

A Case of Ventricular Fibrillation in a Frail Patient with Chronic Obstructive Pulmonary Disease, Obstructive Sleep Apnea Syndrome, and Electrolytic Disorder

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Keywords

Chronic obstructive pulmonary disease · Ventricular fibrillation · Cardiovascular risk · Frailty · Obstructive sleep apnea syndrome · Electrolytic disorders

Abstract

Chronic obstructive pulmonary disease (COPD) is associated with an increased cardiovascular risk, although the pathophysiological mechanisms responsible for this interdependence are not completely known. For instance, the increased sympathetic activity may be implied. The severity of COPD correlates with various arrhythmic manifestations such as atrial fibrillation, atrial flutter, and either sustained or nonsustained ventricular tachycardia. COPD and obstructive sleep apnea syndrome may increase the overall cardiovascular risk, especially in elderly patients. Additionally, electrolytic disorders may precipitate cardiac rhythm disturbances and thus cause important arrhythmic consequences such as ventricular fibrillation, as reported in our clinical case. We discuss here the possible treatment of this association of pathological conditions on the basis of a single case we have successfully treated, and provide a brief review of the available literature regarding cardiovascular comorbidities in COPD patients.

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Introduction

The correlation between respiratory disease and increased cardiovascular risk, mainly secondary to a neurovegetative dystonia with orthosympathetic system imbalance, is well established [1]. Chronic obstructive pulmonary disease (COPD) is a common comorbidity complicating the hospital course of patients experiencing cardiovascular disorders, especially heart failure. Pharmacological strategies aim to reduce exacerbations and control symptoms in order to improve the overall quality of life of COPD patients.

We report our clinical experience of a case of ventricular fibrillation in a frail patient with COPD and electrolyte disorder secondary to gastroenteric disease and present a brief review of the available literature.

Case Description

An 82-year-old man came to our attention in March 2015 with diarrhea due to gastroenteritis which appeared resistant to pharmacological treatment. His medical history revealed several comorbidities, such as COPD, congestive heart failure secondary to ischemic heart disease, with chronic pleural effusion and perma-

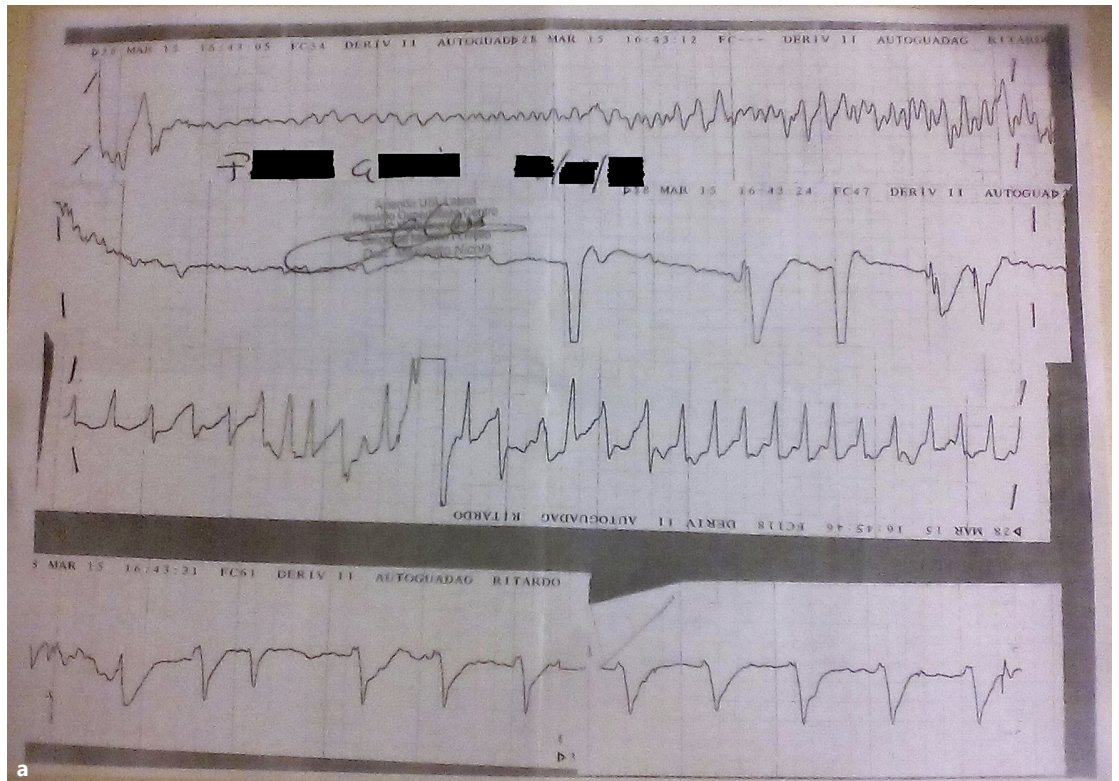


Fig. 1. a, b Continuous ECG trace during ventricular fibrillation and subsequent treatment with DC shock.

Table 1. Laboratory tests of the patient over the course of his hospitalization

Laboratory test	Emergency room	Internal medicine ward	Reference value
Potassium, mEq/L	2.3	3.9	3.5–5.2
Creatinine, mg/dL	3.25	2.92	0.5–1.1
pH	7.31	7.32	7.38–7.42
PCO ₂ , mm Hg	41.6	41.1	34–46
PO ₂ , mm Hg	67	167	80–100
HCO ₃ , mmol/L	18.5	24.3	22–26

ventricular fibrillation, chronic kidney disease, diabetes mellitus, and low-mobility syndrome.

Due to markedly reduced potassium plasma levels (Table 1), the patient received electrolyte supplementation with 40 mEq of KCl in saline solution twice per day; additionally, aerosol therapy with ipratropium bromide 15 drops, beclomethasone 15 drops, and salbutamol 15 drops b.i.d. was instituted; large-spectrum antibiotic therapy for intestinal disease and diarrhea (cephalosporin and quinolone) was initiated. During hospitalization, an episode of new-onset high-frequency arrhythmia was recorded, with the subsequent need for continuous electrocardiographic monitoring. The patient remained conscious, although the ECG showed ventricular fibrillation immediately treated with a double 360 J DC shock external defibrillation (the first shock was ineffective to restore a valid heart rhythm; Fig. 1). Amiodarone was thus initiated as antiarrhythmic prophylaxis and the patient was switched to the combination indacaterol/glycopyrronium, given its favorable safety profile [2]. Moreover, basal spirometry revealed a restrictive pattern, whereas polysomnographic examination showed obstructive sleep apnea syndrome with oxygen desaturation and concomitant supraventricular tachycardia. Of note, further cardiological investigations were planned in order to evaluate the feasibility of implanting an internal cardioverter defibrillator for secondary prevention of ventricular arrhythmias.

Discussion

The presence of several concomitant comorbidities makes it difficult to evaluate the real arrhythmic and cardiovascular risk of old patients with electrolytic disorders. In a recent paper, Konecny et al. [3] have highlighted the association of ventricular and supraventricular arrhythmic disturbances in COPD patients. Moreover, Men et al. [4] also underlined the importance of the association of heart conduction abnormalities (such as sinus bradycardia and supraventricular arrhythmias) and severe-to-moderate sleep apnea.

Cardiovascular comorbidities negatively impact the morbidity and mortality rates of COPD patients [5, 6], mainly due to the chronic proinflammatory state associated with these diseases, and the subsequent microcirculation disturbances [7, 8].

Long-acting beta-agonists (LABA) and long-acting muscarinic antagonists (LAMA) are useful in controlling COPD symptoms and prevent/reduce COPD exacerbations requiring hospital admission [9]. Recently, a new combination drug formed of a LABA (indacaterol) and a LAMA (glycopyrronium) in a fixed association has been developed. This drug has demonstrated a synergistic effect of the two molecules in improving bronchodilation [10], and moreover it also seems to be safe in patients with cardiovascular comorbidities. No cardiovascular events were recorded in the SHINE study, evaluating this drug combination versus both each single drug compound and placebo, over a 26-week follow-up period [11].

Conclusion

COPD and obstructive sleep apnea may increase the cardiovascular risk profile of elderly patients. Moreover, electrolytic disorders may further raise the arrhythmic rate and thus precipitate a long-standing unstable condition in a frail patient with several comorbidities.

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Financial Disclosure and Conflicts of Interest

The authors have no conflicts of interest to declare.

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