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Pediatric obstructive sleep apnea – current concepts in diagnosis and management

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Abstract

Pediatric obstructive sleep apnea (OSA) is a common sleep-related breathing disorder causing recurrent upper airway obstruction during sleep and multiple neurocognitive, behavioural, and cardiovascular complications in children. *The aim* of this review was to summarise current evidence regarding the diagnosis and management of pediatric obstructive sleep apnea. *Material and methods.* A narrative literature review with PRISMA-informed methodology was conducted using PubMed, Scopus, Web of Science and Google Scholar. Literature published between 2016 and 2026 concerning pediatric OSA, polysomnography, adenotonsillectomy, CPAP therapy and multidisciplinary treatment was analysed. *Results.* Pediatric OSA is associated mainly with adenotonsillar hypertrophy, obesity, craniofacial abnormalities, allergic rhinitis and neuromuscular disorders. Common manifestations include snoring, witnessed apneas, restless sleep, impaired concentration, hyperactivity and daytime sleepiness. Polysomnography remains the diagnostic gold standard. Adenotonsillectomy is the first-line treatment, while additional therapies include CPAP, anti-inflammatory treatment, orthodontic interventions, weight reduction and myofunctional therapy. Persistent OSA after surgery remains common in obese and syndromic patients. *Conclusions.* Pediatric OSA requires early diagnosis and individualised multidisciplinary management. Appropriate treatment may reduce long-term complications and improve quality of life in affected children.

Key words: pediatric obstructive sleep apnea, adenotonsillectomy, polysomnography, pediatric otolaryngology, sleep-disordered breathing, upper airway obstruction.

1. Introduction

Pediatric obstructive sleep apnea (OSA) is a common sleep-related breathing disorder characterised by recurrent episodes of partial or complete upper airway obstruction during sleep, resulting in intermittent hypoxia, hypercapnia, sleep fragmentation and impaired ventilation [1,2]. The prevalence of pediatric OSA is estimated to range from 1% to 5% in the general pediatric population, although the true prevalence may be significantly underestimated due to underdiagnosis and variability in clinical presentation [1,3]. Habitual snoring, one of the most common symptoms of pediatric sleep-disordered breathing, may affect up to 10% of children, while clinically significant OSA occurs less frequently but is associated with substantial morbidity [4,5,6].

The pathophysiology of pediatric OSA is multifactorial and differs significantly from adult OSA. Adenotonsillar hypertrophy remains the leading anatomical cause of airway obstruction in children, particularly in preschool-aged patients [1,7]. However, obesity, craniofacial abnormalities, neuromuscular disorders, allergic rhinitis, genetic syndromes and impaired neuromuscular control of the upper airway also play important roles in disease development [2,8,9]. Recent studies have demonstrated a growing association between pediatric obesity and sleep-disordered breathing, emphasising the increasing importance of metabolic factors in the pathogenesis of OSA [10,11]. Additionally, adenoid hypertrophy combined with allergic rhinitis may significantly worsen airway obstruction and contribute to increased disease severity [9].

Clinical manifestations of pediatric OSA are often heterogeneous and may include nocturnal as well as daytime symptoms. Typical nighttime manifestations include habitual snoring, witnessed apneas, restless sleep, mouth breathing, frequent awakenings, diaphoresis and abnormal sleep positions [1,12]. Daytime symptoms may involve excessive daytime sleepiness, behavioural disturbances, impaired concentration, hyperactivity, poor school performance, emotional dysregulation and reduced quality of life [11,13]. In some children, neurobehavioural

manifestations may resemble attention-deficit/hyperactivity disorder (ADHD), which may further complicate diagnosis [8]. Untreated pediatric OSA has also been associated with cardiovascular complications, metabolic dysfunction, growth impairment, systemic inflammation and adverse neurocognitive outcomes [1,13].

Accurate diagnosis of pediatric OSA remains challenging due to variability in symptom severity and limited access to specialised sleep laboratories. Polysomnography (PSG) is currently considered the gold-standard diagnostic modality and enables objective assessment of respiratory disturbances during sleep [1,4]. Nevertheless, clinical history, physical examination, pulse oximetry, validated questionnaires, imaging studies and drug-induced sleep endoscopy (DISE) may provide additional diagnostic value in selected cases [2,5,12]. Contemporary guidelines emphasise the importance of early diagnosis and multidisciplinary evaluation in children presenting with symptoms suggestive of sleep-disordered breathing [4,14].

Management strategies for pediatric OSA depend on disease severity, anatomical factors, age, obesity status and the presence of comorbidities. Adenotonsillectomy remains the first-line treatment for most pediatric patients with adenotonsillar hypertrophy and moderate-to-severe OSA [7,15,16]. However, residual OSA after surgery remains a significant clinical problem, particularly in obese children and patients with craniofacial abnormalities or syndromic disorders [14,17]. Alternative or adjunctive therapies may include continuous positive airway pressure (CPAP), anti-inflammatory pharmacotherapy, weight reduction, orthodontic interventions and myofunctional therapy [7,17,18]. Recent literature highlights the growing importance of individualised and multidisciplinary therapeutic approaches in optimising long-term outcomes [2,19].

Given the increasing prevalence of pediatric sleep-disordered breathing and its potentially serious systemic consequences, comprehensive understanding of current diagnostic and therapeutic concepts is essential for clinicians involved in pediatric care. Therefore, the aim of this narrative review was to summarise contemporary evidence regarding the epidemiology, pathophysiology, clinical presentation, diagnostic evaluation and management of pediatric obstructive sleep apnea.

2. Materials and methods

2.1. Literature search strategy

A narrative literature review with PRISMA-informed methodology was conducted using PubMed, Scopus, Web of Science and Google Scholar databases. Literature published between January 2016 and May 2026 was analysed in order to summarise current evidence regarding pediatric obstructive sleep apnea (OSA). The search strategy included combinations of the following keywords and Medical Subject Headings (MeSH): “pediatric obstructive sleep apnea”, “sleep-disordered breathing”, “adenotonsillectomy”, “polysomnography”, “drug-induced sleep endoscopy”, “continuous positive airway pressure”, “adenoid hypertrophy”, “sleep quality” and “pediatric otolaryngology”.

The review primarily focused on clinical studies, systematic reviews, meta-analyses, narrative reviews and international clinical guidelines related to pediatric OSA. Additional references were identified through manual screening of bibliographies from selected articles.

2.2. Eligibility criteria

Studies published in English between 2016 and 2026 involving pediatric populations with obstructive sleep apnea or sleep-disordered breathing were considered eligible for inclusion. Priority was given to high-quality evidence, including international guidelines, systematic reviews, meta-analyses and multicentre clinical studies.

Articles focusing exclusively on adult populations, studies lacking scientific relevance, conference abstracts without full-text availability and publications with insufficient methodological quality were excluded from the analysis.

2.3. Data collection and narrative synthesis

Relevant publications were independently screened according to their titles, abstracts and full-text content. The selected studies were analysed with emphasis on epidemiology, pathophysiology, clinical manifestations, diagnostic approaches, therapeutic management and long-term consequences of pediatric OSA.

Due to heterogeneity among the included studies, a narrative synthesis approach was applied. The collected evidence was organised into thematic sections to provide a comprehensive overview of current concepts in pediatric OSA diagnosis and management.

2.3.1. Use of artificial intelligence

Artificial intelligence tools were used exclusively for linguistic refinement, structural organisation of the manuscript and improvement of academic English readability. All scientific content, interpretation of evidence, critical analysis and final conclusions were independently reviewed and verified by the authors. AI-assisted technologies were used solely as supportive instruments and did not replace human scientific judgement.

3. Results

3.1. Epidemiology and risk factors

Pediatric OSA is one of the most common sleep-related breathing disorders in children and represents an important public-health concern due to its growing prevalence and potential long-term systemic consequences [1,3,8]. Current epidemiological studies estimate that clinically significant pediatric OSA affects approximately 1–5% of children, whereas habitual snoring may occur in up to 10% of the pediatric population [1,4]. The prevalence appears to be increasing, partially due to rising childhood obesity rates and improved awareness of sleep-disordered breathing among clinicians [8,10,18].

The incidence of pediatric OSA varies according to age, body mass index, craniofacial anatomy and the presence of comorbidities. Two prevalence peaks are commonly observed: the first in preschool-aged children due to adenotonsillar hypertrophy, and the second during adolescence, often associated with obesity and metabolic dysfunction [1,2]. Boys and girls appear to be affected relatively equally before puberty, unlike adult OSA, where male predominance is more pronounced [1].

Adenotonsillar hypertrophy remains the primary anatomical risk factor for pediatric OSA and is strongly associated with upper-airway narrowing during sleep [7,15]. Enlargement of adenoids and palatine tonsils contributes to increased airway resistance, turbulent airflow and recurrent episodes of airway collapse. Recent studies additionally demonstrated that adenoid hypertrophy associated with allergic rhinitis may significantly worsen respiratory obstruction and increase disease severity [9].

Obesity is another major risk factor contributing to pediatric sleep-disordered breathing. Excess adipose tissue surrounding the upper airway may increase pharyngeal collapsibility and impair respiratory mechanics [8,10]. Obese children with OSA are also more likely to develop residual disease after adenotonsillectomy and frequently require additional therapeutic interventions [14,17]. Furthermore, obesity-related systemic inflammation and metabolic dysregulation may exacerbate upper-airway dysfunction and sleep fragmentation [10].

Several additional conditions have been associated with increased pediatric OSA risk, including craniofacial abnormalities, Down syndrome, neuromuscular disorders, prematurity and genetic syndromes [2,20]. Children with Down syndrome are particularly susceptible to severe OSA due to macroglossia, midfacial hypoplasia, generalised hypotonia and obesity [20]. Neuromuscular diseases may further impair airway stability and respiratory control mechanisms during sleep.

Environmental and inflammatory factors may also contribute to pediatric OSA development. Allergic rhinitis, chronic nasal obstruction, recurrent upper respiratory infections and exposure to tobacco smoke may worsen upper-airway inflammation and increase airway resistance [9]. Additionally, insufficient sleep quality and sleep fragmentation may negatively affect neurocognitive function, emotional regulation, metabolic health and behavioural development in pediatric patients [11]. The principal risk factors and their mechanisms contributing to pediatric OSA are presented in Table 1.

Table 1. Major risk factors for pediatric obstructive sleep apnea

Risk factor	Mechanism contributing to OSA
Adenotonsillar hypertrophy	Upper-airway narrowing and increased airflow resistance
Obesity	Increased pharyngeal collapsibility and systemic inflammation
Allergic rhinitis	Nasal obstruction and chronic airway inflammation
Craniofacial abnormalities	Reduced airway dimensions
Down syndrome	Macroglossia, hypotonia, midfacial hypoplasia
Neuromuscular disorders	Impaired upper-airway muscle control
Prematurity	Abnormal respiratory regulation
Chronic nasal obstruction	Increased airway resistance during sleep

Source: own elaboration.

Recent literature emphasises that pediatric OSA should be considered a multifactorial and heterogeneous disorder requiring individualised assessment of anatomical, inflammatory, metabolic and neurological contributors [2,8,19]. Early identification of risk factors may improve diagnostic accuracy and facilitate timely implementation of appropriate therapeutic strategies.

3.2. Pathophysiology

The pathophysiology of pediatric OSA is complex and involves dynamic interactions between anatomical abnormalities, neuromuscular control dysfunction, inflammatory processes and systemic metabolic factors [1,2,8]. Unlike adult OSA, where obesity and pharyngeal collapsibility predominate, pediatric OSA is frequently associated with adenotonsillar hypertrophy and developmental anatomical factors affecting upper-airway patency [1,7].

Upper-airway obstruction during sleep occurs primarily due to repetitive collapse or narrowing of the pharyngeal airway. During sleep, physiological reduction in upper-airway muscle tone increases airway susceptibility to collapse, especially in children with enlarged tonsils and adenoids [1,15]. This results in intermittent hypoxia, hypercapnia, sleep fragmentation and recurrent arousals, which may negatively affect multiple organ systems [13].

Adenoid and tonsillar hypertrophy are considered the major anatomical contributors to pediatric OSA. Hypertrophied lymphoid tissue narrows the nasopharyngeal and oropharyngeal airway lumen, increasing airflow resistance and promoting turbulent breathing during sleep [7,9]. The severity of airway obstruction may be further exacerbated by chronic nasal obstruction associated with allergic rhinitis or recurrent upper respiratory infections [9].

Obesity significantly contributes to the pathogenesis of pediatric OSA through several mechanisms. Fat deposition surrounding the upper airway increases pharyngeal collapsibility and impairs respiratory mechanics [8,10]. Additionally, obesity-related systemic inflammation may influence neuromuscular control of the upper airway and contribute to sleep fragmentation and altered ventilatory responses [10]. Obese children are also at increased risk of persistent OSA following adenotonsillectomy [14,17].

Neuromuscular dysfunction represents another important pathophysiological component. Impaired activation of pharyngeal dilator muscles during sleep may reduce airway stability and predispose to repetitive airway collapse [2]. Children with neuromuscular diseases or genetic syndromes frequently demonstrate reduced airway-muscle tone and impaired respiratory regulation mechanisms [20].

Craniofacial abnormalities may additionally contribute to airway narrowing and impaired airflow dynamics. Midfacial hypoplasia, retrognathia, macroglossia, high-arched palate and mandibular deficiency may reduce upper-airway dimensions and increase obstruction risk [20]. Such abnormalities are commonly observed in children with Down syndrome and other craniofacial disorders [20].

Intermittent hypoxia and sleep fragmentation associated with pediatric OSA may induce systemic inflammatory responses, oxidative stress, endothelial dysfunction and autonomic nervous system dysregulation [1,11,13,18]. These mechanisms are believed to contribute to cardiovascular complications, neurocognitive impairment, metabolic disturbances and behavioural abnormalities observed in affected children [11,13].

Recent evidence suggests that pediatric OSA should be considered a heterogeneous disorder with variable underlying mechanisms depending on age, obesity status, anatomical factors and associated comorbidities [2,8]. Understanding the multifactorial pathophysiology of pediatric OSA is essential for selecting individualised therapeutic strategies and optimising long-term clinical outcomes.

3.3. Clinical presentation

Clinical manifestations of pediatric OSA are highly variable and may involve both nocturnal and daytime symptoms [1,12,13,17]. The severity of symptoms often depends on the degree of airway obstruction, patient age, obesity status and the presence of associated comorbidities [8].

Habitual snoring is the most common presenting symptom and is frequently reported by caregivers [1,3]. Snoring is typically loud, persistent and may be accompanied by witnessed apneas, gasping episodes, choking during sleep or laboured breathing [6]. Additional nighttime manifestations may include restless sleep, frequent awakenings, diaphoresis, parasomnias, enuresis, abnormal sleeping positions and mouth breathing [1,13].

Children with severe OSA may demonstrate increased respiratory effort during sleep, paradoxical chest movements and prolonged respiratory pauses [2]. Infants and very young children may present atypically with feeding difficulties, failure to thrive or developmental delay rather than classic snoring symptoms [2].

Daytime symptoms are frequently underestimated but may significantly affect cognitive and psychosocial functioning [8]. Excessive daytime sleepiness, fatigue, morning headaches, impaired concentration, memory disturbances, irritability, emotional dysregulation and reduced school performance are commonly reported [11,13]. Hyperactivity and behavioural disturbances resembling ADHD may also occur, particularly in younger children [8].

Untreated pediatric OSA may negatively influence growth, cardiovascular health and metabolic regulation [1,13]. Recurrent nocturnal hypoxia and sleep fragmentation are associated with systemic inflammation, endothelial dysfunction, elevated blood pressure, insulin resistance and impaired neurodevelopment [13]. Long-term consequences may include reduced quality of life, impaired academic performance and psychosocial difficulties [11].

Children with obesity, Down syndrome, craniofacial abnormalities or neuromuscular disorders often present with more severe symptoms and increased risk of persistent disease [17,20]. Additionally, allergic rhinitis and chronic nasal obstruction may worsen sleep quality and intensify upper-airway symptoms [9].

Current guidelines emphasise the importance of comprehensive clinical evaluation in children presenting with habitual snoring or symptoms suggestive of sleep-disordered breathing [4,14]. Early recognition of clinical

manifestations remains essential for timely diagnosis and prevention of long-term systemic complications associated with pediatric OSA.

3.4. Diagnostic evaluation

Accurate diagnosis of pediatric OSA requires comprehensive clinical assessment combining medical history, physical examination and objective sleep evaluation methods [1,4]. Due to the heterogeneity of clinical manifestations and variability in symptom severity, diagnosis may often be delayed or underestimated [12].

Clinical history remains the first and essential diagnostic step. Particular attention should be paid to habitual snoring, witnessed apneas, restless sleep, mouth breathing, nocturnal awakenings, daytime sleepiness, behavioural disturbances and impaired school performance [1,13]. Caregiver-reported symptoms are particularly important in younger children who may be unable to accurately describe sleep-related complaints [3].

Physical examination should include assessment of upper-airway anatomy, craniofacial abnormalities, body mass index, nasal obstruction and tonsillar hypertrophy [1,7]. Enlarged tonsils and adenoids are among the most common anatomical findings associated with pediatric OSA [7,9]. Evaluation for obesity, allergic rhinitis, retrognathia, macroglossia and neuromuscular disorders is also recommended [2,20].

Polysomnography (PSG) remains the gold-standard diagnostic tool for pediatric OSA [1,4]. Overnight PSG allows comprehensive assessment of respiratory events, oxygen saturation, airflow limitation, respiratory effort, sleep architecture and arousal frequency [4]. The apnea–hypopnea index (AHI) is commonly used to classify disease severity, although pediatric threshold values differ from adult criteria [1]. Current guidelines emphasise the importance of PSG in children with suspected moderate-to-severe OSA, obesity, craniofacial abnormalities, Down syndrome or persistent symptoms after adenotonsillectomy [4,14]. Diagnostic methods used in pediatric OSA are summarised in Table 2.

Table 2. Diagnostic methods in pediatric obstructive sleep apnea

Diagnostic method	Advantages	Limitations
Polysomnography (PSG)	Gold standard, comprehensive assessment	Limited availability, high cost
Pulse oximetry	Accessible and inexpensive	Lower sensitivity
Pediatric Sleep Questionnaire	Useful screening tool	Subjective assessment
Drug-induced sleep endoscopy (DISE)	Dynamic airway evaluation	Requires sedation
Imaging studies	Anatomical visualisation	Limited routine utility
Physical examination	Widely available	Limited specificity

Source: own elaboration based on [1,4,12,14].

Despite its diagnostic value, access to polysomnography may be limited due to high cost, insufficient availability of pediatric sleep laboratories and prolonged waiting times [12]. Consequently, alternative diagnostic methods are increasingly utilised in selected clinical settings.

Pulse oximetry may serve as a useful screening tool, particularly in resource-limited settings [12]. Recurrent oxygen desaturation episodes during sleep may suggest significant sleep-disordered breathing, although normal oximetry results do not exclude OSA [1]. Several questionnaires and screening instruments, including the Pediatric Sleep Questionnaire (PSQ), may additionally support clinical evaluation [12,21].

Drug-induced sleep endoscopy (DISE) has gained increasing importance in the assessment of persistent or complex pediatric OSA [14,17]. DISE enables dynamic visualisation of upper-airway obstruction sites during pharmacologically induced sleep and may help guide individualised surgical planning [14]. This method is particularly valuable in children with residual OSA following adenotonsillectomy or craniofacial abnormalities [17].

Imaging studies such as lateral neck radiography, cephalometry, magnetic resonance imaging (MRI) and computed tomography (CT) may provide additional anatomical information in selected patients [2,5]. However, routine imaging is generally not recommended for all children with suspected OSA and should be reserved for specific clinical indications [4].

Recent literature emphasises the importance of multidisciplinary diagnostic evaluation involving pediatricians, otolaryngologists, pulmonologists, orthodontists and sleep-medicine specialists [2,4,14,18,19]. Early and accurate diagnosis remains crucial for preventing long-term neurocognitive, cardiovascular and metabolic complications associated with untreated pediatric OSA [11,13].

3.5. Therapeutic management

Management of pediatric OSA should be individualised according to disease severity, anatomical abnormalities, obesity status, age and associated comorbidities [1,2,18,19]. Contemporary therapeutic approaches involve both surgical and non-surgical strategies aimed at improving airway patency, sleep quality and long-term systemic outcomes [7]. Current therapeutic strategies for pediatric OSA are presented in Table 3.

Table 3. Current therapeutic strategies in pediatric obstructive sleep apnea

Treatment modality	Main indications	Advantages	Limitations
Adenotonsillectomy	Adenotonsillar hypertrophy	First-line treatment	Residual OSA possible
CPAP therapy	Persistent / severe OSA	High efficacy	Adherence difficulties
Intranasal corticosteroids	Mild OSA / allergic rhinitis	Non-invasive	Limited efficacy
Montelukast	Mild inflammatory OSA	Adjunctive therapy	Variable response
Weight reduction	Obesity-associated OSA	Improves metabolic profile	Requires long-term compliance
Orthodontic therapy	Craniofacial abnormalities	Improves airway dimensions	Limited indications
Myofunctional therapy	Mild / moderate OSA	Supportive treatment	Limited evidence

Source: own elaboration based on [7,8,10,14,17,19].

3.5.1. Adenotonsillectomy

Adenotonsillectomy remains the first-line treatment for most children with moderate-to-severe OSA associated with adenotonsillar hypertrophy [7,15]. Surgical removal of enlarged tonsils and adenoids significantly improves upper-airway patency and reduces obstructive respiratory events during sleep [11,22].

Numerous systematic reviews and meta-analyses have confirmed substantial postoperative improvements in apnea–hypopnea index, oxygen saturation, sleep quality, behavioural symptoms and overall quality of life following adenotonsillectomy [15,16,22]. Tsikopoulos et al. demonstrated that adenotonsillectomy was superior to watchful waiting in improving clinical outcomes among children with obstructive sleep apnea syndrome [22].

Nevertheless, residual OSA following surgery remains a clinically important issue. Persistent disease is particularly common among obese children, patients with Down syndrome, craniofacial abnormalities or severe baseline OSA [14,17,20]. Consequently, postoperative follow-up and repeat polysomnography may be necessary in selected high-risk patients [14].

3.5.2. Continuous positive airway pressure (CPAP)

Continuous positive airway pressure therapy is recommended for children with persistent OSA after adenotonsillectomy, contraindications to surgery or severe disease not amenable to surgical treatment [10,11,14,17]. CPAP maintains upper-airway patency by delivering positive pressure during sleep and effectively reduces respiratory events and nocturnal hypoxia [1].

Although CPAP demonstrates high therapeutic efficacy, adherence remains a major challenge in pediatric populations due to discomfort, mask intolerance and psychosocial factors [17]. Multidisciplinary support and caregiver education are essential for improving long-term compliance.

3.5.3. Pharmacological treatment

Anti-inflammatory pharmacotherapy may be beneficial in children with mild OSA or associated allergic rhinitis [1,9]. Intranasal corticosteroids and leukotriene receptor antagonists such as montelukast may reduce upper-airway inflammation and improve nasal airflow [8].

Pharmacological therapy is most commonly used in mild disease or as adjunctive treatment following adenotonsillectomy [1]. However, current evidence suggests that medication alone is generally insufficient for moderate-to-severe pediatric OSA [7].

3.5.4. Orthodontic and myofunctional therapy

Orthodontic interventions, including rapid maxillary expansion, may improve airway dimensions in selected children with craniofacial abnormalities and maxillary constriction [8]. Additionally, myofunctional therapy aimed at strengthening oropharyngeal muscles has recently gained interest as a potential supportive treatment modality [8,19].

Although current evidence remains limited, these approaches may provide additional benefit in carefully selected patients, particularly when combined with other therapeutic strategies.

3.5.5. Weight reduction and lifestyle modification

Weight management represents an essential component of therapy in obese children with OSA [8,10]. Lifestyle interventions involving dietary modification, physical activity and behavioural counselling may improve respiratory parameters and reduce disease severity [10].

Recent literature highlights the close relationship between obesity, sleep quality and metabolic dysfunction in pediatric populations [10,11]. Consequently, multidisciplinary obesity management may significantly contribute to long-term treatment success.

3.5.6. Management of persistent OSA

Persistent OSA after adenotonsillectomy represents an increasingly recognised clinical problem [14,17]. Current American Thoracic Society guidelines recommend individualised management strategies depending on the underlying mechanism of residual airway obstruction [14].

Potential treatment options include CPAP therapy, revision surgery, orthodontic interventions, anti-inflammatory therapy, weight reduction and DISE-guided surgical procedures [14,17,19]. Comprehensive multidisciplinary evaluation is particularly important in children with complex or refractory disease.

3.6. Complications and long-term outcomes

Untreated pediatric OSA may lead to significant short-term and long-term systemic complications affecting cardiovascular, neurocognitive, metabolic, behavioural and psychosocial health [1,13]. Increasing evidence

suggests that recurrent nocturnal hypoxia and sleep fragmentation may adversely influence multiple organ systems during critical periods of childhood development [8,11].

Neurocognitive and behavioural disturbances are among the most commonly reported consequences of pediatric OSA [13]. Children with untreated disease frequently demonstrate impaired attention, memory deficits, learning difficulties, emotional dysregulation, irritability and reduced academic performance [8,11]. Hyperactivity and behavioural manifestations resembling ADHD are also commonly observed, particularly in younger children [8,11]. Chronic sleep disruption may negatively affect brain maturation and neurodevelopmental processes.

Cardiovascular complications represent another important concern. Intermittent hypoxia and increased sympathetic nervous system activity may contribute to endothelial dysfunction, elevated blood pressure, systemic inflammation and altered cardiac remodelling [1,13]. Severe or prolonged OSA may increase the risk of pulmonary hypertension and cardiovascular morbidity later in life [13].

Metabolic dysfunction is increasingly recognised in pediatric patients with OSA, particularly among obese children [10]. Sleep fragmentation and chronic hypoxia may contribute to insulin resistance, dyslipidemia, altered glucose metabolism and obesity progression [8,10]. The relationship between sleep quality and metabolic health appears bidirectional, further complicating disease management [10].

Growth impairment may additionally occur in children with severe untreated OSA. Increased nocturnal respiratory effort, disrupted sleep architecture and altered growth-hormone secretion may negatively influence somatic development and weight gain [1]. Some children demonstrate improvement in growth parameters following effective treatment.

Persistent OSA after adenotonsillectomy remains an important clinical issue requiring long-term monitoring [14,17]. Residual disease occurs more frequently in obese patients, children with Down syndrome, craniofacial abnormalities, neuromuscular disorders or severe baseline OSA [14,20]. These patients may require additional interventions such as CPAP therapy, orthodontic treatment or revision surgical procedures [17,19]. Major complications associated with untreated pediatric OSA are summarised in Table 4.

Table 4. Major complications of untreated pediatric obstructive sleep apnea

System affected	Potential complications
Neurocognitive	Attention deficits, impaired concentration, learning difficulties
Behavioural	Hyperactivity, irritability, emotional dysregulation
Cardiovascular	Hypertension, endothelial dysfunction
Metabolic	Insulin resistance, obesity progression
Growth and development	Growth impairment, developmental delay
Psychosocial	Reduced quality of life, poor school performance

Source: own elaboration based on [1,8,10,11,13].

Pediatric OSA may also significantly reduce quality of life for both patients and caregivers [11]. Chronic sleep disruption may contribute to daytime fatigue, impaired social functioning, emotional stress and caregiver burden. Early diagnosis and effective treatment may substantially improve overall well-being and psychosocial functioning.

Recent studies emphasise that pediatric OSA should not be considered an isolated respiratory disorder but rather a systemic condition with potentially lifelong health consequences [8,19]. Comprehensive multidisciplinary management and long-term follow-up remain essential for reducing morbidity and optimising clinical outcomes.

4. Discussion

Pediatric OSA represents a complex and increasingly prevalent clinical disorder associated with substantial systemic morbidity and reduced quality of life [1,8]. The growing incidence of obesity, improved awareness of sleep-disordered breathing and advances in diagnostic techniques have contributed to increased recognition of pediatric OSA in recent years [10]. The increasing prevalence of childhood obesity may further contribute to the global burden of pediatric OSA in the coming years.

The present narrative review summarises current evidence regarding epidemiology, pathophysiology, clinical manifestations, diagnostic approaches and therapeutic management of pediatric OSA. Contemporary literature consistently emphasises the multifactorial nature of the disease, involving anatomical, inflammatory, neuromuscular and metabolic mechanisms [2,8]. Although adenotonsillar hypertrophy remains the primary anatomical contributor to airway obstruction, obesity and associated metabolic dysfunction increasingly influence disease severity and long-term outcomes [8,10].

Early diagnosis remains essential due to the potentially serious neurocognitive, cardiovascular and metabolic consequences of untreated pediatric OSA [1,13]. However, diagnosis may still be challenging because clinical manifestations vary significantly depending on patient age, obesity status and comorbid conditions [12]. Many children present with behavioural disturbances, impaired concentration or poor school performance rather than the classic excessive daytime sleepiness observed in adults [8].

Polysomnography continues to represent the gold-standard diagnostic modality and remains indispensable for objective assessment of disease severity [1,4]. Nevertheless, limited access to pediatric sleep laboratories may delay diagnosis and treatment initiation [12]. Consequently, there is growing interest in alternative screening methods including pulse oximetry, validated questionnaires and drug-induced sleep endoscopy [12,14]. DISE has become particularly valuable in identifying persistent obstruction sites in children with residual disease after adenotonsillectomy [14,17].

Adenotonsillectomy remains the cornerstone of treatment in children with adenotonsillar hypertrophy and moderate-to-severe OSA [7,15]. Meta-analyses have consistently demonstrated significant improvements in respiratory parameters, sleep quality and behavioural outcomes following surgery [16,22]. However, residual OSA remains common in obese children and patients with syndromic or craniofacial abnormalities [14,17,20]. This highlights the necessity for individualised and multidisciplinary treatment approaches.

Non-surgical therapies such as CPAP, anti-inflammatory pharmacotherapy, orthodontic interventions, weight reduction and myofunctional therapy may provide additional benefit in selected patients [8,17,19]. Weight management appears particularly important due to the close relationship between obesity, sleep quality and metabolic dysfunction [10]. Recent literature additionally suggests that comprehensive management of allergic rhinitis and nasal obstruction may improve upper-airway function and reduce disease severity [9].

Current international guidelines emphasise the importance of multidisciplinary cooperation involving pediatricians, otolaryngologists, pulmonologists, sleep specialists, orthodontists and nutrition experts [4,14,18,19]. Such an approach may optimise long-term treatment outcomes and reduce the risk of persistent disease and systemic complications.

Despite significant progress in recent years, several limitations remain within the current literature. Many studies demonstrate methodological heterogeneity, variable diagnostic criteria and inconsistent definitions of treatment success [19]. Further prospective multicentre studies are necessary to establish standardised management protocols and evaluate long-term outcomes of emerging therapeutic strategies.

This review has several limitations. As a narrative review, the study may be associated with selection bias and heterogeneity among included publications. Additionally, variability in diagnostic criteria, study populations and treatment protocols across the analysed studies may limit direct comparison of results. Nevertheless, the review summarises current evidence and highlights important contemporary concepts in pediatric obstructive sleep apnea diagnosis and management.

5. Conclusions

Pediatric OSA is a multifactorial sleep-related breathing disorder associated with significant neurocognitive, cardiovascular, metabolic and psychosocial consequences. Early diagnosis and individualised treatment remain essential for reducing morbidity and improving long-term quality of life.

Polysomnography remains the gold-standard diagnostic method, while adenotonsillectomy continues to represent the first-line treatment in most children with adenotonsillar hypertrophy. However, persistent disease following surgery remains a major clinical challenge, particularly among obese patients and children with craniofacial or syndromic abnormalities.

Contemporary management of pediatric OSA increasingly requires a multidisciplinary and personalised approach involving surgical, pharmacological, behavioural, orthodontic and supportive therapeutic strategies. Further high-quality studies are needed to optimise diagnostic pathways and evaluate the long-term effectiveness of emerging treatment modalities.

Declarations

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