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Special Article

Tracheobronchomalacia

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ABSTRACT

Tracheobronchomalacia is a central airway disease characterised by weakness of the wall and dynamic decrease in the tracheal lumen and the large bronchi, particularly while exhaling. It is more common in middle age and the elderly with previous exposure to cigarettes. It causes chronic symptoms such as cough, dyspnea, increase in recurrent infections, and poor secretion management, but it can also progress to chronic respiratory failure and death. It is usually confused with other common diseases like chronic obstructive pulmonary disease (COPD) or asthma. Its causes can be congenital or acquired and its diagnosis involves the dynamic assessment of the airway with tomography and fibrobronchoscopy. It is classified as mild, moderate or severe depending on the degree of collapse of the airway when exhaling. Management consists of a primary phase, in which concomitant diseases must be controlled, such as COPD, asthma or gastro-oesophageal reflux. In diffuse moderate to severe symptomatic tracheobronchomalacia tracheobronchoplasty must be considered with strengthening of the posterior wall. Silicone and "Y" stents can be used to identify patients who could potentially benefit from surgical treatment as well as being used for the definitive symptomatic treatment with high surgical risk. More prospective studies need to be done in order to standardise certain common criteria for the management of this usually under-diagnosed disease.

Traqueobroncomalacia

Palabras clave:
Broncoscopio
Traquebroncomalacia
Stents
Traquebroncoplastia
Traqueomalacia
Colapso dinámico de la vía aérea

RESUMEN

La traqueobroncomalacia es una enfermedad de la vía aérea central que se caracteriza por debilidad de la pared y disminución dinámica de la luz de la tráquea y los grandes bronquios, especialmente durante la espiración. Es más frecuente en individuos de edad media y ancianos con exposición previa a cigarrillo. Genera síntomas crónicos, como tos, disnea, aumento de las infecciones recurrentes y mal tratamiento de las secreciones, pero también puede evolucionar hasta falla respiratoria crónica y muerte. Usualmente se confunde con otras enfermedades comunes, como enfermedad pulmonar obstructiva crónica (EPOC) o asma. Sus causas pueden ser congénitas o adquiridas y su diagnóstico involucra la evaluación dinámica de la vía aérea con tomografía y fibrobroncoscopia, y se clasifica en leve, moderada o grave, según el grado de colapso espiratorio de la vía aérea. El tratamiento contempla una primera fase, en la que se deben controlar las enfermedades concomitantes, como EPOC, asma o reflujo gastroesofágico. En la traqueobroncomalacia sintomática difusa moderada a grave se debe considerar la traqueobroncoplastia con reforzamiento de la pared posterior. Se pueden utilizar *stents* de silicona en Y para la identificación de pacientes que potencialmente se beneficiarían del tratamiento quirúrgico como también se pueden utilizar para tratamiento sintomático definitivo en pacientes con riesgo quirúrgico alto. Se necesita un mayor número de estudios prospectivos para poder unificar criterios comunes para el tratamiento de esta enfermedad, usualmente subdiagnosticada.

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Introduction

The word malacia comes from the Greek word "malakia", which in medical terms means softening of the bone or cartilage component of a structure of an organism. Tracheobronchomalacia is a disease of the central airway in which weakness develops in the tracheal walls and the bronchi due to softening or destruction of the supporting cartilage. This may or may not be accompanied by degeneration and atrophy of the elastic fibres of the posterior wall which recoil tightly, especially during expiration,[1] and cause a reduction of at least 50% in the transverse area of the tracheal lumen. The cartilage/soft tissue ratio in a normal trachea is about 4.5:1, while in patients with tracheobronchomalacia it can be as low as 2:1. Histopathological studies have found inflammatory infiltrates, T lymphocytes, and class ii human leukocyte antigens expressed in the macrophages.[2] The immunohistochemistry of the damaged cartilage depends on the severity of the damage. In severe malacia the cartilage disappears and it is replaced by collagen. In patients with lesions of the trachea secondary to prolonged mechanical ventilation (MV), a fibrous tissue replaces the cartilage. Furthermore, a polychondritic type of deterioration is described, which is a type of cartilaginous tracheobronchomalacia, but with an excessive inflammatory component, in which there are empty spaces and mixed inflammatory infiltrates in the cartilage.[2]

The prevalence of adult tracheobronchomalacia is unknown because data comes from studies performed on selected populations and not the general population. According to studies by Nuutinen,[3] carried out in the 1970s, it is more common in middle-aged men and the elderly with a history of exposure to cigarette smoke. In a Japanese study involving 4,283 patients who underwent bronchoscopy to assess respiratory diseases, tracheobronchomalacia was described in 12.7% of the cases.[4] In another study, it was diagnosed in 44% of cases on whom bronchoscopy was performed when a patient population with a diagnosis of chronic bronchitis was assessed.[5]

In tracheomalacia, there is dynamic obstruction of the airway and so there may be hyperinflation and air trapping. When expiring normally, the increase in intrathoracic pressure leads to the narrowing of the airway, which is counteracted by the rigidity of the tracheobronchial tree, the intraluminal pressure and the supporting structures.[1] In patients with airway malacia the progressive increase in intrathoracic pressure exceeds the intratracheal pressure and leads to reduced air flow, which brings about symptoms such as dyspnoea, difficulty in expectorating secretions, recurrent infections and, on occasions, chronic respiratory failure.[1]

It is an underdiagnosed disease, in which there are asymptomatic patients and others whose symptoms are often confused with those of other respiratory diseases such as asthma and chronic obstructive pulmonary disorder (COPD) (table 1).[1] Despite being a benign disease, it is important to bear in mind that it can cause progressive morbidity and, on occasions, respiratory failure and death.[1]

At present there is controversy with regards to the difference between normal expiratory airway collapse, excessive dynamic airway collapse (EDAC) and tracheobronchomalacia;[2] some authors call tracheobronchomalacia the weakening of the airway cartilage and refer to EDAC when the posterior wall becomes weak and redundant. However, for the time being, from a practical point of view, the focus of the treatment of tracheobronchomalacia and EDAC is the same when the patient has severe clinical symptoms, and in fact in some patients present a combination of the two.[6]

Table 1
Tracheobronchomalacia symptoms[1]

Frequent symptoms	• Dyspnoea • Orthopnoea • Chronic cough • Difficulty expectorating • Barking cough
Occasional symptoms	• Cough syncope • Stridor • Haemoptysis • Wheezing • Difficulty discontinuing MV

MV: mechanical ventilation.

Classification

The disease can be localised or diffuse, damaging only the trachea (tracheomalacia), the bronchi (bronchomalacia) or the trachea and the large bronchi (tracheobronchomalacia).[1] Several classifications have been proposed for adult tracheobronchomalacia. From morphological findings, tracheas can be identified with coronal narrowing of the lateral walls (known as sabre-sheath trachea), with

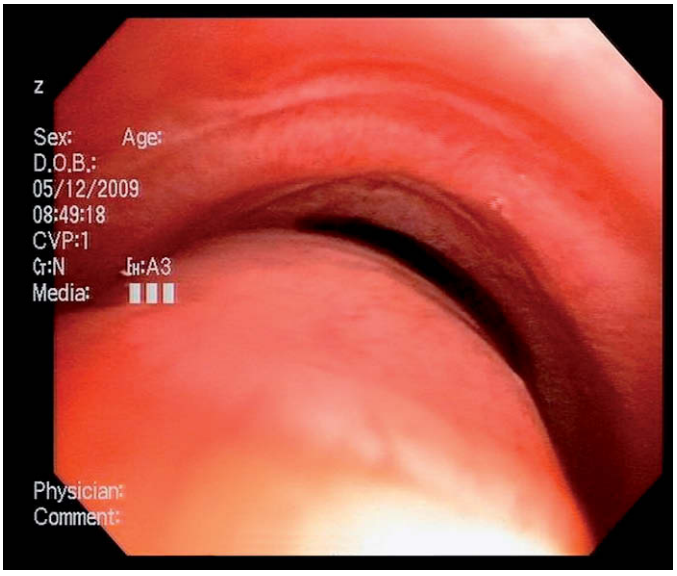

Figure 1. Crescent.

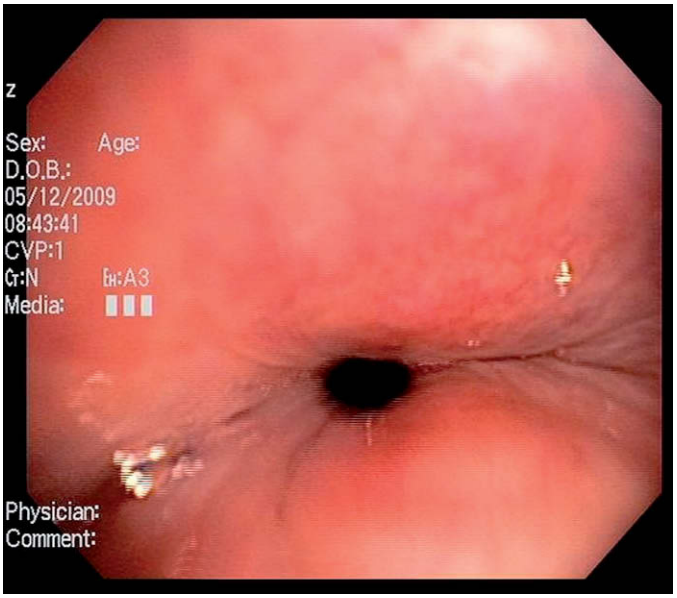

Figure 2. Concentric.

Table 2
FEMOS classification of tracheobronchomalacia by Murgu and Colt[2] *(functional class, aetiology, morphology, origin, severity)*

Functional class	NYHA		I II III IV
Underlying cause	Primary		Genetic idiopathic
	Secondary (acquired)	Post-traumatic	• Post intubation • Post-tracheotomy • Chest trauma • Post lung transplant
		COPD	• Recurrent Infection • Chronic bronchitis
		Chronic inflammation	• Relapsing polychondritis
		Extrinsic compression	• Benign tumours • Malignant tumours • Cysts • Abscesses • Aortic aneurysms • Thyroids
Severity (expiratory collapse)	Mild Moderate Severe	• Between 50 and 75% • Between 76 and 90% • Between 91 and 100%	
Morphology	Sabre-sheath trachea Crescent shape Circumferential		
Location	Tracheomalacia Bronchomalacia and tracheobronchomalacia		

COPD: chronic obstructive pulmonary disorder; NYHA: New York Heart Association.

Table 3
Classification of adult tracheobronchomalacia, by Carden et al[1]

Primary (congenital)	Genetic	
	Idiopathic	Giant trachea or Mounier-Kuhn syndrome
Secondary (acquired)	Post-traumatic	• Post intubation • Post-tracheotomy • Chest trauma: • Post lung transplant
	Emphysema/COPD	
	Chronic infection/bronchitis	
	Chronic inflammation	• Relapsing polychondritis
	Extrinsic compression of the trachea	• Malignancy • Benign tumours • Cysts • Abscesses • Aortic aneurysms
	Vascular rings	

COPD: chronic obstructive pulmonary disease.

anteroposterior narrowing producing a crescent shape, or with a lateral and anteroposterior reduction of the concentric lumen (figs. 1 and 2). This depends on if it is a congenital or acquired disease or whether it is a mild, moderate or severe case.[1]

Murgu and Colt[2] propose a classification called FEMOS *(functional class, etiology, morphology, origin, severity)* (table 2).[2] This system has the disadvantage of not reflecting accurately the clinical course and the prognosis. Carden et al proposed another similar classification which is often used in studies and reviews (table 3).[1]

Aetiology and Pathogenesis

Primary or congenital tracheobronchomalacia: of particular importance in adults is tracheobronchomegaly or Mounier-Kuhn syndrome, which is one of the few trachea abnormalities which causes diffuse dilatation of the airway. The underlying pathogenesis is related to congenital defects or atrophy of the muscle and elastic tissue of the trachea and central bronchi. It is common in middle-aged men, with 75% of diagnoses made in patients over 28 years old. The patients are often asymptomatic, but when there are symptoms they have chronic coughing, excessive sputum production, dyspnoea and haemoptysis, difficulty dealing with secretions with recurrent infections, bronchiectasis and, on occasions, pulmonary fibrosis.[1]

Images show that the trachea and central bronchi are markedly enlarged, with the diameter of the trachea over 3cm, the right bronchus over 2.4cm and the left bronchus over 2.3cm. The weakness of the airway wall between the rings leads to folds or sacculi, which gives it a corrugated appearance with the formation of diverticula and, of course, expiratory airway collapse.[1]

In 1973, Himal Stein et al classified the disease into 3 types: type 1 (relatively subtle, symmetric, diffuse enlargement of the tracheobronchial tree), type 2 (more obvious enlargement with diverticula and bizarre eccentric configurations) and type 3 (diverticula and sacculi in the trachea extending to the bronchial tree).[7]

Post-traumatic Tracheobronchomalacia

Endotracheal tubes and tracheotomy: the use of endotracheal tubes and tracheotomy can produce tracheal stenosis. Recurrent intubation and the duration of intubation can predispose patients to developing tracheobronchomalacia, and this is the most common cause of acquired focal tracheomalacia in adults.[1] Its pathogenesis is connected with the pressure exerted by the cuff of the endotracheal tube which affects blood flow and leads to the destruction and loss of the supporting cartilage with a weakening of the tracheal wall; it is usually a segmental malacia.[8]

Chronic MV: this is described as a cause of malacia in children when positive pressure ventilation is used for only a few weeks or months, for example in patients with muscular atrophy or dystrophy who have undergone many years of positive pressure ventilation via tracheotomy. This occurs despite using cuffless tubes and it is not known if the myopathic impairment of the muscularis mucosa also contributes to the dilatation and weakness of the wall. To this day it is not known what critical pressure in the airway or duration of ventilator use produces the lesions. Secondary tracheobronchomalacia due to chronic, non-invasive, positive pressure MV has not been described, which is important because usually continuous positive airway pressure (CPAP) functions as a pneumatic stent.[8]

Blunt chest trauma: after blunt chest trauma there may be lesions due to unidentified fractures of the trachea, such as those caused by seat belts.[1] The outcome of these lesions may be stenosis or malacia.

Postpneumonectomy or lobectomy: tracheomalacia associated with pneumonectomies or lobectomies, especially of the superior lobes, is usually detected in the segment above the site of anastomosis.[8] Lung transplant patients are susceptible to suffering alterations at the suture site and also distal to the anastomosis, with the possibility of associated malacia. This is basically due to the ischaemia which the bronchial mucosa suffers in the weeks after the transplant, since anastomosis of the bronchial artery is not performed in the usual way.

Chronic inflammation: relapsing polychondritis is an uncommon autoimmune disease which is characterised by recurrent episodes of chondritis and inflammation of the connective tissue of the auricle, nose and tracheobronchial tree. This disease most frequently affects middle-aged women (50% have an impairment of the airway and glottis). Characteristically, destruction and fibrosis of the tracheal and bronchial cartilage is observed with preservation of the normal posterior wall, and infectious respiratory complications are the main cause of death in these patients.[9]

Extrinsic compression: extrinsic tumours can invade, destroy and weaken the airway walls and cause tracheobronchomalacia. Amongst non-malignant entities, it is worth noting the disease's association with intrathoracic goitre and aortic aneurysms.[1]

Associated with COPD: some authors consider tracheobronchomalacia to be an extension of the peripheral obstruction of the airway. A substantial proportion of patients with severe emphysema have some degree of malacia of the central airways. The weakening of the walls could be related to chronic lesions secondary to exposure to cigarette smoke, or it may be connected with hypermobility of the airways, which is usual in emphysema.[8]

In 1958, in a study of patients with emphysema, Herzog demonstrated exaggerated invagination of the posterior walls of the stem bronchi, which caused expiratory occlusion of the superior lobes, and he suggested that this phenomenon could be related to the formation of bullae.[1]

In 1972, COPD was found in 24 of 35 patients with tracheobronchomalacia and it was shown that tracheobronchomalacia accelerated the progression of emphysema and chronic bronchitis.[10] Later studies described the association between tracheobronchomalacia and COPD in 57% of the patients under study. However, not all collapses of the airway are tracheobronchomalacia and it is important to differentiate it from normal dynamic collapses of the airway.[8]

Loring et al tried to establish what the relationship is between central collapses of the airway and their contribution to the flow limitations observed in patients with COPD and asthma.[11] In 80 patients with suspected or proven tracheobronchomalacia, 40% were found to have COPD and 24% asthma, and no correlation was found between the degree of obstruction, expressed as the forced expiratory volume in the first second (FEV_1), and collapse of the central airway. Other similar studies have also identified that tracheal collapse only makes a modest contribution to the total resistance of the airway.[11] Recent studies show that patients with COPD can benefit more from therapy aimed at treating severe tracheobronchomalacia when it is concomitant.[8]

Diagnosis

Patients with tracheobronchomalacia have non-specific symptoms or symptoms which overlap with those of more prevalent diseases, such as COPD and asthma.[1] Therefore, its diagnosis may go unnoticed.[1]

On the one hand, the usual assessment with chest x-rays does not detect it, as it is a dynamic process demanding more elaborate imaging with special dynamic manoeuvring.[12]

Images: studies of traditional images taken at the end of inspiration do not assess the changing collapse of the airway during the respiratory cycle, so it is necessary to take images during the 2 phases of the cycle and not at the end of each one.[12] Computed tomography (CT) is ideal for non-invasive assessment of this entity, especially the images taken with multidetector CT, which now enables us to identify the central airway in only a few seconds and create three-dimensional reconstructions with excellent spatial resolution in real time. It also makes it possible, with manoeuvring, to measure the area of the airway at different moments of the respiratory cycle, such as during coughs or deep expiration[13,14] (fig. 3).[17]

The protocol for dynamic CT scans of the central airway followed by the radiology department of the Beth Israel Deaconess Medical Center includes images at the end of inspiration and dynamic images during expiration taken in a caudal-cranial direction. To calculate the percentage of luminal collapse, the dynamic expiratory area (DEA) is subtracted from the area at the end of inspiration (AEI) and this is divided by the AEI, and then multiplied by 100.[6,15,15] There is considered to be malacia if the percentage of luminal collapse during dynamic expiration is above 50%.[6,15,16]

$$\frac{\text{AEI} - \text{DEA}}{\text{AEI}} \times 100$$

In a recent study, 29 patients with a diagnosis of tracheobronchomalacia were assessed, and the dynamic CT scanning of the airway was compared with the gold standard: dynamic bronchoscopy. With CT, the correct diagnosis was made in 97% of the cases (28 out of 29 patients), showing that dynamic CT of the airway is a highly sensitive method for detecting airway malacia.[18]

Nuclear magnetic resonance imaging of the central airway, with or without respiratory effort, can also successfully measure the collapsibility index of the airway, according to recent studies.[8]

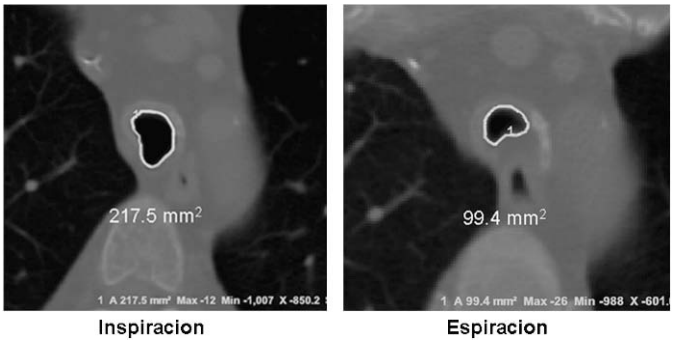

Figure 3. Dynamic tomography.

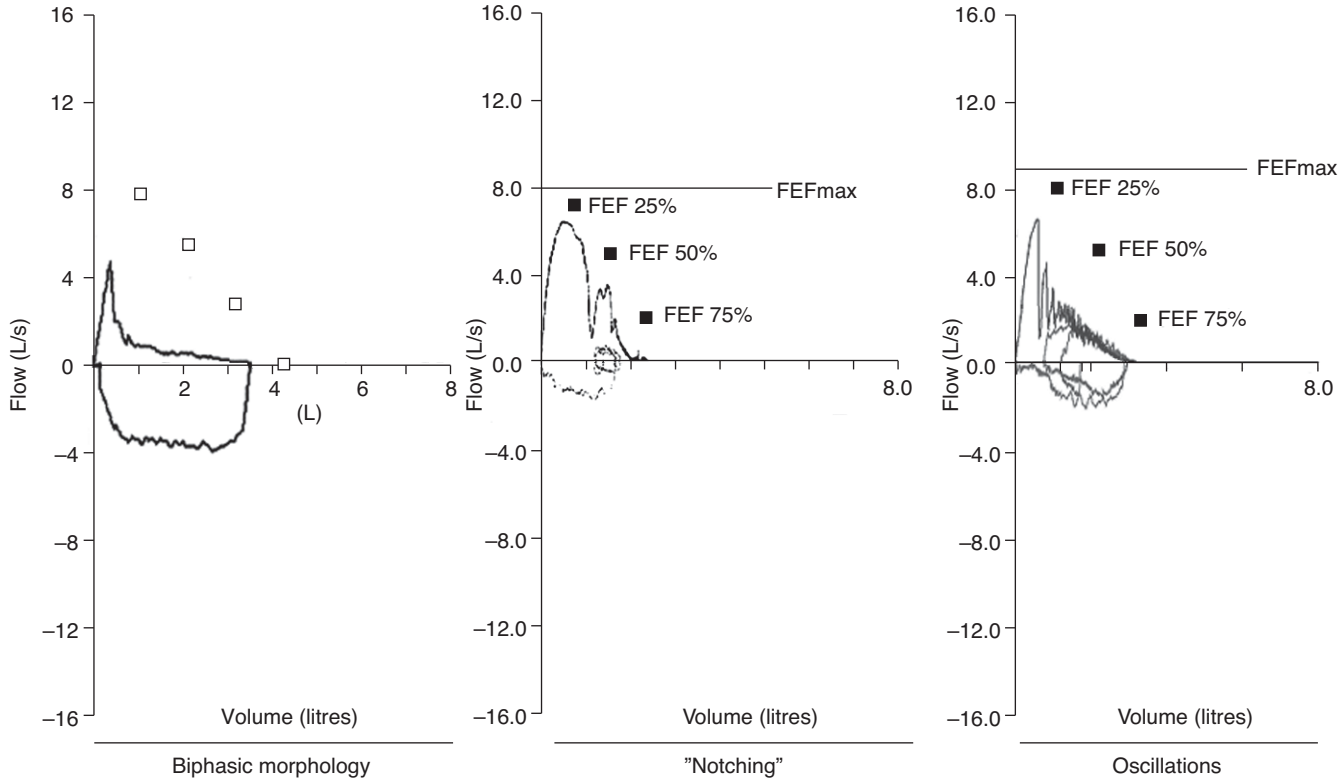

Figure 4. Changes in the flow volume curve. FEF: forced expiratory flow.

Tests of lung function: the use of tests of lung function in the diagnosis of tracheobronchomalacia is limited. A biphasic morphology is described, characterised by: a rapid reduction in maximum expiratory flow after the initial peak, related to the collapse of the central airway secondary to the negative transmural pressure;[1] notching of the expiratory descent; oscillations in the curve (fig. 4);[1] and a reduction in maximum voluntary ventilation. The findings described in tracheobronchomalacia can not be differentiated from those in cases of increased dynamic collapse of the airway, obstructive sleep apnoea and neuromuscular disorders of the larynx.[13]

It was reported recently that there is no correlation between the collapse of the central airway and the degree of obstruction, expressed by FEV_1; there may be deterioration of the trachea and large bronchi independent of the obstruction identified in the FEV_1.[6,11]

Bronchoscopy: visualization of the dynamic collapse of the airway is the gold standard for the diagnosis of tracheobronchomalacia.[1,6] The preferred method is fibrobronchoscopy in which the patient can breathe spontaneously with mild sedation and follow instructions, allowing the behaviour of the trachea to be identified during spontaneous breathing.[6,15]

Tracheobronchomalacia is considered to be mild if the narrowing of the lumen is between 50 and 75%, moderate if it is between 76 and 90% and severe between 91 and 100%, meaning the anterior and posterior walls make contact.[6,15] Respiratory manoeuvres, such as deep inspiration and forced expiration are used, and images and measurements are taken at different points of the bronchial tree under examination.[6,15]

The protocol for dynamic fibrobronchoscopy followed in the Beth Israel Deaconess Medical Center to diagnose tracheobronchomalacia is described in table 4.[6,15] It includes a study of the superior trachea at the level of the cricoid cartilage, in the mid-trachea 5cm from the carina, in the distal trachea 2cm from the carina, in the stem bronchi, and in the intermediary bronchus during the breathing cycle.

Tracheobronchomalacia Treatment

There are no well-defined guidelines for treating tracheobronchomalacia. Asymptomatic patients with an incidental diagnosis do not need treatment. Identifying patients with symptoms who would benefit from the different treatment alternatives is still a challenge.[15]

Initial treatment: the first line of treatment is controlling the symptoms of the concomitant underlying diseases. In most cases initial measures of support are enough. These include: stopping cigarette smoking; effective treatment of the respiratory infections; rehabilitation and breathing and relaxation techniques; non-pharmacological measures for gastro-oesophageal reflux; treatment of the disorders in the upper respiratory tract; and identification and treatment of obstructive sleep apnoea.[1]

Pharmacological treatment: before trying a more intensive treatment, it is particularly important to provide the best pharmacological treatment for the associated diseases. The treatment of bronchospasms in COPD and asthma is especially important as they cause important variations in intrathoracic pressure and worsening of the collapse of the trachea and large bronchi with malacia.

The treatment of gastro-oesophageal reflux and diseases such as polychondritis, using non-steroidal anti-inflammatory drugs in mild diseases and progressive doses of steroids in the more severe kinds, reduces the frequency and severity of recurrences, but does not detain the late loss of supporting cartilage or influence survival.[8]

Table 4
Dynamic fibrobronchoscopy protocol

Larynx				
Oedema		Mild		
	Yes	Moderate		
		Severe		
	No			
Trachea				
Proximal trachea at the level of the cricoid cartilages				
	Normal (< 50%).	Mild (51-75%)	Moderate (76-90%)	Severe (> 90%).
Inspiration				
Expiration				
Mid-trachea 5cm from the carina				
Expiration				
Distal trachea 2cm from the carina				
Expiration				
Right stem bronchus				
Expiration				
Proximal intermediate bronchus				
Expiration				
Left stem bronchus				
Expiration				
Others				
Inspiration				
Expiration				

Following the recommendations of the Division of Interventional Pulmonology of the Beth Israel Deaconess Medical Center, Boston, MA.

Non-invasive positive pressure airway ventilation: when the other measures do not work or exacerbations become more frequent, CPAP can be tried, which acts as a pneumatic stent. The positive pressure decreases pulmonary resistance and the work or breathing, maintaining the airway open and improving expiratory flow.

Controlled studies are needed to confirm the benefits of this procedure; intermittent nasal CPAP is recommended during the day, and continuous use at night. Positive pressure stabilizes patients and acts as a stepping stone treatment before using other more specific alternatives, such as placing a stent in the airway or surgery.[1]

Stents: there are 3 types of stents: metal, silicon and hybrids. Unfortunately, at the present time the ideal stent does not exist.

Stents restore the endoluminal structure of the airway, keeping it open and improving symptoms and ventilatory function. The constant changes in the size and shape of the obstruction in tracheobronchomalacia mean that stents often migrate or fracture. Furthermore, there are other potential complications, such as the formation of granulation tissue and mucous plugs. In general, there has been a reduction in the complications with silicon stents to treat diffuse tracheobronchomalacia with the implementation of Y stents and the concomitant use of N-acetylcysteine and guaiphenesin.[6,15]

In a recent study, Ernst et al[15] performed a prospective assessment of the effect of silicon Y stents on 75 patients diagnosed with severe diffuse tracheobronchomalacia. They assessed symptoms, quality of life, lung function and exercise tolerance before and 14 days after intervention. In some cases the stent was used as the sole alternative treatment or as a stepping stone treatment before the definitive tracheobronchoplasty.[6,15] Improvements were observed in quality of life, functional tests and the dyspnoea, but a high frequency of complications were found connected with the stent.[6,15]

Stents must only be used in the airways of patients with severe symptoms and with a suitable follow-up able to anticipate and correct any complications related to their use. In individuals with severe disease who are candidates for surgical correction, the stent is used as a first step to identify the benefits of stabilization and normalization of the airway.

Also, stents are acceptable for long-term treatment of those patients with improvements in their symptoms who are not candidates for surgical treatment, but they must be monitored closely to identify complications and obtain an improvement in their quality of life.[1,6,15]

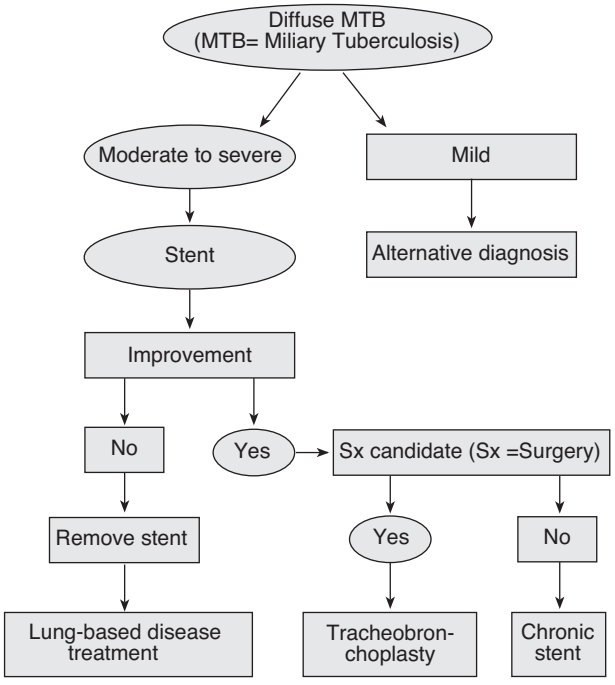

Figure 5. Treatment algorithm for diffuse tracheobronchomalacia.

Surgery: the objective of surgical treatment is to stabilize the membranous walls of the intrathoracic trachea, the stem bronchi and the intermediate bronchus. At present, for those cases selected the recommended surgery is tracheobronchoplasty. This procedure involves reefing and strengthening of the redundant posterior wall with marlex mesh to prevent expiratory protrusion into the airway lumen. Over time the mesh is incorporated by the wall with subsequent stiffening of the membranous part.[15]

Majid et al[15] recently reported the biggest series of severe diffuse tracheobronchomalacia cases, in which the patients' central airway was stabilised with tracheobronchoplasty using marlex mesh. There was an improvement in symptoms, quality of life, functional state

and exercise capacity, with statistically significant results in the 35 patients selected with severe disease.[15] Furthermore, they proposed a treatment and follow-up algorithm which included using a Y stent to identify the patients who would benefit from surgery (if potentially operable) or failing that, from chronic treatment (fig. 5).[15]

Other surgical treatment alternatives are laryngotracheal reconstruction, tracheal resection with reconstruction, and tracheotomy, which could be an alternative to by-pass the malacic segment and also ease handling of secretions and the use of positive airway pressure.[14,15]

Conclusions

Progress in imaging techniques and the developments in interventional bronchoscopy have improved the understanding, recognition and treatment of adult tracheobronchomalacia.

Progress still has to be made to unify the criteria for the classification, diagnosis and treatment guidelines of the disease, with prospective studies that clarify current doubts. The minimally invasive interventions currently available and surgical procedures can only be used at present with selected cases and in reference medical centres.

Conflict of Interest

The authors affirm that they have no conflicts of interest.

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