017). Chronic insomnia likely heightened somatic vigilance, amplifying bodily perceptions and reducing the threshold for discomfort.

From Hyperarousal to Essential Hypertension

Persistent sympathetic activation disrupts cardiovascular homeostasis. Under normal circumstances, blood pressure decreases at night due to parasympathetic dominance. Chronic insomnia prevents this nocturnal dipping. Individuals with nondipping patterns have significantly higher risks of stroke, renal impairment, and cardiac events (Khan & Aouad, 2022; L. Li et al., 2024).

In this patient, blood pressure reached 173/93 mmHg and remained elevated. There was no prior history of hypertension, indicating that insomnia likely contributed to its emergence. Experimental studies demonstrate that short-term sleep restriction can elevate blood pressure in healthy adults, while chronic insomnia increases renin–angiotensin activation, oxidative stress, and cytokine release (C. Wang et al., 2022). Thus, insomnia functions as an independent cardiovascular risk factor.

Once hypertension developed, symptoms such as palpitations and pulsatile pressure sensations intensified nocturnal arousal. The patient began monitoring physical sensations, anticipating discomfort, and worrying about cardiovascular danger, which further disrupted sleep. The cycle became self-reinforcing: insomnia elevated blood pressure, while hypertension increased nocturnal symptom vigilance and prevented sleep.

Somatic Distress and Interoceptive Hypervigilance

A central feature of this case was the escalation of somatic symptoms. The patient described abdominal pressure, internal trembling, and musculoskeletal heaviness. These sensations reflected interoceptive hypervigilance—heightened awareness of internal bodily signals. In healthy sleepers, most visceral sensations are filtered below conscious awareness. In chronic insomnia, cortical filtering weakens, and neutral sensations become salient or even alarming.

Once sensations are interpreted as dangerous, sympathetic activation increases and the discomfort intensifies. This leads to a somatic amplification loop:

1. A neutral internal sensation appears
2. The sensation captures attention
3. The brain interprets it catastrophically
4. Autonomic activation increases
5. The sensation becomes stronger and more distressing
6. The intensified sensation confirms fear, reinforcing the cycle

Sleep deprivation also reduces the pain threshold and suppresses endogenous analgesia, which explains why mild muscular tension becomes experienced as heaviness or pain (Vitale & Varrasi, 2021).

Why Repeated Medical Evaluations Do Not Break the Cycle

Patients often undergo extensive medical examinations. Although normal results may temporarily reassure clinicians, they rarely reduce patient hypervigilance. In some cases, negative medical findings increase anxiety because patients assume the problem is hidden or serious. Anxiety-driven monitoring of bodily sensations—checking heartbeats, blood pressure, or gastric movements—activates sympathetic pathways, reinforcing insomnia.

A more effective clinical approach is validating the symptoms while explaining the functional mechanism: the sensations are real, but caused by autonomic dysregulation and hypersensitivity rather than organ damage.

Failure of Medication-Only Approaches

Throughout this case, hypnotics provided temporary sedation but did not solve the underlying hyperarousal. Long-term use of benzodiazepines and Z-drugs is associated with tolerance, reduced slow-wave sleep, rebound insomnia, and psychological dependence (Wichniak et al., 2017). When medication ceases to work, patients often increase dosage or combine drugs, further worsening physiological instability.

Because insomnia is a learned and conditioned disorder, behavioral interventions are required to retrain sleep regulation.

Why CBT-I Fits the Pathophysiology

CBT-I is considered the first-line treatment for chronic insomnia and provides sustained improvement across medical populations (Wu et al., 2020). It directly addresses the three core mechanisms of chronic insomnia:

Mechanism	Manifestation in Patient	CBT-I Intervention
Conditioned hyperarousal	The bedroom triggers tension and alertness instead of sleep	Stimulus control
Excessive time awake in bed	Lying awake for long periods while restless	Sleep restriction therapy
Catastrophic beliefs	"If I cannot sleep, my blood pressure will rise and my heart will fail"	Cognitive restructuring

CBT-I reduces nocturnal sympathetic activity, restores circadian stability, increases homeostatic sleep pressure, and eliminates maladaptive sleep behaviors. Unlike pharmacotherapy, CBT-I improves sleep architecture and remains effective long after treatment ends.

Integrated Management Framework

Optimal management in this case would involve:

- Gastroenterological care for dyspepsia and microbiome regulation
- Cardiology evaluation for nondipping hypertension
- CBT-I and psychoeducation to reduce hyperarousal
- Gradual reduction of hypnotics to prevent rebound insomnia
- Lifestyle modifications including caffeine reduction, exercise, and light exposure therapy

Recent literature recommends multidisciplinary integration because insomnia increases morbidity across gastrointestinal and cardiovascular domains.

Clinical Implications

This case illustrates that insomnia should be screened in patients presenting with persistent gastrointestinal symptoms, fluctuating blood pressure, unexplained bodily discomfort, or dependence on sleep medication. A brief sleep assessment may prevent unnecessary diagnostic procedures and reduce emotional and financial burden.

This patient's condition can be understood as a psychophysiological cascade:

1. Functional dyspepsia initiated sleep disruption
2. Repeated awakenings developed into chronic insomnia
3. Insomnia triggered sustained hyperarousal
4. Hyperarousal produced hypertension and somatic amplification
5. Medication dependence maintained the cycle

6. Symptoms persisted despite normal medical findings

Insomnia, therefore, functioned as a causal driver of systemic dysfunction—not a secondary complaint. Early identification and non-pharmacological treatment are essential to prevent long-term morbidity.

CONCLUSION

This case illustrates how insomnia can function as a central disorder mediating and intensifying systemic illness. Initiated by visceral discomfort, the patient's chronic insomnia sustained physiological hyperarousal, which contributed to essential hypertension and somatic distress. The psychophysiological cascade observed here emphasizes that insomnia is not merely a sleep complaint but a systemic dysregulation with both cardiovascular and psychiatric implications.

Clinical Implications

Clinicians should recognize insomnia as a potential gateway condition linking physical and psychological morbidity. Early identification and comprehensive management—integrating gastroenterological, psychiatric, and behavioral perspectives—are essential to prevent chronicity. Non-pharmacological interventions, particularly Cognitive Behavioral Therapy for Insomnia (CBT-I), should be prioritized over long-term hypnotic use. Screening for insomnia is recommended among patients with functional gastrointestinal disorders, essential hypertension, and unexplained somatic complaints, as timely intervention may prevent downstream systemic complications.

Research Implications

Future studies should explore the bidirectional mechanisms connecting the gut–brain axis, sleep regulation, and cardiovascular function. Longitudinal designs and biomarker assessments (e.g., cortisol rhythm, autonomic variability) could clarify causal pathways between insomnia and systemic dysregulation. Broader case series may also help delineate cultural variations in the expression of somatized insomnia and masked depression.

Limitations

As a single case report, the findings cannot be generalized to wider populations. However, the detailed documentation of temporal and psychosomatic dynamics offers valuable insight into the integrative understanding of insomnia as a systemic disorder.

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