International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
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ARTICLE TITLE	IMPACT OF CHRONIC ADENOID HYPERTROPHY AND RECURRENT ANTIBIOTIC THERAPY ON COGNITIVE FUNCTION, SLEEP QUALITY, AND HEARING OUTCOMES IN PRESCHOOL CHILDREN – REVIEW
DOI	https://doi.org/10.31435/ijitss.2(50).2026.5638
RECEIVED	07 March 2026
ACCEPTED	10 June 2026
PUBLISHED	22 June 2026
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IMPACT OF CHRONIC ADENOID HYPERTROPHY AND RECURRENT ANTIBIOTIC THERAPY ON COGNITIVE FUNCTION, SLEEP QUALITY, AND HEARING OUTCOMES IN PRESCHOOL CHILDREN – REVIEW

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ABSTRACT

In recent years, increasing attention has been paid to the broader developmental consequences of chronic adenoid hypertrophy (AH) in early childhood, particularly in relation to sleep, cognitive performance, and auditory function. Although adenoid enlargement is often regarded as a common pediatric condition, growing evidence indicates that its clinical impact may extend far beyond upper airway obstruction alone. In preschool children, chronic AH is strongly associated with sleep-disordered breathing, recurrent middle ear dysfunction, and repeated courses of antibiotic therapy, all of which may adversely affect critical stages of neurodevelopment.

The aim of this review is to analyze the impact of chronic adenoid hypertrophy and recurrent antibiotic therapy on cognitive function, sleep quality, and hearing outcomes in preschool-aged children, with particular emphasis on pathophysiological mechanisms, developmental implications, and current therapeutic approaches. Special attention is given to the relationship between upper airway obstruction and sleep fragmentation, the role of intermittent hypoxia in neurocognitive impairment, and the contribution of adenoid-related Eustachian tube dysfunction to otitis media with effusion and conductive hearing loss.

Available evidence indicates that chronic AH may significantly impair sleep architecture, leading to reduced sleep quality, behavioral disturbances, attention deficits, and impaired executive functioning. At the same time, persistent nasopharyngeal obstruction may predispose children to recurrent middle ear disease and fluctuating hearing loss, which in turn can negatively affect speech and language development. Although antibiotic therapy is frequently used in children with recurrent infections associated with AH, its benefits are usually limited to short-term symptom control and do not address the underlying anatomical and functional pathology. Furthermore, repeated antibiotic exposure raises concerns regarding overtreatment and the persistence of disease despite temporary symptomatic improvement.

In summary, chronic adenoid hypertrophy should not be considered solely a localized otolaryngological problem but rather a condition with potentially important systemic and developmental consequences. Early diagnosis, careful clinical monitoring, and appropriately selected treatment strategies are essential to reduce the risk of long-term impairment in preschool children.

KEYWORDS

Adenoid Hypertrophy, Preschool Children, Sleep-Disordered Breathing, Cognitive Impairment, Hearing Loss, Otitis Media with Effusion, Antibiotic Therapy, Child Development

CITATION

Wiktoria Goździejewska, Patrycja Szczygielska, Sylwia Hejna, Martyna Lipiarz, Michał Niespodziewański, Łukasz Jaworek, Gabriela Zimka, Krystian Bjorgen, Lilianna Jasińska, Magdalena Roman. (2026) Impact of Chronic Adenoid Hypertrophy and Recurrent Antibiotic Therapy on Cognitive Function, Sleep Quality, and Hearing Outcomes in Preschool Children – Review. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5638

1. Introduction

The early years of life represent a critical phase of human development, characterized by rapid maturation of the central nervous system, acquisition of language skills, and establishment of behavioral and cognitive patterns that may persist into later childhood and adulthood. During this period, physiological processes such as sleep regulation and sensory input integration play a fundamental role in shaping neural architecture and functional outcomes. Consequently, chronic disturbances affecting these processes may have disproportionate and lasting effects on development.

Adenoid hypertrophy (AH) is one of the most common conditions encountered in pediatric otolaryngology and is particularly prevalent among children aged 3 to 6 years. The adenoids, as part of Waldeyer's ring, contribute to mucosal immunity by participating in the recognition and response to inhaled and ingested antigens. However, repeated exposure to pathogens and environmental stimuli may lead to chronic inflammation and excessive lymphoid tissue proliferation, resulting in persistent enlargement.

While physiological hypertrophy is expected during early childhood, pathological enlargement may lead to significant functional impairment. Enlarged adenoids can obstruct the nasopharyngeal airway, alter normal breathing patterns, and disrupt ventilation of the middle ear. As a result, children may present with a

constellation of symptoms including nasal obstruction, mouth breathing, habitual snoring, restless sleep, and recurrent episodes of otitis media.

In recent decades, the clinical perspective on adenoid hypertrophy has evolved. Rather than being viewed solely as a structural issue affecting the upper airway, AH is increasingly recognized as a condition that may influence multiple domains of child development. Of particular importance is its association with sleep-disordered breathing (SDB), which ranges from primary snoring to obstructive sleep apnea. SDB has been linked to a variety of neurocognitive and behavioral disturbances, suggesting that chronic disruption of sleep may play a central role in mediating the broader effects of AH [1,2,5].

In parallel, the relationship between AH and middle ear disease has been extensively documented. Mechanical obstruction of the Eustachian tube and chronic inflammation of the nasopharynx contribute to the development of otitis media with effusion (OME), which is a leading cause of hearing impairment in preschool children. Because auditory input is essential for language development, even mild or fluctuating hearing loss may interfere with the acquisition of speech and communication skills [3,4].

An additional challenge in the management of chronic AH is the frequent use of antibiotic therapy. Children with recurrent upper respiratory symptoms or middle ear infections are often treated with multiple courses of antibiotics, despite the fact that such treatment does not address the underlying anatomical cause. This raises important questions regarding the effectiveness and appropriateness of current therapeutic strategies.

Given these considerations, there is a growing need to adopt a more integrative approach to the evaluation and management of adenoid hypertrophy. The aim of this review is to synthesize current evidence regarding the impact of chronic AH and recurrent antibiotic therapy on sleep quality, cognitive function, and hearing outcomes in preschool children, with a focus on both biological mechanisms and clinical implications.

2. Methodology

To provide a comprehensive overview of the clinical and developmental consequences of chronic adenoid hypertrophy in preschool children, a narrative literature review was conducted. The methodological approach was designed to identify and synthesize relevant data regarding the relationship between adenoid hypertrophy, recurrent antibiotic therapy, sleep quality, cognitive outcomes, and hearing impairment.

The literature search was based primarily on peer-reviewed publications indexed in major biomedical databases, with particular emphasis on studies addressing pediatric otolaryngology, sleep medicine, developmental pediatrics, and audiology. The search strategy included combinations of terms such as *"adenoid hypertrophy," "sleep-disordered breathing," "preschool children," "cognitive function," "hearing loss," "otitis media with effusion,"* and *"antibiotic therapy."* Preference was given to clinical guidelines, systematic reviews, meta-analyses, cohort studies, and interventional studies that contributed to understanding both clinical outcomes and underlying biological mechanisms [1–8].

Particular attention was paid to studies involving preschool-aged children, as this age group represents a critical developmental window during which chronic disturbances in sleep and hearing may have disproportionately strong effects on language acquisition, emotional regulation, and school readiness. The data were analyzed qualitatively, with emphasis on integrating findings from different but interconnected clinical domains.

3. Mechanisms Linking Chronic Adenoid Hypertrophy to Developmental Outcomes

3.1 Upper airway obstruction and sleep fragmentation

One of the most clinically significant consequences of chronic adenoid hypertrophy is persistent upper airway obstruction. Enlarged adenoid tissue narrows the nasopharyngeal space, increases airway resistance, and predisposes the child to chronic mouth breathing, snoring, and disturbed nocturnal ventilation. In more advanced cases, AH contributes to sleep-disordered breathing, including partial upper airway obstruction and obstructive sleep apnea [1,5].

The resulting disruption of normal sleep architecture has important physiological consequences. Frequent arousals, fragmented sleep, reduced slow-wave sleep, and altered rapid eye movement sleep may interfere with restorative sleep processes that are essential for memory consolidation, emotional regulation, and neural plasticity. In young children, these disturbances may not always present as overt daytime sleepiness; instead, they often manifest as irritability, hyperactivity, reduced frustration tolerance, poor concentration, and behavioral instability [1,2,5].

3.2 Intermittent hypoxia and neurocognitive dysfunction

Beyond sleep fragmentation, chronic obstruction of the upper airway may also lead to intermittent hypoxia, which is increasingly recognized as an important contributor to neurocognitive dysfunction. Recurrent oxygen desaturation, even when mild, may affect neuronal metabolism and increase oxidative stress in vulnerable brain regions involved in executive function, attention, and memory processing. Clinical studies of children with sleep-disordered breathing have shown associations with poorer performance in domains such as sustained attention, working memory, verbal learning, and behavioral self-regulation [2,5].

3.3 Eustachian tube dysfunction, otitis media, and hearing impairment

A second major mechanism through which AH affects child development involves dysfunction of the Eustachian tube and the consequent development of middle ear pathology. Because the adenoids are located near the nasopharyngeal opening of the Eustachian tube, their enlargement may mechanically obstruct normal middle ear ventilation. Chronic inflammation in the surrounding tissue may further worsen mucosal dysfunction and promote the accumulation of fluid in the middle ear cavity [3,4].

This process contributes to otitis media with effusion, one of the most common causes of conductive hearing loss in preschool children. Although the degree of hearing impairment is often mild to moderate and may fluctuate over time, its developmental significance should not be underestimated. Even temporary reductions in auditory input during early childhood may impair phonological processing, speech discrimination, language acquisition, and social communication [3,4].

3.4 Recurrent infections and repeated antibiotic exposure

Children with chronic adenoid hypertrophy frequently experience recurrent upper respiratory tract symptoms and repeated episodes of otitis media, often leading to multiple courses of antibiotic therapy. While antibiotics may temporarily reduce bacterial burden or improve acute inflammatory symptoms, they do not remove the underlying anatomical obstruction or restore normal upper airway and middle ear function. Consequently, symptomatic improvement is often partial and short-lived [7]. This reinforces the need to distinguish between temporary control of infectious exacerbations and definitive management of the underlying chronic condition.

4. Impact on Sleep Quality

Sleep quality is one of the most consistently affected domains in children with chronic adenoid hypertrophy. The relationship between nasopharyngeal obstruction and disordered sleep is well established, and AH remains one of the most common structural contributors to pediatric sleep-disordered breathing. In preschool children, the clinical significance of this relationship is especially important because adequate sleep is essential for healthy somatic growth, neurological maturation, behavioral balance, and cognitive integration [1,5].

Children with chronic AH frequently present with symptoms such as habitual snoring, restless sleep, mouth breathing, frequent awakenings, sweating during sleep, and non-restorative sleep patterns. In more severe cases, witnessed apneas, labored breathing, and gasping episodes may occur. However, even in the absence of full obstructive sleep apnea syndrome, partial obstruction may be sufficient to alter sleep architecture and compromise sleep efficiency [1,5].

The consequences of poor sleep quality in this age group are often multidimensional. Daytime manifestations may include fatigue, irritability, inattention, emotional lability, and reduced adaptive functioning. Importantly, children may respond to chronic sleep disruption not with overt lethargy but with paradoxical hyperactivity and behavioral dysregulation [2,5]. From a developmental standpoint, chronic impairment of sleep quality may affect the processes by which children consolidate newly acquired information, regulate emotional responses, and engage effectively in learning environments.

5. Cognitive and Behavioral Outcomes

The impact of chronic adenoid hypertrophy on cognition is closely related to the effects of sleep fragmentation, intermittent hypoxia, and chronic physiological stress. Although AH itself is not a neurological disorder, the mechanisms through which it disturbs sleep and oxygenation may create conditions unfavorable for optimal brain development. This is particularly relevant in preschool children, whose cognitive functions are still rapidly evolving and are therefore more sensitive to chronic disruption [1,2,5].

Studies examining children with sleep-disordered breathing consistently report impairments in attention, executive function, inhibitory control, memory, and language-related performance. In the context of chronic AH, these deficits may present subtly at first, for example as difficulties with concentration, impulsivity, slower acquisition of new material, reduced persistence in structured tasks, or diminished school readiness. Behavioral changes, including oppositional behavior, emotional irritability, and poor self-regulation, may occur alongside cognitive difficulties and further complicate the child's functioning in educational and social settings [2,5].

The importance of these findings lies in the fact that the observed impairments may be partially reversible when the underlying cause is recognized and appropriately treated. Improvement in sleep quality following medical or surgical intervention has been associated in many reports with better daytime behavior, improved attention, and enhanced quality of life [1,6]. This suggests that chronic AH may act as a modifiable contributor to cognitive and behavioral dysfunction rather than merely coexist with it.

6. Hearing Outcomes and Middle Ear Disease

Hearing outcomes constitute another major area of concern in preschool children with chronic adenoid hypertrophy. Due to the close anatomical and functional relationship between the adenoids and the Eustachian tube, persistent enlargement of adenoid tissue may compromise middle ear ventilation and promote the development of otitis media with effusion [3,4].

The resulting conductive hearing loss is often mild to moderate, bilateral or fluctuating, and therefore may remain unnoticed for long periods. Nevertheless, its developmental relevance is substantial. Preschool children rely heavily on consistent auditory input for the development of speech perception, vocabulary, grammar, and pragmatic communication. Even intermittent hearing impairment may reduce the clarity of speech signals reaching the developing brain and interfere with normal language processing [3,4].

In clinical practice, the combined presence of chronic mouth breathing, recurrent ear symptoms, and speech delay should prompt careful evaluation for AH-related middle ear dysfunction. Delayed recognition may result in persistent listening difficulties, reduced participation in group learning, and impaired communicative confidence. The burden of hearing impairment may be particularly pronounced when it coexists with poor sleep quality, as both factors may jointly undermine attention, memory, and classroom engagement.

7. Recurrent Antibiotic Therapy: Benefits, Limitations, and Clinical Concerns

Repeated antibiotic therapy remains a common feature of the clinical management of children with chronic adenoid hypertrophy, especially in the context of recurrent upper respiratory tract infections and middle ear disease. In many clinical settings, antibiotics are prescribed in response to symptoms such as fever, purulent nasal discharge, or suspected bacterial complications, despite the absence of clear evidence that such treatment modifies the long-term course of adenoid-related pathology [3,4,7].

From a pathophysiological perspective, the limitations of antibiotic therapy in this context are evident. While antimicrobial agents may temporarily reduce bacterial load and alleviate acute inflammatory symptoms, they do not address the underlying structural abnormalities associated with adenoid hypertrophy. Persistent enlargement of lymphoid tissue continues to promote airway obstruction, impaired drainage, and chronic inflammation, thereby predisposing to symptom recurrence [7].

One of the key factors contributing to the limited effectiveness of antibiotic therapy is the presence of bacterial biofilms within adenoid tissue. Biofilms provide a protective microenvironment that enhances bacterial survival and reduces susceptibility to antimicrobial agents. This phenomenon may explain why symptoms often recur shortly after the completion of antibiotic treatment, leading to repeated cycles of therapy [7,8].

In addition to limited therapeutic efficacy, repeated antibiotic exposure raises several important clinical concerns. Frequent use of antibiotics may contribute to the development of antimicrobial resistance, which represents a significant public health challenge. Although this issue extends beyond the individual patient, it underscores the need for more judicious prescribing practices in pediatric populations [7].

Another consideration is the potential impact of antibiotics on the developing microbiome. Early childhood is a critical period for the establishment of microbial communities that play a role in immune regulation and mucosal homeostasis. Disruption of these communities through repeated antibiotic exposure may have unintended consequences, although the long-term implications remain an area of ongoing research [7,8].

Furthermore, reliance on antibiotic therapy may delay the implementation of more appropriate and definitive management strategies. When symptoms are repeatedly treated pharmacologically without

addressing the underlying anatomical cause, opportunities for early intervention—such as targeted medical therapy or surgical treatment—may be missed [3,4].

From a clinical standpoint, these findings highlight the importance of distinguishing between acute infectious episodes that may warrant antibiotic treatment and chronic conditions driven primarily by structural and inflammatory factors. A more selective and evidence-based approach to antibiotic use is therefore recommended, with greater emphasis on identifying and addressing the root causes of disease [7,8].

8. Treatment Approaches and Clinical Implications

The management of chronic adenoid hypertrophy in preschool children requires a nuanced and individualized approach that takes into account not only anatomical findings but also the functional and developmental consequences of the condition. Given the multifactorial impact of AH on sleep, cognition, and hearing, therapeutic strategies should be guided by symptom severity, duration of disease, and the presence of associated complications such as sleep-disordered breathing, recurrent otitis media, and hearing impairment [1,3,4].

Conservative management remains an appropriate initial approach in selected cases, particularly in children with mild symptoms and minimal functional impairment. Non-invasive strategies may include watchful waiting, especially in younger children in whom spontaneous regression of adenoid tissue is possible. In addition, treatment of coexisting conditions, such as allergic rhinitis, may contribute to symptom improvement. Intranasal corticosteroids have been shown to reduce adenoid size and alleviate nasal obstruction in some patients, likely through their anti-inflammatory effects on lymphoid tissue [8].

However, the effectiveness of conservative treatment is often limited in cases of significant anatomical obstruction or persistent functional impairment. In such situations, surgical intervention becomes a key consideration. Adenoidectomy, either as a standalone procedure or in combination with tonsillectomy or tympanostomy tube insertion, is widely regarded as an effective treatment for children with moderate to severe symptoms [1,6].

The benefits of adenoidectomy extend beyond the relief of nasal obstruction. Numerous studies have demonstrated improvements in sleep quality following surgery, including reductions in snoring, normalization of breathing patterns, and restoration of more stable sleep architecture. These changes are often accompanied by improvements in daytime functioning, including enhanced attention, reduced behavioral disturbances, and better overall quality of life [1,6].

In the context of middle ear disease, adenoidectomy may also play a role in reducing the incidence of otitis media with effusion. By removing the source of mechanical obstruction and inflammation near the Eustachian tube, the procedure may facilitate improved ventilation of the middle ear and reduce fluid accumulation. When combined with tympanostomy tube placement, this approach may be particularly effective in children with recurrent or persistent OME [3,4].

Despite these benefits, surgical intervention is not without limitations. As with any procedure, there are risks associated with anesthesia and postoperative complications, although these are generally low in pediatric populations. Additionally, not all children experience complete resolution of symptoms, particularly in cases where multiple contributing factors are present [1,6].

From a broader clinical perspective, the decision to proceed with surgery should be based on a comprehensive assessment that includes not only anatomical findings but also the child's sleep quality, hearing status, behavioral profile, and overall developmental trajectory. This underscores the importance of a multidisciplinary approach involving pediatricians, otolaryngologists, sleep specialists, and audiologists [1,3,4].

9. Discussion

The findings presented in this review highlight the complex and multifaceted nature of chronic adenoid hypertrophy as a condition that extends beyond its traditional characterization as a localized upper airway disorder. Rather than acting through a single isolated mechanism, AH appears to exert its effects through a network of interrelated physiological processes, including airway obstruction, sleep disruption, intermittent hypoxia, and auditory dysfunction. These processes, when persistent, may collectively influence the developmental trajectory of affected children [1–5].

One of the most important insights emerging from the literature is the concept of cumulative burden. While individual factors such as mild sleep fragmentation or transient hearing loss may have limited impact in isolation, their co-occurrence and persistence over time may result in significant functional impairment. This

is particularly relevant in preschool children, whose developing nervous systems are highly sensitive to environmental and physiological influences [2–5].

The relationship between sleep quality and neurocognitive development represents a central theme in understanding the broader implications of AH. Sleep is not merely a passive state but an active process essential for brain maturation. Chronic disruption of sleep architecture may interfere with synaptic plasticity, reduce the efficiency of neural processing, and impair the consolidation of learning. Over time, these effects may manifest as deficits in attention, executive functioning, and behavioral regulation [1,2,5].

Importantly, the behavioral manifestations of sleep disruption in children often differ from those observed in adults. Rather than presenting with overt fatigue, children may exhibit hyperactivity, impulsivity, and emotional instability. This paradoxical response can obscure the underlying cause and lead to misinterpretation of symptoms. As a result, sleep-related disorders may remain underdiagnosed, and children may receive treatment for behavioral conditions without addressing the root of the problem [2,5].

The contribution of intermittent hypoxia further complicates the clinical picture. Although the degree of oxygen desaturation in children with AH is often less severe than in adults with obstructive sleep apnea, the developing brain may be particularly vulnerable to even mild but repeated hypoxic events. Experimental and clinical evidence suggests that such exposure may induce oxidative stress, alter neurotransmitter systems, and promote neuroinflammatory responses, all of which may affect cognitive and behavioral outcomes [2,5].

In parallel, the role of hearing impairment in shaping developmental outcomes cannot be overlooked. Otitis media with effusion, commonly associated with AH, may result in fluctuating conductive hearing loss that interferes with the acquisition of language and communication skills. Unlike more severe forms of hearing loss, which are often readily identified, mild or intermittent impairment may go unnoticed, particularly in young children who are still developing expressive language abilities [3,4].

The interaction between hearing loss and sleep disruption represents an important area of consideration. Both factors independently affect attention, learning, and behavior, and their coexistence may produce additive or synergistic effects. For example, a child experiencing both fragmented sleep and reduced auditory input may face greater challenges in processing information and maintaining engagement in learning environments [2–5].

Another critical aspect of this discussion concerns current patterns of clinical management, particularly the use of antibiotic therapy. While antibiotics are frequently prescribed in children with recurrent symptoms associated with AH, their role in long-term disease modification appears limited. This reflects a broader issue in clinical practice, where treatment is often directed toward acute symptoms rather than underlying pathophysiology [7].

The repeated use of antibiotics in this context may be seen as a response to diagnostic uncertainty or as an attempt to provide symptomatic relief in the absence of definitive intervention. However, this approach may inadvertently contribute to a cycle of recurrent symptoms and repeated treatment, without addressing the structural factors driving disease. In addition, concerns regarding antimicrobial resistance and the potential impact on the microbiome further highlight the need for more targeted therapeutic strategies [7,8].

The concept of early intervention emerges as a key theme in addressing these challenges. Because the effects of AH are cumulative and may become more pronounced over time, timely recognition and management are essential. Early identification of children at risk—based on symptoms such as persistent snoring, mouth breathing, recurrent ear infections, or behavioral changes—may allow for intervention before significant developmental consequences occur [1,3,4,6].

Despite the growing body of evidence, several limitations must be acknowledged. Many studies in this field are heterogeneous in terms of methodology, diagnostic criteria, and outcome measures, making direct comparisons difficult. In addition, the majority of research has focused on relatively short-term outcomes, and there is a need for longitudinal studies examining the long-term effects of chronic AH and its treatment [1,2,5].

Furthermore, the relationship between AH and developmental outcomes is influenced by a range of confounding factors, including socioeconomic status, environmental exposures, and access to healthcare. These variables may affect both the severity of symptoms and the likelihood of timely diagnosis and treatment [3,4].

Taken together, the available evidence supports a shift toward a more integrated and multidisciplinary approach to the management of chronic adenoid hypertrophy. Rather than focusing solely on anatomical findings, clinicians should consider the broader functional and developmental context in which the condition occurs [1–4,6].

10. Conclusions

Chronic adenoid hypertrophy in preschool children is associated with important consequences for sleep quality, cognitive functioning, and hearing outcomes. Its clinical significance lies not only in nasopharyngeal obstruction itself but in the downstream effects of chronic sleep fragmentation, intermittent hypoxia, Eustachian tube dysfunction, and recurrent middle ear disease during a critical phase of development [1–5].

The available evidence indicates that children with persistent AH may be at increased risk of behavioral dysregulation, impaired attention, reduced learning readiness, and delayed speech and language development, particularly when hearing impairment and poor sleep coexist [2–5]. Recurrent antibiotic therapy, although commonly used, provides only limited short-term benefit in many cases and does not resolve the underlying pathology [7]. Early diagnosis, multidisciplinary follow-up, and appropriately selected conservative or surgical treatment may reduce the risk of long-term functional impairment and improve overall quality of life in affected children [1,3,4,6,8].

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