Chronic obstructive pulmonary disease and atrial fibrillation: An unknown relationship

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A B S T R A C T

Chronic obstructive pulmonary disease (COPD) is independently associated with atrial fibrillation (AF). Decreased oxygenation, hypercapnia, pulmonary hypertension, diastolic dysfunction, oxidative stress, inflammation, changes in atrial size by altered respiratory physiology, increased arrhythmogenicity from nonpulmonary vein foci commonly located in the right atrium, and respiratory drugs have been implicated in the pathogenesis of AF in COPD. The understanding of the relationship between COPD and AF is of particular importance, as the presence of the arrhythmia has significant impact on mortality, especially in COPD exacerbations. On the other hand, COPD in AF is associated with AF progression, success of cardioversion, recurrence of AF after catheter ablation, and increased cardiovascular and all-cause mortality. Treatment of the underlying pulmonary disease and correction of hypoxia and acid-base imbalance represents first-line therapy for COPD patients who develop AF. Cardioselective β-blockers are safe and can be routinely used in COPD. In addition, AF ablation was proved to be efficient and safe, and improves quality of life in these patients. This review presents the association between COPD and AF, describes the pathophysiological mechanisms implicated in AF development in COPD, underlines the prognostic significance of AF in COPD patients and vice versa, and highlights emerging therapeutic approaches in this setting.

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Introduction

Chronic obstructive pulmonary disease (COPD) is a leading cause of morbidity and mortality worldwide [1]. The overall prevalence in men aged 30 years or more is 14.3% (95% CI: 13.3–15.3%) compared to 7.6% (95% CI: 7.0–8.2%) in women [2]. COPD is currently rated the fourth most common specific cause of death globally and predicted to be the third by 2030, in the absence of interventions that address the risks – especially tobacco smoking, exposures to combustion products of biomass fuels, and environmental pollution [2]. Atrial fibrillation (AF) is the commonest arrhythmia in clinical practice and is associated with increased cardiovascular morbidity and mortality [3,4]. COPD has been independently associated with AF [5–16], but the precise pathophysiological mechanisms are complex and not completely understood. The presence of AF in COPD patients impairs prognosis, while the presence of COPD in AF patients also seems

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to have a specific impact on AF progression, success of cardioversion, ablation outcomes, and mortality. Specific medications and therapeutic implications may improve prognosis in this population. The present review provides a concise overview of the association between COPD and AF, and describes the arrhythmogenic mechanisms in this setting. Prognosis, as well as therapeutic implications, is also discussed.

Chronic obstructive pulmonary disease and atrial fibrillation

An accumulating body of evidence indicates that COPD is associated with AF [5–16]. The Copenhagen City Heart Study reported that reduced forced expiratory volume in one second (FEV1) is an independent predictor of AF onset [5]. In the ARIC study, after multivariable adjustment for traditional cardiovascular disease risk factors and height, hazard ratios (HRs) of AF comparing the lowest with the highest quartile of FEV1 were 1.37 (95% CI: 1.02–1.83) for white women, 1.49 (95% CI: 1.16–1.91) for white men, 1.63 (95% CI: 1.00–2.66) for black women, and 2.36 (95% CI: 1.30–4.29) for black men [9]. The Malmo Preventive Project, the largest to date population-based cohort study reported that after adjustment for age, height, weight, current smoking status, systolic blood pressure, erythrocyte sedimentation rate, and fasting blood glucose, FEV1 was inversely related to incidence of AF [HR: 1.39 (95% confidence interval [CI]: 1.16–1.68; p = 0.001) for women, and HR: 1.20 (95% CI: 1.13–1.29; p < 0.0001) for men] [10]. Forced vital capacity (FVC) was also inversely related to incidence of AF [HR: 1.20 (95% CI: 1.03–1.41; p = 0.020) for women, and HR: 1.08 (95% CI: 1.02–1.14; p = 0.01) for men] [10].

Pathophysiological mechanisms implicating chronic obstructive pulmonary disease in atrial fibrillation

Studies investigating the effects of hypoxia on atrial electrophysiology have yielded divergent results. In experimental models, Krause et al. observed no atrial electrophysiologic effects and no increase in AF vulnerability in the setting of profound hypoxemia [17]. Similar findings were reported by Stevenson et al. who failed to show an effect of hypoxia on either atrial refractoriness or atrial conduc tion. Their findings point against a direct electrophysiologic

Table 1

<table>
<thead>
<tr>
<th>Author (year)</th>
<th>No. of pts</th>
<th>Type of study</th>
<th>Follow-up</th>
<th>Results/findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Buch et al. [5] (2003)</td>
<td>13,430</td>
<td>Prospective</td>
<td>5 yrs</td>
<td>Risk of new AF at re-examination was 1.8-times higher for FEV1 between 60–80% of predicted compared with FEV1 ≥80% after adjustment for sex, age, smoking, blood pressure, diabetes and body mass index</td>
</tr>
<tr>
<td>Mapel et al. [7] (2005)</td>
<td>70,679</td>
<td>Retrospective</td>
<td></td>
<td>Among COPD patients, the prevalence of AF was very high (14.3%) and significantly higher than among the matched non-COPD cohort (10.4%; p = 0.001)</td>
</tr>
<tr>
<td>Shibata et al. [8] (2011)</td>
<td>2917</td>
<td>Prospective</td>
<td>1 yr</td>
<td>FEV1 and FVC are independent risk factors for AF (independent of age, gender, left ventricular hypertrophy and serum levels of B-type natriuretic peptide)</td>
</tr>
<tr>
<td>Johnson et al. [10] (2014)</td>
<td>29,744</td>
<td>Prospective</td>
<td>24.8 yrs</td>
<td>FEV1 was inversely related to incidence of AF [HR: 1.39 (95% CI: 1.16–1.68; p = 0.001) for women, and HR: 1.20 (95% CI: 1.13–1.29; p &lt; 0.0001) for men]. FVC was also inversely related to incidence of AF [HR: 1.20 (95% CI: 1.03–1.41; p = 0.020) for women, and HR: 1.08 (95% CI: 1.02–1.14; p = 0.01) for men]</td>
</tr>
<tr>
<td>Li et al. [9] (2014)</td>
<td>15,004</td>
<td>Prospective</td>
<td>17.5 yrs</td>
<td>After multivariable adjustment for traditional cardiovascular disease risk factors and height, HRs of AF comparing the lowest with the highest quartile of FEV1 were 1.37 (95% CI: 1.02–1.83) for white women, 1.49 (95% CI: 1.16–1.91) for white men, 1.63 (95% CI: 1.00–2.66) for black women, and 2.36 (95% CI: 1.30–4.25) for black men. Moderate/severe airflow obstruction (FEV1/FVC &lt; 0.70 and FEV1 &lt; 80% of predicted value) was also associated with higher AF incidence</td>
</tr>
<tr>
<td>Konceny et al. [12] (2014)</td>
<td>7,441</td>
<td>Retrospective</td>
<td>9 yrs</td>
<td>The presence and severity of COPD were associated with increased likelihood of AF/AFL (23.3% vs. 11.0%, respectively, p &lt; 0.0001), compared to pts without COPD. COPD remained a significant predictor of AF/AFL (p = 0.001) after adjusting for age, gender, tobacco use, obesity, hypertension, coronary artery disease, heart failure, diabetes, anemia, cancer, chronic kidney disease, and rate/rhythm control medications</td>
</tr>
<tr>
<td>Terzano et al. [16] (2014)</td>
<td>193</td>
<td>Prospective</td>
<td>1 yr</td>
<td>In pts with COPD exacerbations, the prevalence of AF was higher with lower FEV1 (57.1% vs. 7.6% vs. 0.3%; p &lt; 0.05). In addition, AF was more frequent in pts with higher values of PCO2 (70.6 mmHg vs. 5.3 vs. 5.01 mmHg vs. 3.5; p &lt; 0.05) and higher values of PAP (45.3 mmHg vs. 3.5 vs. 35.2 mmHg vs. 2.3; p &lt; 0.05)</td>
</tr>
<tr>
<td>Chahal et al. [11] (2015)</td>
<td>6,814</td>
<td>Prospective</td>
<td>4.1 yrs</td>
<td>Lower levels of FEV1 and FVC were associated with a higher risk for AF (HRs 1.21 and 1.19 per 500 ml, respectively, p &lt; 0.001) after adjustment for demographic and cardiovascular risk factors</td>
</tr>
<tr>
<td>Mita et al. [15] (2016)</td>
<td>116</td>
<td>Prospective</td>
<td></td>
<td>Multivariate analysis revealed COPD as risk factor for POAF</td>
</tr>
<tr>
<td>Geçmen et al. [13] (2016)</td>
<td>94</td>
<td>Prospective</td>
<td></td>
<td>To evaluate the value of SYNTAX score to predict POAF in pts undergoing CABG. An independent association was identified with COPD and POAF [β: 2.222; p: 0.003; OR: 9.228, 95% CI (2.150–39.602)]</td>
</tr>
<tr>
<td>Cerit et al. [14] (2016)</td>
<td>106</td>
<td>Retrospective</td>
<td></td>
<td>To assess the relationship between SYNTAX score and development of AF after CABG. In logistic regression analysis COPD [OR = 19.313, 95% CI: 2.416–154.407; p = 0.005] appeared as an independent variable [OR = 20.005] predicting the development of POAF</td>
</tr>
</tbody>
</table>

COPD, chronic obstructive pulmonary disease; AF, atrial fibrillation; FEV1, forced expiratory volume in one second; FVC, forced vital capacity; POAF, postoperative atrial fibrillation; SYNTAX, synergy between percutaneous coronary intervention with Taxus and cardiac surgery (scoring system evaluating the complexity of the lesions in coronary angiography); CABG, coronary artery bypass grafting; HR, hazard ratio; OR, odds ratio; CI, confidence interval; AFL, atrial flutter; pts, patients.
effect of hypoxia, but an indirect effect on arrhythmogenesis through sympathetic activation cannot be excluded [18]. On the other hand, Lamers et al. reported that in isolated superfused rabbits, the higher vulnerability of the atrium for reentrant arrhythmias by hypoxia is based on a combination of a moderate shortening of the wavelength and an increase in inhomogeneity in conduction of premature wavefronts [19]. Hypoxia-induced vascular endothelial growth factor (VEGF) expression is strongly regulated by hypoxia-inducible factor-1α (HIF-1α), the transcriptional factor for VEGF, which is a critical modulator for sensing and responding to changes in the oxygen concentration [20]. MMP-9 expression increases in fibrillating atria, and may contribute to atrial structural remodeling of AF [20]. It is possible that upregulation of HIF-1α/VEGF is involved in the enhancement of MMP-9 expression under hypoxic conditions [20]. Xu et al. reported increased levels of Toll-like receptor 2 (TLR2), HIF-1α, and MMP-9 in patients with persistent and permanent AF, and suggested that TLR2 and HIF-1α may promote left atrial structural remodeling [21]. In the same line, Su et al. reported that HIF-1α promotes the expression of TGF-β1 and MMP-9 protein, and thus is involved in atrial fibrosis [22]. QTc was also found to be prolonged in COPD patients compared to controls [23,24]. Sievi et al. reported that one third of a typical COPD population has altered cardiac repolarization and is at risk for increased arrhythmogenicity [25]. Several mechanisms may be related to hypoxia [24]. The ARIC study examined prolonged QT interval corrected by using the Framingham formula (QTc) as a predictor of incident AF, and showed that prolonged QTc predicts a roughly 2-fold increased risk of AF [25]. QTc interval reflects the atrial effective refractory period (AERP), suggesting that QTc interval may be used as a marker of atrial refractoriness relevant to assessing AF risk and mechanism-specific therapeutic strategies [26].

Hypercapnia has also been implicated in AF occurrence in COPD. Hypercapnia produces a marked and uniform increase in atrial refractoriness and a significant slowing in atrial conduction [18]. Return of CO₂ to normal values in rapid return of refractoriness to baseline levels but conduction slowing persists, suggesting that hypercapnia may create the substrate for AF after correction [18]. Moreover, hypercapnia and hypoxemia produce pulmonary arteriolar constriction leading to pulmonary arterial and right ventricular hypertension. Right ventricular hypertension may induce arrhythmias by leading to right atrial dilatation and increasing transmural pressure on endocardial vessels altering the distribution of blood flow [16]. In COPD exacerbations, AF is more frequent with higher levels of PaCO₂ and higher values of pulmonary artery systolic pressure [16].

The notion that prolonged and inhomogeneous propagation of depolarization could be present in the atria of patients with COPD has recently emerged. Right atrial electromechanical delay is significantly prolonged and negatively correlated with FEV₁, while the duration of atrial depolarization is significantly prolonged in COPD patients [27]. In a recent study, Acar et al. showed that prolongation of atrial electromechanical delay measured from lateral tricuspid annulus is independently related with FEV₁/FVC ratio [28]. P wave dispersion (PWD) – the difference in maximum and minimum duration of the P wave – was also found to be an independent risk factor for AF development [29]. PWD was more increased in the acute phase than in stable phase and was greater in patients with more frequent exacerbations suggesting that the PWD could be a target for prediction, prevention, and therapy of acute exacerbations of COPD [30]. P-wave duration and PQ interval were also reported to be risk factors for AF development in patients with P pulmonale, and P-wave duration is highly correlated with PQ interval >150 ms [31]. On the contrary, Buzea et al. reported that in patients with acute COPD exacerbations, P-wave signal averaged electrocardiography (SAECG) analysis and atrial late potentials detection seem to have little value in the arrhythmic risk evaluation [32].

COPD contributes to ventricular diastolic dysfunction [33–35]. Left ventricular diastolic dysfunction is associated with disease severity, and may serve as another possible pathophysiologic mechanism for AF initiation and perpetuation [33–35]. In addition, right ventricular systolic dysfunction and elevated systolic pulmonary artery pressure are independently associated with right atrium volume index in patients with pulmonary hypertension due to COPD [36]. This increase in right atrium volume index possibly promotes AF [36]. Furthermore, hemodynamic overloading or stretching of the right atrium due to pulmonary hypertension may contribute to a higher prevalence of non-pulmonary vein foci from the right atrium in chronic lung disease patients [37].

Oxidative stress and inflammation represent major pathogenetic mechanisms in COPD [38,39], but have been implicated in AF initiation and perpetuation as well [40,41]. It is therefore tempting to speculate that COPD-related oxidative and inflammatory responses may promote AF development. Regarding inflammation, the C allele of polymorphic marker GI−174C of interleukin-6 gene has been considered to be independently associated with development of AF in patients with COPD [42].

Respiratory drugs and atrial fibrillation

Respiratory drugs have been shown to precipitate AF. The new use of short- and long-acting β-agonists may slightly increase the risk of AF in COPD [43]. β-Agonist medications have a potential for arrhythmogenicity because of effects on chronotropy, depolarization, repolarization, and cellular potassium distribution mediated through β-adrenergic receptors [44,45]. A single, regular dose of inhaled salbutamol was reported to enhance AV nodal conduction reflected by shortening of the AH interval and decreased atrial refractoriness [45]. The shortening of atrial refractoriness may facilitate AF induction and explain the increased incidence of the arrhythmia in COPD [46].

Anticholinergic drugs suppress parasympathetic control of heart rate, which is associated with an increased incidence of tachyarrhythmias [47]. The Lung Health Study investigators reported a relative risk for hospitalizations due to supraventricular tachycardia of 4.5 (95% CI: 0.97–20.8) associated with use of short-acting inhaled ipratropium versus placebo [48]. Ogale et al. also reported an elevated risk of dysrhythmia associated with the use of ipratropium [49], however these findings were not substantiated in the randomized controlled trials of tiotropium [50–53].

Methylxanthine agents, such as theophylline and aminophylline, have also been shown to precipitate AF [54]. The responsible mechanisms include depolarization effects and electrolyte depletion with increased excretion of potassium and magnesium through the urine [55]. Theophylline causes significant reductions in AV and His–Purkinje conduction intervals, sinotriatal conduction time, corrected sinus node recovery time, shortest atrial pacing interval producing 1:1 AV conduction and atrial effective refractory period (ERP), but does not affect intraatrial conduction interval or ventricular ERP [56]. In a case control study, short-term use of theophylline was associated with increased risk of AF [54]. Doxofylline has similar efficacy to theophylline, but appears to have a better safety profile [57].

Current glucocorticoid use was associated with an almost 2-fold increased risk of AF [58]. Despite the fact that no increased risk of arrhythmias overall was found among users of inhaled steroids, oral steroids were associated with AF [54]. Several mechanisms seem to be involved in the development of AF in patients treated with corticosteroids. Long-term glucocorticoid use has been associated with increased risk of atherosclerosis, hypertension,
diabetes mellitus, left atrial enlargement, heart failure, and ischemic heart disease, conditions that are well-known risk factors for AF [58]. It has also been postulated that high-dose corticosteroids mediate potassium efflux via a direct effect on the cell membrane inducing arrhythmias [59]. Moreover, the increased AF risk, especially in new high-dose users, suggests that corticosteroids may have a direct arrhythmogenic effect [59].

The pathophysiological mechanisms implicating COPD in AF are presented in Fig. 1.

**Prognostic significance of atrial fibrillation in chronic obstructive pulmonary disease and vice versa**

It is of utmost importance to take into account the incidence of AF in COPD patients. BODE Index is a multidimensional grading system that evaluates body mass index, measure of airflow obstruction, dyspnea score, and exercise capacity in COPD patients, and predicts mortality and long-term outcomes [60]. Subjects with higher scores in BODE index have a significantly greater prevalence of arrhythmias, namely supraventricular tachycardia, AF and atrial flutter [60]. Increased BODE index has been associated with impaired left ventricular and right ventricular mechanics in patients with COPD [61,62]. Five strongest predictors of mortality (extended MRC Dyspnoea Score, ecosinopenia, consolidation, acidemia, and AF) were combined to form the Dyspnoea, Ecosinopenia, Consolidation, Acidaemia, and atrial Fibrillation (DECAF) Score in COPD patients [63]. DECAF Score shows excellent discrimination for mortality and has performed more strongly than other clinical prediction tools [63].

On the other hand, the presence of COPD in AF patients seems to have a specific impact on AF progression, success of cardioversion, ablation outcomes, and mortality. Hypertension, age, previous stroke or transient ischemic attack, COPD, and heart failure are independent predictors of AF progression [64]. Based on these parameters, de Vos et al. developed the HATCH score (an acronym for Hypertension, Age, previous Transient ischemic attack or stroke, COPD, and Heart failure), that independently predicts AF progression [64]. As the HATCH score correlates with the occurrence of AF, it may be used to select patients for intensified ECG monitoring [65]. HATCH score can also be used as predictor of new-onset AF after typical atrial flutter ablation [66] or after coronary artery bypass grafting [67], and is useful in predicting short-term success of AF electrical cardioversion at early stages [68]. Notably, the score can be used for stroke risk assessment, as none of the patients with a low HATCH score was proved to suffer a stroke [65]. In a recent study, however, Kochhäuser et al. reported that HATCH score is a poor predictor of AF progression, whereas left atrial diameter of more than 45 mm and heart failure are strong and independent predictors [69]. The Euro Heart Survey on AF aimed to provide insight into the present-day cardioversion of AF in “real life” patients and identify predictors for both immediate and long-term success. Absence of COPD predicts successful electrical cardioversion and sinus rhythm maintenance at 1-year follow-up [70]. Studies have also evaluated the efficacy and safety of managing AF with radiofrequency catheter ablation in COPD patients. Roh et al. investigated the electroanatomic alterations in pulmonary veins in chronic lung disease patients with AF and assessed their effect on the outcomes of catheter ablation [37]. They reported that catheter ablation has a comparable success rate to that of patients with normal lung function [37]. In another study, Gu et al. aimed to investigate the impact of COPD on outcomes of catheter ablation in patients with AF in terms of recurrence and quality of life [71]. Despite the fact that the presence of COPD predicted higher recurrence after single-catheter ablation in AF patients, significant improvements in quality of life were observed in the postablation COPD population [71]. The presence of COPD may also increase the risk of ischemic stroke in subjects with AF [72]. Nadeem et al. reported that AF plus COPD is a stronger predictor of ischemic cerebrovascular accident than AF only or COPD only [72]. In the EURObservational Research Programme-ATRial Fibrillation General Registry Pilot Phase (EORP-AF Pilot), COPD was proved to be highly prevalent in European AF patients, and is associated with higher rates of cardiovascular death, all-cause death, and the composite outcome of any thromboembolic event/bleeding/cardiovascular death [73]. Similarly, Huang et al. reported that the presence of COPD in patients with AF is an independent risk factor for 1-year all-cause and cardiovascular mortality [74]. In addition, data from the ROCKET AF [75] and the ARISTOTLE trial [76] showed that COPD is strongly associated with all-cause mortality in AF. The Arrhythmic Fibrillation in the Emergency Room (AFER) Study, sought to derive and validate a complex and a simplified model that predicts mortality in emergency department patients with AF [77]. Both the complex and simplified instrument that included COPD as a risk variable have been shown to predict mortality after an emergency visit for AF [77].
Antiarrhythmic therapy and interventions for atrial fibrillation in chronic obstructive pulmonary disease

Treatment of COPD in patients with concomitant AF should be the same as those without AF [78]. Available evidence suggests that an overall acceptable safety profile for using long-acting β-agonists, anticholinergics, and inhaled corticosteroids [79,80]. On the contrary, caution is advised when using short-acting β-agonists [45,81] and theophylline [82,83], which may precipitate AF and worsen ventricular rate control.

In patients with COPD who develop AF, treatment of the underlying pulmonary disease and correction of hypoxia and acid-base imbalance are of primary importance and represent first-line therapy [84]. Propafenone and adenosine should be used with caution in patients with significant bronchospasm, while they can safely be used in patients with COPD [84]. Diltiazem and verapamil are often tolerated and effective [84]. Amiodarone [85] or digoxin in combination with calcium channel blockers [86] can also be used. Early prophylactic amiodarone administration in patients with COPD subjected to coronary artery bypass grafting has been shown to reduce AF [87]. The use of β-blockers in patients with COPD and co-morbid cardiovascular disease has been controversial, and as a result they are under-prescribed despite increasing evidence to support their use as safe and efficacious [88]. Current evidence supports that COPD is not a contraindication for cardioselective β-blocker therapy [89,90]. β-Blocker use in patients with COPD may not decrease the risk of overall mortality, but also reduce the risk of COPD exacerbations [91]. Direct-current cardioversion should be attempted in patients with pulmonary disease who become hemodynamically unstable as a consequence of new-onset AF [92]. In patients refractory to drug therapy, AV nodal ablation and ventricular pacing may be considered in order to control ventricular rate [92]. AF ablation was proved to be efficient and safe, and improves quality of life in COPD [71,93]. However, more studies with large sample size and long-term follow-up are needed to evaluate the efficacy and safety in managing AF with catheter ablation, as well as with different types of operation in COPD [94].

Conclusion

An increasing body of evidence indicates that COPD is associated with AF. The presence of AF in COPD has significant impact on mortality, while the presence of COPD in AF has a specific impact on AF progression, success of cardioversion, ablation outcomes, and mortality. A complete elucidation of the pathophysiological mechanisms implicating COPD in AF, will offer the possibility for better therapeutic options and reduction of mortality in these patients. Treatment of the underlying pulmonary disease and correction of hypoxia and acid-base imbalance represents first-line therapy for COPD patients who develop AF. β-Blockers have been traditionally contraindicated in COPD, however, recent evidence suggests that cardioselective β-blockers are safe and reduce risk of atrial fibrillation and mortality. AF ablation can be performed safely in COPD and improves quality of life, but large studies are missing.

Conflict of interest

None.

References


