after patients attained spontaneous breathing and conditions for tracheal extubation were satisfied. No hypoxia or airway obstruction occurred.

2. Anesthetic method: In previous reports, T-tube implantation was performed with spontaneous breathing or mechanical ventilation under different anesthetic methods.[8,10] Although there are a few studies describing successful T-tube insertion while retaining spontaneous breathing, the obvious cough reflex and vigorous involuntary movements may complicate T-tube insertion and necessitate administration of muscle relaxants.[11] We chose RB because of its advantages, such as airway control and provision for HFV, which can help avoid hypoxemia, and availability of operation channels convenient for using forceps to assist placement of T-tube. During this period, the stimulation caused by RB is sufficiently serious that it cannot be suppressed by sedation with topical anesthesia, and the patients' obvious cough reflex and vigorous involuntary movements must be avoided, as they may complicate T-tube insertion. As the airway was open, we chose total intravenous anesthesia (TIVA), including muscle relaxants, instead of sedation and topical anesthesia. Based on observations from clinical practice, during T-tube insertion for tracheotomy patients with severe or complete subglottic stenosis, TIVA can help maintain an appropriate depth of anesthesia while avoiding intraoperative awareness and severe fluctuations of vital parameters, facilitate RB for HFV, and provide good operative conditions and avoid coughing.

The five patients all had stable vital parameters, depth of anesthesia, and no hypoxemia. This proves that during T-tube insertion for subglottic complete stenosis, TIVA can ensure anesthesia depth, and controlled ventilation or HFV by the tracheotomy tube, RB, T-tube, and LMA on demand can ensure oxygen supply. Thus, this approach is safe and effective.

Funding

This work was supported by a grant from Basic-Clinical Medicine (Tiantan project) Cooperation Fund of Capital Medical University [Grant number: 17JL(TTZX)06], Beijing, China. The funding agency had no role in the study design, the collection, analysis, or interpretation of data, the writing of the report, or the decision to submit the article for publication.

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https://doi.org/10.1016/j.arbres.2019.08.015

Adjusting the Level of Intervention in Patients with Chronic Obstructive Pulmonary Disease According to the Risk Stratification Proposed by the Spanish COPD Guidelines (GesEPOC) Version 2017☆

Adecuación del nivel de intervención en pacientes con enfermedad pulmonar obstructiva crónica (EPOC) según la estratificación de riesgo propuesta por la Guía española de la EPOC (GesEPOC) versión 2017

To the Editor:

The most recent update of the GesEPOC Spanish COPD guidelines recommends an integrated approach to classifying patients with chronic obstructive pulmonary disease (COPD) according to their level of risk, in order to make a multidimensional assessment in high-risk individuals.[1]

Multiple studies have shown that forced expiratory volume in 1 second (FEV_1) is a good predictor of morbidity and mortality,[2] although this parameter alone is not sufficient to correctly classify patients. For this reason, new composite indices have been created to improve the prognostic information provided by FEV_1.[3] The first version of GesEPOC, published in 2012, proposed an alternative to FEV_1 in the form of a new classification with 5 severity (or risk) levels, based essentially on the BODE and BODEx indices. However, its use in clinical practice was found to be very low.[4] For this reason, the new 2017 edition of GesEPOC responded to the need to simplify the risk stratification and proposed a new classification which is based on functional (percentage of FEV_1 after bronchodilation) and clinical (dyspnea grade measured by the modified Medical Research Council [mMRC] and exacerbations) criteria.

We conducted an observational study of the cohort of patients being followed up by the dedicated COPD clinic of the respiratory medicine department the Hospital Universitario de La Princesa, Madrid (Spain). The primary objective of the study was to char-

☆ Please cite this article as: Erro Iribarren M, et al. Adecuación del nivel de intervención en pacientes con enfermedad pulmonar obstructiva crónica (EPOC) según la estratificación de riesgo propuesta por la Guía española de la EPOC (GesEPOC) versión 2017. Arch Bronconeumol. 2020;56:183–185.

Table 1
Demographic and clinical characteristics according to GesEPOC risk level.

	Low risk n = 144 (33.5%)	High risk n = 286 (66.5%)	p
Sex, n (%)			0.791
Men	96 (66.7%)	187 (65.4%)	
Women	48 (33.3%)	99 (34.6%)	
Age, mean ± SD	69.79 ± 9.86	71.55 ± 9.54	0.076
BMI (kg/m²), mean ± SD	27.03 ± 5.25	26.21 ± 5.10	0.120
Smoking habit (PYI), mean ± SD	52.41 ± 26.83	56.86 ± 28.15	0.092
Charlson index, mean ± SD	4.17 ± 2.25	4.20 ± 2.26	0.880
FEV_1 (ml), mean ± SD	1705 ± 572	1165 ± 465	< 0.05
FEV_1%, mean ± SD	62.90 ± 16.59	44.61 ± 15.25	< 0.05
mMRC dyspnea grade, n (%)			< 0.05
0	28 (19.4%)	21 (7.4%)	
1	116 (80.5%)	47 (16.4%)	
2	–	122 (42.6%)	
3	–	75 (26.2%)	
4	–	21 (7.3%)	
No. of exacerbations, mean ± SD	0.47 ± 0.78	1.81 ± 1.79	< 0.05
BODE, mean ± SD	1.5 ± 1.4	4 ± 2.3	< 0.05
BODEx, mean ± SD	1.5 ± 1.3	3.9 ± 1.8	< 0.05
Treatment, n (%)			< 0.05
Monotherapy	45 (31.6%)	16 (5.6%)	
LAMA + LABA	51 (35.4%)	68 (23.7%)	
LABA + ICS	9 (6.3%)	11 (3.8%)	
LAMA + LABA + ICS	39 (27.1%)	189 (66.1%)	
Clinical phenotype, n (%)			< 0.05
Non-exacerbator	130 (90.3%)	117 (81.5%)	
Mixed	14 (9.7%)	25 (8.7%)	
Exacerbator with emphysema	–	79 (27.6%)	
Exacerbator with chronic bronchitis	–	65 (22.7%)	

BMI, body mass index; BODE: body mass index, airflow obstruction, dyspnea and exercise capacity; BODEx: body mass index, airflow obstruction, dyspnea and severe exacerbations; FEV_1: forced expiratory volume in 1 second; ICS: inhaled corticosteroids; LABA: long-acting β-2 adrenoceptor agonist; LAMA: long-acting muscarinic agonist; m: mean; mixed: asthma-COPD; mMRC: modified Medical Research Council; n: number; PYI: pack-year index; SD: standard deviation.

acterize these patients according to the new risk classification, and to evaluate the appropriateness of care and treatment based on the recommendations proposed by GesEPOC 2017.

We performed a descriptive statistical analysis (relative and absolute frequencies; means and standard deviations depending on the type of variable) using the SPSS statistical package version 22.0 (IBM, USA).

Our cohort comprised a total of 430 patients. Of these, 144 (33.5%) met the criteria for low risk and 286 (66.5%) were classified as high risk. In total, 57 (19.9%) of the high-risk population presented the 3 criteria, and 33 (11.5%) met a single criterion. The most common high-risk criterion was mMRC dyspnea grade ≥2, present in 76.2% of the patients. Twelve (4.2%) patients were classified as high risk due to presence of mMRC dyspnea grade ≥2 as a single criterion.

Demographic and clinical characteristics, and the distribution by clinical phenotype according to level of risk, are shown in Table 1. No statistically significant differences were found in our cohort between the 2 groups according to age, sex or comorbidities. All high-risk patients were classified according to their clinical phenotype, the exacerbator phenotype being the most common (50.3%) and the mixed phenotype the least common (8.7%), in line with findings from previous studies.[5–7]

High-risk patients presented a significantly higher number of exacerbations, more dyspnea, and worse lung function than low-risk individuals ($p < 0.05$), as was to be expected. These patients also had a higher level of severity according to the BODE and BODEx indices (4 ± 2.3 and 3.9 ± 1.8, respectively).

With regard to treatment by risk level, most high-risk patients were treated with triple therapy (long-acting muscarinic antagonist/long-acting ®-2 adrenoceptor agonist/inhaled corticosteroids [LAMA/LABA/ICS]), with no differences between exacerbators with chronic bronchitis vs. exacerbators with emphysema (66.1% vs. 67.1%), similar to reports from larger published series.[8,9] A total of 62.4% of high-risk non-exacerbators were also receiving triple therapy. Furthermore, despite the fact that in low-risk patients the most commonly used option was dual bronchodilation, 27.1% of the patients were receiving triple therapy. The percentage of low-risk COPD patients in our series who are receiving triple therapy cannot be fully justified by the frequency of mixed phenotype (9.7% of patients). As observed in other studies, this high number must correspond to overtreated patients.[10]

With regard to non-pharmacological treatment, 61.2% of high-risk patients in our cohort were included in a pulmonary rehabilitation program, 46.9% had chronic domiciliary oxygen therapy, and 22.2% used portable oxygen therapy.

In terms of the diagnostic process, 94.4% of low-risk patients and 93.7% of high-risk patients underwent spirometry with bronchodilator challenge for diagnosis and arterial oxygen saturation was evaluated in all patients. In contrast, alpha 1-antitrypsin deficiency determination was not requested in the vast majority of patients (23.3% of the low-risk group and 25.9% of the high-risk group). With regard to other more specific complementary tests, a CT scan of the chest was performed in 99.6%, a walk test in 80.4%, lung volumes in 87.1%, and diffusing capacity in 81.5% of the high-risk patients. Diagnostic tests were performed in our patients according to the recommendations of the GesEPOC 2017 guidelines, except for alpha 1-antitrypsin deficiency screening.

One of the limitations of our study is that all patients were from a single hospital cohort, so the results cannot be applied to the general COPD population, which may have more comorbidities and other factors associated with high risk.

In conclusion, most of the patients in follow-up in our dedicated clinic are high-risk and present an exacerbator phenotype. Patients are managed in line with the recommendations proposed by GesEPOC 2017, although there are significant areas for improvement with respect to alpha 1-antitrypsin deficiency screening and adherence to the recommendations for drug treatment.

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https://doi.org/10.1016/j.arbr.2019.09.005

Spontaneous Pneumomediastinum: Rare Complication of Tracheomalacia

Neumomediastino espontáneo: una complicación infrecuente de la traqueomalacia

Dear Editor:

We would like to commend Gutierrez-Morales et al. on their interesting case presentation of spontaneous pneumomediastinum in an asthmatic patient published in your esteemed journal.[1] We wish to extend the spectrum of this rare phenomenon presenting with perplexing clinical and radiological findings which make the management and decision making a challenging task.

A 62-year-old man in the medical ICU was being treated for alleged asthma exacerbation. He has had no past admissions for asthma exacerbation. On clinical examination his blood pressure was 114/86 mm HG with heart rate of 96 beats per minute. He was saturating 97% on 50% FiO_2 on bi-level non-invasive ventilation for his work of breathing. Chest auscultation revealed prolonged expiratory phase with shortness of breath worse in supine position. There was equal but diminished air entry on both sides with equal chest wall movement. There was no subcutaneous crepitus on palpitation. During treatment with non-invasive ventilation patient developed subcutaneous emphysema (extensive subcutaneous crepitus) and progressive shortness of breath requiring intubation and mechanical ventilation. Extensive subcutaneous crepitus was noted on palpation as well as on auscultation of the chest and precordium (Hammond's crunch).

Chest X-ray done at the time showed extensive subcutaneous emphysema. Due to progressive shortness of breath and hypoxia, patient was intubated and pharmacological paralysis was induced to aid effective ventilation. Computed tomography (CT) of the chest showed severe subcutaneous emphysema (Fig. 1A/B/C white arrows), moderate to severe pneumomediastinum (Fig. 1A/B/C red arrows) and moderate sized left basal pneumothorax (Fig. 1B yellow arrows). Interestingly reporting radiologist also noted complete collapse of the trachea distal to the endotracheal tube (ETT) (Fig. 1C green arrow) and suggested tracheal/bronchial rupture as a differential diagnosis in the current clinical context. However, there was no loss of peak or plateau airway pressures on the mechanical ventilator. At this point, patient's oxygen requirements had increased requiring 100% FiO2 for optimal oxygen saturation with hypotension with systolic blood pressure of 90 mm Hg. Urgent surgical consultation was sought and patient was anticipated to undergo extra corporeal membrane oxygenation (ECMO) prior to surgical correction of suspected tracheal rupture.

Patient underwent emergency bronchoscopy which showed collapsed trachea distal to the ETT tip classic for tracheomalacia (Fig. 1D blue arrows). Focused bronchoscopic examination until the second generation bronchi bilaterally did not reveal any airway wall rupture.

It was concluded that the patient most likely had tracheomalacia which was probably diagnosed as acute asthma exacerbation. The positive airway pressure ventilation most likely caused barotrauma and leading to pneumothorax, pneumomediastinum and extensive subcutaneous emphysema leading to hypoxic respiratory failure. Our patient subsequently underwent tube thoracostomy for left sided pneumothorax and conservative management for pneumomediastinum. He was extubated 7 days later and made complete recovery.

Spontaneous pneumomediastinum (SPM) is also known as Hamman's syndrome. SPM is a rare entity and is characterized by air leak into the mediastinum, not secondary to any underlying disease.[2] It was first described by Macklin as "*the transference of air along sheaths of pulmonic blood vessels from alveoli to mediastinum*" it can be shortened as follows: alveolar ruptures leading to air dissection along bronchovascular sheaths, and spreading into the mediastinum.[3] Hamman's syndrome is not a life-threatening condition and, once diagnosed, may require only supportive and symptomatic therapy, which includes oxygen, analgesics and sedatives as necessary, unless it is associated with tension pneumothorax and/or hemodynamic instability as noted in our patient.[4–7] Secondary pneumomediastinum can be differentiated from SPM when a causative factor is identified. It could be iatrogenic (endoscopic procedures, airway manipulation such as during endotracheal intubation, pleural cavity instrumentation, central venous access procedures, blunt or penetrating trauma, inhalation of toxic fumes etc.). Addressing the underlying cause usually should suffice and conservative management would be the choice of treatment. However, in rare cases the simple pneumomediastinum can progress to a malignant one and can lead to hemodynamic instability due to cardiac tamponade and occasionally airway compromise. In these cases video-assisted thoracoscopic surgery becomes an indispensable tool for decompression and attaining hemodynamic stability.

Due to its acute presentation and often other concomitant illnesses, spontaneous pneumomediastinum creates a cause of worry for the treating physicians. Quest for diagnosis and underlying cause is of prime importance, since it has an impact on overall prognosis and management plans. Chest X rays and CT scans are the investigative modalities of choice for a conclusive diagnosis.[8–10] Despite better visualization of structures on CT chest, there are instances where the source of free air in the mediastinum has been misdiagnosed. Brussa et al. described a case of spontaneous pneumomediastinum in a pregnant patient, CT scan of the chest was over-read as possible tracheal rupture but bronchoscopy was able to avert the unnecessary surgical exploration.[11] Bronchoscopy often provides precious diagnostic information in critically ill patients where the cause of hypoxia is elusive, such as lobar torsion after surgery, mucus plugging and lung collapse.[12] Similar mistake occurred in the radiological assessment of our patient which triggered a need for a major surgical intervention and possibly ECMO. In our case too, bronchoscopy was able to provide a more accurate diagnosis and eliminate the doubts of tracheal rupture. It is understandable though, that in a subset of critically ill patients, requiring high fraction of inspired oxygen and positive end expira-