Vol. 4(3), 133-141, 2026 PEDIATR RESPIR J

NARRATIVE REVIEW

Respiratory involvement in inflammatory bowel disease in children: a case-based narrative review

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ABSTRACT

Pediatric inflammatory bowel disease (IBD) is increasingly recognized as a systemic inflammatory disorder, although its respiratory manifestations remain frequently overlooked. While clinically apparent pulmonary involvement occurs in fewer than 1% of patients, subclinical abnormalities may affect up to 40% of children and adolescents with IBD. We present the case of a 15-year-old boy with Crohn's disease (CD) who developed chronic tracheobronchitis, highlighting the diagnostic challenges involved in distinguishing primary extraintestinal manifestations (EIMs) from treatment-related pulmonary toxicity and opportunistic infections. We also review the spectrum of respiratory involvement in pediatric IBD, in which airway disease and interstitial lung disease (ILD) may evolve independently of intestinal disease activity. Current evidence suggests that impulse oscillometry (IOS) and measurement of the diffusing capacity of the lung for carbon monoxide (DLCO) are more sensitive than conventional spirometry for the early detection of pulmonary abnormalities. A multidisciplinary approach combined with structured longitudinal follow-up is essential to ensure early diagnosis, individualized management, and prevention of long-term respiratory sequelae.

IMPACT STATEMENT

Pulmonary involvement in pediatric IBD is uncommon but probably underrecognized, with subclinical abnormalities occurring far more frequently than clinically overt disease. This narrative review highlights the central role of the gut-lung axis in the pathogenesis of respiratory involvement and emphasizes the value of sensitive functional assessments, including DLCO and IOS, for the early detection of pulmonary abnormalities. Greater clinical awareness and a multidisciplinary approach are essential to improve the timely diagnosis and management of these potentially irreversible EIMs in children and adolescents.

INTRODUCTION

Inflammatory bowel disease (IBD), encompassing Crohn's disease (CD), ulcerative colitis (UC), and IBD-unclassified (IBD-U), is a chronic immune-mediated inflammatory disorder with well-recognized systemic manifestations (1-3). The global incidence of pediatric IBD has increased steadily over recent decades, particularly in Europe and North America, and approximately one-quarter of all patients are diagnosed before 18 years of age (2, 4). Compared with adult-onset disease,

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10.56164/PediatrRespirJ.2026.13

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KEY WORDS
Pediatric inflammatory bowel disease; pulmonary manifestations; extraintestinal manifestations; gut-lung axis; pulmonary function tests.

pediatric IBD presents unique clinical challenges, including impaired growth, delayed puberty (5), and a greater burden of extraintestinal manifestations (EIMs) (3, 4). Although EIMs affecting the skin, joints, eyes, and hepatobiliary system are well recognized, pulmonary manifestations (PMs) remain considerably underdiagnosed (6). Increasing evidence indicates that the gastrointestinal and respiratory tracts constitute a functionally integrated mucosal immune system linked through the gut-lung axis (4). Nevertheless, it remains uncertain whether pulmonary involvement represents a primary extraintestinal manifestation of IBD or results from immunosuppressive therapies or opportunistic infections (7). Distinguishing between these mechanisms is of major clinical importance because an incorrect diagnosis of drug-induced lung toxicity may lead to the unnecessary discontinuation of highly effective IBD treatments.

In this narrative review, we use a representative clinical case to discuss the spectrum of pulmonary involvement in pediatric IBD, focusing on current pathogenetic concepts, diagnostic challenges, and implications for clinical practice.

METHODS

A comprehensive literature search was performed using PubMed to identify relevant publications from January 1982 through April 2026. The search strategy combined Medical Subject Headings (MeSH) and free-text keywords, including “inflammatory bowel disease”, “Crohn’s disease”, “ulcerative colitis”, “pulmonary manifestations”, “extraintestinal manifestations”, and “gut-lung axis”, together with pediatric age-related filters.

Eligible studies included original research articles, systematic reviews, narrative reviews, and clinically relevant case series involving pediatric patients (<18 years of age). Only articles published in English were considered. Studies exclusively involving adult populations and conference abstracts without full-text availability were excluded.

CASE PRESENTATION

A 15-year-old boy with a one-year history of CD was followed at our institution for severe ileocolonic disease. Owing to persistent disease activity despite conventional therapy, he sequentially received mesalamine, azathioprine combined with systemic corticosteroids, methotrexate, and subsequently the anti-tumor necrosis factor (TNF) agent infliximab. Despite intensive medical treatment, his intestinal disease continued to progress, ultimately requiring surgical resection of the terminal ileum, cecum, and ascending colon. The postoperative course was initially uneventful.

Shortly after hospital discharge, the patient developed fever, sore throat, and symptoms consistent with acute bronchitis. Empirical treatment with azithromycin was prescribed by his primary care physician. Although the fever resolved promptly, a persistent productive cough prompted referral to our pediatric pulmonology clinic.

At presentation, the patient was afebrile, in no respiratory distress, and maintained an oxygen saturation of 98% while breathing room air. Chest auscultation revealed diffuse bilateral rhonchi with occasional wheezing. Laboratory investigations showed mildly elevated inflammatory markers. Spirometry demonstrated a mild obstructive ventilatory defect with significant bronchodilator responsiveness. Sputum culture yielded *Staphylococcus aureus*, while chest radiography showed bilateral hilar prominence with increased perihilar bronchovascular markings. Treatment with inhaled bronchodilators, airway clearance therapy, and targeted antibiotic therapy with amoxicillin-clavulanate resulted in partial clinical improvement; however, the productive cough persisted.

Given the persistence of respiratory symptoms, a comprehensive diagnostic workup was undertaken. Repeat pulmonary function testing demonstrated a mild restrictive ventilatory pattern, whereas bronchodilator responsiveness, fractional exhaled nitric oxide (FeNO), diffusing capacity of the lung for carbon monoxide (DLCO), and methacholine challenge testing were all within normal limits. Extensive investigations excluded alternative causes of chronic cough, including allergic disease, gastroesophageal reflux, primary immunodeficiency, cystic fibrosis, tuberculosis, and atypical respiratory infections.

High-resolution computed tomography (HRCT) of the chest demonstrated diffuse bronchial wall thickening with intraluminal mucus plugging but no evidence of bronchial stenosis, bronchiectasis, or parenchymal lung disease. Flexible bronchoscopy revealed diffusely inflamed tracheobronchial mucosa characterized by marked vascular congestion and abundant thick whitish secretions. Cytological analysis of bronchoalveolar lavage (BAL) fluid demonstrated a predominance of granulo-

cytes. Based on the combination of persistent respiratory symptoms, exclusion of infectious and alternative systemic causes, and characteristic bronchoscopic and radiological findings, chronic tracheobronchitis was diagnosed as an extraintestinal manifestation of CD. Treatment with inhaled corticosteroids resulted in complete resolution of respiratory symptoms and normalization of pulmonary function at follow-up.

A major diagnostic challenge was distinguishing persistent infection, drug-induced pulmonary toxicity, and a primary pulmonary extraintestinal manifestation of CD. The initial isolation of *Staphylococcus aureus* together with partial improvement following antibiotic therapy initially suggested prolonged bacterial tracheobronchitis. However, the persistence of symptoms despite appropriate antimicrobial treatment, the absence of radiological evidence of pneumonia or bronchiectasis, and the lack of sustained systemic inflammatory activity argued strongly against ongoing infection.

Drug-induced pulmonary toxicity was also carefully considered. Methotrexate-associated lung disease typically manifests as diffuse interstitial involvement with restrictive impairment accompanied by radiological infiltrates, none of which were observed in our patient. Likewise, pulmonary complications associated with anti-TNF therapy, including opportunistic infections, granulomatous disease, and hypersensitivity pneumonitis, were systematically excluded through microbiological, radiological, and bronchoscopic investigations. No imaging findings suggestive of interstitial lung disease (ILD) or pulmonary granulomatosis were identified.

The diagnosis of chronic tracheobronchitis as a pulmonary manifestation of IBD was ultimately supported by several converging findings: persistent productive cough despite adequate anti-infective therapy, bronchoscopic evidence of diffuse tracheobronchial inflammation with excessive airway secretions, exclusion of alternative etiologies, and rapid clinical response to inhaled corticosteroid therapy. Furthermore, the predominance of granulocytes in BAL fluid supported ongoing neutrophilic airway inflammation rather than persistent infection. Notably, respiratory symptoms developed during a period of severe, refractory intestinal disease requiring surgical intervention, consistent with previous observations that pulmonary EIMs may occur during periods of heightened systemic inflammatory activity.

Although chronic tracheobronchitis is a recognized pulmonary manifestation of IBD, pediatric cases remain exceedingly rare, particularly among patients receiving multiple immunomodulatory and biologic therapies. The principal strength of this case lies in the detailed diagnostic approach adopted to distinguish inflammatory airway disease from infectious and treatment-related pulmonary complications in an immunosuppressed adolescent. It also emphasizes the complementary role of bronchoscopy and HRCT in evaluating persistent respiratory symptoms, even in the absence of significant parenchymal abnormalities.

Overall, this case underscores the importance of considering pulmonary involvement in any child or adolescent with IBD who presents with persistent respiratory symptoms and illustrates the multidisciplinary diagnostic approach required to establish this uncommon but clinically significant extraintestinal manifestation.

Written informed consent for publication was obtained from the patient's parents before submission of this manuscript.

Pathophysiology: the gut-lung axis

The pathogenesis of PMs in IBD is complex and remains incompletely understood. Current evidence suggests that respiratory involvement results from the bidirectional immunological communication between the gastrointestinal and respiratory tracts, commonly referred to as the gut-lung axis (3, 8, 9). Although both organs originate from the primitive foregut during embryonic development, their relationship appears to be driven primarily by shared immune mechanisms rather than by their common embryological origin alone (10, 11).

Genetic susceptibility plays a pivotal role in this process. Variants in genes such as *NOD2*, *ATG16L1*, and *IL23R* predispose individuals to an exaggerated mucosal immune response and may increase susceptibility to EIMs (8, 12). Environmental factors, including intestinal dysbiosis, smoking, medications, and microbial exposures, may subsequently trigger immune activation in genetically susceptible individuals (3).

Disruption of the intestinal epithelial barrier facilitates the translocation of microbial products and inflammatory mediators into the systemic circulation, thereby influencing the pulmonary microenvironment (9). This mechanism may explain why subclinical pulmonary abnormali-

ties are frequently detected even during periods of gastrointestinal remission (9, 11).

The recruitment of inflammatory cells is believed to represent a key pathogenic mechanism. Neutrophils activated within the inflamed intestinal mucosa migrate into the bloodstream and accumulate within the pulmonary microvasculature, where they contribute to local tissue injury (3, 13). Likewise, gut-primed memory T lymphocytes may aberrantly home to the bronchus-associated lymphoid tissue (BALT) through chemokine-mediated trafficking involving receptors such as CCR4 and CCR6 (11). This process is amplified by a pro-inflammatory cytokine network dominated by tumor necrosis factor-α (TNF-α), interleukin (IL)-1, IL-6, and the Th17/IL-17 pathway (3, 4).

Collectively, these mechanisms support the concept that the lung functions as an immunological extension of the intestine rather than as an independent target organ, providing the biological basis for pulmonary involvement in pediatric IBD (**Figure 1**).

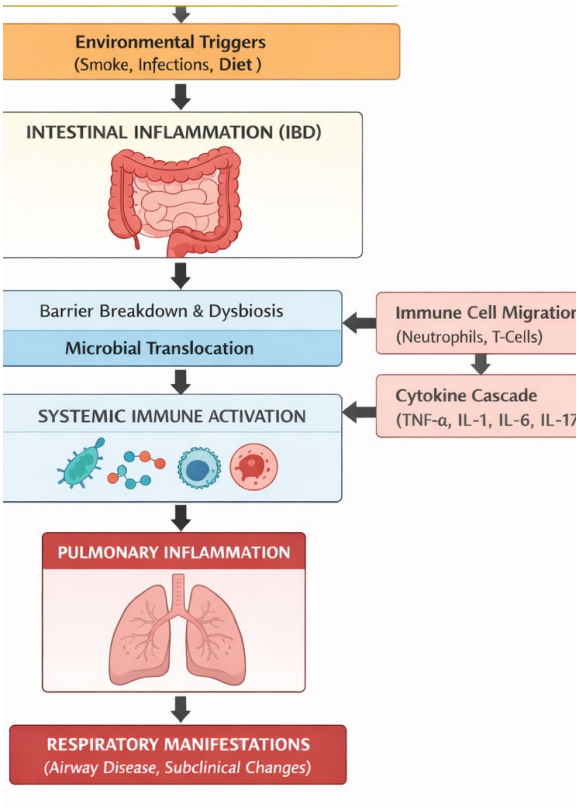

Figure 1. *Simplified model of gut-lung axis-mediated pulmonary involvement in pediatric IBD.*

Epidemiology

The reported prevalence of PMs in IBD varies considerably according to study design and diagnostic methodology. Clinically overt respiratory disease has traditionally been estimated to occur in fewer than 1% of patients. However, systematic screening using pulmonary function tests (PFTs) and high-resolution imaging has revealed subclinical pulmonary abnormalities in up to 40% of pediatric patients (4), indicating that respiratory involvement is substantially more common than previously recognized.

Importantly, prevalence estimates approaching 75% reported in some studies refer to the overall frequency of EIMs rather than to pulmonary disease specifically (14), and should therefore not be interpreted as reflecting respiratory involvement alone.

Children with pediatric-onset IBD appear to have a greater burden of EIMs than adults, possibly owing to longer cumulative disease duration, earlier immune dysregulation, and differences in disease phenotype (15). Although airway disease predominates in adults, particularly among patients with UC, available pediatric evidence suggests that CD is more frequently associated with interstitial pulmonary involvement (4, 15). Nevertheless, the rarity of pediatric cases and the predominance of retrospective studies continue to limit accurate estimates of disease prevalence and risk factors.

Spectrum of pulmonary manifestations and clinical presentation

Pulmonary involvement in pediatric IBD may affect virtually every anatomical component of the respiratory tract, including the upper airways, lower airways, lung parenchyma, pleura, and pulmonary vasculature (3, 5). Clinical presentation is highly heterogeneous, ranging from completely asymptomatic disease detected only through functional testing to severe, life-threatening respiratory failure (7).

The most common respiratory symptoms include chronic cough, dyspnea, wheezing, reduced exercise tolerance, sputum production, and, less frequently, chest pain or stridor (16). Importantly, respiratory manifestations often show little or no correlation with intestinal disease activity. Pulmonary involvement may precede the diagnosis of IBD, occur simultaneously with gastrointestinal symptoms, or develop years later despite complete intestinal

remission (7, 8). Consequently, clinicians should maintain a high index of suspicion whenever persistent respiratory symptoms occur in patients with established IBD. A substantial proportion of pediatric patients remain asymptomatic despite demonstrable abnormalities on pulmonary function testing or thoracic imaging (20). This silent clinical course contributes significantly to delayed diagnosis and probably explains why pulmonary involvement remains underrecognized in routine clinical practice. An additional diagnostic challenge is differentiating primary PMs of IBD from secondary pulmonary disorders related to immunosuppressive therapy, including opportunistic infections and drug-induced lung toxicity (16). This distinction has major therapeutic implications because inappropriate discontinuation of effective biologic therapy may adversely affect intestinal disease control.

Large-airway involvement includes chronic tracheobronchitis, chronic bronchitis, suppurative bronchitis, and glottic or subglottic stenosis (8). Bronchiectasis represents the most frequently reported structural airway abnormality, accounting for up to two-thirds of confirmed airway cases (21). Patients commonly present with productive cough, dyspnea, wheezing, excessive sputum production, or stridor. Because these symptoms frequently resemble asthma or recurrent respiratory infections, diagnosis may be delayed.

Persistent inflammation of the tracheobronchial tree may eventually result in irreversible structural remodeling, including fibrotic stenosis, airway webs, and fixed airflow obstruction. Early recognition is therefore essential to prevent permanent respiratory impairment.

Small-airway disease, including bronchiolitis obliterans and granulomatous bronchiolitis, usually follows a more insidious course. Patients often report only chronic dry cough or progressive exercise intolerance, while PFTs may reveal subtle abnormalities before overt symptoms become evident (8).

Parenchymal lung disease represents another important category of pulmonary involvement. Organizing pneumonia is the most frequently reported interstitial pattern, whereas nonspecific interstitial pneumonia, necrobiotic pulmonary nodules, eosinophilic pneumonia, pulmonary vasculitis, and diffuse alveolar damage occur less commonly (8, 22). Clinical presentation may include fever, progressive dyspnea, non-productive cough, pleuritic chest pain, hypoxemia, and, in severe cases, acute respiratory failure requiring hospitalization.

Less frequently, clinicians should also consider pleural disease, pulmonary thromboembolism, and the rare occurrence of enteropulmonary fistulas (19, 23). Although uncommon, these complications are poten-

Table 1. *Respiratory manifestations in pediatric inflammatory bowel disease.*

Respiratory site	Typical manifestations	Clinical presentation / symptoms	Subclinical findings	Notes / frequency
Large airways	Tracheobronchitis, chronic or suppurative bronchitis, glottic/ subglottic stenosis	Dyspnea, wheezing, productive cough, stridor, hoarseness	Airway narrowing detectable on imaging or bronchoscopy before symptoms	Bronchiectasis is common (up to 66% of cases); irreversible if untreated
Small airways	Bronchiolitis obliterans, granulomatous bronchiolitis	Non-specific dry cough, mild dyspnea, exercise intolerance	Reduced FEV1 or DLCO, subtle imaging changes	Often insidious; may remain asymptomatic for years
Lung parenchyma	Organizing pneumonia (OP), non-specific interstitial pneumonia (NSIP), necrobiotic nodules, pulmonary vasculitis	Fever, dyspnea, dry cough, chest pain, acute respiratory failure	Abnormal HRCT patterns, mild PFT changes	OP most common in children; other patterns rarer
Pleura	Pleural effusion	Chest pain, dyspnea	Often asymptomatic if small	Rare
Rare / severe complications	Thromboembolic events, enteric–pulmonary fistulas	Acute respiratory compromise, hypoxemia	N/A	Life-threatening; requires high clinical suspicion

DLCO: Diffusing capacity of the lung for carbon monoxide; FEV1: Forced expiratory volume in 1 second; HRCT: High-resolution computed tomography; NSIP: Non-specific interstitial pneumonia; OP: Organizing pneumonia; PFT: Pulmonary function tests.

tially life-threatening and require prompt recognition and multidisciplinary management.

A comprehensive overview of PMs associated with pediatric IBD is summarized in **Table 1**.

Management and prognosis

Management of PMs in pediatric IBD remains particularly challenging because of the absence of randomized controlled trials (RCTs). Consequently, current therapeutic strategies are largely extrapolated from adult studies or based on retrospective case series and expert opinion, resulting in a relatively low level of evidence (35). Corticosteroids remain the cornerstone of treatment and are effective in approximately two-thirds of reported cases (35). The choice between inhaled and systemic corticosteroids should be guided by the anatomical distribution and severity of pulmonary involvement (7). Inhaled corticosteroids are generally recommended for isolated airway disease, whereas systemic therapy is reserved for diffuse parenchymal or interstitial lung involvement (5). However, important pediatric-specific issues remain unresolved, particularly regarding the optimal duration of treatment and the minimum effective dosage required to prevent irreversible pulmonary damage while minimizing corticosteroid-related adverse effects in growing children. In patients with severe or refractory disease, biologic agents, particularly anti-TNF therapies such as infliximab, have expanded the available therapeutic options (5). Although infliximab has shown encouraging results in patients unresponsive to conventional treatment, evidence supporting its efficacy for PM remains limited to isolated pediatric case reports and small case series, as these agents are generally introduced to control intestinal disease rather than PMs (35, 36). A major therapeutic challenge arises from the dual role of anti-TNF agents: while they may successfully treat severe pulmonary inflammation, they have also been implicated in drug-induced ILD (37). Distinguishing between primary pulmonary involvement requiring treatment escalation and drug-related toxicity requiring treatment withdrawal therefore represents a critical step in clinical decision-making.

Macrolide antibiotics are frequently prescribed in patients with bronchiectasis complicated by chronic bacterial colonization (7). In addition to their antimicrobial activity, macrolides exert well-recognized immunomodulatory effects on neutrophilic airway inflammation, contributing to a reduction in exacerbation frequency and improvement in respiratory symptoms (38). Supportive interventions, including pulmonary rehabilitation and airway clearance techniques, should also be considered in symptomatic patients (39), although their long-term impact on disease progression has not yet been systematically evaluated in pediatric IBD.

One of the most debated aspects of management concerns the high prevalence of subclinical pulmonary involvement in children. Because respiratory abnormalities frequently remain asymptomatic, it is still unclear whether isolated functional alterations, such as a reduced diffusing capacity for carbon monoxide (DLCO), should prompt early pharmacological intervention to prevent irreversible pulmonary sequelae later in life (20, 24). This represents one of the major unresolved questions in pediatric clinical practice. Until stronger evidence becomes available, structured multidisciplinary follow-up combined with longitudinal assessment of pulmonary function should be regarded as an essential component of patient care to detect disease progression and preserve long-term respiratory health during the transition to adulthood (6, 40).

Future directions

Despite increasing recognition of PMs in pediatric IBD, substantial gaps remain in their early detection, pathophysiological understanding, and clinical management. Future research should prioritize the development of standardized screening strategies for children at increased risk, particularly those with persistent respiratory symptoms, severe intestinal inflammation, or prolonged disease duration.

The identification of reliable non-invasive biomarkers, including advanced pulmonary function parameters such as DLCO and impulse oscillometry (IOS), together with circulating inflammatory biomarkers, may facilitate the early detection of subclinical pulmonary involvement. Furthermore, additional prospective studies are required to better define the role of emerging imaging techniques, including lung magnetic resonance imaging (MRI) and low-dose HRCT, in longitudinal disease monitoring while minimizing cumulative radiation exposure.

A more comprehensive understanding of the immunological crosstalk between the gastrointestinal tract and the respiratory system may ultimately lead to novel therapeutic strategies. Targeted biologic therapies and pre-

cision medicine approaches directed at specific inflammatory pathways represent particularly promising areas for future investigation.

Finally, large prospective multicenter pediatric studies are urgently needed to clarify the natural history of PMs, identify predictors of disease progression, evaluate treatment efficacy, and define long-term respiratory outcomes in children with IBD.

CONCLUSIONS

PMs represent an uncommon but clinically significant extraintestinal complication of pediatric IBD and remain substantially underrecognized in routine clinical practice. As illustrated by the present case, respiratory involvement may evolve independently of intestinal disease activity and frequently presents with subtle or nonspecific symptoms, such as persistent cough, leading to delayed diagnosis.

From a clinical perspective, pediatricians should increasingly consider the lung as an immunologically integrated component of the gut-lung axis rather than an isolated organ. Persistent respiratory symptoms, even during periods of gastrointestinal remission, should prompt consideration of pulmonary involvement as an extraintestinal manifestation and warrant a structured diagnostic evaluation based on sensitive pulmonary function testing, particularly DLCO, complemented by targeted imaging and bronchoscopy whenever clinically indicated. Equally important is the careful differentiation between primary PMs and drug-induced pulmonary toxicity, as this distinction has major therapeutic implications and may prevent the unnecessary discontinuation of highly effective IBD therapies.

Ultimately, greater awareness among pediatric gastroenterologists, pulmonologists, and general pediatricians, together with a multidisciplinary approach and structured longitudinal follow-up, will be essential to optimize individualized patient management, facilitate early diagnosis, and reduce the risk of irreversible long-term pulmonary sequelae.

COMPLIANCE WITH ETHICAL STANDARDS

Conflicts of interest

The authors declare no conflicts of interest.

Financial support

None.

Author contributions

CC, IP: conceptualization, formal analysis, data curation, writing – original draft. AA, IP: methodology, supervision. AA, IP, ZM: validation, writing – review & editing. CC, IP, AA, ZM: investigation. IP, ZM: visualization. AA: project administration.

Ethical approval

Human studies and subjects

Specific ethical approval was not required for this case report in accordance with institutional guidelines.

Data sharing and data accessibility

N/A.

Publication ethics

Plagiarism

Authors declare no potentially overlapping publications with the content of this manuscript and all original studies are cited as appropriate.

Data falsification and fabrication

All data reported are accurate and authentic.

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