

Tracheobronchopathia Osteochondroplastica: An Underrecognized Benign Airway Disorder - Pathogenesis, Clinical Spectrum, and Advances in Management

Guillermo Andrés Moreno Cortes¹, Sonia Maya-Fajardo², Katherine Sthefania Trejos Guzman³, Maria Luisa Earls⁴, Rishita Dave⁵, Philip Obiri Ankomah⁶, Perla Carolina García-Hernandez⁷, Frederick Aniagyei Bonsafo⁸ and Maria Isabel Gomez-Coral^{9*}

¹Universidad del Rosario, Escuela de Medicina y Ciencias de la Salud, Bogotá, Colombia

²Universidad del Rosario, Escuela de Medicina y Ciencias de la Salud, Bogotá, Colombia

³Universidad Nacional Autónoma de Nicaragua UNAN Managua

⁴Latin American School of Medicine (ELAM), Cuba

⁵University of Medicine and Health Sciences, Saint Kitts

⁶Takoradi Technical University Medical Center, Ghana

⁷Universidad Nacional Autónoma de Nicaragua, León

⁸Ivano Frankivsk Medical University, Ukraine

⁹Universidad del Valle, México

Submission: October 03, 2025; **Published:** October 22, 2025

***Corresponding author:** Maria Isabel Gomez-Coral, Universidad del Valle, México

Abstract

Introduction: Tracheobronchopathia osteochondroplastica (TPO) is a rare, benign, and underdiagnosed central airway disorder characterized by cartilaginous and/or osseous nodules in the submucosa. Its nonspecific clinical presentation, overlapping with common respiratory conditions like asthma and chronic bronchitis, often leads to diagnostic delays and mismanagement. This review provides an updated overview of TPO's historical background, clinical features, diagnostic challenges, and management strategies.

Methods: A comprehensive review of the literature was conducted, encompassing historical case reports, clinical studies, and recent advances in diagnostic and therapeutic approaches.

Results: TPO is more prevalent in middle-aged to older males and is commonly discovered incidentally. The exact etiology is unknown, but chronic inflammation and genetic predisposition are suspected. Symptomatic patients often present with chronic cough, dyspnea, and recurrent infections. Diagnosis relies on characteristic CT findings of calcified nodules sparing the posterior tracheal wall and a cobblestone appearance on bronchoscopy, with histopathology confirming the presence of osseous and cartilaginous metaplasia. Management is primarily conservative, with interventional bronchoscopic debulking, laser therapy, or stenting reserved for severe symptomatic cases. The prognosis is generally favorable, but some patients may experience progressive airway obstruction affecting their quality of life.

Conclusion: TPO is a critical differential diagnosis for chronic respiratory symptoms. Increased awareness of its distinctive imaging and endoscopic features is essential for accurate diagnosis, preventing unnecessary interventions for suspected malignancy, and ensuring appropriate long-term management.

Keywords: Tracheobronchopathia osteochondroplastica; Airway disorder; Bronchoscopy; Inflammation; Asymptomatic

Abbreviations: TPO: Tracheobronchopathia Osteochondroplastica; CT: Computed Tomography

Introduction

Tracheobronchopathia osteochondroplastica (TPO) is a rare, benign disorder of the central airways characterized by the presence of multiple cartilaginous and/or osseous submucosal nodules projecting into the tracheobronchial lumen [1]. These nodules typically spare the posterior membranous wall of the

trachea, a key feature that helps distinguish TPO from other airway diseases. The exact etiology remains unclear, although chronic inflammation, mechanical irritation, and genetic predisposition have been proposed as contributing factors [1-6]. Most cases are diagnosed incidentally during bronchoscopy or imaging studies, as patients may remain asymptomatic or present with nonspecific

respiratory symptoms such as chronic cough, wheezing, recurrent infections, or dyspnea [1].

TPO was first described in the 19th century by Rokitsansky in 1855, and later more precisely characterized by Wilks in 1857 and by Luschka in 1861. Since then, sporadic case reports and small case series have gradually contributed to the understanding of this rare entity. Despite its benign nature, the condition has remained relatively obscure in clinical practice due to its low prevalence and nonspecific clinical manifestations [1-3]. TPO is considered underdiagnosed, largely because its symptoms overlap with those of more common airway disorders such as asthma, chronic bronchitis, or tracheal stenosis [4]. Moreover, its characteristic endobronchial nodules may be mistaken for malignancy or other infiltrative tracheal diseases during imaging or bronchoscopy. This under recognition can lead to diagnostic delays, mismanagement, and unnecessary procedures. This review aims to provide an updated overview of tracheobronchopathia osteochondroplastica, highlighting its historical background, clinical features, diagnostic challenges, and management strategies, with special emphasis on differentiating it from other tracheal pathologies.

Epidemiology and Demographics

Tracheobronchopathia osteochondroplastica (TPO), a benign condition involving bone and cartilage formation in the airways, is exceedingly rare [7]. The first documented case was identified by Wilks in 1857 [5]. To date, approximately 500 cases have been reported worldwide [8]; however, the true incidence is likely underestimated. This underdiagnosis is primarily due to the disease's nonspecific clinical presentation and ambiguous symptoms, leading many cases to be discovered incidentally during autopsies [8]. In Japanese literature, around 140 cases have been documented since the first report by Kidokoro in 1938, and 80 cases have been reported in Chinese literature [8]. The condition is most commonly observed in patients between the fifth and seventh decades of life [9]. TPO also exhibits a higher prevalence and/or severity in males compared to females [10].

Etiology and Pathogenesis

The exact cause and mechanism of Tracheobronchopathia Osteochondroplastica (TPO) are still not fully understood, but several hypotheses have been proposed regarding its development. One leading theory suggests that chronic airway inflammation plays a central role in its pathogenesis [11]. This persistent inflammation, which may be a response to environmental factors like smoking or long-term infections, is thought to trigger a metaplastic process in the submucosa. Another hypothesis involves an abnormality in calcium and phosphate metabolism, which could lead to the deposition of these minerals in the cartilaginous rings of the trachea and bronchi [12]. There is also speculation that a genetic predisposition may be involved, as some cases have been reported in families, suggesting an inherited component to the disease [3].

Histopathologically, TPO is characterized by the presence of numerous cartilaginous and osseous nodules located within the submucosa of the trachea and main bronchi [10]. These nodules are typically firm and can vary in size. A key diagnostic feature is that these calcified and bony deposits are found only in the cartilaginous rings, sparing the posterior membranous tracheal wall [3,11-12]. The nodules may project into the lumen of the airway, leading to the clinical manifestations. The presence of mature bone with a fatty marrow is a hallmark of the advanced stage of the disease, confirming the osseous metaplasia [11].

Clinical Manifestations

Tracheobronchopathia osteochondroplastica (TPO) often presents as a benign and asymptomatic condition, with many cases being discovered incidentally during bronchoscopy or autopsy for other reasons [11-17]. The severity of the disease, as observed on imaging or during an endoscopic exam, may not correlate with the presence or absence of symptoms [12]. However, when the condition does become symptomatic, the clinical presentation is often nonspecific, which can lead to misdiagnosis [11,14,17]. The insidious and slow-progressing nature of the disease also contributes to it being underrecognized by clinicians [11,14].

In symptomatic patients, the clinical picture is typically characterized by respiratory complaints. A chronic cough is the most common symptom, but other frequent manifestations include dyspnea, or shortness of breath, and wheezing [11,15-17]. Hemoptysis, the coughing up of blood, can occur due to ulceration of the nodules or an acute infection [11,15,16]. Recurrent respiratory infections are also a common issue, as the nodular lesions alter the mucosal surface and reduce mucociliary clearance, making the airways more susceptible to infection [15,16]. In more advanced stages of the disease, the nodules can grow and become confluent, leading to significant airway obstruction and stiffness [12,14,15]. These complications can result in severe dyspnea and, in very rare cases, acute respiratory failure [11]. The proliferation of the nodules and subsequent airway narrowing can also create difficulty with intubation [13].

Diagnostic Approaches

The diagnosis of TPO is primarily based on a combination of imaging and endoscopic findings, with histopathology providing definitive confirmation. While chest X-rays are generally not sensitive enough to detect the characteristic nodules, a computed tomography (CT) scan of the chest is a crucial diagnostic tool [13,17]. CT scans typically reveal multiple, small, calcified or ossified submucosal nodules that project into the lumen of the trachea and main bronchi [4,13,14]. A key finding on CT is the sparing of the posterior membranous wall of the trachea, which helps distinguish TPO from other conditions [12,13,17]. Bronchoscopy is considered the gold standard for the diagnosis of TPO [12,13,17]. The endoscopic appearance is highly characteristic, showing a cobblestone or beaded-like pattern of

hard, pale, or whitish submucosal nodules that are often firm to the touch [4,13]. These nodules can be sessile or have a broad base and are generally located along the cartilaginous rings of the trachea and proximal bronchi, again, sparing the posterior wall. A biopsy is usually performed during bronchoscopy to confirm the diagnosis and rule out other diseases.

Histopathology provides the definitive diagnosis by confirming the presence of cartilaginous and osseous tissue in the submucosa [13,14,17]. The microscopic examination reveals metaplastic changes with the formation of mature bone, sometimes with fatty marrow, within the submucosal layers [4,13]. The integrity of the overlying respiratory epithelium is usually preserved, though it may be thinned or ulcerated in areas of severe nodule formation. Given its rarity and the nonspecific nature of its symptoms, TPO must be differentiated from several other airway diseases. The differential diagnoses include tracheobronchial amyloidosis, which involves amorphous protein deposits that can also narrow the airway but typically affect the posterior wall [14,17]. Relapsing polychondritis is an inflammatory condition that can cause tracheal stenosis, but it is characterized by cartilage destruction and is not associated with calcified nodules [12]. Other conditions to consider are papillomatosis and primary or metastatic malignancy, which can be distinguished from TPO by their distinct histopathological features and location [13].

Classification and Severity Assessment

TBO is classified into three stages based on bronchoscopic and histopathologic features. Stage I (mild) demonstrates scattered plaque-like nodules on the mucosal surface with inflammatory cells and occasional cartilaginous elements, typically causing minimal lumen narrowing. Stage II (moderate) presents with diffuse cartilaginous nodules and sessile spicules protruding into the lumen, often described as cobblestones or stalactitic caves, resulting in partial airway narrowing. Stage III (severe) involves a deformed and rigid tracheal wall causing significant lumen narrowing, with protrusions containing lamellated bone and fatty marrow with hematopoiesis [18].

An additional classification, proposed by Dutau et al., emphasizes the extent of endoscopic lesions, defining Stage A as few scattered nodules with large areas of normal mucosa, Stage B as multiple nodules affecting the entire mucosa, and Stage C as confluent lesions causing substantial mechanical obstruction [19]. Integrating morphological and extent-based classifications provides a comprehensive framework for assessing TBO, combining structural changes with functional implications for airway patency.

Clinically, TBO may present as asymptomatic or symptomatic. Asymptomatic cases are often detected incidentally, ranging from mild nodularity to severe airway narrowing, and are usually managed conservatively with observation. Symptomatic TBO results from airway obstruction and inflammation, manifesting

as chronic cough, wheezing, exertional dyspnea, recurrent respiratory infections, or hemoptysis [20]. Notably, the degree of endoscopic severity and airway narrowing does not always predict symptom presence. Management of symptomatic cases focuses on symptom relief and complication prevention, with severe instances potentially requiring interventions such as laser ablation, mechanical debulking, or stenting [21]. This variability underscores the importance of integrating endoscopic findings, lumen narrowing, and symptomatology when assessing and managing TBO.

Management and Treatment

The management of tracheobronchopathia osteochondroplastica (TPO) is primarily conservative, focusing on symptomatic relief as the disease is benign and its progression is often slow [18,19]. Asymptomatic patients do not require specific treatment, but regular follow-up is recommended to monitor for any changes. For symptomatic patients, the goal is to alleviate respiratory complaints. This often involves the use of medications such as bronchodilators and mucolytics to help with symptoms like dyspnea and chronic cough, respectively [19]. The treatment of recurrent respiratory infections is also a key component of management, often requiring appropriate antibiotic therapy.

Interventional approaches are considered for patients with severe symptoms or significant airway narrowing. Rigid bronchoscopy is a common therapeutic modality that allows for the mechanical debulking of large or obstructive nodules [19,21]. This procedure can restore airway patency and significantly improve a patient's breathing. Other endoscopic techniques include laser therapy or cryotherapy, which can be used to ablate or reduce the size of the nodules [18,21]. For cases with extensive and severe airway stenosis that cannot be managed with debulking, the placement of an airway stent may be necessary to maintain a clear lumen and prevent collapse [18,19]. These interventional procedures are typically performed by experienced interventional pulmonologists.

Surgical management is rarely required due to the diffuse nature of the disease and the high risk of complications [19]. It is reserved for very select, localized cases of severe stenosis, but this is an extremely rare occurrence. Anesthetic considerations are critical for patients with TPO undergoing any procedure that requires intubation [7,18]. The hardened and irregular airway caused by the calcified nodules can make intubation challenging or even impossible with a standard endotracheal tube [7]. Anesthesiologists must be aware of the condition and may need to use smaller-sized tubes, fiberoptic bronchoscopy for guidance, or a laryngeal mask airway to secure the airway and prevent trauma to the tracheobronchial mucosa [7,18].

Prognosis and Long-Term Outcomes

The prognosis for patients with TPO is generally favorable, as the disease is considered benign and slowly progressive [18,20].

Many patients remain asymptomatic throughout their lives, with the condition having minimal impact on their daily activities [15,18]. For these individuals, the disease may be an incidental finding during a medical examination for an unrelated reason. The course of the disease is highly variable; some patients experience a stable condition without any significant changes in symptoms or airway caliber over many years, while others may develop a more aggressive course with progressive growth of nodules that can lead to significant airway narrowing and increasing respiratory distress [2,20].

Long-term follow-up is crucial for all patients, whether they are symptomatic or asymptomatic [15,19]. Regular monitoring with imaging and bronchoscopy can help track the progression of the nodules and determine if interventional management is necessary. While the disease itself does not typically affect life expectancy, its long-term impact on the quality of life can be substantial in symptomatic individuals [18]. Patients with severe airway obstruction may experience chronic dyspnea, recurrent infections, and other complications that require ongoing medical attention and can limit physical activity. For these individuals, the goal of management is to maintain airway patency and minimize symptoms to preserve a good quality of life [2,15,18-20].

Conclusion

Tracheobronchopathia osteochondroplastica is a rare and often underrecognized benign airway disorder that presents a unique diagnostic and management challenge for clinicians. Although its pathogenesis remains unclear, chronic inflammation and genetic factors are hypothesized to play a role. The condition is often asymptomatic, but when symptoms do arise, they are nonspecific and can mimic more common respiratory diseases, leading to diagnostic delays. The diagnostic cornerstone is the characteristic finding of calcified submucosal nodules on CT and a classic cobblestone appearance on bronchoscopy, with histopathology providing definitive confirmation. Management is largely conservative for asymptomatic individuals, while symptomatic patients may benefit from bronchodilators, mucolytics, and targeted interventional procedures like bronchoscopic debulking or stent placement in severe cases. Given its variable clinical course and potential for significant airway compromise, TPO underscores the need for increased awareness among healthcare professionals to ensure timely diagnosis and appropriate long-term follow-up to optimize patient outcomes and quality of life.

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DOI: [10.19080/IJOPRS.2025.07.555730](https://doi.org/10.19080/IJOPRS.2025.07.555730)

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