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ORIGINAL RESEARCH

Clinical Characteristics and Factors Associated with One-Year Smoking Cessation Among Smokers with COPD: A Subgroup Analysis of a Multicentre Randomised Trial

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Background: Smoking cessation is the most effective intervention for modifying the clinical course of chronic obstructive pulmonary disease (COPD), yet many patients continue to smoke after diagnosis. We evaluated the characteristics associated with cessation at one year among smokers with COPD in a multicentre randomised cessation trial.

Methods: This prespecified subgroup analysis included patients with COPD randomised to immediate referral to a smoking cessation clinic or to usual care. Smoking status one year later was determined through a structured telephone interview, and patients who had quit were contacted and their cessation was verified by exhaled carbon monoxide measurement; 48 of the 220 patients (21.8%) were lost to follow-up, leaving 172 patients for analysis. A prespecified six-variable logistic regression model was fitted; Firth penalised regression and an analysis classifying patients lost to follow-up as continuing smokers were prespecified sensitivity analyses.

Results: Of 172 patients, 32 (18.6%) had quit at one year: 24 of 93 (25.8%) allocated to immediate referral versus 8 of 79 (10.1%) allocated to usual care (p = 0.010). Quitters were older and had a lower baseline FEV1 in unadjusted comparisons. In the multivariable model, usual care was associated with lower odds of cessation (OR, 0.25; 95% CI, 0.09–0.64), whereas absence of comorbidity (OR, 2.64; 95% CI, 1.06–6.60) and more than 1 exacerbation-related hospitalisation (OR, 9.32; 95% CI, 1.97–44.06) were associated with higher odds; the latter estimate rests on 10 patients and is imprecise. Baseline FEV1 and Fagerström score were not independently associated with cessation.

Conclusion: Immediate referral to a cessation clinic was the factor most strongly associated with one-year abstinence, together with absence of comorbidity and, tentatively, repeated hospitalisation; greater disease severity was associated with quitting only in unadjusted comparisons. These exploratory findings support structured cessation pathways in routine COPD care.

Keywords: smoking cessation, chronic obstructive pulmonary disease, intervention, comorbidities, hospitalisation

Introduction

Chronic obstructive pulmonary disease (COPD) remains a major cause of morbidity and mortality worldwide, and cigarette smoking is its dominant preventable cause. Continued tobacco exposure after diagnosis accelerates the decline in lung function and increases symptom burden, exacerbation risk, and health care use.[1–4] Smoking cessation is the most

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effective way to alter the natural history of the disease: unlike inhaled therapies, which chiefly relieve symptoms and reduce exacerbations, cessation slows disease progression by removing ongoing airway and systemic injury, and is associated with slower lung-function decline, fewer exacerbations, and lower mortality.[3,4]

Despite these benefits, many patients keep smoking after a COPD diagnosis. Persistent smoking in this population has been linked to high nicotine dependence, psychological factors, socioeconomic disadvantage, and insufficient access to evidence-based cessation support,[5] and recent work has emphasised the gap between guideline recommendations and the cessation support that COPD patients actually receive.[6]

Health-system interventions can narrow that gap. In a multicentre randomised trial of patients with chronic airway disease who smoke, immediate scheduling of a smoking cessation clinic appointment improved clinic attendance and increased quit rates, an effect sustained at 12 months.[7–9] How individual patient factors relate to long-term cessation in COPD, however, is less well understood, and existing prediction models remain preliminary.[10] Disease severity, nicotine dependence, comorbidity burden, and previous quit experience may all influence success, and acute exacerbations or hospitalisations may act as teachable moments that increase readiness to quit.[11]

The parent trial reported cessation outcomes for the whole chronic airway disease cohort and did not examine COPD-specific characteristics.[9] That distinction matters clinically, because patients with COPD differ from other smokers with airway disease in age, cumulative tobacco exposure, comorbidity burden, and frequency of health care contact, and because cessation advice in COPD is delivered in the context of an established, progressive diagnosis. Which patients with COPD benefit from a proactive referral pathway, and which remain refractory to it, therefore cannot be inferred from the pooled results.

We therefore undertook this prespecified subgroup analysis to evaluate the demographic, smoking-related, clinical, and treatment characteristics associated with successful smoking cessation at 1 year among patients with COPD enrolled in the trial. Disease severity, nicotine dependence, comorbidity, and exacerbation-related hospitalisation were specified in advance as the candidate factors of primary interest, together with allocated study group and age.

Methods

Study Design and Ethical Approval

This was a prespecified subgroup analysis of a multicentre randomised trial evaluating immediate smoking cessation support during routine outpatient care for patients with chronic airway disease who actively smoke.[7–9] The subgroup of interest (patients with COPD) and the candidate factors examined in the multivariable model were defined before the present analysis was undertaken; no further variable selection was performed on the basis of the observed results. The parent trial first reported the short-term (one-week) outcomes of immediate appointment scheduling to smoking cessation clinics,[7] while subsequent publications reported the 3-month and 12-month smoking cessation outcomes in the overall chronic airway disease cohort.[8,9] The present manuscript differs from these previous reports in that it focuses exclusively on patients with COPD and presents COPD-specific clinical characteristics, treatment patterns of smoker patients, and factors associated with successful smoking cessation that were not included in the previous publications. Ethical approval for this analysis was obtained from the Ethics Committee for Non-Interventional Clinical Trials, Kutahya Health Sciences University (approval No. 2026/02-58; 02/09/2026). The parent trial and its ethical approval have been described previously.[7–9] The present analysis used anonymized existing data and involved no additional intervention, and was conducted in accordance with the Declaration of Helsinki.

Study Population

Patients attending chest diseases outpatient clinics between November 2022 and June 2023 were screened. Eligible patients had physician-diagnosed COPD of at least 6 months' duration, confirmed by post-bronchodilator spirometry showing a FEV1/FVC ratio below 0.70 in accordance with GOLD criteria,[1] were actively smoking cigarettes at baseline, and had baseline and follow-up data available. Eligibility was based on current cigarette smoking; exclusive users of other tobacco products or electronic cigarettes were not enrolled, although concurrent use of other tobacco products was recorded at follow-up.

Written informed consent had been obtained from all participants during enrolment into the parent randomised trial, including consent for the scientific analysis of anonymised data. No additional informed consent was required for this secondary subgroup analysis. The parent protocol is registered at ClinicalTrials.gov (NCT05764343);[8] in brief, adults with chronic airway disease who smoked were randomised to usual support or to immediate cessation support. The allocation recorded at randomisation was used for all analyses reported here.

Smoking Cessation Care Context

The content of care in each arm was standardised across participating centres by a common written protocol. All patients, in both arms, received brief cessation advice from the treating chest physician during the routine visit, together with information on locally available cessation services, and were encouraged to book an appointment at a dedicated smoking cessation clinic through the national appointment system; this constituted usual support. Patients allocated to immediate cessation support received the same brief advice plus an appointment at a smoking cessation clinic that was scheduled during the index visit by the study team. In Türkiye, smoking cessation clinics operate under a single national programme, so behavioural counselling and pharmacological treatment (varenicline, bupropion, or nicotine replacement therapy) were delivered according to the same national protocol and were accessible free of charge at every participating centre. No cessation treatment was administered by the study team itself.

Follow-Up and Outcome Definitions

One-year follow-up was undertaken by telephone between May 2 and June 15, 2024, using a structured questionnaire administered by trained investigators. Patients were asked whether they had made a quit attempt, whether they were smoking at the time of the call, how many cigarettes they currently smoked per day, whether they had used any other tobacco product, and, if they had stopped, the date of the quit attempt and the number of months since their last cigarette. The target quit date was the date on which the patient made the quit attempt that followed the index visit, whether or not a cessation clinic was attended; it was not the date of randomisation. Successful cessation was defined as complete abstinence from the target quit date until the one-year call. Any resumption of smoking, including a small number of cigarettes or a brief period of smoking, was classified as non-cessation: 32 patients (18.6%) reported a quit attempt followed by relapse (median abstinence before relapse, 60 days; interquartile range, 29–128 days) and were counted as non-quitters, as were the 108 patients who never stopped. Smoking status was based on the participants' self-reports. Participants who reported that they had not smoked at all since their target quit dates were invited to the clinic for biochemical verification via an exhaled carbon monoxide (CO) test; a CO level below 7 ppm was accepted as confirmation of abstinence from smoking. The measurement was completed in all 32 patients who reported having quit, and all measured values were 3 ppm or below. Patients lost to follow-up were excluded from the primary analysis and were addressed in a sensitivity analysis (below).

Study Outcomes

The primary outcome was the comparison of clinical characteristics according to smoking cessation status at 1 year. Secondary outcomes included exacerbation frequency, emergency department use, symptom burden, treatment patterns and adherence, and the factors independently associated with successful cessation.

Data Collection and Variables

Data were obtained from medical records and the parent-trial database. Demographic variables included age, sex, body mass index (BMI), marital status, education, income, household size, occupation, and occupational exposure. Smoking-related variables included pack-years, daily cigarette consumption, smoking duration, age at initiation, the Fagerström Test for Nicotine Dependence,[12] previous quit attempts, depression, and anxiety. Disease-related variables included GOLD category, blood eosinophil count, FEV1/FVC, FEV1 (litres and percent predicted), modified Medical Research Council (mMRC) dyspnoea score, COPD Assessment Test (CAT) score, exacerbation-related emergency, outpatient, and hospitalisation events, long-term oxygen therapy, noninvasive ventilation, previous pulmonary infections, and comorbidities.

Statistical Analysis

Analyses were performed in R version 4.6.0 (R Foundation for Statistical Computing). Continuous variables are summarised as mean (SD) and categorical variables as number (percentage). Quitters and non-quitters were compared using the Welch t test for continuous variables and the Fisher exact test for categorical variables. Because only 32 cessation events were available, the primary multivariable model was deliberately parsimonious and prespecified: a binary logistic regression including allocated study group, age, baseline FEV1 (litres), Fagerström score, presence of other comorbidity, and more than 1 exacerbation-related hospitalisation, giving approximately 5 events per candidate variable. These six factors correspond to the domains identified a priori in the Introduction. Results are reported as odds ratios (ORs) with 95% confidence intervals (CIs). Multicollinearity was assessed with the variance inflation factor (all values <2.5).

Baseline characteristics of patients lost to follow-up and of those analysed are compared in Supplementary Table S1. Three sensitivity analyses were prespecified. First, the same model was refitted using Firth penalised maximum likelihood with profile-likelihood confidence intervals, which reduces small-sample bias and improves the behaviour of estimates for sparse categories. Second, all patients lost to follow-up were classified as continuing smokers and the model refitted in the full cohort of 220 patients (Supplementary Table S2). Third, the exploratory model containing all 15 baseline variables that was examined initially is reported in Supplementary Table S3, in both unpenalised and penalised form, so that its instability can be compared with the primary model. The distribution of inhaler regimens across GOLD categories in the analysed cohort was displayed as a grouped bar chart. A 2-sided p value <0.05 was considered statistically significant.

Results

Demographic Characteristics

Of 220 patients with COPD randomised in the parent trial, 48 (21.8%) were lost to follow-up, leaving 172 patients; 32 (18.6%) had quit smoking at 1 year and 140 (81.4%) continued to smoke (Figure 1). Loss to follow-up did not differ significantly between arms (21 of 114 [18.4%] with immediate referral vs 27 of 106 [25.5%] with usual care; p = 0.25). Patients lost to follow-up had higher Fagerström scores (7.5 [2.3] vs 6.3 [2.8]; p = 0.003), higher CAT scores (19.5 [7.6] vs 14.0 [7.5]; p < 0.001) and fewer pack-years (40.1 [17.3] vs 51.5 [24.5]; p < 0.001) than those analysed, but did not differ in age, sex, lung function, comorbidity, or hospitalisation history (Supplementary Table S1). Among the 172 analysed patients, 24 of 93 (25.8%) allocated to immediate referral had quit versus 8 of 79 (10.1%) allocated to usual care (p = 0.010). Quitters were older than non-quitters (63.5 [7.1] vs 59.9 [8.8] years; p = 0.018). Sex, BMI, marital status, education, income, household size, occupation, and occupational exposure did not differ between groups (Table 1).

Smoking Dependence Characteristics

Smoking exposure was comparable between groups: pack-years, daily cigarette consumption, smoking duration, age at initiation, previous quit attempts, and longest prior cessation period did not differ significantly. The mean Fagerström score was lower among quitters (5.4 [2.9] vs 6.5 [2.8]) but reached only a non-significant trend (p = 0.056), and depression and anxiety were similarly distributed (Table 2).

Disease Characteristics

GOLD category distribution was similar between groups, as were CAT score, mMRC dyspnoea score, eosinophil count, long-term oxygen therapy, noninvasive ventilation, and previous pulmonary infections. Quitters had a lower baseline FEV1 than non-quitters (1.5 [0.6] vs 1.9 [0.7] L; p = 0.010); FEV1/FVC was numerically lower among quitters (61.8 [14.9] vs 67.6 [12.9] %) but was interpreted as a trend (p = 0.050). Exacerbation-related emergency and outpatient visits were similar between groups, whereas more than 1 COPD exacerbation–related hospitalisation was more frequent among quitters (5 of 32 [15.6%] vs 5 of 140 [3.6%]; p = 0.021) (Table 3).

Attendance at a smoking cessation clinic during follow-up was more common in the immediate-referral arm (62 of 87 [71.3%] vs 18 of 79 [22.8%] with available data), but 18 patients allocated to usual care did access cessation services on their own initiative, consistent with the encouragement given to all participants. Cessation pharmacotherapy was started

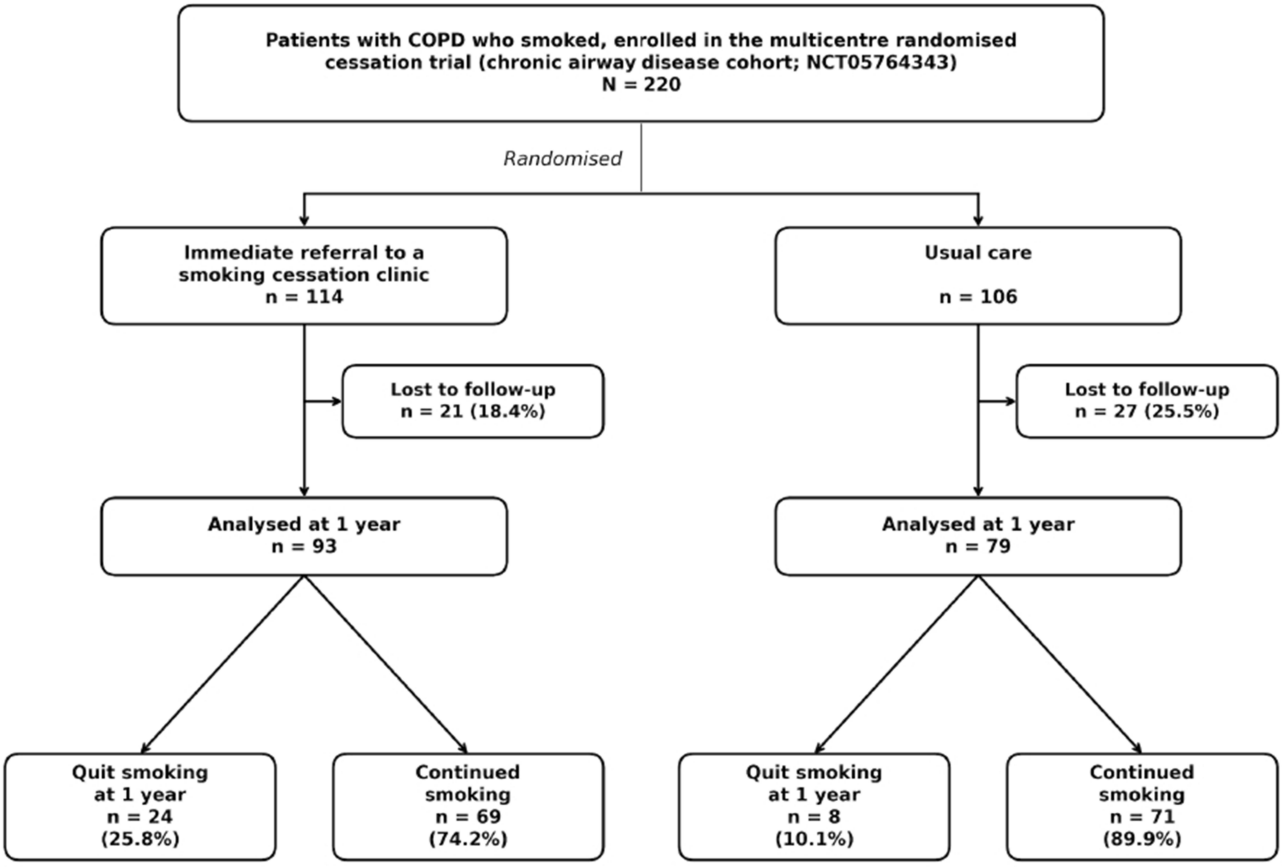


Figure 1 Flow of patients through the COPD subgroup analysis Flow of the 220 patients with COPD randomised in the parent trial, by allocated study group, showing loss to follow-up in each arm, the analysed population, and 1-year smoking cessation status. Patients recorded as having stopped smoking from the target quit date until the 1-year interview were classified as quitters; those who relapsed after a quit attempt and those who made no quit attempt were classified as non-quitters. Self-reported cessation was confirmed by exhaled carbon monoxide measurement in all 32 quitters. Percentages are calculated within the corresponding study arm.

at a cessation clinic in 62 patients and purchased independently by 5 further patients; 5 attended a clinic without receiving pharmacotherapy. Quit rates were 19 of 80 (23.8%) among clinic attenders and 12 of 86 (14.0%) among non-attenders (p = 0.12) (Table 4).

Table 1 Demographic Characteristics According to 1-Year Smoking Cessation Status

Characteristic	Overall (N = 172)	Quit (n = 32)	Not Quit (n = 140)	p value
Study group (randomised)				0.010
Immediate referral	93 (54.1)	24 (75.0)	69 (49.3)	
Usual care	79 (45.9)	8 (25.0)	71 (50.7)	
Age, mean (SD), y	60.6 (8.6)	63.5 (7.1)	59.9 (8.8)	0.018
Sex				0.5
Female	20 (11.6)	5 (15.6)	15 (10.7)	
Male	152 (88.4)	27 (84.4)	125 (89.3)	
BMI, mean (SD)	26.2 (4.3)	25.9 (3.4)	26.3 (4.5)	0.6
Marital status				0.3
Married	152 (88.4)	29 (90.6)	123 (87.9)	
Single	14 (8.1)	1 (3.1)	13 (9.3)	
Separated	6 (3.5)	2 (6.3)	4 (2.9)	

(*Continued*)

Table 1 (Continued).

Characteristic	Overall (N = 172)	Quit (n = 32)	Not Quit (n = 140)	p value
Education				0.8
< High school	122 (70.9)	22 (68.8)	100 (71.4)	
≥ High school	50 (29.1)	10 (31.3)	40 (28.6)	
Income				>0.9
≤ Minimum wage	114 (66.3)	21 (65.6)	93 (66.4)	
> Minimum wage	58 (33.7)	11 (34.4)	47 (33.6)	
Occupation				0.4
Retired	85 (49.4)	20 (62.5)	65 (46.4)	
Housewife	13 (7.6)	1 (3.1)	12 (8.6)	
Employed	56 (32.6)	8 (25.0)	48 (34.3)	
Unemployed	18 (10.5)	3 (9.4)	15 (10.7)	
Occupational exposure	51 (31.3)	10 (32.3)	41 (31.1)	>0.9

Notes: Data are presented as No. (%) unless otherwise indicated. Study group is the allocation recorded at randomisation. p values are from the Welch *t* test (continuous variables) or Fisher exact test (categorical variables). Household-size data are not shown. BMI indicates body mass index.

Table 2 Smoking Dependence Characteristics According to 1-Year Smoking Cessation Status

Characteristic	Overall (N = 172)	Quit (n = 32)	Not Quit (n = 140)	p value
Pack-years, mean (SD)	51.5 (24.5)	49.0 (23.9)	52.1 (24.7)	0.5
Daily cigarettes, mean (SD)	23.1 (12.1)	21.6 (15.9)	23.4 (11.0)	0.5
Smoking duration, mean (SD), y	40.9 (11.0)	43.2 (9.7)	40.4 (11.2)	0.2
Smoking onset age, mean (SD), y	18.1 (6.1)	17.6 (6.7)	18.3 (6.0)	0.6
Fagerström score, mean (SD)	6.3 (2.8)	5.4 (2.9)	6.5 (2.8)	0.056
Past quit attempts, mean (SD)	1.4 (2.5)	1.6 (1.7)	1.4 (2.6)	0.6
Longest cessation, mean (SD), d	157.9 (584.3)	290.6 (995.0)	123.3 (416.6)	0.4
Depression	34 (19.8)	5 (15.6)	29 (20.7)	0.6
Anxiety	33 (19.2)	6 (18.8)	27 (19.3)	>0.9

Notes: Data are presented as mean (SD) or No. (%). p values are from the Welch *t* test (continuous variables) or Fisher exact test (categorical variables).

Table 3 Disease Characteristics According to 1-Year Smoking Cessation Status

Characteristic	Overall (N = 172)	Quit (n = 32)	Not Quit (n = 140)	p value
Disease severity and symptoms				
GOLD category				0.547
A	59 (34.7)	9 (28.1)	50 (36.2)	
B	65 (38.2)	12 (37.5)	53 (38.4)	
E	46 (27.1)	11 (34.4)	35 (25.4)	
FEV1/FVC, mean (SD), %	66.5 (13.5)	61.8 (14.9)	67.6 (12.9)	0.050
FEV1, mean (SD), L	1.8 (0.7)	1.5 (0.6)	1.9 (0.7)	0.010
FEV1, mean (SD), % predicted	59.4 (20.3)	54.7 (16.6)	60.5 (21.0)	0.100
Eosinophil count, mean (SD),/μL	77.6 (129.3)	52.4 (92.1)	84.0 (136.8)	0.132
mMRC dyspnoea score				0.167
0	11 (6.4)	2 (6.3)	9 (6.4)	
1	64 (37.2)	8 (25.0)	56 (40.0)	
2	70 (40.7)	17 (53.1)	53 (37.9)	
3	23 (13.4)	3 (9.4)	20 (14.3)	
4	4 (2.3)	2 (6.3)	2 (1.4)	

(*Continued*)

Table 3 (Continued).

Characteristic	Overall (N = 172)	Quit (n = 32)	Not Quit (n = 140)	p value
CAT score, mean (SD)	14.0 (7.5)	14.7 (8.2)	13.8 (7.4)	0.578
Inhaled treatment				
LAMA only	26 (15.1)	3 (9.4)	23 (16.4)	0.418
LABA + LAMA	55 (32.0)	11 (34.4)	44 (31.4)	0.834
LABA + LAMA + ICS	57 (33.1)	13 (40.6)	44 (31.4)	0.405
LABA + ICS	31 (18.0)	6 (18.8)	25 (17.9)	>0.999
ICS dose				0.350
Low	9 (10.5)	2 (10.5)	7 (10.4)	
Moderate	47 (54.7)	13 (68.4)	34 (50.7)	
High	30 (34.9)	4 (21.1)	26 (38.8)	
SABA/SAMA	32 (18.6)	3 (9.4)	29 (20.7)	0.207
SABA/SAMA as needed	80 (46.5)	17 (53.1)	63 (45.0)	0.437
Exacerbation-related health care use, previous year				
>1 Emergency visit	32 (18.6)	8 (25.0)	24 (17.1)	0.318
>1 Outpatient visit	44 (25.7)	10 (31.3)	34 (24.5)	0.501
>1 Hospitalisation	10 (5.8)	5 (15.6)	5 (3.6)	0.021
Other clinical characteristics				
Long-term oxygen therapy	13 (7.6)	3 (9.4)	10 (7.1)	0.711
Noninvasive ventilation	9 (5.2)	2 (6.3)	7 (5.0)	0.674
Previous pulmonary infections	59 (34.3)	9 (28.1)	50 (35.7)	0.537
Other comorbidities present	93 (54.1)	14 (43.8)	79 (56.4)	0.239

Notes: Data are presented as No. (%) unless otherwise indicated. Variables are grouped under bold subheadings, which are not data rows. p values are from the Welch *t* test (continuous variables) or Fisher exact test (categorical variables). GOLD category was missing for 2 non-quitters. ICS dose percentages are among ICS users. CAT indicates COPD Assessment Test.

Abbreviations: FEV1, forced expiratory volume in 1 second; FVC, forced vital capacity; GOLD, Global Initiative for Chronic Obstructive Lung Disease; ICS, inhaled corticosteroid; LABA, long-acting β2-agonist; LAMA, long-acting muscarinic antagonist; mMRC, modified Medical Research Council; SABA, short-acting β2-agonist; SAMA, short-acting muscarinic antagonist.

Table 4 One-Year Follow-Up Outcomes According to Smoking Cessation Status

Outcome at 1 Year	Quit	Not Quit	p value
Use of cessation services			
Attended a cessation clinic	19/31 (61.3)	61/135 (45.2)	0.12
Received cessation pharmacotherapy	17/24 (70.8)	50/99 (50.5)	0.11
Symptom burden			
CAT score, baseline, mean (SD)[a]	18.5 (7.3)	15.3 (7.7)	—
CAT score, 1 year, mean (SD)[a]	13.9 (6.9)	13.3 (7.3)	—
Within-group change in CAT score[a]	−4.5 (3.0); p = 0.002	−2.0 (6.9); p = 0.19	—
CAT score <10 at 1 year	11/30 (36.7)	59/119 (49.6)	0.23
Inhaler adherence at 1 year			0.065
Never misses a dose	21/30 (70.0)	60/116 (51.7)	
Sometimes forgets	6/30 (20.0)	26/116 (22.4)	
Stops when feeling well (own decision)	0/30 (0)	18/116 (15.5)	
Stops when feeling well (physician advice)	3/30 (10.0)	12/116 (10.3)	
Health care use during follow-up year			
Exacerbation-related emergency visits, mean (SD)	1.39 (2.30)	0.96 (1.49)	0.56
Exacerbation-related hospitalisations, mean (SD)	0.32 (0.60)	0.22 (0.50)	0.35

(*Continued*)

Table 4 (Continued).

Outcome at 1 Year	Quit	Not Quit	p value
Tobacco use and verification			
Concurrent use of another tobacco product	0/12 (0)	9/117 (7.7)	>0.9
Exhaled carbon monoxide measured	32/32 (100.0)	0/140 (0)	<0.001

Notes: Data are presented as No./total with available data (%) unless otherwise indicated. Denominators vary because follow-up items were not recorded for every patient. p values are from the Fisher exact test, the Fisher–Freeman–Halton exact test (inhaler adherence), the Mann–Whitney *U*-test (health care use), or the paired *t* test (within-group change in CAT score). [a]CAT scores at 1 year were recorded as an exact value in 11 quitters and 58 non-quitters and as a category (<10 or ≥10) in the remainder; the paired comparison uses only patients with exact values at both time points. All exhaled carbon monoxide values were ≤3 ppm. CAT indicates COPD Assessment Test.

Inhaler regimens and inhaled corticosteroid (ICS) dose did not differ by cessation status (all $p > 0.05$). Follow-up outcomes are summarised in Table 4. Follow-up CAT scores were recorded as an exact value in only 69 patients, and as a category (<10 or ≥10) in the remainder. In the subgroup with paired numerical values, the CAT score fell from 18.5 (7.3) to 13.9 (6.9) among 11 quitters (mean change, −4.5 [3.0]; $p = 0.002$) and from 15.3 (7.7) to 13.3 (7.3) among 58 non-quitters (mean change, −2.0 [6.9]; $p = 0.19$); using all available data, a follow-up CAT score below 10 was reached by 11 of 30 quitters (36.7%) and 59 of 119 non-quitters (49.6%) ($p = 0.23$). Full adherence to inhaler therapy was reported by 21 of 30 quitters (70.0%) and 60 of 116 non-quitters (51.7%), a difference that did not reach statistical significance ($p = 0.099$; $p = 0.065$ for the distribution across all four adherence categories). Concurrent use of another tobacco product was reported by 9 patients, all of them non-quitters. Exhaled CO was measured in all 32 quitters and in none of the 140 non-quitters (Table 4). Successful cessation was not associated with a significant between-group difference in exacerbation-related hospitalisations (0.32 [0.60] vs 0.22 [0.50]; $p = 0.35$) or emergency visits (1.39 [2.30] vs 0.96 [1.49]; $p = 0.56$) during the follow-up year.

Inhaler Treatment Patterns

Inhaler treatment intensity generally increased with disease severity in the analysed cohort of 172 patients (Figure 2). Dual bronchodilation with a long-acting muscarinic antagonist (LAMA) plus a long-acting β2-agonist (LABA) was common in GOLD A and B, whereas ICS–containing triple therapy predominated in GOLD E, broadly consistent with GOLD recommendations.[1] Contrary to those recommendations, ICS-containing combinations were also prescribed in GOLD A and B, and short-acting (rescue) bronchodilators were used across all categories, most often in advanced disease. These patterns suggest that prescribing reflected symptom burden, exacerbation risk, and clinical judgment beyond GOLD category alone.

Predictors of Smoking Cessation: Multivariable Analysis

In the prespecified multivariable model, three factors were independently associated with smoking cessation at one year among the 167 patients with complete covariate data (Table 5). Patients allocated to usual care were significantly less likely to quit than those who received immediate referral to a cessation clinic (OR, 0.25; 95% CI, 0.09–0.64; $p = 0.004$). Patients without another comorbidity were more likely to quit than those with comorbidity (OR, 2.64; 95% CI, 1.06–6.60; $p = 0.038$). Patients with more than 1 COPD exacerbation–related hospitalisation were also more likely to quit (OR, 9.32; 95% CI, 1.97–44.06; $p = 0.005$); this estimate was based on only 10 such patients and remains imprecise, and should therefore be regarded as hypothesis-generating. Age (OR, 1.03; 95% CI, 0.97–1.10; $p = 0.32$), baseline FEV1 (OR, 0.65; 95% CI, 0.32–1.33; $p = 0.24$) and the Fagerström score (OR, 0.86; 95% CI, 0.74–1.01; $p = 0.060$) were not independently associated with cessation.

The findings were stable across the prespecified sensitivity analyses. Firth penalised estimation of the same model gave closely similar results (usual care: OR, 0.27; 95% CI, 0.10–0.65; comorbidity absent: OR, 2.51; 95% CI, 1.06–6.28; >1 hospitalisation: OR, 8.11; 95% CI, 1.90–36.98) (Table 5). When all 48 patients lost to follow-up were classified as continuing smokers, the overall quit rate was 32 of 220 (14.5%), the difference between arms persisted (24 of 114 [21.1%] vs 8 of 106 [7.5%]; $p = 0.007$), and the model, refitted in the 211 patients with complete covariate data, again

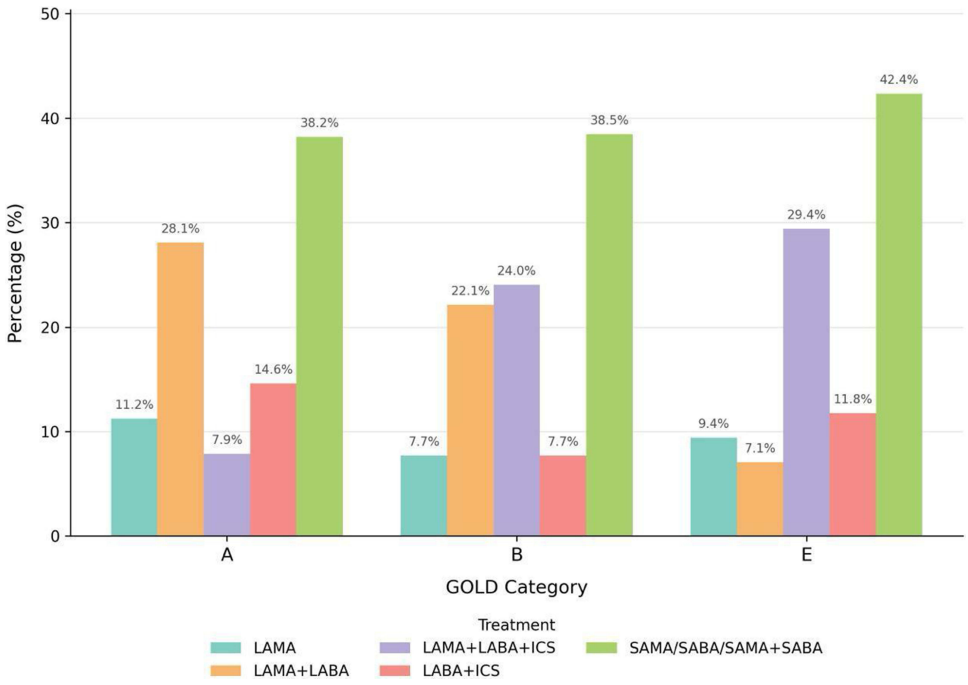


Figure 2 Distribution of inhaler treatment regimens across GOLD categories among patients with COPD who smoked Data are for the analysed cohort (N = 172). Within each GOLD category, bars show the relative distribution across the 5 displayed regimen categories, normalised to 100%; patients receiving more than one regimen component contribute to more than one category, and percentages therefore describe the mix of prescribed regimens rather than the proportion of patients. ICS indicates inhaled corticosteroid; LABA, long-acting β2-agonist; LAMA, long-acting muscarinic antagonist; SABA, short-acting β2-agonist; SAMA, short-acting muscarinic antagonist.

identified allocated study group (OR, 0.22; 95% CI, 0.09–0.56), absence of comorbidity (OR, 2.49; 95% CI, 1.03–6.04) and repeated hospitalisation (OR, 7.12; 95% CI, 1.78–28.45) as the factors associated with cessation (Supplementary Table S2). In the exploratory 15-variable model, the same three factors reached significance, but the estimates were markedly less precise, most conspicuously for hospitalisation (OR, 15.03; 95% CI, 1.76–128.55), illustrating the instability expected with 32 events and 15 covariates (Supplementary Table S3).

Discussion

In this prespecified subgroup analysis of current smokers with COPD enrolled in a multicentre randomised cessation trial, immediate referral to a smoking cessation clinic was the factor most strongly associated with abstinence at 1 year, whereas baseline demographic and disease characteristics had more limited value. Older age and lower baseline FEV1

Table 5 Prespecified Multivariable Model of Factors Associated with 1-Year Smoking Cessation

Variable	Unpenalised Model			Firth Penalised Model		
	OR	95% CI	p value	OR	95% CI	p value
Age, per 1 year	1.03	0.97–1.10	0.320	1.03	0.97–1.10	0.337
Usual care (vs immediate referral)	0.25	0.09–0.64	0.004	0.27	0.10–0.65	0.003
Fagerström score, per point	0.86	0.74–1.01	0.060	0.87	0.75–1.01	0.062
FEV1, per litre	0.65	0.32–1.33	0.239	0.67	0.33–1.33	0.260
Comorbidity absent (vs present)	2.64	1.06–6.60	0.038	2.51	1.06–6.28	0.037
>1 Hospitalisation (vs 0–1)	9.32	1.97–44.06	0.005	8.11	1.90–36.98	0.005

Notes: Prespecified binary logistic regression in 167 patients with complete covariate data and 32 cessation events; all six variables were entered simultaneously and no variable selection was performed. The unpenalised model reports Wald confidence intervals; the Firth penalised model reports profile-likelihood confidence intervals and penalised likelihood ratio p values. The hospitalisation estimate is based on 10 patients, remains imprecise, and is hypothesis-generating.
Abbreviations: CI indicates confidence interval; OR, odds ratio.

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distinguished quitters from non-quitters in unadjusted comparisons but were not independently associated with cessation after adjustment. Absence of comorbidity was independently associated with success, and repeated COPD-related hospitalisation showed a strong but imprecise association that we regard as hypothesis-generating.

Quitting smoking remains the cornerstone of COPD treatment. Although inhaled therapies alleviate symptoms and reduce exacerbations, continued smoking accelerates airway inflammation, hastens the decline in lung function, and worsens the prognosis.[1,3] Our findings reinforce the case for structured cessation pathways as part of routine COPD care rather than reliance on brief advice alone.

The strongest factor associated with smoking cessation was being assigned to the intervention group. Patients who were referred immediately quit smoking much more frequently than those who received usual care, and this association persisted even after adjustment. A similar pattern has been reported internationally: immediate cessation support delivered during a lung health check increased quit rates, suggesting that reducing the delay between identifying tobacco use and starting treatment improves outcomes.[13] It is worth noting that 18 patients allocated to usual care attended a cessation clinic on their own initiative, as all participants were encouraged to do; the difference between arms observed here is therefore likely to understate the difference between proactive referral and no cessation support at all. These data suggest that the organisation and accessibility of cessation services may be more important than individual patient characteristics alone.

The association between older age and cessation in the unadjusted comparison is consistent with prior COPD studies; older patients may have greater awareness of smoking-related harm, more frequent health care contact, and stronger motivation to prevent further decline.[5] Age was not independently associated with cessation after adjustment, which suggests that any effect is shared with disease severity, health care engagement, or intervention exposure. Successful quitters also had a lower baseline FEV1 in the unadjusted comparison, but this association did not persist in the multivariable model, and FEV1/FVC differed only borderline ($p = 0.050$). Greater disease severity should therefore not be interpreted as an independent predictor of quitting in this cohort. More severe airflow limitation generally signals greater disease burden, and it has been suggested that more severe dyspnoea and functional limitation may strengthen the willingness to stop smoking, with worsening lung function described as a teachable moment for cessation;[3,4] our data are compatible with that hypothesis but do not establish it.

A notable association was that between COPD-related hospitalisation and cessation: patients with repeated admissions were more likely to quit, and this association persisted after adjustment. Hospital admissions increase patients' awareness of disease severity and may provide opportunities for intensive intervention, and have been described as a teachable moment for cessation.[11] The finding must nevertheless be treated as hypothesis-generating rather than established: it rests on only 10 patients, the confidence interval remains wide even after penalisation, and greater health care contact among more severely affected patients may itself contribute to cessation rather than the admission alone. Confirmation in a cohort with a larger number of admitted patients is required before exacerbation-related admission is used to target cessation resources.

Unlike many previous studies, nicotine dependence was not significantly associated with cessation in the primary analysis; Fagerström scores were only nominally lower among quitters ($p = 0.056$), and the adjusted estimate approached but did not reach significance ($p = 0.060$). Notably, the association did reach significance when patients lost to follow-up were classified as continuing smokers, which is consistent with their higher baseline Fagerström scores and suggests that attrition attenuated this association in the primary analysis. Dependence is a well-established barrier in the general smoking population,[12] but among patients with COPD, disease-related motivation and structured support may reduce its effect, and the universal provision of advice and wide access to pharmacotherapy in the parent trial may have further narrowed baseline differences.[14]

The absence of comorbidity was the other independent factor associated with cessation. Multimorbidity can complicate quitting through psychological stress, treatment and pill burden, polypharmacy, and competing health priorities, and COPD frequently coexists with cardiovascular, metabolic, and psychiatric disease, conditions that may reduce adherence to behavioural change.[15–18] Depression and anxiety themselves were not associated with cessation, which may reflect the beneficial effect of structured programmes that combine counselling and pharmacotherapy, as well as the small number of patients with recorded psychiatric diagnoses.[19]

Inhaler prescribing broadly followed disease severity, with LAMA/LABA common in GOLD A and B and ICS-containing triple therapy more frequent in GOLD E, consistent with GOLD guidance.[1] The overlap between categories, particularly ICS use in early disease and the widespread use of rescue medication, illustrates the complexity of real-world COPD management, in which clinicians weigh exacerbation history, eosinophil counts, and patient preference alongside GOLD category. Inhaler adherence was numerically higher among quitters, and CAT scores fell in both groups, but neither difference was statistically significant and both were measured after cessation had occurred; they should be read as coexisting health behaviours rather than as consequences of quitting.

The lack of between-group differences in exacerbations and health care use should be interpreted with caution: many benefits of cessation become apparent over years rather than months,[4,20] and a single year with few quitters limited the power to detect them. These findings are particularly relevant in Türkiye, where cessation support has been insufficiently integrated into routine care for chronic airway disease,[6,8] and where the same gap has been reported for other high-risk groups such as patients with cardiovascular disease.[21] These findings suggest that cessation in COPD depends not only on patient motivation and disease characteristics but also, to a considerable extent, on how cessation care is delivered. Ensuring timely access to dedicated services may therefore be one of the most effective ways to improve long-term outcomes.

Strengths and Limitations

Strengths of this analysis include its origin in a multicentre randomised trial, a full year of follow-up, a clinically coherent COPD subgroup, and the breadth of demographic, smoking-related, disease, and treatment variables examined.

Several limitations apply. This was a prespecified but exploratory subgroup analysis, and, apart from the randomised study-group allocation, comparisons by cessation status are observational and should not be read as causal. Only 32 patients quit, so even the parsimonious model provides approximately 5 events per variable; some estimates, particularly for repeated hospitalisation, remain imprecise and hypothesis-generating, and the wider intervals of the exploratory 15-variable model (Supplementary Table S3) illustrate the limits of what these data can support. Smoking cessation status was obtained from the patients' self-reports, and although exhaled CO verification was completed in all 32 patients who reported having quit, it was performed only at the one-year contact, so smoking earlier in the follow-up period cannot be excluded, and the reports of non-quitters were not biochemically checked. Loss to follow-up was substantial (21.8%) and, although it did not differ significantly between arms, patients who were lost were more nicotine dependent and more symptomatic at baseline; the sensitivity analysis classifying them as continuing smokers preserved every principal finding, but residual attrition bias cannot be excluded. Comorbidity was recorded only as present or absent, without disease-specific severity or a validated index. Reasons for relapse were not recorded, so we cannot say why patients who stopped resumed smoking; prospective collection of relapse triggers would inform the design of future interventions. Attrition was also numerically higher in the usual-care arm, which may reflect disengagement among patients who had hoped to receive the intervention, although the difference was not significant. Finally, the data come from a single health care system and may not generalise to all COPD-care settings.

Conclusion

Among current smokers with COPD, immediate referral to a smoking cessation clinic was the factor most strongly associated with abstinence at 1 year, together with the absence of comorbidity. Repeated COPD-related hospitalisation was also associated with quitting, but this estimate is based on few patients and remains hypothesis-generating, and greater disease severity was associated with cessation only in unadjusted comparisons. Given the exploratory nature of this subgroup analysis and the fact that biochemical verification was performed only at the one-year contact, these associations need to be confirmed in larger COPD cohorts with biochemical verification at scheduled follow-up visits. However, the findings support the inclusion of structured, proactive smoking cessation processes in routine COPD care.

Abbreviations

BMI, body mass index; CAT, COPD Assessment Test; CI, confidence interval; COPD, chronic obstructive pulmonary disease; FEV1, forced expiratory volume in 1 second; FVC, forced vital capacity; GOLD, Global Initiative for Chronic Obstructive Lung Disease; ICS, inhaled corticosteroid; LABA, long-acting β2-agonist; LAMA, long-acting muscarinic antagonist; mMRC, modified Medical Research Council; OR, odds ratio; SABA, short-acting β2-agonist; SAMA, short-acting muscarinic antagonist; SD, standard deviation.

Data Sharing Statement

The deidentified dataset analysed for the present study is not publicly available because of participant-privacy considerations but may be available from the corresponding author on reasonable request and subject to institutional approval.

Ethics Approval and Consent to Participate

Ethical approval for this analysis was obtained from the Ethics Committee for Non-Interventional Clinical Trials, Kutahya Health Sciences University (approval No. 2026/02-58; 02/09/2026). The analysis used anonymized existing data from the parent trial; informed consent for the parent trial was obtained as previously described.[7,8]

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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