Mechanical Ventilation for the Treatment of Severe Excessive Dynamic Airway Collapse

To the Editor:

We read with interest the case report by Ismael et al[1] describing a patient with Sjögren's syndrome and cystic lung disease who could not be weaned from a ventilator due to severe central excessive dynamic airway collapse (EDAC) of the lower part of the trachea and proximal bronchi. EDAC corresponds to the expiratory bulging of the tracheobronchial wall without known airway structural abnormalities, leading to a decrease of at least 50% in internal diameter.[2] It is a rare and underdiagnosed entity, commonly confused with other respiratory diseases such as asthma and COPD. Although noninvasive procedures such as cervicothoracic computed tomography scan on inspiration and expiration may suggest the disorder, the accepted standard method for diagnosis is bronchoscopy.[3-7]

There is no consensus on the best treatment. The authors stated that stenting could resolve the collapsibility in that case.[1] In our practice, endotracheal or endobronchial stenting is effective when the collapse affects only a small area of the trachea or main bronchi. In some patients with diffuse airway involvement, stenting is frequently useless. Murgu and Colt[2] proposed an algorithm for the management of EDAC. Preceding invasive measures, the authors recommend the appropriate pharmacologic treatment of comorbidities, including COPD, asthma, and gastroesophageal reflux disease, because they may be predisposing factors for airway collapse. When treatment is not sufficient or the exacerbations become more frequent, noninvasive ventilation (NIV) may be tried. There are reports of significant improvement with CPAP $>$ 6 cm H_2O.[8]

We want to report our case of a female patient with scleroderma and a longstanding history of poorly controlled asthma who presented with severe and diffuse EDAC. Bronchoscopy (Fig. 1) showed a striking expiratory bulging (75–100%) of the larynx, posterior wall of the trachea, and bronchial tree bilaterally (main, lobar, and segmental bronchi), with some parts of the airway walls making contact. Pulmonary involvement with connectivitis was excluded, and histological examination of bronchial and hypopharynx biopsies was inconclusive (chronic nonspecific inflammation). Following consecutive hospitalizations requiring invasive mechanical ventilation support, the patient started nocturnal CPAP with 10 cm H_2O (see Fig. 1).

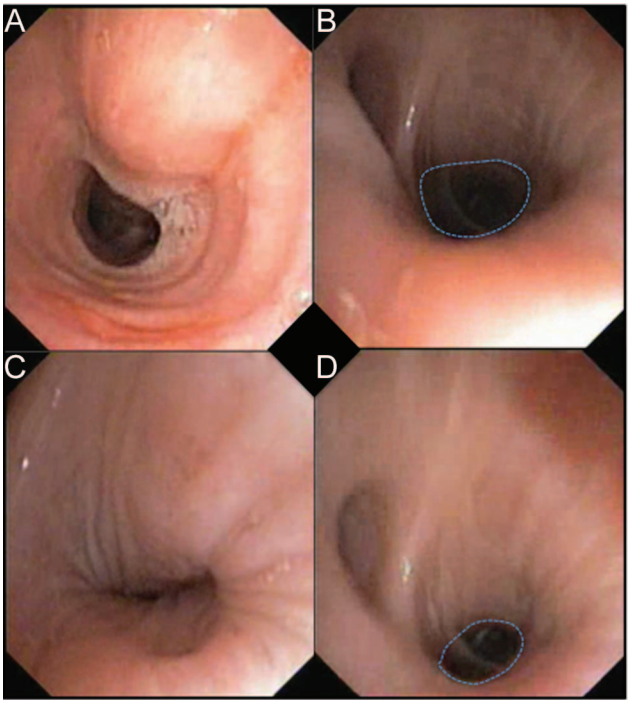

Fig. 1. Bronchoscopic images of the trachea. A: The beginning of the bulging of the posterior wall over its lumen in expiration. B: Intermediate bronchus in inspiration. C: Intermediate bronchus in expiration with complete luminal collapse. D: The diameter of the intermediate bronchus is shown in expiration with the application of CPAP.

The patient remained relatively stable for ~1 y, when she was again invasively ventilated in 2 successive admissions. Due to weaning difficulties, NIV with bi-level positive airway pressure was introduced after extubation. The patient became increasingly dependent on ventilatory support and gradually began to require continuous NIV with a full-face mask (aiming to prevent skin lesions). At this stage, the patient could not tolerate more than brief periods without NIV, just enough to feed herself. Due to the high degree of dependence on the ventilator and the continued clinical worsening, a tracheotomy was proposed. Her ventilator settings were adjusted according to the presence of a fenestrated inner cannula and cuff inflation. Thus, during the day using the fenestration and a deflated cuff, the parameters were: BiPAP S/T with AVAPS (tidal volume of 500 mL guaranteed), maximum and minimum inspiratory positive airway pressures of 18 and 13 cm H_2O, expiratory positive airway pressure of 8 cm H_2O, breathing frequency of 12 breaths/min, and inspiratory time of 1.1 s. At night, without the fenestra and with an inflated cuff, the parameters were: BiPAP S/T, inspiratory positive airway pressure of 14 cm H_2O, expiratory positive airway pressure of 8 cm H_2O, breathing frequency of 12 breaths/min, and inspiratory time of 1.1 s. Because the patient was tracheostomized, she had several microbial isolates in bronchial secretions. After a few months of stability, she was again admitted with a new episode of unstoppable cough and respiratory failure. New bronchoscopic examination revealed that the patient had increased tracheal collapsibility with cuff deflation. Keeping the cuff continuously inflated and sedating the patient were the only solutions for ventilation, ending the cough, and improving gas exchange. She finally died due to septic shock with nosocomial pneumonia ~5 y after diagnosis.

We want to emphasize the need for more research to elucidate the underlying pathogenesis and optimum management of dynamic airway collapse. As highlighted by the 2 cases, this is a condition with significant morbidity and mortality, and treatment

must be adapted to the severity and extension of the disease.

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