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Dyspepsia as a Trigger for Sleep Disorders: A Case Study of Gut–Brain Axis Interaction

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ABSTRACT: Functional dyspepsia is an upper gastrointestinal complaint without organic abnormalities that may interact with sleep through gut–brain axis mechanisms. We report the case of a 49-year-old woman with persistent insomnia occurring concurrently with epigastric pain for approximately one year. The patient relied on hypnotic medication and experienced early-morning awakenings. Psychiatric, somatic, and psychosocial evaluations indicated that the sleep disturbance was related to dyspeptic complaints. Management included psychoeducation, supportive psychotherapy, and Cognitive Behavioral Therapy for Insomnia (CBT-I), along with dyspepsia therapy (sucralfate and omeprazole) and a spiritual approach according to the patient's preferences. Following integrated therapy, sleep patterns improved and the Insomnia Severity Index score decreased in parallel with the resolution of dyspepsia symptoms. This case demonstrates that dyspepsia can trigger sleep disturbances through gut–brain axis mechanisms and highlights the importance of a multidisciplinary approach in managing insomnia with gastrointestinal comorbidity. The report also emphasizes the clinical relevance of addressing biopsychosocial factors and incorporating culturally sensitive, patient-centered care to enhance therapeutic outcomes and prevent symptom recurrence.

Keywords: Gut–Brain Axis, Functional Dyspepsia, Insomnia, CBT-I

INTRODUCTION

Sleep represents a fundamental physiological process indispensable for cognitive performance, immune regulation, emotional stability, and systemic homeostasis. Emerging research increasingly highlights sleep not as an isolated neurological function, but as a complex process deeply intertwined with peripheral physiological systems, most notably through the gut-brain axis (GBA)(Annisa Pratiwi et al., 2022). This bidirectional communication network, facilitating constant crosstalk between the central nervous system and the gastrointestinal tract, plays a pivotal role in maintaining both sleep architecture and digestive health. Disruptions within this axis,

particularly those originating from functional gastrointestinal disorders like dyspepsia, can profoundly dysregulate sleep patterns. The high prevalence of sleep complaints among individuals with functional dyspepsia (FD) underscores a significant clinical interplay, suggesting shared underlying mechanisms beyond mere discomfort. Consequently, comprehending the multifactorial origins of sleep disturbances, especially through the lens of the GBA, is paramount for developing nuanced and effective therapeutic interventions for patients presenting with this comorbidity.

Recent studies highlight the role of the gut–brain axis (GBA) in modulating sleep quality through neuroimmune, metabolic, and neuroendocrine signaling pathways. The GBA represents a bidirectional communication network that integrates neural, hormonal, and immunological signals between the gastrointestinal tract and the central nervous system. This axis involves multiple components, including the vagus nerve, the enteric nervous system, the hypothalamic-pituitary-adrenal (HPA) axis, and various neuroactive compounds produced by gut microbiota(Santos & Galiè, 2024). Alterations in the gut microbiota can influence neurotransmitter production, vagal activity, and circadian rhythm regulation, contributing to sleep disturbance (Cryan et al., 2019; Wang et al., 2022). This bidirectional system underscores the close relationship between gastrointestinal function and central nervous system activity, suggesting that disturbances in one domain can significantly impact the other. Functional dyspepsia often coexists with psychological symptoms such as anxiety and depression (Nelvita Sari et al., 2017; Rezeki et al., 2021), which may further exacerbate gastrointestinal discomfort and disrupt sleep regulation.

Functional dyspepsia (FD) is a common gastrointestinal disorder frequently associated with sleep problems, including difficulty initiating and maintaining sleep. Clinical evidence shows that poor sleep quality is strongly correlated with symptom severity in FD (Wuestenberghs et al., 2022), and similar findings have also been reported in Indonesian populations (Nurjanah et al., 2024; Damayanti et al., 2025). Furthermore, emerging research suggests that gut microbiota dysbiosis may be a shared underlying mechanism, with a recent population-based study confirming that specific alterations in microbial composition are independently associated with both FD symptomatology and poorer sleep quality (Zhu et al., 2023). These observations suggest that sleep disturbance in FD may be related not only to abdominal discomfort but also to underlying neurophysiological mechanisms within the GBA. The relationship appears to be bidirectional, with sleep disturbances exacerbating gastrointestinal symptoms and vice versa, creating a self-perpetuating cycle that can significantly impair quality of life.

However, despite emerging evidence linking dyspepsia and sleep disturbances, case-based explorations that integrate biopsychosocial interpretation and therapeutic outcomes remain limited. Most research has focused on epidemiological associations rather than detailed mechanistic explorations in individual cases. Furthermore, there is a scarcity of literature describing integrated treatment approaches that address both gastrointestinal and sleep symptoms simultaneously. This case report aims to describe a patient with dyspepsia-related insomnia and explore the potential role of GBA dysregulation in symptom development, as well as discuss a multidimensional treatment approach that addresses the complex interplay between biological, psychological, and social factors(Walker et al., 2022). By providing a detailed account of clinical presentation, diagnostic reasoning, and therapeutic interventions, this report seeks to contribute to a more nuanced understanding of the GBA in clinical practice.

METHOD

This case study involved a 49-year-old woman presenting with persistent insomnia associated with dyspeptic symptoms. The study was conducted in accordance with ethical principles for case reporting, and written informed consent was obtained from the patient for publication of her clinical information. The methodological approach combined comprehensive clinical assessment with targeted interventions and longitudinal follow-up to evaluate treatment response.

Clinical evaluation included detailed psychiatric interviews, physical examination, and assessment of psychosomatic and gastrointestinal complaints(Murni, 2020). The psychiatric interview employed a semi-structured format to explore the onset, course, and characteristics of sleep disturbances, as well as associated psychological factors. Particular attention was paid to the temporal relationship between gastrointestinal symptoms and sleep problems, the patient's emotional state, cognitive patterns related to sleep, and behavioral responses to symptoms. Physical examination focused on identifying potential organic causes of both dyspeptic symptoms and sleep disturbances, with special consideration given to neurological, endocrine, and gastrointestinal systems.

Sleep disturbance was assessed using the Insomnia Severity Index (ISI), a validated 7-item self-report questionnaire that evaluates the severity of both nighttime and daytime components of insomnia. The ISI provides a quantitative measure of insomnia severity, with scores ranging from 0 to 28, categorized as no insomnia (0-7), subthreshold insomnia (8-14), moderate insomnia (15-21), and severe insomnia (22-28). This instrument was administered at baseline and following intervention to objectively document treatment response.

The case was analyzed through the gut–brain axis framework based on current literature. This involved examining how alterations in gastrointestinal function might influence sleep regulation through neural, immune, and endocrine pathways, and conversely, how sleep disturbances might exacerbate gastrointestinal symptoms. The analysis considered multiple dimensions of the GBA, including visceral hypersensitivity, gut microbiota composition, HPA axis activity, vagal tone, and neuroinflammatory processes (Xu et al., 2025).

The patient received a multimodal treatment approach that integrated biological, psychological, and social interventions. Psychological interventions included psychoeducation about the gut–brain axis and its relationship to sleep, supportive psychotherapy to address emotional factors related to her symptoms (Harkomah et al., 2021; Nasution et al., 2021), and Cognitive Behavioral Therapy for Insomnia (CBT-I).CBT-I was delivered over six weekly sessions and focused on stimulus control (strengthening the bed as a cue for sleep), sleep restriction (limiting time in bed to actual sleep time), cognitive restructuring (addressing maladaptive beliefs about sleep), and relaxation techniques. Pharmacological therapy for dyspepsia (sucralfate and omeprazole) was continued in collaboration with internal medicine, with regular monitoring of treatment adherence and response. Cultural and spiritual preferences were accommodated as supportive measures, including incorporating prayer and mindfulness practices that aligned with the patient's values and beliefs.

Treatment response was monitored clinically through regular follow-up appointments, with emphasis on changes in sleep patterns (sleep onset latency, wake after sleep onset, total sleep time, sleep efficiency) and gastrointestinal symptoms (frequency and severity of epigastric pain, bloating, discomfort). The ISI was readministered at 4 and 8 weeks post-intervention to quantitatively assess changes in insomnia severity. Additional outcome measures included subjective reports of sleep quality, daytime functioning, and gastrointestinal symptom burden.

RESULT AND DISCUSSION

Patient Information. A 49-year-old married woman presented with complaints of persistent insomnia for approximately one year. She reported difficulty initiating sleep without medication and frequent early-morning awakenings. The patient described her insomnia as having a gradual onset, with symptoms worsening progressively over time. She expressed significant distress about her sleep difficulties, particularly concerning their impact on her energy levels and overall well-being (Chichlowski et al., 2022). Despite non-restorative sleep, her daily functioning, household activities, and social relationships remained preserved, though she reported having to exert more effort to maintain her usual level of functioning. The patient had no prior personal or family history of psychiatric disorders, and she did not report any significant medical conditions beyond the current complaints.

Gastrointestinal History. Sleep disturbance began after the onset of recurrent epigastric pain accompanied by bloating and discomfort. The patient previously received analgesic treatment for a neurological complaint at another hospital, after which dyspeptic symptoms gradually appeared. She could not recall the specific medication but remembered that it was prescribed for headaches that had since resolved. Dyspepsia symptoms fluctuated in intensity and were consistently followed by sleeplessness, whereas symptom-free days were associated with normal sleep. This temporal relationship was particularly notable, with the patient reporting that on evenings when she experienced significant epigastric discomfort, she would inevitably struggle with sleep initiation and maintenance. The gastrointestinal symptoms were described as a burning or gnawing sensation in the upper abdomen, occasionally accompanied by bloating and early satiety. Symptoms typically worsened throughout the day and were most pronounced in the evening.

Sleep History. The patient required a hypnotic (zolpidem 10 mg) to initiate sleep, usually taken around 10:00 p.m., resulting in sleep onset within 30 minutes. She routinely awakened at approximately 3:00 a.m. and was unable to return to sleep, spending the remaining nighttime hours in a state of restless wakefulness. This pattern resulted in approximately 4-5 hours of total sleep per night, which she perceived as insufficient and non-restorative. Attempts to improve sleep independently—such as increasing daytime activity—were unsuccessful, as she reported an absence of drowsiness at night despite physical fatigue. The patient had developed various compensatory behaviors, including spending excessive time in bed (9-10 hours) in an attempt to maximize sleep opportunity, and using her smartphone during nighttime awakenings. The Insomnia Severity Index (ISI) indicated moderate insomnia with a baseline score of 18.

Psychosocial and Mental Status Findings. The patient denied depressive mood, anxiety, anhedonia, auditory hallucinations, or interpersonal stressors. She described her marriage as stable and supportive and reported good relationships with her children and extended family. Her occupational functioning was unaffected, as she worked in a family business with flexible hours. No history of trauma or recent psychosocial conflict was reported. Mental status examination revealed clear consciousness, coherent thought processes, appropriate affect, and no psychotic features. Although the patient did not endorse overt psychological distress, subtle elements of health anxiety were noted, particularly concerning the meaning and potential implications of her symptoms. She expressed concerns about possible serious underlying medical conditions despite reassurances from previous physicians.

Physical Examination and Diagnosis. No significant abnormalities were found on general physical examination. Vital signs were within normal limits, and abdominal examination revealed mild epigastric tenderness without guarding, rebound, or organomegaly. Neurological examination was unremarkable. Basic laboratory investigations, including complete blood count, comprehensive metabolic panel, thyroid function tests, and inflammatory markers, were within normal limits. Abdominal ultrasonography showed no structural abnormalities. Dyspepsia was diagnosed by internal medicine evaluation based on Rome IV criteria for functional dyspepsia, and insomnia was diagnosed based on DSM-5 criteria. The working diagnosis was:

- Functional Dyspepsia (ICD-10: K30)
- Insomnia (ICD-10: G47.0)

Treatment Course and Outcomes. The integrated treatment approach was implemented over an eight-week period. Psychoeducation focused on explaining the gut–brain axis and the bidirectional relationship between gastrointestinal symptoms and sleep, which helped normalize the patient's experience and reduce catastrophic interpretations of her symptoms. Supportive psychotherapy provided a space to explore emotional factors that might be contributing to symptom maintenance, with particular attention to the patient's tendency to minimize psychological distress. CBT-I techniques included establishing a consistent wake time, limiting time in bed to actual sleep time, eliminating non-sleep activities from the bedroom, and developing a pre-sleep wind-down routine. Pharmacological management of dyspepsia with sucralfate (1g twice daily) and omeprazole (20mg once daily) was maintained throughout the treatment period.

By the fourth week of treatment, the patient reported gradual improvements in both sleep and gastrointestinal symptoms. Sleep onset latency decreased to approximately 15-20 minutes without hypnotic medication, and nighttime awakenings became less frequent and of shorter duration. Epigastric pain and bloating decreased in both frequency and intensity, with the patient reporting approximately 3-4 symptom-free days per week. By week eight, the patient had discontinued hypnotic use entirely and reported sleeping 6-7 hours per night with only occasional brief awakenings. The ISI score decreased to 8, indicating subthreshold insomnia. Follow-up at three months showed maintained improvements in both sleep and gastrointestinal symptoms, with the patient reporting significantly improved quality of life and daytime functioning.

The complex interaction between gastrointestinal function and sleep regulation can be understood through the gut–brain axis (GBA), in which neural, immune, and endocrine pathways mediate bidirectional communication between the gut and the central nervous system. This case illustrates

how dyspepsia can trigger and perpetuate sleep disturbances through multiple interconnected mechanisms within this axis. Dysbiosis and visceral hypersensitivity may contribute to heightened arousal and sleep fragmentation through altered vagal signaling and neuroinflammatory mechanisms (Cryan & Dinan, 2012; Smith et al., 2019). In this patient, the temporal relationship between dyspeptic symptoms and sleep disruption suggests that visceral signals from the gastrointestinal tract directly influenced central nervous system arousal, creating a state of hypervigilance incompatible with sleep initiation and maintenance.

The role of the vagus nerve deserves particular attention in understanding the connection between dyspepsia and sleep disturbances. As a major component of the parasympathetic nervous system, the vagus nerve plays a crucial role in promoting sleep and regulating gastrointestinal function. Altered vagal tone has been implicated in both functional dyspepsia and insomnia, suggesting a possible shared pathophysiology. In this case, it is plausible that dyspepsia-related discomfort led to increased sympathetic activation and decreased vagal tone, thereby disrupting the autonomic balance necessary for sleep initiation and maintenance. This mechanism would explain why the patient experienced sleep difficulties specifically on evenings when dyspeptic symptoms were prominent.

Stress-related activation of the hypothalamic–pituitary–adrenal (HPA) axis further amplifies this interaction by increasing cortisol secretion, impairing gastric motility, and enhancing visceral pain perception. These pathways can exacerbate dyspeptic symptoms and perpetuate autonomic hyperarousal, even in the absence of overt anxiety (Konturek et al., 2011; Qin et al., 2014; Rao & Gershon, 2016). Such findings are consistent with this case, in which "silent stress" likely contributed to the maintenance of symptoms despite minimal conscious anxiety reported by the patient. The concept of "silent stress" refers to physiological stress responses that occur without subjective awareness of psychological distress. In this patient, the discrepancy between her denial of anxiety and the presence of stress-related physical symptoms suggests that she may have exhibited alexithymic traits or employed defensive mechanisms that minimized awareness of psychological distress.

A reciprocal and reinforcing relationship between pain and sleep disturbance also plays a significant role. Disrupted sleep heightens pain sensitivity, lowers pain thresholds, and worsens gastrointestinal discomfort, creating a self-sustaining cycle that may explain the relapsing course observed in functional dyspepsia (Finan et al., 2013). In this context, insomnia is not merely a consequence of visceral symptoms, but also a driver of symptom perpetuation. This bidirectional relationship creates a vicious cycle wherein dyspepsia disrupts sleep, and poor sleep in turn exacerbates dyspeptic symptoms. Breaking this cycle requires interventions that address both components simultaneously, as was attempted in this case through the integrated treatment approach.

The patient's clinical progression reflects this biopsychophysiological model, in which dyspeptic episodes preceded sleep disruption and subsequently led to early awakenings and non-restorative sleep. The pattern of early morning awakening is particularly noteworthy, as it may reflect disturbances in the circadian regulation of the sleep-wake cycle or abnormalities in the ultradian rhythm of sleep stages. Early morning awakening has been associated with alterations in cortisol rhythm and proinflammatory cytokine activity, both of which are implicated in GBA dysfunction.

A comprehensive understanding of these mechanisms is essential to avoid underestimating sleep disturbance as a secondary feature of dyspepsia and to develop targeted interventions that address the underlying pathophysiology.

Cognitive Behavioral Therapy for Insomnia (CBT-I) has demonstrated consistent efficacy in reducing sleep latency, improving sleep maintenance, and restoring sleep continuity, and is therefore recommended as the first-line intervention for chronic insomnia (Edinger & Carney, 2009; Sateia et al., 2021; Hapsari & Kurniawan, 2019). In this case, the application of CBT-I effectively addressed maladaptive sleep behaviors and cognitive arousal, complementing gastrointestinal management without relying on long-term hypnotic pharmacotherapy. The success of CBT-I in this patient can be attributed to its multifaceted approach, which targets the cognitive, behavioral, and physiological factors that perpetuate insomnia. Specifically, sleep restriction helped consolidate sleep by increasing homeostatic sleep pressure, while stimulus control techniques strengthened the association between the bed and sleep. Cognitive restructuring addressed the patient's performance anxiety about sleep and catastrophic thinking about the consequences of insomnia.

The integration of spiritual approaches according to patient preferences represents an important aspect of culturally sensitive care that may enhance treatment engagement and outcomes. For this patient, incorporating prayer and mindfulness practices that aligned with her spiritual beliefs provided a sense of agency and meaning that complemented the technical components of CBT-I. This holistic approach acknowledges the importance of addressing existential dimensions of suffering that may not be fully captured by biomedical or psychological frameworks alone.

This case highlights the need for clinicians to consider GBA dysregulation in patients presenting with dyspepsia and persistent insomnia. An evidence-based, multidisciplinary approach that prioritizes sleep-focused therapy and targets physiological as well as behavioral contributors may optimize outcomes in similar cases. The successful management of this patient required collaboration between psychiatry and internal medicine, with treatment addressing both the gastrointestinal and sleep components of her presentation. Future research should explore whether specific subtypes of functional dyspepsia are more strongly associated with sleep disturbances and whether targeting the GBA with probiotics, dietary modifications, or vagal nerve stimulation might enhance treatment outcomes for patients with comorbid dyspepsia and insomnia.

CONCLUSION

This case illustrates how functional dyspepsia can trigger and perpetuate insomnia through gut–brain axis mechanisms, creating a self-sustaining cycle that impairs quality of life. The successful integration of gastrointestinal and sleep-focused interventions highlights the importance of a multidisciplinary approach that addresses the biological, psychological, and social dimensions of these comorbid conditions. Clinicians should maintain a high index of suspicion for sleep disturbances in patients with functional dyspepsia and vice versa, as timely recognition and treatment of both conditions may prevent the establishment of chronic symptom patterns. The gut–brain axis provides a useful framework for understanding and treating these complex

interactions, pointing toward future integrated treatment models that target shared physiological pathways.

The findings from Zhu et al. (2023) further reinforce the importance of considering gut microbiota factors in understanding the relationship between functional dyspepsia and sleep disorders. A holistic approach that accounts for the complex interactions within the gut-brain axis proved effective in managing this case of comorbid gastrointestinal and sleep disturbances. Therefore, assessment of gut microbiota and targeted interventions should be considered important components in the management of similar cases in the future.

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