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**Review Article**

# Systematic assessment and management of obesity-related asthma in children: a practical clinical framework

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## ABSTRACT

Childhood obesity significantly worsens asthma risk, symptom burden, and clinical outcomes, yet consistent, practical guidance for managing this common comorbidity is lacking in routine care. This review outlines a clinically actionable framework for assessing and treating obesity-related asthma in children, emphasizing Body Mass Index percentile classification, age-appropriate lung function testing, structured asthma control evaluation, and screening for comorbidities such as sleep-disordered breathing, reflux, metabolic dysfunction, and mental health concerns. Ongoing management should include regular monitoring of respiratory status, weight trajectory, and comorbidity burden. Effective care requires a dual-target approach that pairs standard asthma pharmacotherapy with comprehensive, family-centered weight-management strategies, including nutrition improvement, increased physical activity, behavioral support, and reduced sedentary time, with pharmacologic weight-management options reserved for select adolescents with severe obesity. Integrating systematic identification, structured follow-up, and coordinated treatment can meaningfully improve lung function, asthma control, cardiometabolic health, and overall quality of life for affected children.

**Keywords:** Obesity-related asthma, Pediatric obesity, Childhood asthma, Clinical algorithms, Body mass index monitoring, Pediatric weight management

## INTRODUCTION

Asthma and obesity represent two prevalent chronic childhood diseases. Asthma affects approximately 10%-15% of school-aged children worldwide, with 5.8% U.S. prevalence among children aged 0-18 years.[1] Childhood obesity affects 19.7% of U.S. children aged 2-19 years, representing over 14 million individuals.[1,2] Obesity rates demonstrate particularly alarming increases among certain demographic groups, with Hispanic and non-Hispanic Black children experiencing disproportionately higher prevalence.[2]

The relationship between obesity and asthma in children is complex, multifactorial, and bidirectional.[3] Epidemiological studies demonstrate that children with overweight or obesity face substantially elevated risk, with meta-analyses suggesting 23%-27% of new asthma diagnoses may be attributable to preexisting obesity.[1,4] Beyond increased incidence, children with established asthma who develop overweight or obesity experience markedly greater disease severity, poorer day-to-day symptom control, more frequent and severe exacerbations requiring urgent care or hospitalization, reduced lung function, diminished response to standard controller medications particularly inhaled corticosteroids, and substantially diminished health-related quality of life.[5-7]

Despite robust epidemiological evidence and growing recognition of obesity-related asthma as distinct clinical

phenotype, significant gaps persist in translating research into practical clinical care.[8] Many pediatric primary care clinicians face challenges in implementing structured frameworks for obesity assessment and management. Recent surveys suggest that well-child visits may not consistently include comprehensive obesity evaluation despite guideline recommendations, with commonly cited factors including time constraints, variable confidence in counseling effectiveness, limited referral resources, concerns about therapeutic relationships, and reimbursement considerations.[8,9] Furthermore, children with obesity-related asthma demonstrate attenuated responsiveness to conventional therapies, necessitating integrated approaches simultaneously addressing airway inflammation and metabolic dysfunction.[3,10,11] This review presents evidence-based strategies supported by practical algorithms that outline assessment schedules, validated tools across stages of care, and defined criteria for clinical escalation.

## EPIDEMIOLOGY AND CLINICAL SIGNIFICANCE

### *Prevalence and demographic patterns*

The parallel rise of childhood asthma and obesity has created steadily expanding populations simultaneously affected. Extensive epidemiologic analyses demonstrate consistent dose-dependent positive association between excess BMI and asthma risk across all pediatric age groups.[1,2,9] Large-scale population-based studies including systematic reviews and meta-analyses indicate children with overweight or obesity are 1.2 to 6.8 times more likely to develop new-onset asthma compared to age-matched normal-weight peers, with risk magnitude increasing proportionally with obesity severity.[4,12] Recent NHANES analyses (2007-2020) demonstrate statistically significant interaction effects, with children affected by both conditions producing synergistically worse health outcomes including higher hypertension rates, increased healthcare utilization, and lower self-reported health compared to either condition alone.[13]

### *Public health impact*

The intersection represents substantial growing public health burden with wide-ranging implications.[6,7] Children simultaneously affected demonstrate markedly worse outcomes across multiple domains. Specifically, these children experience 30%-50% higher asthma exacerbation rates, 2-3 times more emergency department visits annually, significantly reduced response to standard controller medications with some studies suggesting response rates below 40% compared to over 80% in normal-weight asthmatic children, substantially lower health-related quality of life across physical functioning, emotional well-being, and social domains, and higher school absenteeism with associated negative impacts on academic achievement.[3,6,14,15] Understanding these patterns is critical for targeting prevention and intervention efforts.

### *Pathophysiological mechanisms*

Mechanisms causally linking obesity to asthma are complex, multifactorial, and incompletely understood, involving intricate interactions among inflammatory pathways, mechanical respiratory effects, metabolic dysfunction, dietary factors, and genetic predisposition. Figure 1 provides comprehensive overview.

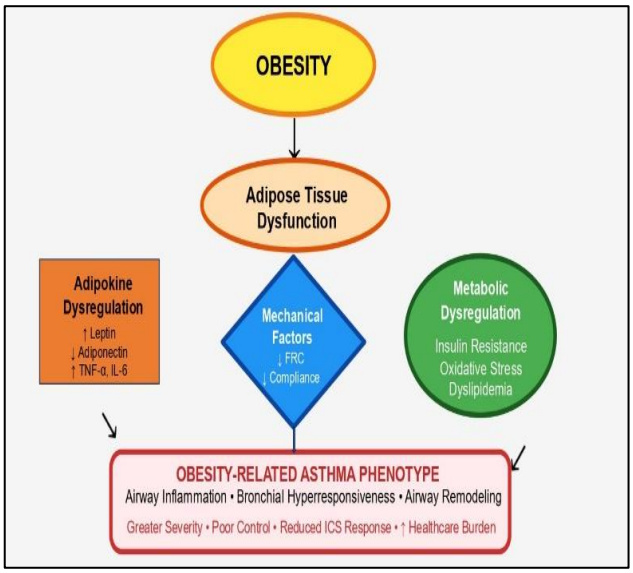


**Figure 1: Pathophysiological mechanisms linking obesity to asthma.**

*Three primary mechanisms: (1) adipokine-mediated systemic inflammation with elevated leptin, decreased adiponectin, increased TNF-alpha/IL-6/IL-1beta, (2) mechanical respiratory compromise reducing lung volumes and chest wall compliance, (3) metabolic dysregulation including insulin resistance and oxidative stress. These lead to airway inflammation, bronchial hyperresponsiveness, and remodeling, creating obesity-related asthma phenotype with greater severity, poor control, reduced ICS responsiveness and increased healthcare burden. Abbreviations: FRC=functional residual capacity; TNF-α=tumor necrosis factor alpha; IL-6=interleukin-6; ICS=inhaled corticosteroids

### *Adipokine-mediated inflammation*

Adipose tissue functions as active endocrine organ secreting bioactive adipokines modulating immune function.[10,11] In obesity, adipokine production becomes dysregulated, fostering chronic pro-inflammatory states.[16] Children with obesity and asthma demonstrate elevated leptin levels correlating with severity.[16-18] Leptin promotes pro-inflammatory cytokines, enhances T-helper responses, and increases airway smooth muscle contractility.[16,19] Conversely, adiponectin with anti-inflammatory properties is reduced, associating with increased airway inflammation and diminished lung function.[18,20] Beyond adipokines, expanded adipose tissue secretes elevated TNF-alpha, IL-6, IL-1beta, perpetuating

inflammation and potentially interfering with glucocorticoid receptor signaling, contributing to reduced corticosteroid responsiveness.[11,16,20]

### *Mechanical factors affecting respiratory function*

Independent of inflammation, obesity exerts direct mechanical effects.[3,21] Increased thoracic and abdominal adipose tissue reduces chest wall compliance, requiring greater inspiratory effort and increasing work of breathing.[16,21] These changes result in reduced functional residual capacity and expiratory reserve volume while promoting premature small airway closure.[3] Studies demonstrate mixed obstructive-restrictive patterns with pronounced small airway dysfunction.[21] Increased mechanical load impairs diaphragmatic excursion and limits bronchodilator response.[21]

### *Metabolic dysregulation and dietary factors*

Metabolic abnormalities including insulin resistance, impaired glucose tolerance, dyslipidemia, and altered glucose-fatty acid metabolism contribute to asthma pathogenesis.[21-23] Insulin resistance promotes airway smooth muscle proliferation and contractility.[20,23] Hyperinsulinemia and elevated free fatty acids enhance reactive oxygen species production and oxidative stress, amplifying inflammatory cascades.[20,21] Furthermore, high consumption of ultra-processed foods correlates with increased obesity risk through hyperpalatable formulations promoting overconsumption and may exert direct pro-inflammatory effects through increased intestinal permeability and gut dysbiosis.[5]

### *Genetic and epigenetic contributions*

Genetic studies including genome-wide association analyses have identified numerous loci associated with increased susceptibility to both obesity and asthma, with some polymorphisms including variants in FTO and PRKCA genes demonstrating associations with both phenotypes.[21] Additionally, emerging epigenetic research suggests that environmental factors including maternal obesity and early-life dietary patterns may induce stable alterations in DNA methylation patterns and histone modifications that influence both metabolic programming and immune programming.[21]

## PHENOTYPE CLASSIFICATION

Asthma represents heterogeneous syndrome encompassing distinct phenotypes differing in pathophysiology, clinical characteristics, natural history, and therapeutic responses.[3,10,11,21] Contemporary understanding recognizes useful categorization based on age of onset, obesity status, allergic sensitization, and inflammatory pattern. Table 1 provides comprehensive comparison of three clinically important phenotypes.

**Table 1: Asthma phenotype comparison.**

| Features | Classical allergic | Early allergic+obesity | Late non-allergic+obesity |
|---|---|---|---|
| **Onset age (in years)** | 2-5 | 2-5 | >8 |
| **Type 2 inflammation** | High (eosinophilic) | Moderate-High (mixed) | Low (neutrophilic) |
| **ICS response** | Excellent >80% | Moderate 50-70% | Poor <40% |
| **Weight loss response** | Not applicable | Significant benefit | Dramatic benefit |
| **Comorbidities** | Allergic rhinitis, eczema | Rhinitis, OSA, GERD | OSA, GERD, metabolic syndrome, depression |
| **Treatment** | ICS+allergen avoidance | ICS/LABA+weight management | LTRA/ICS/LABA+intensive weight mgmt+biologics |

*Comparison showing obesity-related phenotypes with progressive severity and reduced responsiveness, necessitating integrated approaches. Abbreviations: ICS=inhaled corticosteroids; LABA=long-acting beta-agonist; LTRA=leukotriene receptor antagonist; OSA=obstructive sleep apnea; GERD=gastroesophageal reflux disease.

Recognition of distinct phenotypes carries important therapeutic implications.[3,10,11] Classical allergic asthma responds excellently to standard ICS therapy. In contrast, obesity-related phenotypes, particularly late-onset non-allergic pattern, demonstrate attenuated ICS responses and require aggressive multifaceted interventions combining higher-dose or combination respiratory medications, intensive weight management, and systematic comorbidity treatment.

## CLINICAL MANIFESTATIONS AND SYSTEMATIC ASSESSMENT

Children with obesity-related asthma exhibit more severe manifestations.[3,6,7] They experience higher symptom frequency, more nighttime awakenings, increased rescue medication use, greater emergency department utilization, reduced exercise tolerance, lower quality-of-life scores, and higher school absenteeism.[3,6,9,15] Initial assessment requires comprehensive approach including BMI classification using CDC growth charts, spirometry with bronchodilator response for children aged 5 or older, asthma control evaluation using validated asthma control test or childhood asthma control test, and systematic comorbidity screening for obstructive sleep apnea, gastroesophageal reflux, metabolic syndrome components, and mental health conditions.[8,24,25]

Comprehensive history should elicit respiratory symptoms including frequency, severity, and triggers,

assess sleep quality and daytime somnolence, evaluate dietary patterns emphasizing ultra-processed food consumption, quantify physical activity levels, and screen for mental health concerns.[8,24] Physical examination includes anthropometric measurements, vital signs with blood pressure percentiles, respiratory examination, and signs of comorbidities including acanthosis nigricans suggesting insulin resistance.[22] Diagnostic testing includes baseline spirometry establishing presence of airway obstruction and bronchodilator responsiveness, fractional exhaled nitric oxide measurement guiding phenotype classification, and metabolic screening for children with BMI at or above 95$^{th}$ percentile.[22,24,25]

## MONITORING AND FOLLOW-UP

Structured follow-up is essential for successful outcomes.[14,24,25]

During active management phases encompassing the first 6 months, visits should occur every 3 months incorporating asthma control assessment via validated instruments with ACT score below 20 indicating poor control, anthropometric measurements with BMI trajectory plotting on growth charts targeting 5%-10% reduction or stabilization, medication adherence review through refill histories and direct observation of inhaler technique, brief lifestyle assessment evaluating dietary changes and physical activity, and comorbidity screening particularly for sleep-disordered breathing symptoms.[14,24,25]

Every 6 months during active phase, spirometry should be repeated to evaluate treatment response with expected 5%-10% FEV1 improvement with successful weight reduction, and metabolic screening repeated if indicated.[22,24,25] Annually, comprehensive systems review should be performed with action plan updates. For stable patients achieving asthma control scores 20 or higher and demonstrating healthy BMI trajectory, maintenance phase visits can extend to every 3-6 months while continuing systematic monitoring.[24,25] Escalation triggers prompting return to every-3-month visits include asthma control score below 20, two or more exacerbations requiring systemic corticosteroids within 6 months, emergency department visit or hospitalization, worsening BMI trajectory, emergence of new comorbidity, or medication adherence below 80%.[24,25]

## STEPWISE CLINICAL ALGORITHM

Figure 2 illustrates systematic approach from initial presentation through long-term maintenance with clearly defined decision points and escalation criteria.

## EVIDENCE-BASED MANAGEMENT STRATEGIES

Effective management requires dual-target approach simultaneously addressing asthma and obesity. Figure 3 illustrates integrated framework with specific interventions for each domain.

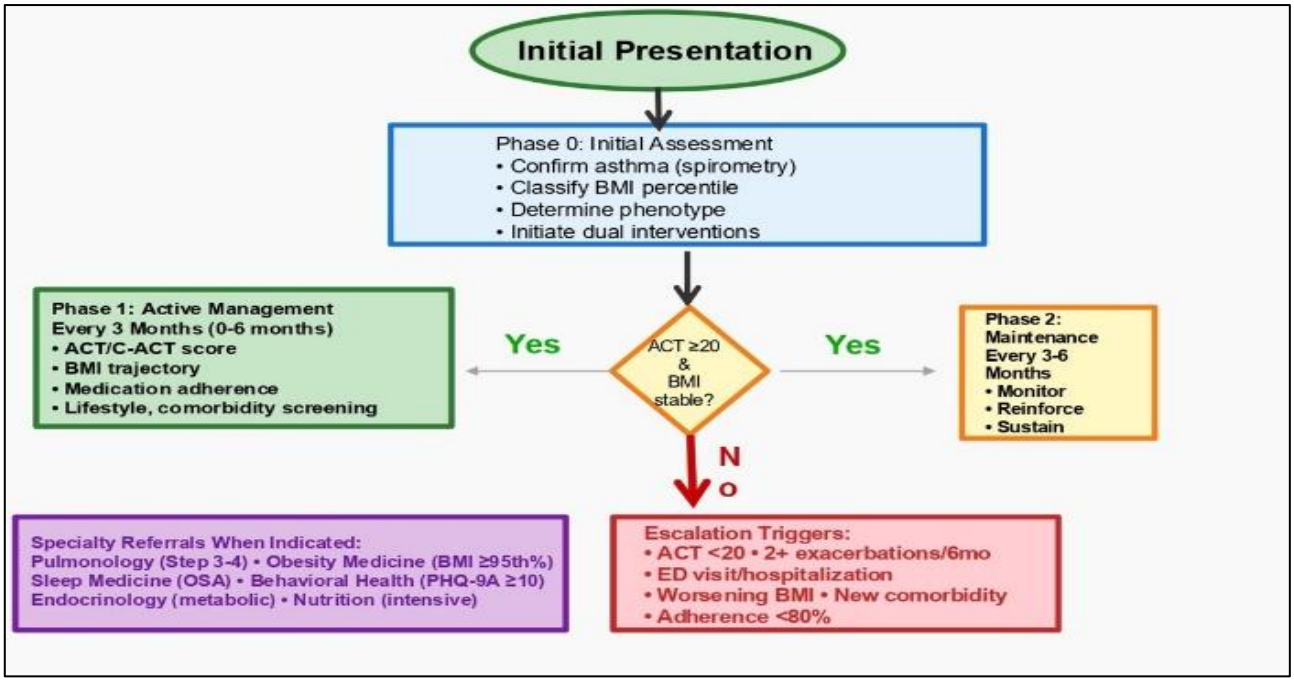


**Figure 2: Stepwise clinical algorithm.**

*Algorithm showing progression from initial assessment through Phase 1 active management every 3 months with decision points for asthma control and weight trajectory to phase 2 maintenance every 3-6 months for stable patients. Includes clearly defined escalation triggers and specialty referral criteria. Abbreviations: BMI=body mass index; ACT=Asthma Control Test; ICS=inhaled corticosteroids; FeNO=fractional exhaled nitric oxide; OSA=obstructive sleep apnea; PHQ-9A=Patient Health Questionnaire for Adolescents; FEV1=forced expiratory volume; PAQLQ=Pediatric Asthma Quality of Life Questionnaire.

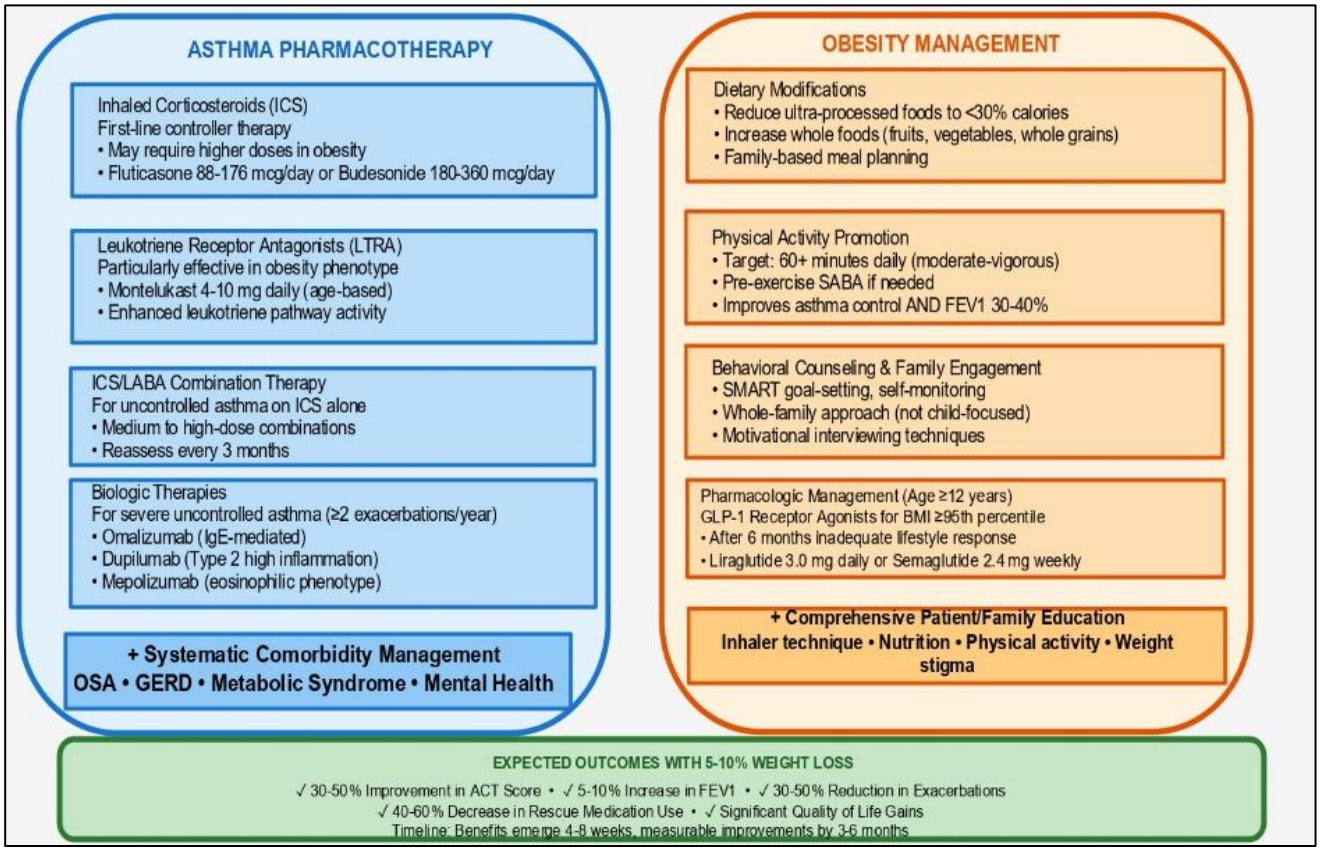


**Figure 3: Integrated management framework.**

*Dual-column framework showing asthma pharmacotherapy alongside obesity management with central integration emphasizing comorbidity management, comprehensive education, and care coordination. Expected outcomes with 5-10% weight loss include 30-50% ACT improvement, 5-10% FEV1 increase, 30-50% reduction in exacerbations. Abbreviations: ICS=inhaled corticosteroids; LABA=long-acting beta-agonist; LTRA=leukotriene receptor antagonist; OSA=obstructive sleep apnea; GERD=gastroesophageal reflux disease; SABA=short-acting beta-agonist; GLP-1=glucagon-like peptide-1; ACT=Asthma Control Test; FEV1=forced expiratory volume in 1 second.

## ASTHMA PHARMACOTHERAPY

### *Inhaled corticosteroids*

Inhaled corticosteroids remain the cornerstone of controller therapy, although children with obesity may require greater treatment intensity to achieve asthma control comparable to normal-weight peers.[3,10,24,25] Treatment is initiated at guideline-recommended doses and adjusted based on clinical response.[24,25] The 2024 GINA (Global Initiative for asthma) guidelines specifically highlight that asthma in obesity is more difficult to control and recommend integrating weight reduction as routine care component.[24]

### *Leukotriene receptor antagonists*

Leukotriene receptor antagonists demonstrate effectiveness in obesity-related asthma phenotype, likely reflecting enhanced leukotriene pathway activity in obesity.[3,10,11] Montelukast may be used as add-on therapy or as an alternative in children with contraindications to inhaled corticosteroids.[24,25] Careful monitoring for neuropsychiatric side effects is essential.[24]

### *Combination therapy and step-up approaches*

For uncontrolled asthma despite adequate ICS adherence and technique, ICS/LABA combination therapy should be employed with careful reassessment every 3 months.[24,25] Medium to high-dose ICS/LABA combinations may be required for children with obesity-related asthma who demonstrate inadequate response to standard doses.[10,11]

### *Biologic therapies*

Biologic therapies, including omalizumab for IgE-mediated disease, dupilumab for type 2 inflammatory asthma, and mepolizumab for eosinophilic asthma, may be considered in pediatric patients with severe asthma that remains uncontrolled despite optimized high-dose controller therapy, with recurrent exacerbations or persistent symptoms.[10,24,25]

Data specifically in obesity-related asthma remain limited but emerging evidence suggests potential benefits particularly for dupilumab which targets multiple type 2 inflammatory pathways.[10]

## OBESITY MANAGEMENT

### *Dietary modifications*

Dietary modifications should emphasize reducing ultra-processed foods to below 30% of daily calories while increasing whole foods including fruits, vegetables, whole grains, and lean proteins.[5,14,26] Practical strategies include family-based meal planning, label reading education, home cooking emphasis, and portion control education.[14,26] Crucially, interventions must be family-based involving parents as role models and promoting whole-family changes rather than singling out affected child.[8,14,26]

### *Physical activity promotion*

Physical activity recommendations target 60 or more minutes daily of moderate-to-vigorous activity combining aerobic and strength-building components.[14,26,27] For children with asthma, special considerations include pre-exercise short-acting beta-agonist use 15-20 minutes before activity if needed, adequate warm-up period, and environmental adjustments.[24,25,27] Evidence demonstrates physical activity improves asthma control and FEV1 while reducing exacerbation frequency by 30-40%.[27]

### *Behavioral counseling and family engagement*

Behavioral counseling incorporating SMART goal setting, self-monitoring through food and activity logs, positive reinforcement emphasizing efforts over outcomes, and problem-solving skills training proves essential.[14,26] Multicomponent programs integrating diet, activity, and behavioral strategies demonstrate superior efficacy to single-modality interventions.[14,26,27] Family engagement represents critical success factor with evidence demonstrating that interventions involving parents achieve significantly greater weight loss and asthma improvement compared to child-only interventions.[14]

### *Pharmacologic obesity management*

For adolescents aged 12 years or older with BMI at or above 95$^{th}$ percentile who demonstrate inadequate response to intensive lifestyle interventions after 6 months, pharmacologic obesity management using glucagon-like peptide-1 receptor agonists should be considered per 2023 AAP clinical practice guidelines.[26]

Liraglutide and semaglutide have demonstrated clinically meaningful BMI reductions of approximately 5-15% in pediatric clinical trials.[26,28] These agents may offer benefits for asthma through direct anti-inflammatory effects in addition to weight-loss-mediated improvements.[28] Pharmacotherapy must always be combined with ongoing lifestyle interventions, requires close monitoring monthly initially then every 3 months, and necessitates shared decision-making regarding long-term safety.[26,28]

### *Comorbidity management*

Comprehensive care requires systematic attention to multiple comorbidities that substantially influence asthma outcomes. Figure 4 illustrates common conditions requiring screening and treatment.

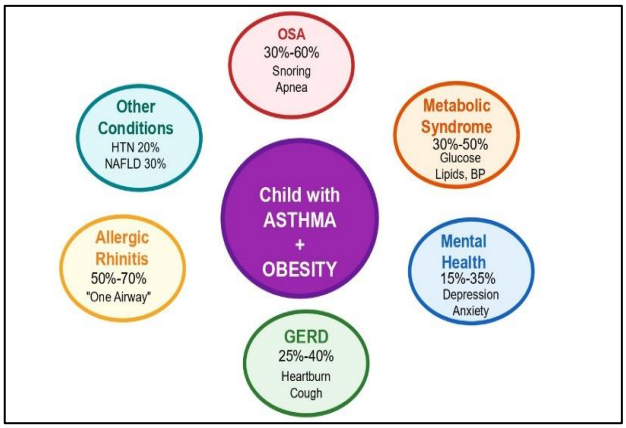


**Figure 4: Common comorbidities.**

*Hub-and-spoke design showing six major comorbidity categories requiring systematic screening: OSA affecting 30-60%, metabolic syndrome 30%-50%, mental health issues including depression 15-30% and anxiety 20%-35%, GERD 25%-40%, allergic rhinitis 50%-70%, and other conditions. Central message emphasizes systematic screening with treatment improving asthma outcomes. Many improve with weight loss. Abbreviations: OSA=obstructive sleep apnea; BP=blood pressure; GERD=gastroesophageal reflux disease; HTN=hypertension; NAFLD=non-alcoholic fatty liver disease.

### *Obstructive sleep apnea*

Obstructive sleep apnea affects 30%-60% of children with both obesity and asthma, worsening nocturnal asthma symptoms and increasing exacerbation risk 2-3-fold.[21,22] Screening should occur at every visit using validated questionnaires assessing snoring frequency, witnessed apneas, gasping, morning headaches, and daytime sleepiness.[22,24] Positive screening warrants polysomnography.[22] Management includes weight loss as primary intervention, adenotonsillectomy if tonsillar hypertrophy present, and continuous positive airway pressure for moderate-severe disease.[22] OSA treatment demonstrably improves asthma control and reduces exacerbation frequency.[21,22]

### *Gastroesophageal reflux disease*

Gastroesophageal reflux disease occurs in 25%-40%, with increased intra-abdominal pressure from obesity promoting lower esophageal sphincter dysfunction.[8,24] Symptoms include heartburn, regurgitation, nocturnal cough, and chest pain.[24] Management emphasizes lifestyle modifications including weight loss, avoiding

late-night eating at least 2 hours before bedtime, head-of-bed elevation, and trigger food avoidance, with pharmacologic therapy using H2-receptor blockers or proton pump inhibitors for symptomatic patients.[24,25]

### *Metabolic syndrome components*

Metabolic syndrome components including insulin resistance, dyslipidemia, and hypertension occur in 30%-50% of children with severe obesity.[21-23] Screening includes fasting glucose, hemoglobin A1c, lipid panel, and blood pressure measurement at every visit.[22,23,26] Management emphasizes lifestyle modification as first-line with endocrinology referral for multiple abnormalities.[22,26] Insulin resistance independently correlates with reduced lung function and greater asthma severity even after adjusting for BMI.[21,23]

### *Mental health conditions*

Mental health issues including depression affecting 15%-30% and anxiety disorders affecting 20-35% significantly impact medication adherence, symptom perception, quality of life, and promote emotional eating.[3,8,15] Screening using PHQ-9 Modified for Adolescents and SCARED anxiety screener should occur at baseline and every 6 months with behavioral health referral for positive screens.[24-26] Addressing mental health represents critical component of integrated care given bidirectional relationships between psychological distress, obesity, and asthma severity.[15]

### *Allergic rhinitis*

Allergic rhinitis affects 50%-70% of children with allergic asthma, with unified airway concept recognizing upper and lower airway inflammation linkage.[24,25] Management with intranasal corticosteroids as first-line therapy demonstrably improves lower airway control when treated.[24,25] Combination therapy with oral antihistamines may be required for moderate-severe symptoms.[24]

### *Other associated conditions*

Additional comorbidities requiring attention include cardiovascular manifestations with hypertension affecting 20% and early atherosclerotic changes, musculoskeletal complications including Blount disease and slipped capital femoral epiphysis limiting physical activity, non-alcoholic fatty liver disease affecting 30-40% of children with severe obesity, and dermatologic manifestations including acanthosis nigricans serving as clinical marker of insulin resistance.[22,23]

## PATIENT AND FAMILY EDUCATION

Comprehensive education is central to effective management of obesity-related asthma.[8,14,24] Asthma education should focus on symptom recognition, correct inhaler technique with demonstration and return demonstration, identification and avoidance of triggers, understanding written asthma action plans, and distinguishing controller from rescue medications.[14,24,25] Obesity and weight management education should present obesity as a chronic condition requiring longitudinal care, outline associated health risks, and emphasize the benefits of modest weight reduction. Practical counseling may include guidance on nutrition, meal planning, label reading, and safe physical activity participation tailored to asthma symptoms, with realistic goals such as a 5%-10% reduction framed as clinically meaningful.[14,26] Addressing weight stigma is essential and includes the use of person-first language, a focus on health-promoting behaviors rather than appearance, promotion of positive body image, and engagement of the family unit in care.[8,14,26] Motivational interviewing strategies that assess readiness for change, explore ambivalence, and support self-efficacy can further enhance patient and family engagement.[14,26]

## EXPECTED OUTCOMES AND TIMELINE

Evidence suggests that modest weight loss of approximately 5-10% is associated with clinically meaningful improvements in children with obesity-related asthma.[7,27,29] Studies report improvements in asthma control, as measured by ACT or C-ACT scores, along with modest gains in lung function and reductions in exacerbations requiring systemic corticosteroids.[6,7,27,29] Decreased reliance on rescue medications, improved quality of life scores, and reductions in healthcare utilization, including emergency department visits and hospitalizations, have also been observed.[6,7,27,29] Additional benefits include favorable changes in systemic inflammation, such as reductions in C-reactive protein and leptin with increases in adiponectin, as well as improvements in metabolic parameters including insulin sensitivity and lipid profiles.[17,18,23]

Improvement in symptoms may emerge within weeks of initiating integrated interventions, with measurable changes in asthma control and lung function typically observed within several months and more sustained benefits evident over 6-12 months.[7,27,29] Achieving and maintaining these outcomes is supported by systematic assessment, coordinated management of asthma and obesity, family engagement, regular follow-up with goal adjustment, proactive identification of barriers, and multidisciplinary collaboration when available.[14,26]

## IMPLEMENTATION BARRIERS AND SOLUTIONS

Several barriers can limit implementation in routine clinical practice. Time constraints may be addressed through structured frameworks that use tables and algorithms, focus on one to two achievable goals per visit, involve allied health professionals such as registered dietitians or health educators, and provide

written resources for families.[8,14] Challenges with patient and family adherence may be mitigated through motivational interviewing, identification of practical barriers such as cost, access, transportation, and health literacy, frequent early follow-up, and reinforcement of incremental progress.[14,26] Medication-related barriers can be managed by prioritizing generic options when available, utilizing patient assistance programs, supporting insurance navigation, and aligning care with step-therapy requirements.[26]

Limited access to subspecialty care may be partially addressed through telemedicine, project ECHO based models that extend specialist support to primary care clinicians, and clear algorithms that support management of mild to moderate disease in primary care settings.[8] Addressing social determinants of health is essential and includes routine screening for social needs, connection to community resources, realistic goal setting that reflects family circumstances, and broader advocacy to address food insecurity and neighborhood safety.[14,26]

## FUTURE DIRECTIONS AND RESEARCH PRIORITIES

Despite increasing recognition of obesity-related asthma, substantial knowledge gaps persist. Research priorities include targeted multidisciplinary interventions, biomarker-guided therapy, long-term outcome studies, comparative effectiveness of pharmacotherapies, identification of novel biologic targets, and improved implementation strategies for diverse clinical settings.[11,15,30]

Emerging therapies of interest include dual GLP-1/GIP receptor agonists, such as tirzepatide, with pediatric trials currently underway; novel anti-inflammatory agents targeting obesity-related pathways beyond classical allergic inflammation; personalized nutrition strategies informed by genomic and metabolomic profiling; microbiome-based approaches addressing gut-lung axis dysbiosis; and digital health interventions with remote monitoring to support ongoing assessment without increasing clinic burden.[11,15,28]

Health equity remains a critical consideration, highlighting the need for research that addresses social and cultural determinants of obesity and asthma, develops culturally responsive interventions, and improves equitable access to multidisciplinary care and emerging therapies across diverse populations.[2,15]

## CONCLUSION

Childhood obesity and asthma represent overlapping chronic conditions creating complex management challenges with substantially greater morbidity, lower quality of life, and increased healthcare utilization compared to either condition alone. This comprehensive review addresses critical gaps by providing evidence-based, actionable frameworks specifically designed for real-world implementation including systematic assessment protocols, structured monitoring schedules with specific tools and action thresholds, stepwise algorithms with defined decision points and escalation triggers, integrated management emphasizing parallel interventions for both conditions, and practical strategies overcoming common implementation barriers. Evidence demonstrates that modest weight reduction produces clinically meaningful improvements in asthma control, lung function, exacerbations, and quality of life, with benefits emerging within weeks and becoming measurable by three to six months. Recognition that obesity-related asthma represents a distinct phenotype with late onset, non-allergic inflammation, reduced corticosteroid responsiveness, and high comorbidity burden guides appropriate phenotype-specific therapeutic strategies. The frameworks outlined offer a practical, evidence-informed approach to integrating airway and metabolic considerations in the care of children with obesity-related asthma.

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## REFERENCES

1. Zhou W, Tang J. Prevalence and risk factors for childhood asthma: a systematic review and meta-analysis. BMC Pediatr. 2025;25(1):50.
2. Fryar CD, Carroll MD, Afful J. Prevalence of Overweight, Obesity, and Severe Obesity Among Children and Adolescents Aged 2-19 Years: United States, 1963-1965 Through 2017-2018. NCHS Health E-Stats. 2020.
3. Chamarthi VS, McCauley JL, Nadler EP, Ro S. Childhood obesity management in primary care: Bridging guidelines and practice. Obesity Pillars. 2025;100225.
4. Chen Z, Salam MT, Alderete TL, Rima H, Theresa MB, Kiros B, et al. Effects of Childhood Asthma on the Development of Obesity among School-aged Children. Am J Respir Crit Care Med. 2017;195(10):1181-8.
5. Chamarthi VS, Shirsat P, Sonavane K, Saketh P, Usha R, Harikrishna CP, et al. The impact of ultra-processed foods on pediatric health. Obesity Pillars. 2025;16:100203.
6. Ahmadizar F, Vijverberg SJH, Arets HGM, de Boer A, Lang JE, Kattan M, et al. Childhood obesity in relation to poor asthma control and exacerbation: a meta-analysis. Eur Respir J. 2016;48(4):1063-73.
7. Lang JE, Hossain J, Dixon AE, David S, Robert AW, Stephen PP, et al. Does age impact the obese asthma phenotype? Chest. 2011;140(6):1524-33.
8. Chamarthi VS. The 15-Minute Conundrum: Strategies for Effective Pediatric Obesity Counseling in Primary Care. Eur J Med Health Sci. 2025;7(6):9-14.

9. Gunasekaran V, Kapoor V, Chamarthi VS. Childhood obesity prevalence and prevention strategies in primary care: A comprehensive review. Cureus. 2025;17(12):e98456.
10. Chamarthi VS, Chamarthi S, Gunasekaran V. Biologic Therapies in Pediatric Asthma: A Clinical Review of Current Advances. Eur J Clin Med. 2025;6(5):1-10.
11. Forno E, Han YY, Mullen J, Celedón JC. Overweight, Obesity, and Lung Function in Children and Adults-A Meta-analysis. J Allergy Clin Immunol Pract. 2018;6(2):570-81.
12. Black MH, Smith N, Porter AH, Steven JJ, Corinna K. Higher prevalence of obesity among children with asthma. Obesity (Silver Spring). 2012;20(5):1041-7.
13. Ma Y, Wang L, Tao M, Zhidan B, Renqiang Y, Guihua L, et al. Interaction between obesity and asthma in children and adolescents with hypertension based on NHANES 2007-2020. Front Public Health. 2025;13:1526832.
14. Martin MA, Rothschild SK, Lynch E, Katherine KC, Militza MP, Jose LR, et al. Addressing asthma and obesity in children with community health workers. BMC Pediatrics. 2016;16(1):198.
15. Vila G, Zipper E, Dabbas M, Catherine B, Jean JR, Claude R, et al. Mental disorders in obese children and adolescents. Psychosom Med. 2004;66(3):387-94.
16. Peters U, Dixon AE, Forno E. Obesity and asthma. J Allergy Clin Immunol. 2018;141(4):1169-79.
17. Sánchez-Ortega H, Jiménez-Cortegana C, Novalbos-Ruiz JP, Gómez-Bastero A, Soto-Campos JG, Sánchez-Margalet V, et al. Role of Leptin as a Link between Asthma and Obesity: A Systematic Review and Meta-Analysis. Int J Mol Sci. 2022;24(1):546.
18. Sood A, Shore SA. Adiponectin, Leptin, and Resistin in Asthma: Basic Mechanisms through Population Studies. J Allergy (Cairo). 2013;2013:785835.
19. Shore SA, Terry RD, Flynt L. Adiponectin attenuates allergen-induced airway inflammation and hyperresponsiveness in mice. J Allergy Clin Immunol. 2006;118(2):389-95.
20. Taylor EB. The complex role of adipokines in obesity, inflammation, and autoimmunity. Clin Sci (Lond). 2021;135(6):731-52.
21. Mazzotta C, Barkai L. Obesity and Asthma in Children-Coexistence or Pathophysiological Connections? Biomedicines. 2025;13(5):1114.
22. Shirsat P, Balachandran M, Chamarthi VS, Sonavane K. Obesity and chronic kidney disease: A comprehensive review. J CardioRenal Med. 2025;1(1):4.
23. Guerrero-Romero F, Simental-Mendía LE, González-Ortiz M, et al. The product of triglycerides and glucose, a simple measure of insulin sensitivity. Comparison with the euglycemic-hyperinsulinemic clamp. J Clin Endocrinol Metab. 2010;95(7):3347-51.
24. Global Initiative for Asthma. 2024 GINA Main Report. 2024.
25. National Heart, Lung, and Blood Institute. 2020 Focused Updates to the Asthma Management Guidelines. 2020.
26. Hampl SE, Hassink SG, Skinner AC, Sarah CA, Sarah EB, Christopher FB, et al. Clinical Practice Guideline for the Evaluation and Treatment of Children and Adolescents With Obesity. Pediatrics. 2023;151(2):e2022060640.
27. Okoniewski W, Lu KD, Forno E. Weight Loss for Children and Adults with Obesity and Asthma. Ann Am Thorac Soc. 2019;16(5):613-25.
28. Kelly AS, Auerbach P, Barrientos-Perez M, Inge G, Paula MH, Claude M, et al. A Randomized, Controlled Trial of Liraglutide for Adolescents with Obesity. N Engl J Med. 2020;382(22):2117-28.
29. Eslick S, Jensen ME, Collins CE, Peter GG, Jodi H, Lisa GW. Characterising a Weight Loss Intervention in Obese Asthmatic Children. Nutrients. 2020;12(2):507.
30. Lang L, Ma M, Zhao H, Jialin Z, Sheng L, Hua L. Global research trends in obesity-related asthma (2004-2023): a bibliometric analysis. Front Nutr. 2025;12:1528366.

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