Excessive Dynamic Airway Collapse or Tracheobronchomalacia: Does It Matter?

Colapso dinámico excesivo de la vía aérea o traqueobronquomalacia: ¿Es relevante?

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Excessive central airway collapse (ECAC) describes two different pathophysiologic entities, excessive dynamic airway collapse (EDAC) and tracheobronchomalacia (TBM). Although the prevalence is not known precisely, it is estimated that approximately 13% with known lung disease had evidence of ECAC.1 Although some experts advocate thinking about these two entities separately, others prefer to lump them together. In this editorial, we will highlight the clinical presentation, diagnostic evaluation and therapeutic approach of TBM and EDAC and discuss whether it is clinically relevant to distinguish between both entities.

TBM and EDAC are an increasingly recognized conditions that affect the trachea and main bronchi leading to symptoms of airway obstruction.2 Several morphological types of ECAC have been described in the literature.2–4

1 EDAC: is characterized by excessive forward displacement of the membranous wall due to weakness and atrophy of the longitudinal elastic fibers of the posterior wall.

2 Cartilaginous type TBM: is characterized by weakness of the lateral and anterior cartilaginous walls of the airways. This type can have a crescent or saber-sheath appearance depending on whether the anterior or lateral walls of the airway are weakened.

3 Circumferential or concentric type TBM: due to combined anterior and lateral airway wall collapse and is usually associated with inflammatory conditions such as relapsing polychondritis.

4 A combination of EDAC and any type of TBM.

Clinical symptoms in patients with EDAC and TBM are similar. While some may be asymptomatic, other may present with severe dyspnea, intractable cough, recurrent infections and respiratory failure.

Although ECAC is usually diagnosed reliably by dynamic flexible bronchoscopy and/or dynamic airway computed tomography (CT),5,6 it is important to emphasize the fact that such diagnosis should not be based solely on that. Two studies in normal healthy volunteers with normal pulmonary function tests (PFT) have shown that expiratory collapse can be present with >50% reduction in cross-sectional area, respectively, on CT scan.5,7 Moreover, up to 20% of patients with ECAC have normal PFT.8 Flow-volume loop patterns that might suggest ECAC are: low maximum forced expiratory flow, biphasic expiratory curve, flow oscillations and notching.9 However, around 17% of patients with ECAC have normal flow volume pattern.8 Thus, the diagnosis of ECAC should be based on: (1) objective data (dynamic flexible bronchoscopy and/or dynamic CT scan) and (2) clinical symptoms that are not fully explained by another disease.

Initial treatment of symptomatic ECAC includes treatment of recurrent infections, maximize medical therapy for concomitant airway diseases, pulmonary physiotherapy, airway clearing device, pursed lip breathing, continuous positive airway pressure (CPAP) and pulmonary rehabilitation. Furthermore, patients should undergo evaluation for vocal cord dysfunction and gastroesophageal reflux disease since both are highly prevalent in this patient population and might negatively impact the outcome of surgical central airway stabilization (CAS).9 CPAP can be an effective treatment option for ECAC patients while awaiting definitive surgical options or as destination therapy for patients who cannot undergo surgery.

Surgical CAS is the definitive treatment for patients with symptomatic ECAC despite optimal medical management. Tracheobronchoplasty (TBP) is a technically demanding surgical technique that achieves airway stabilization by suturing a knitted polypropylene mesh to the posterior membrane of the trachea and bilateral bronchi.10 In experienced hands, TBP is a safe operation with low mortality rate (1%) despite significant risk for perioperative complications (47%).10,11 TBP has been shown to improve quality of life, exercise capacity and dyspnea symptoms, though not FEV1, in carefully selected adults with severe symptomatic ECAC.10

In our opinion, before surgical intervention, symptomatic ECAC patients should undergo a short-term stent trial to determine
whether they are candidates for TBP. Patients are evaluated at baseline and 7–14 days following stent placement using the following parameters:

1. Respiratory symptoms (dyspnea, cough and ability to clear secretions)
2. Objective assessment of dyspnea and cough with the modified Medical Research Council and Cough Quality of Life Questionnaire
3. Lung function using spirometry (FEV1)
4. Exercise capacity using a 6-min walk test

While some groups are forgoing this step,11 we feel that it is important to perform a stent trial in order to test the hypothesis that CAS will ameliorate symptoms. In our experience, 75% of patients undergoing stent trial will experience subjective and/or objective improvement, and 65% will ultimately undergo surgery.

Silicone Y-stent placement has been suggested as a trial option for patients with severely symptomatic ECAC. Two observational studies including COPD patients had reported improved respiratory symptoms, quality of life and functional status when silicone stent is used for ECAC.12,13 However, silicone stents were associated with significant irreversible complications (severe cough, mucus obstruction, stent migration and infection) despite using a standardized protocol leading occasionally to equivocal results.12–14 In order to circumvent such adverse events, our group have recently published our experience using uncovered self-expanding metallic stents (USEMAS) as a short-term trial to evaluate patients with symptomatic ECAC.15 USEMAS achieved clinical improvement with few complications in patients with severe symptomatic ECAC.15

Following clinical improvement with stent placement, patients with EDAC and TBM cartilaginous type may become candidates for surgical CAS with TBP. Although both are amenable to surgical stabilization, it becomes important to distinguish between both morphologies in regards to technical surgical approach. There are adjustments that may be made in the degree of tensioning created by the suture placement. In most cases of TBP for EDAC, the transverse diameter of the airways is not excessively large, and thus lateral downsizing should not be undertaken to the same degree as what would be aimed for when repairing a TBM airway with cartilaginous involvement where there has been spaying apart of the cartilaginous ends, causing an increased transverse diameter and decrease of the anteroposterior diameter. In that case more downsizing of the airway diameter would be attempted. In EDAC, tension may be predominantly developed by suture placement to achieve axial tension instead.

In conclusion, both EDAC and TBM are heterogeneous entities of ECAC, and are characterized by a diverse non-specific symptom profile that may overlap with other more common airway diseases. Although the pathophysiology of airway mechanics as well as morphology in both entities is different, current evidence so far show no practical benefit in making such distinction since both have similar symptoms, diagnostic and therapeutic work. Airway stenting with silicone or USEMAS is the only reliable way to identify the subgroup of patients who will benefit from surgical CAS. Once surgical intervention is decided, the morphology of dynamic airway collapse becomes clinically relevant in the technique used for a successful surgical approach.

References