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CASE REPORT

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Asthma or not asthma? That is the question

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wet cough

Abstract
A 12-year-old boy with a family history of atopy and no relevant personal medical history was referred to the Pediatric Allergy Out-Clinic due to a persistent cough lasting 1 year, refractory to multiple therapeutic interventions. He presented with a daily productive cough, occasionally leading to vomiting, associated with exertional dyspnea during moderate physical activity. He denied any clear seasonality or identifiable triggers. He reported episodes of low-grade fever 2-3 days per month, dysphagia, and occasionally experienced a subjective sensation of food impaction. Blood tests performed at another center showed sensitization to house dust mites (*Dermatophagoides)*, olive pollen, pellitory (*Parietaria*), and animal dander. Physical examination was unremarkable. Spirometry performed in the Pediatric Allergy outpatient clinic revealed an obstructive ventilatory pattern with a negative bronchodilator response. High-resolution chest CT scan revealed multiple bilateral cylindrical bronchiectasis, predominantly in the upper lobes, as well as diffuse dilation of the thoracic esophagus up to 6 cm in diameter, extending to the esophagogastric junction, suggestive of stenosis at this level. The patient was urgently referred to the Pediatric Pulmonology and Pediatric Gastroenterology department. Further gastrointestinal studies—including an esophagogastroduodenal transit study, upper digestive endoscopy, and esophageal high-resolution manometry—confirmed a diagnosis of type II achalasia. Surgical intervention (Heller myotomy technique and Dor anterior fundoplication) was performed, and 30 months post-surgery, the patient remains asymptomatic, pulmonary function has normalized, and chest CT scan revealed no pleuroparenchymal abnormalities. Achalasia should be considered in the differential diagnosis of chronic productive cough unresponsive to treatment in children.

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Introduction

Cough remains a common reason for pediatric evaluation in both primary and specialty care. Recent studies estimate the prevalence of chronic cough (lasting more than 4 weeks) at 5–12% in the general pediatric population, highlighting the need for systematic evaluation.[1,2]

A correct anamnesis on when and how it started, characteristics, accompanying symptoms, predominance and triggers, as well as the response to treatment, guide us through the different etiologies (Table 1). Chronic cough, particularly when productive, necessitates ruling out serious respiratory diseases such as bronchiectasis, cystic fibrosis, or chronic aspiration syndrome.[1,2]

Dry cough can be a symptom of asthma, especially when accompanied by wheezing, atopy, or exertional dyspnea. Asthma is diagnosed with pulmonary function tests, which require cooperation or specialized hospital equipment. In such cases, a therapeutic trial with bronchodilators and anti-inflammatory drugs may help assess the clinical response. Although asthma is a leading cause of recurrent cough in children, lack of response to bronchodilator therapy should prompt reconsideration of the diagnosis.[3] The physical examination must be thorough and detailed, always including measurement of heart and respiratory rate, and oxygen saturation (SpO_2). It should assess the general condition and nutritional status, and include examination of the nose (e.g., turbinate hypertrophy, polyps, mouth breathing), ears (e.g., foreign bodies, serous otitis), and nasopharynx (e.g., tonsil size, mucus in the nasopharynx). The chest should be examined for shape (e.g., hyperinflated or barrel-shaped chest suggesting chronic lung disease) and auscultated for symmetrical and pathological cardiopulmonary sounds. The clinical history of persistent cough since the neonatal period should also consider neonatal causes such as aspiration or congenital malformations.[3] In every child with a persistent cough, the evaluation should always include a chest X-ray and baseline spirometry with a bronchodilator test. Based on the initial results, additional complementary tests may be considered, such as blood analysis, Mantoux test, allergy skin prick test, microbiological studies, and further imaging as needed.[3]

Case Report

This case involves a 12-year-old boy with a maternal family history of allergic asthma and no relevant personal history. He had a complete vaccination schedule and had no regular contact with domestic animals. He was referred to the Pediatric Allergy Out-Clinic due to a persistent productive cough lasting for 1 year, sometimes associated with vomiting and dyspnea with moderate exertion. No clear seasonality or identifiable triggers were reported. Episodes of low-grade fever 2–3 days per month and difficulty swallowing solid foods, occasionally with a sensation of impaction, were reported. He was evaluated by the Pediatric Gastroenterology and was awaiting further testing. Previous blood analysis showed sensitization to *Dermatophagoides*, olive pollen, *Parietaria judaica*, and animal epithelium. Physical examination was normal,and spirometry revealed an obstructive ventilatory pattern with no bronchodilator response (FVC 85% [−1.51z], FEV1 56% [−3.85 z] [PBD+ 0%], FEV1/FVC 56 [−3.68 z], FEF 25-75 32% [−3.72 z] (Figure 1). A high-resolution chest CT (HRCT) was performed, which revealed multiple bilateral cylindrical bronchiectasis in the upper lobes and esophageal dilation up to 6 cm in diameter, extending to the esophagogastric junction, compatible with stenosis at that level (Figures 2–4, HRCT). The patient was urgently referred to the Pediatric Pulmonology and Gastroenterology department.The digestive workup was completed with an esophagogastroduodenal contrast study (Figure 5), upper gastrointestinal endoscopy, and esophageal high-resolution manometry, which confirmed a diagnosis of type II achalasia. The patient was screened for related systemic pathologies assessed by the Pediatric Rheumatology team, and they were ruled out due to the lack of other suggestive symptoms. In addition, specific respiratory care measures for chronic aspiration syndrome and bronchiectasis were established, such as measures to prevent food aspiration by adjusting food texture and volume, residual emptying measures to prevent nocturnal aspiration (dinner rules, sleeping in the anti-Trendelenburg position), and respiratory physiotherapy. He was subsequently evaluated by the Pediatric Surgery team, who performed surgical treatment using the Heller myotomy

Table 1 The most common etiology of cough by age.

Below 1 year	1-6 years of age	Above 6 years
• Congenital anomalies:	• ORL infection	• Asthma
– Airway malformation	• Asthma	• SVARS
– Malacia	• Gastroesophageal reflux disease	• Bronchiectasis
– Tracheoesophageal fistula	• Foreign body aspiration	• Lung malformations
– Laryngotracheal cleft	• Infections	• Lung malformations
– Vascular ring	• Persistent bacterial bronchitis	• Tumors
• Infant asthma	• Lung malformations	• Smoking
• Infection: RSV, CMV, Chlamydia	• Cystic fibrosis	
• Swallowing disorders	• Immunodeficiencies	
• Cystic fibrosis	• Passive smoking	
• Passive smoking		

CMV: cytomegalovirus; ENT: otorhinolaryngological; RSV: respiratory syncytial virus; SARS: upper airway cough syndrome.
Adapted from the Aeped protocol Tos persistente. Protoc diagn ter pediatr. 2017;1:1–14.

INFORME DE FVC Maniobra N°: 1

		PRE				POST		
PARÁMETRO		M.	%REF	REF	LLN	M	(%P)	(%R)
Mejor FVC	**(l)**	**2.57**	**85**	**3.03**	**2.30**	**2.78**	**8**	**92**
Mejor FEV1	**(l)**	**1.44**	**56**	**2.59**	**1.97**	**1.44**	**0**	**56**
MFev1/MFvc	**(%)**	**56.03**	**66**	**84.65**	**67.72**	**51.75**	**-7**	**61**
FVC	(l)	2.57	85	3.03	2.30	2.78	8	92
FEV1	(l)	1.44	56	2.59	1.97	1.37	-4	53
FEV1/FVC	(%)	56.15	66	84.65	67.72	49.36	-12	58
PEF	(l/s)	2.16	37	5.77	4.00	2.62	19	45
FEF25%-75%	(l/s)	1.03	32	3.20	1.89	0.69	-39	22
GRADO CALIDAD					B			

Repetibilidad ATS/ERS (PRE): FVC: Si, FEV1: Si
Repetibilidad ATS/ERS (POST): FVC: Si, FEV1: Si
Comentarios:

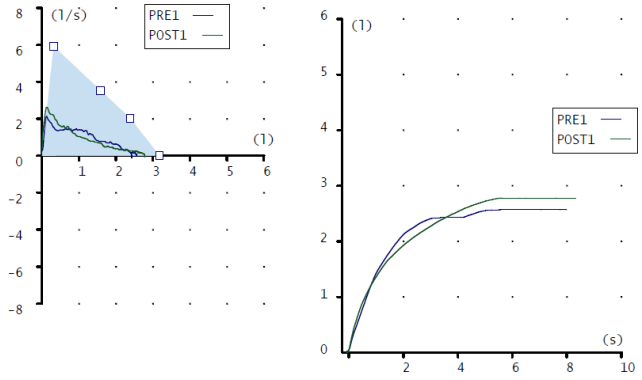

Figure 1 Spirometry before surgery.

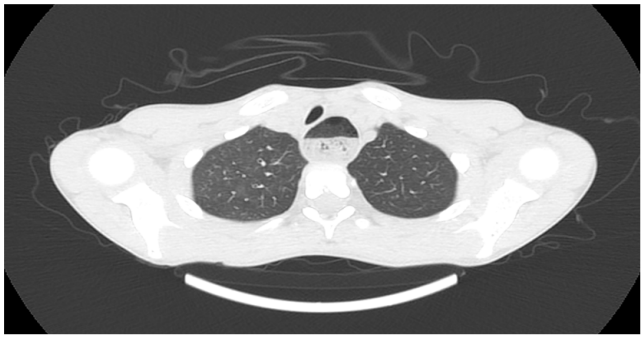

Figure 2 Axial HRCT image showing bilateral cylindrical bronchiectasis predominantly in the upper lobes.

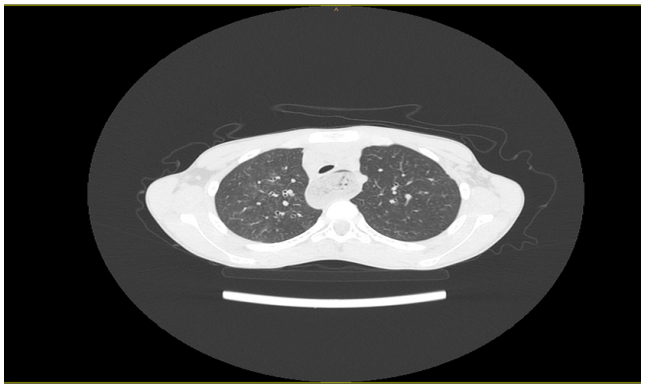

Figure 3 Coronal HRCT image revealing marked esophageal dilation measuring up to 6 cm in diameter.

technique and Dor anterior fundoplication to correct the disorder and prevent gastroesophageal reflux secondary to it. Thirty months post-surgery, the patient remains asymptomatic about both respiratory and digestive symptoms,

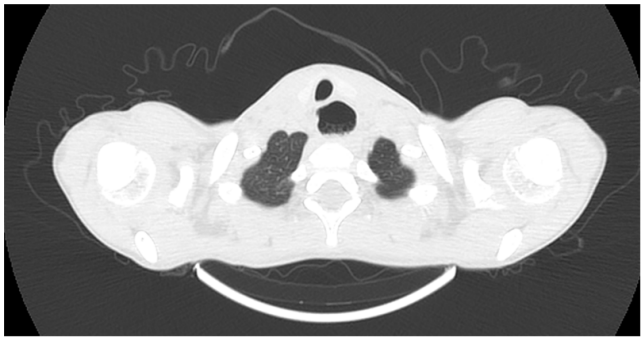

Figure 4 Sagittal HRCT view demonstrating esophageal dilation extending to the esophagogastric junction, consistent with distal stenosis.

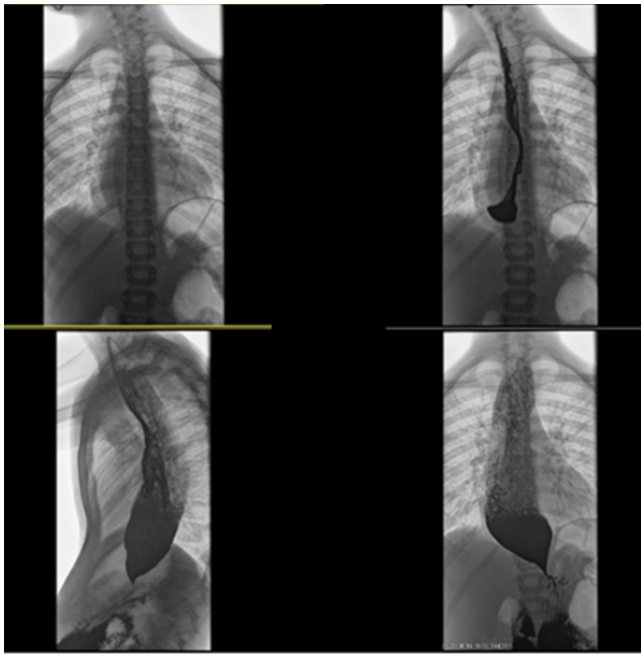

Figure 5. Esophagogastroduodenal contrast study revealing the classic “bird-beak” appearance of the distal esophagus, consistent with achalasia.

with normalized pulmonary function, and chest CT scan revealed no pleuroparenchymal abnormalities.

Discussion

Achalasia is a motor disorder of the esophagus, characterized by the failure of the lower esophageal sphincter (LES) to relax and the aperistalsis of the esophageal body. It has a low incidence in pediatrics (0.1 per 100,000), but should be suspected in the presence of progressive dysphagia, regurgitation, and atypical respiratory symptoms.[4,5]

Chronic productive cough in children that does not respond to standard treatment warrants a thorough etiological investigation to exclude potential differential diagnoses. While asthma typically presents with a persistent dry cough,

the presence of persistent dyspnea on minimal exertion combined with sensitization to environmental allergens in our patient raised the suspicion of a concurrent allergic condition. A thorough anamnesis, complemented by a comprehensive physical examination, is essential for guiding the differential diagnosis. In our patient's case, cough was productive, and spirometry revealed a moderate obstructive pattern with no response to bronchodilators, prompting us to request an HRCT to investigate possible structural lung disease.[6]

Other notable symptoms in the patient included dysphagia, regurgitation, and weight loss, which led to an early referral to a pediatric gastroenterologist. However, this evaluation was conducted without performing complementary diagnostic tests. Gastrointestinal evaluation includes esophagogastroduodenal contrast studies, upper gastrointestinal endoscopy, and esophageal high-resolution manometry.[7,8] Esophageal high-resolution manometry enables the classification of esophageal motility disorders according to the Chicago Classification v4.0, with type II achalasia being the most common subtype in children.[9]

Various gastrointestinal conditions, such as achalasia, fistulas, or diverticula, can cause chronic aspiration syndrome and present with nonspecific or asthma-like symptoms. In achalasia, retained food in a dilated esophagus can lead to microaspirations and secondary bronchiectasis, as in the present case.

The etiology of primary achalasia is unknown, although different mechanisms have been proposed (genetic, infectious, autoimmune, etc.). Currently, the most accepted theory is the destruction of esophageal neuronal plexuses by immunological mechanisms. Achalasia can occur in isolation or be associated with other systemic diseases such as amyloidosis, sarcoidosis, scleroderma, or Sjogren's Syndrome. Treatment options for achalasia include endoscopic and surgical approaches. In children, the standard procedure is extramucosal myotomy with partial Dor fundoplication. Emerging techniques such as peroral endoscopic myotomy (POEM) show promising results but require long-term validation in the pediatric population.[10,11]

A retrospective study published in 2017 reviewed 11 pediatric cases of achalasia. This study included children aged between 6 and 17 years and evaluated different therapeutic approaches: pneumatic dilations, laparoscopic Heller myotomy, and pharmacological treatment. Heller myotomy proved to be the most effective definitive treatment. The importance of long-term follow-up to detect recurrences was emphasized.[12]

In 2024, Asseri et al. reviewed three cases of pediatric patients with achalasia who presented with respiratory symptoms (chronic cough and recurrent pneumonia). All were diagnosed using contrast studies and were initially managed with pneumatic dilation, followed by laparoscopic myotomy with antireflux procedures.[13]

More recently, in 2024, several articles have been published regarding achalasia in the pediatric population. Among them is the case of a 3-month-old infant who presented with symptoms of recurrent pneumonia, feeding difficulties, and persistent regurgitation. Radiological and manometric evaluations confirmed the diagnosis of achalasia. Surgical treatment consisted of a modified Heller myotomy with fundoplication, with good clinical outcomes.[14]

In the same year, Gerçel et al. also published an article about a 2-month-old infant, one of the youngest reported cases, who presented symptoms from birth (dysphagia, regurgitation, failure to gain weight, and recurrent pneumonias). The patient did not respond to antireflux treatment and was ultimately diagnosed with achalasia. Management in this case included Heller myotomy with Dor fundoplication. The authors emphasized the importance of early diagnosis, as this condition can be confused with other neonatal pathologies such as gastroesophageal reflux.[15]

In 2024, a case of a girl diagnosed with Allgrove syndrome (a triad of achalasia, alacrima, and adrenal insufficiency) was reported, who underwent POEM after failed pneumatic dilations. This case highlights the feasibility of POEM as a minimally invasive alternative in pediatric patients.[16] In the same year, a clinical case was reported involving a 16-year-old adolescent who presented with progressive dysphagia and regurgitation. Pneumatic dilation was initially attempted without success, leading to the decision to perform a Heller myotomy with fundoplication, which resulted in an excellent clinical outcome. This case reinforces the importance of individualizing treatment based on the initial therapeutic response.[17]

Another similar case, described by Prado in 2025, reported a 16-year-old adolescent who had an insidious clinical course with predominant dysphagia, which delayed the diagnosis. An initial attempt at pneumatic dilation was unsuccessful, so a Heller myotomy with antireflux fundoplication was performed, with good postoperative outcomes.[17]

This case underscores the importance of a multidisciplinary approach in the evaluation of persistent respiratory symptoms and the need to integrate gastrointestinal findings into the diagnostic process for chronic cough when chronic aspiration syndrome is suspected, and not systematically. In esophageal achalasia, the motility disorder itself can lead to a chronic aspiration syndrome due to reflux of undigested contents, accounting for the respiratory symptoms. Thirty months after surgery, the patient remains asymptomatic with normalized lung function, and chest CT scan revealed no pleuroparenchymal abnormalities. Sensitization to aeroallergens was deemed clinically irrelevant. A detailed, targeted clinical history is essential in cases of persistent cough to help identify alternative diagnoses.

Conclusions

Achalasia should be considered in the differential diagnosis of chronic productive cough unresponsive to treatment in children. The integration of digestive and respiratory symptoms, along with imaging findings, can guide diagnosis. Multidisciplinary collaboration was essential in this case to achieve an accurate diagnosis and for successful treatment.

Mandatory Disclosure on Use of Artificial Intelligence

The authors declare that AI-assisted tools were used as follows: ChatGPT, free version was used to explore and

suggest publications related to achalasia. All references have been manually verified for accuracy and relevance.

Authors Contributions

All authors contributed equally.

Conflicts of Interest

The authors declare no potential conflicts of interest with respect to research, authorship, and/or publication of this article.

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