



Effect of metabolic syndrome on pulmonary dysfunction in children with asthma

Hyo-Bin Kim, MD, PhD*

Department of Pediatrics, Inje University Sanggye Paik Hospital, Seoul, Korea

Key message

- The prevalence of metabolic syndrome increased in Korean children during the coronavirus disease 2019 pandemic owing to reduced physical activity resulting from social distancing.
- Metabolic syndrome impacts pulmonary dysfunction in childhood asthma.
- Further studies are needed to understand the mechanism linking asthma and metabolic syndrome and develop interventions.

Asthma is a well-known chronic airway inflammatory disease, but not much attention has been paid to the fact that it is also a systemic inflammatory disease. Recent studies have focused on the association between asthma and extrapulmonary abnormalities such as metabolic syndrome, depression/anxiety, and cardiovascular abnormalities. It is important to recognize that organ dysfunction may occur in asthma beyond the airway associated with systemic inflammation. The coexistence of systemic abnormalities may contribute to asthma-related morbidity and mortality.

Obesity is a recognized risk factor for the development of and modification of asthma in children. Previous studies reported that obese subjects are at increased risk of asthma, and obese children with asthma experience more frequent asthma symptoms or exacerbations, reduced responses to asthma medications, and more common changes in lung function.¹⁾ In terms of systemic inflammation, metabolic dysfunction is a broader concept than obesity, which is merely a marker of fat mass and only one component of metabolic syndrome. The criteria for diagnosing metabolic syndrome include abdominal obesity (increased waist circumference), high blood pressure, high blood triglyceride, low high-density lipoprotein cholesterol, and high fasting glucose levels. During the coronavirus disease 2019 pandemic, the pre-

valence of metabolic syndrome and obesity among children increased due to reduced physical activity; it has since become a public health burden in Korea that requires attention.²⁾

According to the Childhood Asthma Management Program, which reported a significant effect of childhood obesity on lung function, body mass index (BMI) is associated with higher forced expiratory volume in 1 second (FEV₁) and forced vital capacity (FVC) but a lower FEV₁/FVC ratio.³⁾ A recent meta-analysis also reported that a lower FEV₁/FVC ratio was associated with BMI.⁴⁾ Forno et al.⁵⁾ suggested the concept of "airway dysanapsis," an incongruence between lung parenchymal growth and airway caliber, to explain the higher FEV₁ and FVC but stated that the lower FEV₁/FVC ratio was due to the larger effect of childhood obesity on FVC. However, despite numerous studies exploring the association between obesity and lung function, findings remain conflicting and influenced by factors such as sex and age. Moreover, regarding changes in airway hyperresponsiveness (AHR) in children with obesity, studies have reported inconsistent findings, potentially due to small sample sizes or variations in participant populations. Similar findings were observed in Korean children with asthma, in whom metabolic components including waist obesity and high triglyceride and low-density lipoprotein cholesterol levels were associated with pulmonary dysfunction and a low FEV₁/FVC ratio but not with AHR.⁶⁾

The suggested mechanisms linking asthma and obesity include mechanical alterations (such as airway dysanapsis), genetic overlap between asthma and obesity, and systemic inflammation. Excessive fat accumulation in the thorax and abdomen was previously thought to lead lung compression, resulting in a reduction in lung volume, including functional residual capacity and expiratory reserve volume.⁷⁾ However, the recent concept of

Corresponding author: Hyo-Bin Kim, MD, PhD. Department of Pediatrics, Inje University Sanggye Paik Hospital, 1342 Dongil-ro Nowon-gu, Seoul 01696, Korea

✉ Email: hbkim@paik.ac.kr, <https://orcid.org/0000-0002-1928-722X>

*Current affiliation: Department of Pediatrics, Inha University School of Medicine, Incheon, Korea

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metabolic syndrome suggests that the association with asthma is mediated by systemic inflammation, insulin resistance, and altered lipid metabolism.⁸⁾ Changes in innate immune responses involving innate lymphoid cells and the Th17 pathways also play a crucial role in adipose tissue homeostasis and asthma. Elevated levels of interleukin-6, a strong biomarker in asthma severity and metabolic traits, have been associated with severe asthma symptoms and metabolic dysfunction. Adipokines, such as leptin and adiponectin, produced by the adipose tissue also affect asthma. Moreover, increased oxidative stress has been observed in patients with obesity-related asthma. The production of nitric oxide, an endogenous bronchodilator, may be regulated by reduced arginine bioavailability.

Several studies have provided evidence that weight loss through various interventions improves asthma control and spirometric lung function in adults.⁹⁾ However, pediatric studies in this field are scarce, although some have reported similar improvements to those in adults. In addition, there is current evidence on the ability of weight loss to improve neutrophilic airway inflammation and clinical outcomes,¹⁰⁾ which could be an important future direction for symptom control in obese children with asthma. Further research is required to identify individualized management strategies for controlling both metabolic syndrome and asthma.

In conclusion, the rising prevalence of metabolic syndrome due to reduced physical activity may impact asthma phenotypes in children, including alterations in pulmonary function. Further research is needed to uncover the precise mechanisms linking childhood asthma and metabolic syndrome and develop methods to improve asthma symptoms and pulmonary function.

See the article “Metabolic syndrome and pulmonary dysfunction in asthmatic children during the COVID-19 pandemic” via <https://doi.org/10.3345/cep.2023.01480>.

Footnotes

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ORCID:

Hyo-Bin Kim  <https://orcid.org/0000-0002-1928-722X>

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