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# Prevalence of microspirometry-detected chronic obstructive pulmonary disease in two European cohorts of patients hospitalised for acute myocardial infarction: a cross-sectional study

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## ABSTRACT

**Objectives** To establish the prevalence of clinically significant chronic obstructive pulmonary disease (COPD) and relevant characteristics in individuals with a significant smoking history who are hospitalised for acute myocardial infarction (MI).

**Design** Cross-sectional study.

**Setting** Hospital inpatients at 8 European centres (7 in Sweden, 1 in the UK).

**Participants** 518 men or women (302 in Sweden, 216 in the UK) hospitalised for acute MI, aged 40 years or older, with a smoking history of at least 10 pack-years.

**Primary and secondary outcome measures** The primary outcome was prevalence of detected significant COPD (Global Initiative for Chronic Obstructive Lung Disease stages 2–4), defined as a ratio of forced expiratory volume in 1 and 6 s ($FEV_1/FEV_6$) <0.7 and $FEV_1$ <80% of the predicted value, measured using microspirometry. Secondary outcome measures were prior diagnosis of COPD, prescription of inhaled corticosteroids (ICS), symptom burden (COPD Assessment Test (CAT)) and blood eosinophil count.

**Results** The prevalence of significant COPD was 91/518 (18% (95% CI 14 to 21)) with no difference between the countries. Of those with detected significant COPD, 69 (76%) had no previous COPD diagnosis. A CAT score >10 was found in 65%, and a blood eosinophil count of ≥100/mm$^3$ and ≥300/mm$^3$ was found in 76% and 20%, respectively. Inhaled corticosteroids were used by 15% of the patients.

**Conclusions** In a cohort of patients hospitalised for acute MI in Sweden and the UK, one in five patients with a history of smoking was found to have significant COPD based on microspirometry. Symptom burden was high and treatment rates with ICS low. Among those diagnosed with COPD, three out of four had not been previously diagnosed with COPD.

## STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ A sample of 518 participants was recruited consecutively across two European countries, providing a representative cohort.
⇒ For the first time in this setting, chronic obstructive pulmonary disease (COPD) was diagnosed by systematic measurement of pulmonary function rather than relying solely on clinical features or prior diagnosis.
⇒ A widely accepted existing questionnaire was used to determine symptom burden.
⇒ Though validated in numerous settings, there remains some uncertainty around the reliability of microspirometry compared with conventional spirometry in patients hospitalised for acute myocardial infarction and further work is needed.
⇒ It is unclear if the criteria we used to diagnose for COPD by microspirometry are the most accurate when compared with diagnosis by conventional spirometry, or whether alternative thresholds may perform better.

## INTRODUCTION

Chronic obstructive pulmonary disease (COPD) often coexists with cardiovascular disease (CVD) and shares risk factors such as smoking, ageing and systemic inflammation.[1 2] Following myocardial infarction (MI), mortality and heart failure are around 30–35% higher in those with COPD compared with those without.[3] Conversely, CVD can worsen COPD outcomes by impairing heart and lung function and exacerbating respiratory symptoms. Inhaled pharmacotherapy in COPD includes long-acting ß2 receptor

agonists (LABA), long-acting muscarinic antagonists (LAMA) and inhaled corticosteroids (ICS). ICS have been suggested to have beneficial effects on cardiovascular risk and all-cause mortality, especially in patients with a high blood eosinophil count.[4 5]

The prevalence of COPD in patients with acute MI has not been accurately determined. The diagnosis of COPD is based on spirometry and defined as a forced expiratory volume in 1s ($FEV_1$) to forced vital capacity (FVC) ratio of <0.7 after the administration of a bronchodilator.[6] Though clinical studies of patients with MI have reported the frequency of a COPD diagnosis, for example, recorded in 5.8% of participants with or without a smoking history in the PLATelet inhibition and patient Outcomes trial,[7] this is solely based on self-reported information and not on verified spirometry data. Underdiagnosis and overdiagnosis of COPD have been recognised as significant issues across multiple cohorts and can lead to inaccuracy of estimation.[8] As an alternative to conventional spirometry, microspirometry substituting the FVC with the FEV in 6s ($FEV_6$) and avoiding bronchodilator use has been validated for COPD screening in a range of outpatient settings.[9–11]

We sought to accurately determine the prevalence of COPD using microspirometry in patients with a significant smoking history hospitalised for acute MI and to characterise this population.

## METHODS

### Study design and data collection

The present analysis includes cross-sectional baseline data from two parallel cohort studies: one conducted at a single centre in the UK and one carried out across seven centres in Sweden. The designs of the two projects are provided in the online supplemental appendix.

The inclusion criteria for both studies were men or women 40 years of age or older who were current or ex-smokers with a smoking history of at least 10 pack-years, presently hospitalised for MI (according to the fourth Universal Definition),[12] and able to give written informed consent. Exclusion criteria included inability or unwillingness to provide written informed consent, current ischaemic chest pain, acute pulmonary oedema, cardiogenic shock, systolic blood pressure <90 mmHg, intra-aortic balloon pump/inotropic support or any other practical reason, in the opinion of the investigator, why accurate spirometry may not be possible or safe to perform. In addition, the UK cohort had further exclusion criteria in terms of current treatment for lower respiratory tract infection, supplemental oxygen unless administered via nasal cannula alone, known diagnosis of restrictive lung disease (eg, pulmonary fibrosis), active haemoptysis, known lung malignancy, or active tuberculosis, and positive COVID-19 PCR test in the preceding 10 days.

The inclusion period for the UK study was January to November 2022, and in Sweden from February 2022 until March 2023. All participants admitted under the care of the cardiology team were consecutively screened and, if appropriate, enrolled during their initial hospitalisation for MI. They underwent collection of baseline data, including relevant co-morbidities, medications and blood test results. Microspirometry was performed using the Vitalograph COPD-6 device by trained members of the research teams as per the manufacturer's instructions. The standard procedure includes three exhalations, the best results of $FEV_1$, $FEV_6$ and $FEV_1/FEV_6$, being saved by the device. The COPD-6 microspirometer also provides immediate feedback on the adequacy of the technique for each of the three exhalations.[13]

In patients with a positive COPD-6 result, blood eosinophil count was recorded and the symptom burden assessed using the COPD Assessment Test (CAT).[14] The CAT includes eight domains in which the patient expresses their response on a visual analogue scale from 0 to 5, representing best to worst. The domains include respiratory symptoms such as cough, production of phlegm, chest tightness, and breathlessness, plus more general areas such as limitation of daily activities, confidence, sleep quality, and energy levels. A CAT score of 10 or more is regarded as representing a high burden of symptoms.[6]

In both studies, the primary study endpoint was the prevalence of COPD with GOLD stage 2–4 severity,[6] defined as $FEV_1/FEV_6$<0.7 and $FEV_1$<80% of predicted measured using the Vitalograph COPD-6 microspirometry device without administration of a bronchodilator. For screening, $FEV_1/FEV_6$ below 0.73 has been recommended as a cut-off for further examination with spirometry, but in other studies, $FEV_1/FEV_6$<0.7 has also been used as a surrogate for established obstruction.[15]

This paper summarises the data collected during both studies at the baseline timepoint in order to provide an accurate and reliable estimate of the prevalence of COPD in patients hospitalised for MI across two geographical cohorts and to characterise this population.

### Regulatory approval

The UK study was approved by the National Health Service Yorkshire & the Humber (Bradford/Leeds) Research Ethics committee (Ref 22/YH/0015) and the Swedish study by the Swedish Ethical Review Authority (Dnr 2021-05615-01). All work was compliant with the Declaration of Helsinki. All participants provided written informed consent.

### Statistical analysis

For continuous variables, summary data were prepared as mean and SD where approximately normally distributed and median and IQR where non-normally distributed. Proportions are given as percentages, and where relevant, the estimate of the 95% CI based on a normal approximation of the population value is provided.

The UK sample size of 216 was based on a power calculation estimating that the recruitment target would provide

a 90% CI of less than 10% in determining the population prevalence of protocol-defined COPD. According to the Swedish research plan, it was estimated that for a sample of 307 patients and a prevalence of 15% (both given), a 95% CI would have an estimated width of ±4.0 percentage points. As secondary outcomes, the prevalence of COPD GOLD stage 1–4,[6] and the prevalence of patients with the commonly used screening threshold value of $FEV_1/FEV_6$ <0.73 were calculated.

The group with protocol-defined COPD (stage 2–4) was further characterised by determining the proportions with high symptom burden (CAT ≥10), blood eosinophil count ≥100 cells/$mm^3$ and ≥300 cells/$mm^3$, and those receiving ICS treatment. Cross-tabulations with chi-square tests explored differences in clinical characteristics between patients with and without stage 2–4 COPD.

The proportion of participants with detected stage 2–4 COPD who had a previous diagnosis of COPD was calculated. To gain further insights into the success and feasibility of microspirometry in this population, the proportion of patients with at least one out of three suboptimal exhalations was determined, and, in a subgroup, results of follow-up dynamic spirometry were compared with those from microspirometry.

### Patient and public involvement

Patients were involved in the design of this research by providing feedback on the study plan and participant-facing documents.

## RESULTS

### Recruitment

A total of 518 eligible participants were enrolled, 216 in the UK and 302 in Sweden. Baseline characteristics of the enrolled participants are shown in table 1. 5% of eligible participants declined to participate.

**Table 1** Baseline characteristics of enrolled participants in the two cohorts

| Characteristics and maintenance treatment at baseline | UK cohort (n=216) | Sweden cohort (n=302) |
|---|---|---|
| Demographics | | |
| Age (years), median (IQR) | 60 (53 to 67) | 68 (61 to 76) |
| Female sex, n (%) | 56 (26) | 73 (24) |
| Weight (kg), median±IQR | 83 (71 to 95) | 85 (75 to 95) |
| Body mass index (kg/$m^2$), median±IQR | 27.7 (24.7 to 31.3) | 27.6 (25.1 to 31.2) |
| Smoking history | | |
| Current smoker, n (%) | 123 (57) | 114 (38) |
| Ex-smoker, n (%) | 93 (43) | 188 (62) |
| Pack-years, median (IQR) | 32 (23 to 45) | 26 (15 to 38) |
| Qualifying diagnosis | | |
| STEMI, n (%) | 73 (34) | 147 (53) |
| NSTEMI, n (%) | 143 (66) | 129 (47) |
| Co-morbidities | | |
| Hypertension, n (%) | 84 (39) | 180 (63) |
| Diabetes mellitus, n (%) | 35 (16) | 67 (24) |
| Existing diagnosis of COPD, n (%) | 41 (19) | 21 (7) |
| Drug treatment at enrolment | | |
| Aspirin, n (%) | 214 (99) | 250 (88) |
| $P2Y_{12}$ inhibitor, n (%) | 203 (94) | 248 (88) |
| Oral anticoagulant, n (%) | 13 (6) | 44 (16) |
| Statin, n (%) | 203 (94) | 272 (96) |
| ACE inhibitor/ARB, n (%) | 177 (82) | 236 (83) |
| Beta blocker, n (%) | 186 (86) | 226 (80) |
| Oral corticosteroid, n (%) | 4 (2) | 12 (6) |
| Inhaled corticosteroid, n (%) | 35 (16) | 23 (8) |
| Inhaled long-acting $\beta_2$-agonist or muscarinic antagonists, n (%) | 26 (12) | 6 (4) |

ACE, angiotensin-converting enzyme; ARB, angiotensin II receptor blocker; COPD, chronic obstructive pulmonary disease; NSTEMI, non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction.

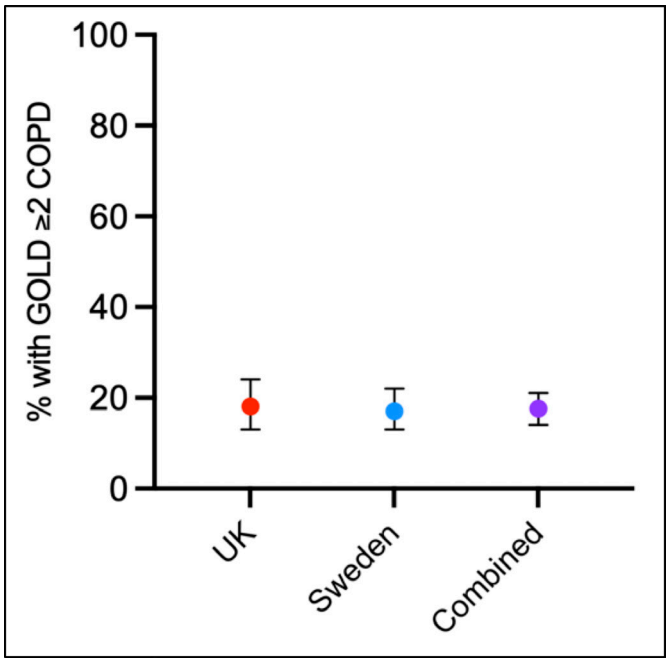


**Figure 1** Proportion of participants in each enrolled cohort with Global Initiative on Chronic Obstructive Lung Disease (GOLD) stage 2–4 chronic obstructive pulmonary disease (COPD). Dots and whiskers indicate proportion with a 95% CI.

### Detection of airflow obstruction by microspirometry

The number of participants meeting the primary study endpoint of GOLD 2–4 COPD assessed using microspirometry was 39 out of 216 in the UK cohort (18% (95% CI 13 to 24)) and 52 out of 302 (17% (13 to 22)) in the Swedish cohort. When the cohorts were combined, the prevalence of GOLD 2–4 COPD was 91 out of 518 (18% (14 to 21)) (figure 1).

When including any evidence of COPD ($FEV_1/FEV_6$ <0.7, ie, GOLD ≥1), the proportion in the combined cohort was 18% (15 to 22), with no significant difference between the countries: 19% (14 to 25) in the UK cohort and 18% (14 to 23) in the Swedish cohort. The prevalence of $FEV_1/FEV_6$<0.73 was 22% (17 to 28) in the UK cohort and 21% (17 to 26) in the Swedish cohort.

### Characteristics of patients with detected significant COPD

Of those in whom GOLD 2–4 COPD was detected, 76% had no prior diagnosis of COPD (figure 2A). Treatment with ICS was infrequent, with 15% receiving these at the time of enrolment (figure 2B). Mean (SD) CAT score was 13.8 (7.6) and 65% had a CAT score of ≥10 (figure 2C). Some 76% had a blood eosinophil count of ≥100/$mm^3$ and 20% ≥300/$mm^3$ (figure 3). These characteristics appeared similar between the two geographical cohorts (table 2).

### Comparison of those with and without detected significant COPD

Those with MI and detected GOLD stage 2–4 COPD were statistically significantly more likely to be ex-smokers rather than current smokers, have a previous diagnosis of COPD and have a diagnosis of heart failure as compared with those in the enrolled cohort without GOLD 2–4 COPD (table 3).

### Feasibility and accuracy of microspirometry in the hospitalised MI population

Across the two cohorts, there were 62 out of 518 (12%) participants with an existing clinical diagnosis of COPD at enrolment. In the UK cohort, 15 out of 41 (37%) with a prior diagnosis of COPD had $FEV_1/FEV_6$ <0.70, and 16 out of 41 (39%) had $FEV_1/FEV_6$ <0.73. In the Swedish cohort, 9 out of 21 (43%) with a previous COPD diagnosis had $FEV_1/FEV_6$ below 0.7, and 12 out of 21 (57%) had $FEV_1/FEV_6$ <0.73. In the combined cohort, 24 (39%) and 28 (46%) had $FEV_1/FEV_6$ below 0.7 and 0.73, respectively.

In the UK cohort, 46 of 216 enrolled participants (21%) performed at least one unsatisfactory exhalation. In the Swedish cohort, data on the microspirometry technique were collected on 95 participants, 49 of whom (52%) performed at least one unsatisfactory exhalation.

In the Swedish cohort, 9 participants with evidence of GOLD ≥2 airflow obstruction on microspirometry also

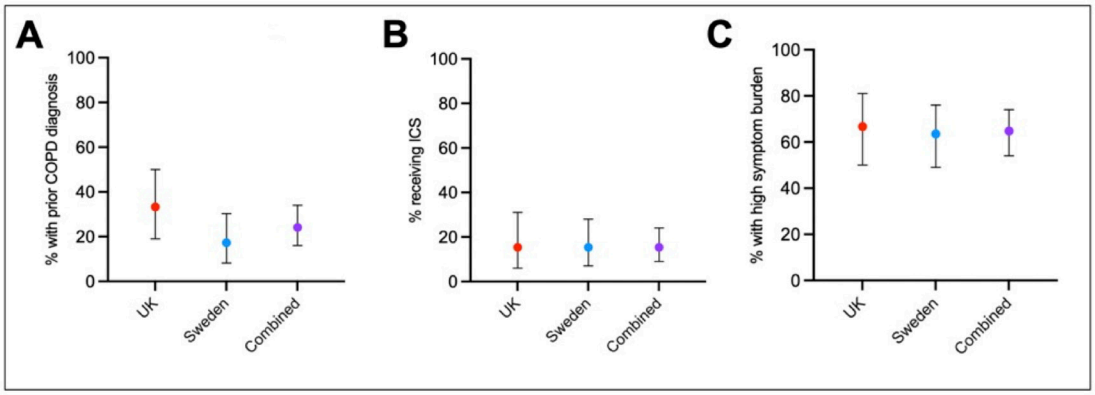


**Figure 2** Presence of (A) prior diagnosis of chronic obstructive pulmonary disease (COPD); (B) receiving inhaled corticosteroid (ICS); and (C) high symptom burden defined as COPD Assessment Test (CAT) score of 10 or greater, in the subgroup of participants with detected significant COPD. Dots and whiskers indicate proportion ±95% CI.

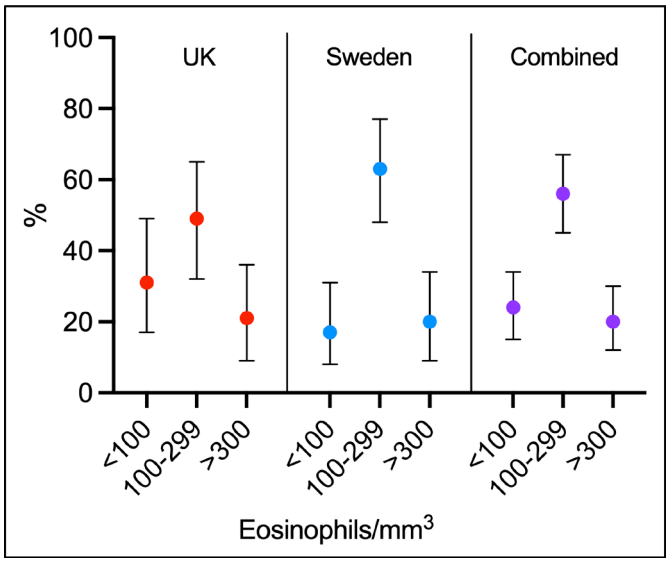


**Figure 3** Peripheral blood eosinophil counts in study participants with detected significant chronic obstructive pulmonary disease. Bars indicate proportion +95% CI.

underwent conventional spirometry after a median (IQR) follow-up time of 77 (71 to 93) days. Of these, spirometry confirmed the diagnosis in 5 (56%), whereas no evidence of significant COPD was found in the remaining 4 (44%).

## DISCUSSION

In this study, we have obtained an accurate microspirometer-derived estimate of the prevalence of clinically significant COPD among those with a significant smoking history hospitalised for acute MI.

Consistent between the two geographical cohorts, we found the prevalence of significant COPD (GOLD 2–4) determined by microspirometry to be around 18%. This is not a trivial prevalence considering that both countries have low smoking rates globally and that the hospitals were large university hospitals with generally relatively healthy and young populations. For comparison, the prevalence of COPD in the adult general European population is around 10%.[16]

Severity of COPD is classified by grade of airflow obstruction (GOLD 1–4), based on $FEV_1$ as a percentage of the predicted value ($FEV_1$%pred).[6] Based on the fact that GOLD stage 1 denotes mild COPD with normal $FEV_1$%pred and that previous mortality studies included moderate and severe COPD,[4 5] we chose to pre-specify GOLD stages 2–4 as the definition of clinically significant COPD. However, including GOLD 1 added only a small number of additional cases and did not substantially change the result. In those with detected COPD, there was evidence of a high burden of symptoms, defined as CAT score ≥10,[6] in around two-thirds of cases.

Notably, a large proportion of those with detected significant COPD had no prior diagnosis and were not receiving any inhaled treatment. This suggests a significant unrecognised and untreated burden of COPD in this population. Similarly, there were participants with a previous diagnosis of COPD in whom no evidence of significant airflow limitation was found. It is not known whether these cases had previously undergone spirometry, but it is recognised that lack of access to spirometry in primary care is an issue that leads to both under-diagnosis and over-diagnosis, a factor that has been exacerbated by the COVID-19 pandemic.[8 17]

There are compelling reasons for detecting COPD in patients who suffer from an acute MI or other cardiovascular diseases, given there are shared risk factors and considerable, multifaceted interplay between the two conditions.[18] The incidence of MI in those with COPD is greater than in those without, and risk further increases significantly after an exacerbation of COPD.[19 20] The presence of COPD in patients with MI confers a worse prognosis than in those without COPD, with evidence suggesting that at least some of this effect may be independent of other factors.[3] Mechanistically, the two conditions share inflammation as an underlying pathological process, which is a potentially powerful target for therapeutic modulation in both COPD and MI.[21]

Moreover, appropriately treating COPD has been shown to reduce cardiovascular risk. In the Informing the Pathway of Chronic Obstructive Pulmonary Disease

**Table 2** Characteristics of those with COPD and GOLD≥2 airflow limitation in the two cohorts and when combined

| Characteristic | UK cohort (n=39) | Sweden cohort (n=52) | Combined cohort (n=91) |
|---|---|---|---|
| Prior diagnosis of COPD, n (%, 95% CI) | 13 (33.3, 19 to 50) | 9 (17.3, 8.2 to 30.3) | 22 (24.1, 16 to 34) |
| Eosinophil count (cells/mm$^3$) | | | |
| <100, n (%, 95% CI) | 12 (30.8, 17 to 48) | 8 (17.4, 8 to 31) | 20 (23.5, 15 to 34) |
| 100 to 299, n (%, 95% CI) | 19 (48.7, 32 to 65) | 29 (63.0, 48 to 77) | 48 (56.5, 45 to 67) |
| ≥300, n (%, 95% CI) | 8 (20.5, 9 to 36) | 9 (19.6, 9 to 34) | 17 (20.0, 12 to 30) |
| CAT score | | | |
| Median (IQR) | 17 (6–23) | 12 (7–18) | 14 (12–17) |
| High symptom burden (CAT≧10), n (%, 95% CI) | 26 (66.7, 50 to 81) | 33 (63.5, 49 to 76) | 59 (64.8, 54 to 74) |
| Receiving inhaled corticosteroid, n (%, 95% CI) | 6 (15.4, 6 to 31) | 8 (15.4, 7 to 28) | 14 (15.4, 9 to 24) |

CAT, COPD Assessment Test; COPD, chronic obstructive pulmonary disease; GOLD, Global initiative on chronic Obstructive Lung Disease.

Table 3 Comparison of characteristics of those with and without GOLD 2–4 COPD in the combined cohort

| Characteristic | With GOLD 2–4 COPD (n=91) | Without GOLD 2–4 COPD (n=427) | Odds ratio (95% CI) | P value |
|---|---|---|---|---|
| Sex, n (%) | | | 0.88 (0.51 to 1.51) | 0.69 |
| Female | 21 (23.1) | 109 (25.5) | | |
| Male | 70 (76.9) | 318 (74.5) | | |
| Age, mean (SD) | 65.2±6.9 | 63.7±4.7 | – | 0.83 |
| Smoking, n (%) | | | 0.61 (0.39 to 0.97) | 0.038 |
| Current | 51 (23.1) | 188 (44.0) | | |
| Ex | 40 (76.9) | 239 (56.0) | | |
| Pack-years, mean±SD | 36.3±3.2 | 32.2±5.9 | – | 0.31 |
| Body Mass Index, mean±SD | 29.4±3.3 | 28.8±5.1 | – | 0.82 |
| Previous COPD diagnosis, n (%) | 22 (24.2) | 40 (9.4) | 3.09 (1.76 to 5.56) | 0.0003 |
| Comorbid conditions, n (%) | | | | |
| Asthma | 6 (6.6) | 37 (8.7) | 0.74 (0.32 to 1.80) | 0.68 |
| Hypertension | 44 (48.4) | 220 (51.5) | 0.88 (0.56 to 1.39) | 0.64 |
| Heart failure | 11 (12.1) | 24 (5.6) | 2.31 (1.05 to 4.87) | 0.036 |
| Atrial fibrillation | 9 (9.9) | 26 (6.1) | 1.69 (0.75 to 3.74) | 0.25 |
| Diabetes | 15 (16.5) | 86 (20.1) | 0.78 (0.42 to 1.40) | 0.47 |
| Previous stroke/TIA | 6 (6.6) | 18 (4.2) | 1.60 (0.64 to 3.97) | 0.41 |
| Type of infarction | | | 1.40 (0.88 to 2.21) | 0.16 |
| STEMI, n (%) | 45 (49.4) | 176 (41.2) | | |
| NSTEMI, n (%) | 46 (50.5) | 251 (58.8) | | |

COPD, chronic obstructive pulmonary disease; GOLD, Global initiative on chronic Obstructive Lung Disease; NSTEMI, non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction; TIA, transient ischaemic attack.

Treatment trial, 10 355 patients with COPD were randomised to triple therapy with LAMA/LABA/ICS or corresponding LABA/ICS or LAMA/LABA, and reported a reduced risk of exacerbation with triple therapy compared with double bronchodilation.[22] In a secondary analysis, all-cause and cardiovascular mortality in patients on triple therapy versus dual bronchodilator were reduced.[4] In the Efficacy and Safety of Triple Therapy in Obstructive Lung Disease trial, 8588 participants with moderate to very severe COPD and at least one exacerbation in the previous year were randomised to receive two times per day inhaled doses of triple therapy with ICS (at one of two doses), LAMA and LABA; or dual therapy with either LAMA+LABA or ICS+LABA. Triple therapy, including either dose of ICS, led to a significantly lower incidence of moderate or severe COPD exacerbations during the 52-week follow-up period.[23] Moreover, those in the triple therapy group receiving the higher ICS dose had reduced all-cause mortality compared with those receiving LAMA+LABA, suggesting ICS may be important in reducing mortality risk in this population. A post hoc analysis further suggested that the mortality reduction was driven by a lower frequency of cardiovascular events in those receiving ICS.[5] This is being prospectively explored in the ongoing Randomized, Double-blind, Parallel Group, Multi-center, Phase III Study to Assess the Efficacy of Budesonide, Glycopyrronium, and Formoterol Fumarate Metered Dose Inhaler Relative to Glycopyrronium and Formoterol Fumarate MDI on Cardiopulmonary Outcomes in Chronic Obstructive Pulmonary Disease (THARROS, NCT06283966). THARROS aims to randomise around 5000 participants and has a composite primary endpoint of time to first severe cardiac event, severe COPD exacerbation or cardiopulmonary death.

ICS may be more beneficial in COPD where eosinophil count is >300 cells/µL, and should also be considered if 100–300/µL, but avoided if <100/µL.[6 24] In our cohort, around three-quarters of those with detected COPD had an eosinophil count that would typically warrant consideration of ICS treatment, but ICS was only being received by 2 out of 15 patients (13%) with eosinophil count >300 cells/µL and 10 out of 56 (18%) with >100 cells/µL. This is likely to be due to both COPD not having been previously diagnosed and under-prescription of ICS in those with a known COPD diagnosis.

Characteristics of enrolled participants with and without detected COPD were also compared. Being an ex-smoker rather than a current smoker was more common in those with detected COPD than those without. This may suggest that developing symptoms of COPD leads to greater motivation (for patients, their support networks, and clinicians) for smoking cessation, while a healthy smoker

effect may be applied to those without COPD.[25] A prior diagnosis of heart failure was more common in those with detected COPD than those without. There is a well-established link between the two conditions, including shared risk factors, symptoms and influence on pulmonary artery pressures.[26]

The studies also sought to demonstrate the feasibility of performing microspirometry in patients hospitalised for MI. A small sub-analysis assessed the correlation of findings of microspirometry during hospitalisation with those from subsequent formal spirometry. While the findings agreed more often than not, in a significant proportion, there was an apparent disparity, which leads to some uncertainty around the value of either microspirometry or in-hospital testing in this population. Even if COPD-6 is validated for use without a post-bronchodilator test,[9] we cannot rule out that some of the patients with a positive screening for COPD may have had normalised values after optimal bronchodilation treatment. In our opinion, due to ease of access and low cost of the COPD-6 device, microspirometry is suitable for screening and making a preliminary diagnosis, but establishing a final diagnosis and making decisions on maintenance treatment should be based on regular dynamic spirometry. However, our work emphasises the importance of active screening for COPD in patients with previous MI, in order to optimise cardiopulmonary outcomes. Compared with<0.7, the $FEV_1/FEV_6$ threshold of 0.73 captured slightly more patients with a previously known COPD diagnosis, and may be preferable for screening.

The major strength of the study was the consecutive recruitment in two study populations from two different countries, where the similar result indicates true robust findings. Interpretation of the study is limited by some uncertainty in the reliability of COPD6 testing in this population, though it has been validated in others. Nevertheless, the findings from the small number that subsequently went on to have conventional spirometry mean that further work is warranted to determine that microspirometry is a valid method for a preliminary COPD diagnosis or only screening in this setting. Similarly, it is possible that conditions related to MI, such as heart failure, even if not clinically apparent, may have affected measurements, though this usually does so in a restrictive rather than obstructive pattern so the prevalence of COPD is unlikely to have been over-estimated.[27] Future work should also focus on longitudinal assessment of the association between microspirometry-determined COPD status and clinical outcomes in patients with MI.

## CONCLUSION

In this cohort of patients hospitalised for acute MI in Sweden and UK, around one in five patients with a history of smoking was found to have significant COPD based on microspirometry. Symptom burden was high and treatment rate with ICS was low. Among those diagnosed with COPD, around three-quarters had not been previously diagnosed with COPD. Detection of COPD in patients with acute MI has clear potential to reduce ongoing cardiopulmonary risk in this population and should be considered as a component of routine practice.

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