Egyptian Journal of Bronchology

Vol 1, No 1, Dec., 2007

REVIEW ARTICLE

EXPIRATORY CENTRAL AIRWAY COLLAPSE: A CONCISE REVIEW

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Expiratory central airway collapse is a clinical syndrome characterized by airflow limitation due to excessive narrowing of the central airways during exhalation. The syndrome consists of two entities, tracheobronchomalacia and excessive dynamic airway collapse, which are different in terms of morphology, physiology, etiology and structure. Treatment alternatives, which include conservative medical management, interventional bronchoscopic procedures and open surgery, should be individualized based on functional status, extent of disease and severity of airway collapse.

DEFINITIONS

Expiratory central airway collapse (ECAC) refers to airflow limitation due to excessive narrowing of the central airway lumen during exhalation. This process has been described in the literature as tracheobronchial collapse,[1] expiratory tracheobronchial collapse, expiratory tracheobronchial stenosis,[2] tracheobronchial dyskinesia,[3] tracheobronchomalacia and dynamic airway collapse.[4,5] Although, these terms suggest a dynamic central airway obstruction, they do not distinguish between a process that involves the cartilaginous rings and one that involves the membranous part of the central airways.

During normal exhalation, increased pleural pressure promotes a small degree of dynamic airway collapse (DAC) that can reduce the cross sectional area of the airway lumen by a maximum of 40%.[6] DAC is a physiologic process characterized by invagination of the posterior membrane of the tracheobronchial tree.

Excessive dynamic airway collapse (EDAC), on the other hand, refers to abnormal and exaggerated bulging of the posterior membrane within the airway lumen during exhalation. This might cause a reduction in cross sectional area of 50 % or

more.[7] It may not be responsible for airflow limitation since it may just represent a consequence of the increased intrathoracic pressure and decreased intraluminal pressure often seen in patients with significant obstructive ventilatory disorders such as asthma and COPD.[8]

Tracheobronchomalacia (TBM) refers to softening of the airway cartilaginous structures. As a result, the trachea and main bronchi lose their stiffness. Airflow limitation is probably caused by the way the airway walls come closer together, especially during exhalation.

CLINICAL PRESENTATION

The excessive reduction of the airway lumen during exhalation may be asymptomatic and detected during routine bronchoscopy or computed tomography obtained for other reasons.[9] Expiratory Central Airway Collapse can also present as a disease state characterized by refractory cough, dyspnea, inability to clear secretions, pneumonia and respiratory failure.[10] These signs and symptoms are nonspecific and similar to those of patients with chronic lung disease. In fact, many patients are diagnosed months or years after being treated for "refractory" asthma or COPD.[11] The cough in patients with ECAC has been described as a characteristic, seal-like barking, presumably because of expiratory collapse and vibration of the floppy airway walls. Wheezing is common and usually occurs early during a forced vital capacity maneuver because it is caused by large airway collapse. In severe cases, both TBM and EDAC can cause respiratory failure requiring mechanical ventilation.[12] Occasionally, ECAC is diagnosed as a cause for inability to wean off the mechanical ventilation.[13] In these patients, ECAC may not be easily appreciated because the endotracheal tube (ETT) partially stents the trachea and prevents the expiratory collapse. Furthermore, positive-pressure ventilation keeps the airway lumen open acting as a pneumatic stent. Once the positive pressure or the ETT is removed, the patient may develop expiratory flow limitation and respiratory failure.

MORPHOLOGY

Both computed tomography and bronchoscopy can be used to diagnose ECAC and determine the configuration of airway lumen during exhalation.[10,14] There is good correlation between the two tests and they are complementary rather than competitive.[15] Paired inspiratory –expiratory CT scanning reveals the airway collapse during expiration, and accurately determines the degree of airway narrowing as well as assesses the adjacent pulmonary parenchyma and vasculature, thus aiding in determining the etiology of the airway collapse.[16] The test is associated, however, with radiation exposure, and requires patient cooperation which may be difficult in the critically ill or dyspneic patient.

Dynamic flexible bronchoscopy, on the other hand, can be performed at the bedside with minimal sedation. The patient is moved through body positions (supine, erect, lateral decubitus) and asked to perform several respiratory maneuvers (deep inspiration, expiration, cough) in order to assess the degree of airway collapse and extent of airway abnormality, as well as to identify associated lesions such as mucosal edema, stenosis or secretions.[17]

Dynamic CT and bronchoscopy demonstrate that EDAC has a crescent configuration because of the bulging of the posterior membrane within the lumen in the presence of intact cartilaginous structures. TBM, on the other hand, has three morphologic types.[10] When the lateral airway walls are weakened, the configuration during exhalation is that of a saber sheath. When the anterior wall is weakened, the airway lumen takes the shape of a crescent. If the anterior and lateral cartilaginous walls are involved, the morphology is circumferential. This is usually seen in patients with relapsing polychondritis and may be accompanied by mucosal edema (Fig. 1).

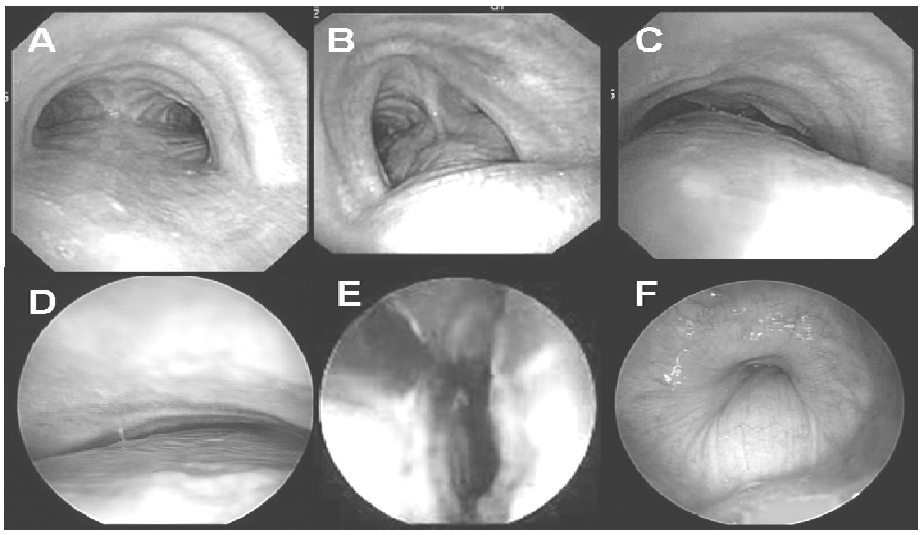

Fig 1A. Normal tracheal lumen during inspiration. B. During exhalation there is a certain degree of dynamic airway collapse (DAC) that reduces the cross sectional area (CSA) by a maximum of 40%. C. A reduction of CSA by 50 % or more defines excessive dynamic airway collapse (EDAC) which is caused by bulging of the posterior membrane within the airway lumen during exhalation in the presence of intact cartilaginous structures. D. In tracheobronchomalacia (TBM), if the anterior wall is weakened, the airway lumen takes the shape of a crescent. E. When the lateral walls are weakened, the configuration during exhalation is that of a saber sheath. F. If both anterior and lateral cartilaginous walls are involved, the type of TBM is called the circumferential type, and is associated with significant edema and seen mainly in relapsing polychondritis.

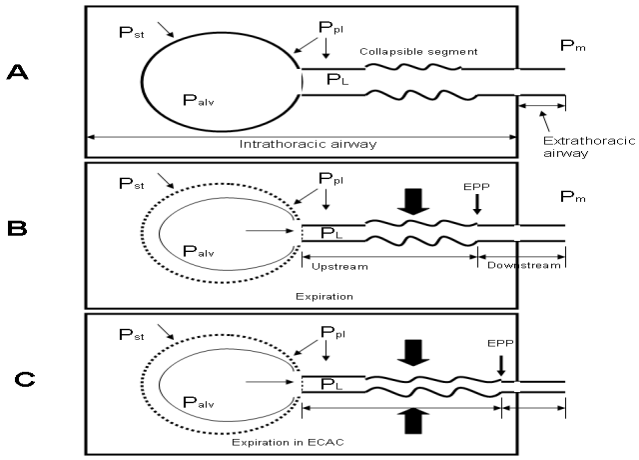

Fig 2A. Schematic diagrams of the thorax and airways. The pressure in the alveoli (Palv) is the sum of total elastic recoil pressure (Pst) and pleural pressure (Ppl): Palv = Ppl + Pst; B. During exhalation, alveolar pressure rises above athmospheric (Pm) to cause air to flow from alveoli to the mouth. Since the pressure at the mouth is zero (athmospheric) and P alv > 0, the intraluminal pressure (PL) at some point has to equal the Ppl; this point is called equal pressure point (EPP) which divides the airway into an upstream and a downstream segment; EPP is not fixed, but migrates from extrathoracic airways into the small airways as the lung volume decreases and Ppl increases during expiration; C. When the compliance of the airways is increased as in expiratory central airway collapse (ECAC), increased airway compressibility causes the EPP to become fixed at a point closer to the thoracic outlet.

PHYSIOLOGY

DAC and Equal Pressure Point Theory: Airways within the lung are surrounded by alveoli. The pressure in these alveoli is the sum total of elastic recoil pressure and pleural pressure (Fig. 2A). During inspiration, pleural pressure is negative relative to atmospheric pressure and so is alveolar pressure in order to cause air to flow from the atmosphere into the alveoli. Hence, the pressure acting on the outside of airways causes them to enlarge and causes diminished resistance to airflow. During quiet expiration, pleural pressure remains negative but alveolar pressure rises above atmospheric pressure so that air flows from the alveoli to the atmosphere. In contrast to what occurs during inspiration, extramural airway pressure is positive and the airways tend to become smaller as the resistance to flow increases. The large, central airways are, somewhat protected from dynamic compression and collapse by the presence of cartilaginous rings. These rings encase the large airway circumferentially, except posteriorly in an area occupied by the membranous structures. During expiration, this area bows slightly inwards causing DAC (Fig. 1). This tendency increases during forced exhalation or cough when pleural and alveolar pressures rise considerably.

The degree of large airway narrowing (DAC) which occurs during exhalation is moderated by the degree of external airway support. The major external support of airways is the elastic tissue of the alveoli which encloses the intraparenchymal airways. As large airways narrow they tend to pull the lung parenchyma inwards. To the extent that intact elastic tissue is normal, airway collapse is opposed and airway narrowing is limited. During expiration, driving alveolar pressure is completely dissipated along airway walls, reaching atmospheric pressure just outside the lips or nose. Because elastic recoil is greatest at total lung capacity and least at residual volume, alveolar pressure becomes progressively dependent upon only pleural pressure as lung volume decreases. However, pleural pressure acts directly not only upon alveoli but also upon the intrathoracic airways (Fig. 2B). At some point in the airways, therefore, pressure inside and outside will be equal and, from that point downstream, only the elastic recoil pressure will driving airflow (Fig. 2B). This point is called the equal pressure point (EPP). It has been shown that, during the forced vital capacity maneuver, EPP is in the extrathoracic airways (e.g., cervical trachea) at high lung volumes but, as lung volume declines, the EPP moves within the thorax progressively towards and into the small airways.

EDAC

In obstructive ventilatory dysfunction (i.e. COPD, asthma), small airways are already narrowed by disease. Airway resistance rises dramatically as these are suddenly compressed to very small diameters. In COPD, decreased elastic recoil at all lung volumes (emphysema) and inflammatory narrowing of small airways (bronchitis) usually coexist. In the presence of emphysema, elastic tissue is decreased. Airways are compressed and may collapse during exhalation and especially during a forced vital capacity maneuver. Airway collapse can also be seen in bronchitis and possibly in asthma when obstruction is severe and occurs primarily in small airways. An "airway collapse" pattern on flow volume loop is seen in 40% of patients with chronic airflow obstruction.[18] This consists of a sudden decrease the peak expiratory flow, defined as a drop of 50% within 10 % of forced volume capacity (Fig. 3A). Results from density dependence of flow studies using heliox showed that for patients with COPD with this flow volume loop pattern, the flow limiting segments (choke points) are peripheral [18] and not central as might be expected when large airway collapse is identified on bronchoscopy or CT. Recent evidence suggests that in bronchoscopically-documented EDAC, central airway collapse is not closely related to airflow obstruction, and expiratory flow limitation at rest often occurs in peripheral airways without central airway collapse.[19] Localizing the choke points to central or peripheral airways, therefore, becomes important in such patients, because if obstruction is located in the peripheral

intrapulmonary airways, these patients might not improve after stent insertion or tracheoplasty, performed to stiffen the central airways.

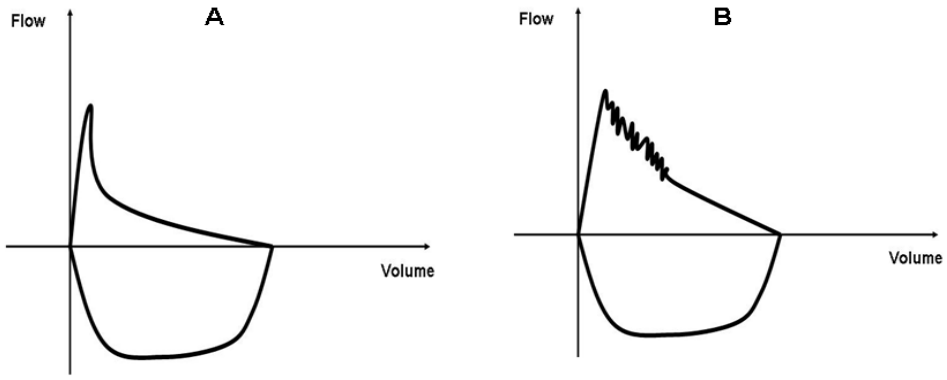

Fig 3. Flow volume loop patterns in patients with expiratory central airway collapse. A. Low maximal flow and airway collapse pattern: maximal flow is reached quickly followed by a sudden large fall in flow (defined as 50% drop within 10% of forced vital capacity) although only a small volume is exhaled. There is subsequently a phase in which flow rate falls very little during the remainder of expiration resulting in a long plateau. B. Flow oscillations or saw–tooth appearance is defined as a reproducible sequence of alternating decelerations and accelerations of flow. This pattern is considered a nonspecific indicator of upper airway dysfunction because it can also be seen in patients with obstructive sleep apnea, structural or functional disorders of the larynx, neuromuscular diseases, Parkinson disease, pedunculated tumors of the upper airway and upper airway burns.

TBM and wave speed theory: Mechanisms responsible for expiratory airflow limitation are not limited, however, to lung elastic recoil pressure, lung volume, and location of the EPP. Also implicated in this process is the density of the gas flowing through the airways, the airway compressibility and smooth muscle tone at the compressibility point described earlier, the overall geometry and mechanical properties of the tracheobronchial tree, the area through which airflow occurs, convective acceleration loss (Bernoulli effect), and the frictional pressure loss during expiration. Interaction of these factors has been described in the wave speed theory of flow limitation which implies that maximum expiratory flow is dependent on airway compliance.[20] Increasing large airway compliance increases airway resistance and decreases maximum expiratory flow, which could contribute to the airway obstruction associated with TBM because flow velocity reaches the speed of wave propagation at the choke point, closely related to EPP, which is now located further downstream towards the airway opening in the central airways (Fig. 2C).

Therefore, it appears that from a physiologic standpoint, the two forms of ECAC are different. Patients with EDAC may have partially reversible

small airway obstruction which may respond to bronchodilator treatment, while patients with TBM have centrally located choke points such that bronchodilators may actually worsen airway obstruction because of increased airway compliance and smooth muscle relaxation.[21]

Pulmonary Function Tests: Pulmonary function tests in ECAC might show diminished expiratory flow, the airway collapse pattern described above on flow-volume loop (FV loop), dynamic airway compression (calculated as Slow Vital Capacity minus Forced Vital Capacity) or flow oscillations[10] (Fig. 3B). Such flow oscillations or saw–tooth appearance, defined as a reproducible sequence of alternating decelerations and accelerations of flow are however, considered a nonspecific indicator of upper airway dysfunction.[22]

Recent evidence suggests that expiratory central airway collapse is not correlated with the degree of obstruction as assessed by FEV1, and that ECAC could be found irrespective of the degree of expiratory flow limitation during quiet breathing.[19] Thus, significant expiratory central airway collapse cannot be assumed in patients with obstructive airway disease, and symptomatic ECAC may exist without significant airflow obstruction as assessed by FEV1. Other physiologic variables such as markers of dynamic hyperinflation (e.g. inspiratory capacity) or density dependence of flow may better identify and predict which patients with symptomatic ECAC will benefit from central airway splinting using airway stents or surgical membranous tracheoplasty.

ETIOLOGY

Both TBM and EDAC can be idiopathic or secondary. While both forms of expiratory central airway collapse may have common risk factors, their etiologies are usually different Table 1. TBM is often a consequence of pressure necrosis from indwelling tracheostomy or endotracheal tubes,[23] chronic inflammatory states of the airways such as relapsing polychondritis,[24] or caused by long standing extrinsic compression that eventually weakens the cartilaginous rings as seen in thyroid goiter[25] or postpneumonectomy syndrome.[26] EDAC, on the other hand, is commonly seen in COPD and asthma but can also be caused by chronic irritation of the airways or atrophy of elastic fibers as seen in congenital tracheobronchomegaly.[27]

STRUCTURE

Histological evidence of structural differences in TBM and EDAC is scarce and usually based on case reports or small case series.[10] It is unclear whether these histopathologic changes are primary or simply end-products of injury from other disorders. In tracheomalacia, the tracheal cartilage-to-soft tissue ratio is sometimes as low as 2:1, while normally it is approximately 4.5:1. In TBM due to relapsing polychondritis, an extensive inflammatory component of the tracheobronchial cartilage showing empty lacunae (empty spaces within the cartilage) and a mixed population of inflammatory cells has been described.[28] In patients with described EDAC, atrophy of longitudinal elastic fibers as well as flaccid and dilated membranous portion of the airway in the absence of cartilaginous abnormalities has been noted.[4]

Endobronchial ultrasound (EBUS) shows differences in airway structural composition in normal airways as well as in airway disease caused by tuberculosis,[29] compression from vascular rings,[30] cancer invasion[31] or relapsing polychondritis.[32] EBUS shows distinct hypo and hyper-echoic layers corresponding to the laminar histological structures of the airway wall[33] (Fig. 4A). Recently, it has been reported that the EBUS findings in DAC, EDAC and TBM are different.[34] In DAC, there are no cartilaginous abnormalities noticed and the posterior membrane is of normal thickness (Fig. 4B). In EDAC caused by COPD, EBUS images reveal intact cartilage of normal thickness while the posterior membrane is thinner than normal (Fig 4B). In TBM caused by chronic inflammation from indwelling tracheostomy, the EBUS image reveals thickened and destroyed

cartilage, thickening of the submucosa overlying the cartilaginous rings and increased thickness of the submucosa in the posterior membrane, while in TBM from relapsing polychondritis EBUS revealed that the cartilage is thickened and destroyed, the submucosa is thickened as well, while the posterior membrane is normal (Fig. 4B). If confirmed in larger studies, these findings would support the hypothesis that the two forms of ECAC are distinct not only morphologically, physiologically and etiologically but also structurally.

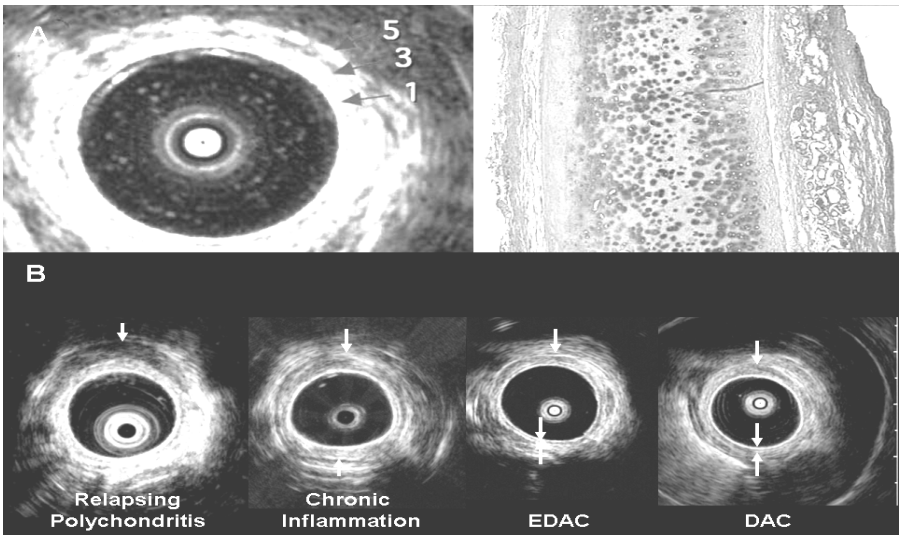

Fig 4A. Endobronchial ultrasound (EBUS) reveals hypo- and hyper-echoic layers (left panel) which correlate with the histological structural layers of the central airways (right panel). B. EBUS findings in various forms of expiratory central airway collapse. In tracheobronchomalacia (TBM) from relapsing polychondritis, the cartilage is thick and the posterior membrane is normal; in TBM from chronic inflammation due to indwelling tracheostomy tube, the cartilage is thick but also the posterior membrane is thick; in excessive dynamic airway collapse (EDAC) the cartilage is normal but the posterior membrane is thin; in dynamic airway collapse (DAC), both the cartilage and the posterior membrane are of normal thickness. Photos are courtesy of Prof. Noriaki Kurimoto, St. Marianna University School of Medicine, Kawasaki, Japan.

TREATMENT

Treatment alternatives for patients with expiratory central airway collapse should depend on the severity of symptoms, the degree and extent of airway collapse, and the underlying cause.[35] Very few studies, however, addressed the treatment of these conditions, and because of a lack of universally accepted definitions and classification, study results cannot be generalized.

In order to facilitate a comprehensive approach to these patients, we have recently proposed a multidimensional classification system that incorporates all of the elements needed to identify and compare patients with various forms of expiratory central airway collapse.[36] This classification system, referred to as FEMOS, addresses Functional status, Extent of abnormalities noted, Morphology of the abnormality, Origin (or etiology) of the abnormality, and Severity of the disease process Table 2. FEMOS can be used during the diagnostic

approach to patients with ECAC, as well as to objectively compare findings after therapeutic interventions. The common language offered by this multidimensional classification system, can help clinicians and researchers to answer existing questions regarding patient selection and outcomes for various therapeutic interventions.

Conservative medical management: Treatment of the underlying condition should be optimized before considering invasive therapies. This is possible however, only in the absence of respiratory distress or critical airway narrowing and impending respiratory failure in which case, the airway has to be urgently stabilized. For stable patients, it is logical that drug therapy be attempted whenever possible before proceeding to more aggressive interventions. This is particularly the case in patients with relapsing polychondritis, for whom non-steroidal anti-inflammatory drugs or high-dose corticosteroids and various immunosuppressants are warranted. Although these drugs are often effective in treating chondritis, they do not stop the late loss of cartilaginous support and may not influence survival.[24] In patients with underlying asthma or COPD, treatment should obviously be in accordance to published guidelines. As mentioned previously, a dramatic fall in peak flow may occur in response to central airway smooth muscle relaxation after bronchodilator administration.[37]

Noninvasive positive pressure ventilation acts as a pneumatic stent and can be used in selected cases to maintain airway patency, facilitate secretion clearance, and improve expiratory flow. Small case series support the addition of nocturnal and intermittent daytime nasal continuous positive airway pressure (10 cm H2 O) to improve spirometry, sputum production, atelectasis, and exercise tolerance.[38]

Interventional Bronchoscopy: The goal of bronchoscopic interventions is to prevent airway collapse responsible for airflow limitation and symptoms. Because it maybe difficult to determine the specific contribution of ECAC to a patient's functional impairment, a comprehensive pretreatment evaluation should be performed especially to distinguish symptoms from those related to underlying airflow obstruction from asthma or COPD. This may include density dependence studies using heliox (HeO2) to determine the choke point location. Physiologic studies showed that the predominant sites of airflow limitations (choke points) in asthmatics can be defined by comparing maximum expiratory flow volume (MEFV) curves after the inhalation of air and after the inhalation of HeO2 (80%-20%).[18] HeO2 is lighter than air, modifies gas density, and subsequently improves the flow in the central airways. HeO2 should not change flow in patients with peripheral choke points because small airway flow is mainly determined by gas viscosity rather than density. A reduced density dependence of flow, therefore, will favor predominantly peripheral choke points, whereas increased density dependence (ΔV E max> 20%) will be consistent with choke points localized in the large airways.[18] The predictive value of these tests, however, has not been tested in patients with ECAC or any other forms of central airway obstruction and will require validation before implementation in clinical practice.

From a practical standpoint, a therapeutic trial of airway stent insertion might result in symptomatic improvement (Fig. 5).[14,35] If objective measures of improvement are also documented using dynamic CT or bronchoscopy and the multidimensional FEMOS classification, for instance, then the stent can be left in place or a decision should be taken regarding a surgical correction by membranous tracheoplasty.

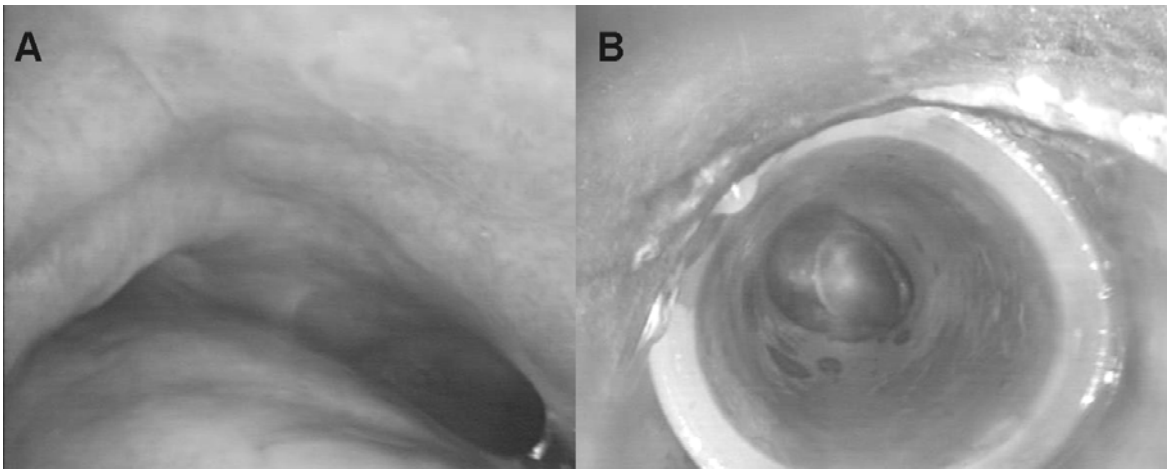

Fig 5. Airway lumen during bronchoscopy before and after stent insertion in a patient with severe idiopathic excessive dynamic airway collapse (EDAC) A. Distal trachea and mainstem bronchi during rigid bronchoscopy before stent insertion: EDAC is characterized by exaggerated bulging of the posterior membrane within the airway lumen. B. Distal trachea and mainstem bronchi during rigid bronchoscopy show complete restoration of the airway lumen after three silicone stents were inserted (one in the lower trachea and two in the mainstem bronchi).

It is difficult, however, to draw conclusions about the efficacy of airway stents pertaining only to ECAC. More than one stent may be required if the symptoms persist, presumably because choke points may migrate distal to the stent and limit airflow.[39] If symptoms do not improve, stent removal is warranted to avoid stent-related complications, including migration, obstruction by mucus or granulation tissue, infection, fracture, and airway perforation.

Silicone stents require rigid bronchoscopy for placement, but have excellent force compression characteristics and are easily removable in case of complications. Using FEMOS system pre and post operatively, we have recently reported that silicone stent insertion improves functional status immediately post intervention in patients with expiratory central airway collapse, but is associated with a high rate of stent-related adverse effects and need for repeat bronchoscopic interventions.[40] A high rate of complications is also reported by others,[41] and is likely explained by the dynamic features and frequently inflamed airway mucosa associated with expiratory central airway collapse. In our study, stent-related adverse events requiring emergent flexible or rigid bronchoscopy usually occurred within the first four weeks after stent insertion. This supports a practice of early surveillance bronchoscopy, close clinical follow-up and immediate bronchoscopy in case of clinical deterioration.

Various results from metal stent insertion for patients with central airway obstruction including malacia have been reported.[35] Metal stents are less likely to migrate or to cause obstruction by mucus plugging because they may preserve mucociliary clearance if uncovered and are capable of a certain degree of dynamic compression during coughing to facilitate mucus clearance. In one case series addressing the role of metal stents in 4 patients with tracheomalacia, however, stents had to be removed because of failure and complications.[42] Metal stents are more difficult to remove, tend to cause recurrent stenosis and are predisposed to fracture and collapse especially in a dynamic process such as ECAC. In accordance with an FDA warning regarding fatalities and stent-related adverse events, metal stents should probably be avoided in patients with benign causes of central airway obstruction.[43]

Bronchoscopists have also attempted to strengthen the posterior membrane in order to prevent ECAC. The injection of sclerosing agents into pericartilaginous tissues resulted in significant peritracheal fibrosis compared with controls.[44] Nd:YAG laser therapy has been used to tighten the airway wall by creating a fibrotic reaction of the posterior membrane.[45] This technique may be hazardous because the posterior wall is only 3-5 mm thick and, to our knowledge, no experimental studies have confirmed feasibility. Although preliminary results in a few patients suggest improvement in symptoms, ventilatory function and bronchoscopic aspects, long term outcomes have not been reported.

SURGERY

The goal of open surgical procedures is to remove or bypass the abnormal airway segment for focal disease or to splint the airway by externally stiffening the airway walls in the setting of multifocal or diffuse ECAC.

A tracheostomy tube may stent the collapsed airway and allows ventilatory support when needed.[46] Tracheostomy, however, should not be considered a first line treatment in elective cases because it can be complicated by secondary tracheomalacia and stenosis at the stoma site[47] and may exacerbate airway collapse because it bypasses the physiologic function of the glottis (the glottis assures positive transmural pressure which keeps the airway lumen patent).

Tracheal resection with end-to-end anastomis has been proposed for focal tracheomalacia. Outcomes are good in 93% of patients, and mortality is about 2 % in experienced centers specialized in tracheal surgery.[48]

Airway splinting via membranous tracheoplasty has been used to consolidate and reshape the airway wall. Outcomes are favorable in uncontrolled studies.[49] This technique involves restoring the proper anatomic configuration of the airway so that cartilage rings are brought into a more normal C shape from their flattened pattern. This is done by reinforcing the posterior membrane with a polypropylene mesh which becomes permanently incorporated into the membranous wall through. Wright et al recently published their 10 year experience during which 14 consecutive membranous wall tracheoplasties were performed.[49] There were no postoperative deaths and the most frequent morbidity was retention of secretions during the immediate postoperative period in 7 of the 14 patients. All patients felt subjective improvement early after surgery. Among ten patients followed long-term, 6 were subjectively judged to have an excellent result, 2 were good, and 2 were judged as failures. This procedure is also limited to specialized centers and has been only offered to a small group of patients judged to be ideal surgical candidates.

Other methods of surgical airway splinting include tying the posterior wall of the trachea with bone chips, fascia grafts or plastic prostheses, performing autologous coastal cartilage grafts and suturing the trachea to dura mater grafts.[10] Surgical placement of external tracheal stents has been advocated for severe cases of malacia.[50] The results of these techniques are encouraging, but their application and efficacy remain to be determined before being widely recommended.

SUMMARY

Excessive central airway collapse consists of two distinct entities, TBM and EDAC, which may be seen separately, but also together. Morphologic differences are noted by bronchoscopic and radiologic imaging. Available evidence suggests that these two entities have different etiologies and pathophysiology. A multidimensional classification system to objectively assess and compare patients with ECAC may assist researchers and clinicians in answering existing questions regarding patient selection for various therapeutic interventions.

Table 1. Risk factors and etiologies for the two forms of expiratory central airway collapse.

Potential risk factor	Underlying disease or process	Form
Pressure necrosis	Tracheostomy and ETT with inflatable cuff	TBM
Unrecognized tracheobronchial fracture	Closed chest trauma	TBM
Chronic irritation of the airways	Smoking	EDAC
	Air pollution	EDAC
	Chronic tracheostomy or endotracheal tubes	TBM
Impairment of blood supply	Tracheostomy and ETT with inflatable cuff	TBM
	Post lung transplantation	TBM
Inflammation	Relapsing polychondritis	TBM
	Chronic, recurrent infections	TBM
	COPD and asthma	EDAC
Destruction of the cartilage: Malignancy	Lung cancer, thyroid cancer	TBM
Iatrogenic	Endobronchial electrosurgery	TBM
Mechanical factors	Post lung transplantation	TBM
	Post thyroid surgery	TBM
	Postpneumenectomy syndrome	TBM
Long standing extrinsic compression	Mediastinal goiter	TBM
	Tumors (carcinoma, teratoma, lymphoma, neuroblastoma)	TBM
	Vascular anomalies (innominate artery, aortic arch ring, pulmonary artery sling, aberrant R subclavian)	TBM
	Cysts (thymic cyst, bronchogenic cyst, lymphatic malformation)	TBM
	Cardiac (enlarged L atrium, enlarged pulmonary arteries or veins)	TBM
Continuous cycling pressures on the airway wall	Chronic ventilation through uncuffed tracheostomy tubes	TBM
Atrophy of the elastic fibers		
Congenital	Mounier Kuhn syndrome	EDAC
Acquired	COPD	EDAC

TBM: tracheobronchomalacia; EDAC: excessive dynamic airway collapse; ETT: endotracheal tube; COPD: chronic obstructive pulmonary disease.

Table 2. FEMOS classification for patients with expiratory central airway collapse.

Criteria	Description
Functional class	Normal 1; Mild 2; Moderate 3; Severe 4
Extent	Normal 1; Focal 2; Multifocal 3; Diffuse 4
Morphology	EDAC (Crescent)
	TBM (Crescent; Saber-sheath; Circumferential)
Origin	Idiopathic; Secondary
Severity	Normal 1; Mild 2; Moderate 3; Severe 4

Functional class refers to the degree of functional impairment as assessed by World Health Organization: 1-no limitation of usual physical activity; ordinary physical activity does not cause symptoms; 2-mild limitation of physical activity; there is no discomfort at rest, but normal physical activity causes increased symptoms; 3-marked limitation of physical activity; there is no discomfort at rest, but less than ordinary activity causes increased symptoms; 4-unable to

perform any physical activity at rest; symptoms may be present at rest, and symptoms are increased by almost any physical activity.

Extent defines the region of the tracheobronchial wall affected as documented by bronchoscopy/ computed tomography: 1-Normal: there is no airway abnormality; 2-Focal: abnormality is present in one main or lobar bronchus or one tracheal region (upper, mid- or lower); 3-Multifocal: abnormality is present in two contiguous or at least two noncontiguous regions; 4-Diffuse: abnormality is present in more than two contiguous regions.

Morphology describes the shape of the airway lumen which is reduced during expiration and respiratory movements as assessed by bronchoscopy/computed tomography: EDAC: reduction in the antero-posterior diameter caused by excessive bulging of the posterior membrane in the absence of cartilaginous abnormalities resulting in a crescent shape airway lumen configuration. TBM: reduction in the airway lumen due to weakness of the cartilaginous structures: Saber-sheath: refers to reduction in the transversal (coronal) diameter; Crescent type: refers to reduction in the antero-posterior (sagittal) diameter; Circumferential: refers to reduction in both transversal and anteroposterior diameters.

Origin describes the underlying mechanism responsible for the abnormality; idiopathic: no underlying etiology is identified; secondary: may be due to known underlying processes.

Severity describes the degree of the airway collapse during expiration as documented by bronchoscopy/ computed tomography: 1-Normal: Expiratory collapse of less than 50%; 2-Mild: Expiratory airway collapse of 50-75%; 3-Moderate: Expiratory airway collapse of 75-100%; 4-Severe: Expiratory airway collapse of 100%; the airway walls make contact.

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