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CLINICAL RESEARCH

Undiagnosed airflow limitation in patients at cardiovascular risk

Troubles ventilatoires obstructifs non-diagnostiqués chez les patients à risque cardiovasculaire

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KEYWORDS
COPD;
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Summary
Background. — Chronic obstructive pulmonary disease (COPD) and cardiovascular diseases (CVD) share risk factors and impair each other's prognosis.
Aims. — To assess the prevalence of airflow limitation (AL) compatible with COPD in a population at cardiovascular risk and to identify determinants of AL.
Methods. — All consecutive patients referred to the cardiovascular prevention unit of a university hospital in 2009 were studied in a cross-sectional analysis. Patients answered questionnaires on socioeconomic status, medical history and lifestyle, and underwent extensive physical examinations, biological measures and spirometry testing. AL was defined as FEV1/FVC < 0.70, without any history of asthma. Determinants of AL were assessed using logistic regression.
Results. — The sample comprised 493 participants (mean age 57.4 ± 11.1 years); 60% were men, 18% were current smokers, 42% were ex-smokers and 10% of patients had a history of CVD.

Abbreviations: AL, airflow limitation; CHD, coronary heart disease; COPD, chronic obstructive pulmonary disease; CVD, cardiovascular disease; FEV1/FVC, forced expiratory volume in 1 s/forced vital capacity; LDL-C, low-density lipoprotein cholesterol.

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Ten-year risk of coronary heart disease (CHD) according to the Framingham equation was intermediate (10—20%) for 25% of patients and high (> 20%) for 10%. Prevalence of AL was 5.9% (95% confidence interval [CI] 4.0—8.3%) in the whole population and 4.3% (2.6—6.6%) among subjects in primary cardiovascular prevention. AL was independently associated with CVD (adjusted odds ratio 4.18, 95% CI 1.72—10.15; $P = 0.002$) but not with Framingham CHD risk. More than 80% of patients screened with AL had not been diagnosed previously and more than one in two patients was asymptomatic.
Conclusion. — Patients with CVD are at increased risk of AL and thus should benefit from AL screening as they are frequently asymptomatic.

MOTS CLÉS
BPCO ;
Troubles ventilatoires obstructifs ;
Spirometrie ;
Prevalence ;
Risque cardiovasculaire

Résumé
Contexte. — La broncho-pneumopathie chronique ostructive (BPCO) et les maladies cardiovasculaires (CV) présentent des facteurs de risques communs et leur association chez un même patient grève le pronostic.
L'objectif. — A été d'évaluer la prévalence des troubles ventilatoires obstructifs (TVO) dans une population à risque CV et d'analyser les déterminants de l'obstruction bronchique.
Méthodes. — Notre étude est basée sur les patients adressés au centre de détection et de prévention de l'athérosclérose du CHU de Toulouse en 2009. Les participants ont répondu à un questionnaire évaluant le niveau socioéconomique, les antécédents médicaux et le style de vie. Ils ont bénéficié d'un examen clinique et d'une spirométrie. Un TVO était diagnostiqué si VEMS/CV était inférieur à 0,70 sans antécédent d'asthme. Les déterminants du TVO ont été évalués par régression logistique.
Résultats. — L'échantillon comprend 493 participants (âge moyen : 57,4 ± 11,1 ans), dont 60 % d'hommes, 18 % des fumeurs actuels, 42 % d'anciens fumeurs et 10 % de sujets atteints de maladies CV. Le risque de maladie coronarienne à dix ans selon l'équation de Framingham était intermédiaire (10—20 %) pour 25 % des sujets et élevé (> 20 %) pour 10 %. La prévalence des TVO était de 5,9 % [IC95 % : 4,0—8,3 %] (4,3 % [IC95 % : 2,6 %—6,6 %] chez les sujets en prévention CV primaire). Le TVO était indépendamment associé aux maladies CV (OR ajusté = 4,18 [1,72—10,15] ; $p = 0,002$) mais pas au score de Framingham. Plus de 80 % des patients dépistés avec un TVO n'en avaient pas connaissance et un patient sur deux était asymptomatique.
Conclusion. — Les patients atteints de maladies CV ont un risque accru de BPCO qui est souvent asymptomatique. Ils devraient donc bénéficier d'un dépistage spirométrique.

Introduction

COPD affects approximately 7.5% of the adult population. By 2020, COPD is expected to be the third most common cause of death and the fifth most common cause of disability in the world [1,2]. Paradoxically, this disease is widely under-diagnosed [3], in part because symptoms appear progressively and become obvious only when lung function is already significantly impaired [4]. Besides, COPD is strongly associated with CVD [5—9] and, among subjects with CVD, the coexistence of COPD is associated with a raised morbidity including more frequent hospitalizations and worsening of symptoms [9—11]. In addition, cardiovascular deaths account for a significant part of the mortality in COPD patients.

COPD prevalence has been estimated in several studies among patients with CVD [12—15], but remains unknown in asymptomatic subjects at cardiovascular risk for whom the combination with COPD may worsen prognosis [9]. Such a situation should be avoided today as COPD is now considered as a preventable and treatable disease [16—18].

The aim of this study was to assess the prevalence of AL in a population at cardiovascular risk and to identify determinants of AL in this population.

Methods

Patients

This report is based on a cross-sectional study comprising 519 consecutive subjects referred to a preventive cardiology unit in a French university hospital between January 2009 and January 2010. Patients were either self-referred or referred by their primary care physician or cardiologist for cardiovascular risk assessment, management of risk factors and routine ambulatory screening for CVD. All of the patients were invited to participate in the study; 502 (97%) accepted. The study was approved by the appropriate ethics committee, in accordance with French law (*Comité de protection des personnes Sud-Ouest et Outre-Mer II*, number 2-08-25) and all participants signed an informed consent form attesting they had received information about the study and agreed to participate.

Data collection

An extensive questionnaire, derived from the questionnaire used in the French MONICA (monitoring trends and determinants in CVD) population surveys [19] carried out in the

same region, was administered to each participant by a trained and certified medical staff member. Data concerning socioeconomic status, personal and family medical history (including family history of premature myocardial infarction, i.e. before 55 years for the father or 65 years for the mother), drug intake, cardiovascular risk factors, pulmonary symptoms (cough, sputum, dyspnoea), lifestyle and quality of life were recorded. Educational level was assessed by the level of graduation or school dropout. Smoking status was described as never smoking (0 pack-years), past smoking (smoking cessation $\geq$ 1 year before inclusion in the study) or current smoking (estimated in pack-years). All subjects underwent a physical examination, blood sample collection, electrocardiography, exercise test and spirometry test. A maximal exercise test was defined as a peak heart rate $\geq$ 85% of the maximal predicted heart rate for a given age [20]. The exercise test was stopped when the participant was unable to continue or in the case of electrocardiographic or blood pressure abnormalities. Height, weight and arterial blood pressure (mean of two measurements performed with an automatic sphygmomanometer in a sitting position after $\geq$ 5 min of rest) were measured according to standardized protocols by the medical staff. Body mass index was calculated as weight divided by height squared (kg/m^2). Dyspnoea was quantified according to the modified Medical Research Council (MRC) scale [21]. Symptoms of chronic bronchitis were assessed using questions derived from the European Community Respiratory Health Survey [22]. Quality of life was measured by the EuroQol 5D scale [23].

Blood samples were taken after $\geq$ 10 h of overnight fast. LDL-C was determined by the Friedewald formula [24]. Diabetes was assessed for people with fasting blood glucose $\geq$ 7 mmol/L (126 mg/dL) or under hypoglycaemic drug treatment. The 10-year risk of CHD (hard event, i.e. myocardial infarction, coronary insufficiency or CHD death) was estimated with the Framingham equation (charts using LDL-C categories) in subjects without CVD (i.e., ischaemic heart disease, history of cerebrovascular disease or atherosclerosis in other arteries such as aorta, renal or lower limb arteries) [25].

Spirometry

Spirometry was conducted according to ATS/ERS guidelines [26,27] by means of a portable spirometer (MINISPIR, MIR Medical, Rome, Italy). All spirometric measurements were reviewed individually and graded for quality by an experienced senior pulmonologist (R.E.).

AL compatible with COPD was defined according to the Global initiative for Obstructive Lung Disease (GOLD) guidelines [16], when forced expiratory volume in 1 s (FEV_1) divided by forced vital capacity (FVC) was < 0.70, without any history of asthma. The severity of AL was staged according to the GOLD guidelines as mild (stage I), moderate (stage II), severe (stage III) or very severe (stage IV), respectively, for percent predicted $FEV_1 \geq 80\%$, $50\% \leq FEV1 < 80\%$, $30\% \leq FEV1 < 50\%$, and $FEV1 < 30\%$ (or $FEV1 < 50\%$ plus chronic respiratory failure), respectively. Because our population was at cardiovascular risk and because a maximal stress test was included in the check-up (performed on a single day) [20], postbronchodilator reversibility to beta-2 agonists could not be tested.

Statistical analysis

The statistical analysis was performed with Stata statistical software (version 9.2, STATA Corporation, College station, TX, USA). Prevalence of AL is given with 95% confidence interval (CI). In bivariate analysis, qualitative variables were compared with the χ^2-test (or bilateral Fisher's exact test, when necessary). Student's *t*-test (or the Mann-Whitney's test when the distribution of continuous variables was skewed or the hypothesis of homoscedasticity was not respected) was used to compare quantitative data according to categories of qualitative variables. As bivariate analyses were performed only to screen variables that should be introduced in multivariable models, no adjustment for multiple comparisons was performed. Independent determinants of AL were assessed with logistic regression. Variables associated with AL in bivariate analysis ($P < 0.20$) were introduced in the multivariable model. A backward procedure was applied to assess variables that were significantly and independently associated with AL ($P < 0.05$). Age and smoking status were kept in the model despite a *P*-value > 0.05 as they are well known risk factors for AL.

Results

Population sample

Overall, 502 of the 519 eligible subjects (97%) agreed to participate. Nine patients with $FEV_1/FVC < 0.70$ and a history of asthma were excluded from the analysis, as asthma and COPD could not be differentiated in these patients due to the lack of reversibility testing. Table 1 describes the 493 patients at cardiovascular risk enrolled in the study. Mean age was 57.4 ± 11.1 years. Of the study population, 60% were men, 18% were current smokers, 42% were ex-smokers and 10% had CVD. The 10-year risk of a CHD event (calculated only in patients without CVD), according to the Framingham equation (LDL-C chart) [25], was intermediate (between 10 and 20%) for 25% of patients and high (> 20%) for 10% of the sample.

Prevalence of airflow limitation

A total of 29 patients exhibited AL. Fig. 1 shows the prevalence of AL according to the GOLD classification [16] and the prevalence of AL or reported COPD. The overall prevalence of $FEV_1/FVC < 0.70$ (AL) was 5.9% (95% CI 4.0—8.3%). Twenty-three patients (79.5%) presented in GOLD stage I, 17% ($n = 5$) in GOLD stage II and 3.5% ($n = 1$) in GOLD stage III. None of the AL patients reached stage IV. The prevalence of $FEV_1/FVC < 0.70$ without previously known COPD (screened AL) was 5.1% (95% CI 3.3—7.4%) (i.e., 86% [$n = 25$] of AL patients had not been diagnosed previously). The prevalence of $FEV_1/FVC < 0.70$ or reported COPD (with $FEV_1/FVC \geq 0.70$) was 7.5% (95% CI 5.3—10.2%) (eight patients reported COPD but exhibited a normal FEV_1/FVC ratio).

Among the individuals in primary prevention of CVD, 19 exhibited AL: 79% ($n = 15$) in stage I, 16% ($n = 3$) in stage II, and 5% ($n = 1$) in stage III. The prevalence of AL was 4.3% (95% CI 2.6—6.6%). As in the whole population, most patients with AL (84%; $n = 16$) had not been diagnosed previously.

Table 1 Description of the study population.

	Patients at cardiovascular risk ($n = 493$)
Age (years)	57.4 ± 11.1
Men	294 (60)
Educational level ≥ high school completion	285 (58)
Smoking status	
Never	200 (41)
Past	206 (42)
Current	87 (18)
Diabetes	37 (8)
Antihypertensive drug treatment	126 (26)
Lipid-lowering drug	216 (44)
Body mass index (kg/m²)	26 ± 4.6
C-reactive protein (mg/L)	2.9 ± 4.6
Family history of premature myocardial infarction[a]	29 (6)
Framingham hard CHD risk[b]	
< 10%	256 (54)
10–20%	120 (25)
> 20%	49 (10)
History of cardiovascular disease[c]	47 (10)
History of COPD[d]	12 (2)

Data are number (%) or mean ± SD. CHD: coronary heart disease; COPD: chronic obstructive pulmonary disease.
[a] Family history of premature myocardial infarction, i.e. before 55 years old for the father or 65 for the mother.
[b] 10-year risk of hard coronary heart disease event according to the Framingham equation (chart using LDL-cholesterol categories) [25] in subjects without any history of cardiovascular disease.
[c] Ischaemic heart disease, history of cerebrovascular disease, atherosclerosis in other arteries such as
[d] Declared or treated COPD.

Determinants of airflow limitation

Table 2 shows the main characteristics of AL and non-AL participants. Male gender, smoking, lipid-lowering treatment and presence of CVD were significantly associated with AL in bivariate analyses. As expected, FEV_1 and FEV_1/FVC were significantly lower in the AL group. Subjects with AL had significantly more frequent mobility problems. Only 10% of AL patients reported cough and sputum production corresponding to the definition of chronic bronchitis, and 38% had dyspnoea (MRC grade I).

Table 3 shows the factors independently associated with AL. After adjustment for age, gender and smoking, the odds ratio for AL was 4.18 (95% CI 1.72–10.15, $P = 0.002$) in patients with, versus without, CVD. Independent determinants of AL among patients in primary prevention of CVD were assessed. The age, gender and Framingham CHD risk-adjusted odds ratio for AL was 3.76 (95% CI 1.33–10.57, $P = 0.012$) in patients with cumulative smoking ≥ 25 pack-years compared with those < 25 pack-years.

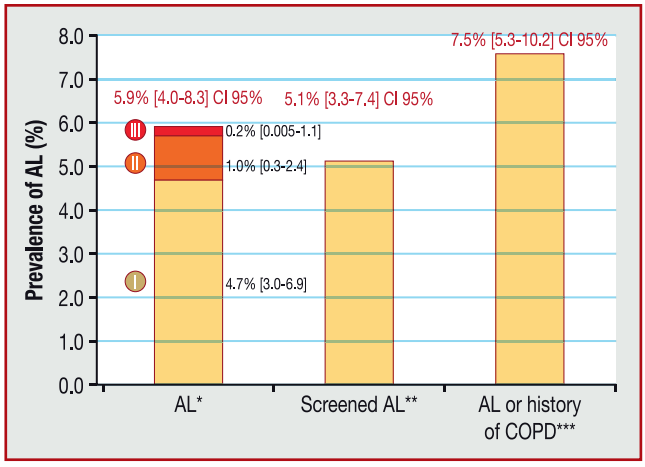

Figure 1. Prevalence of airflow limitation (AL) according to the GOLD classification [16] and history of Chronic obstructive pulmonary disease (COPD).
CI: confidence interval; COPD: chronic obstructive pulmonary disease.
*AL: forced expiratory volume in one second (FEV_1)/forced vital capacity (FVC) < 0.70.
**Screened AL: $FEV_1/FVC < 0.70$ without known COPD.
***AL or history of COPD: $FEV_1/FVC < 0.70$ or history of COPD (with $FEV_1/FVC \geq 0.70$).
I: stage I, mild AL ($FEV_1/FVC < 0.70$ and predicted $FEV_1 \geq 80\%$).
II: stage II, moderate AL ($FEV_1/FVC < 0.70$ and $50\% \leq$ predicted $FEV_1 < 80\%$).
III: stage III, severe AL ($FEV_1/FVC < 0.70$ and $30\% \leq$ predicted $FEV_1 < 50\%$).
No stage IV was observed in the sample: very severe AL ($FEV_1/FVC < 0.70$ and predicted $FEV_1 < 30\%$ or predicted $FEV_1 < 50\%$ plus chronic respiratory failure).

Discussion

Airflow limitation compatible with COPD was present in 5.9% of the patients at cardiovascular risk referred to the cardiovascular prevention unit, and in 4.3% of the patients in primary prevention of CVD. Almost 80% of AL patients exhibited a mild form (stage I) and more than one in every two patients was asymptomatic. After adjustment for age, gender and smoking, patients with CVD had a fourfold higher risk of AL than those without CVD. In the primary prevention population, smoking was the main predictor of AL after adjustment for age, gender and cardiovascular risk. More than 80% of patients had not been diagnosed previously (86% in the whole population and 84% in the subsample in primary prevention).

To the best of our knowledge, our study is the first to assess the prevalence of AL compatible with COPD across the range of patients referred for prevention of CVD, including subjects in primary prevention, those with known cardiovascular risk factors and people with a history of CVD. We also chose to report the prevalence of patients exhibiting a normal FEV_1/FVC ratio but a history of COPD, as these are COPD patients (probably improved by appropriate disease management following diagnosis), and thus had to be taken into account in prevalence estimation. Previously, the prevalence of COPD has been reported to range from 12 to 34% among patients with coronary artery disease [12–15].

Table 2 Main characteristics of airflow limitation (AL) and non-AL participants.

	Patients without AL ($n = 464$)	Patients with AL ($n = 29$)	*P*
Demographic			
Men	269 (58)	25 (86)	0.003
Age (years)	57.2 ± 11.0	59.4 ± 12.4	0.309
Educational level < high school completion	193 (42)	12 (41)	0.959
Cardiovascular risk factor			
Smoking status			
Never	190 (41)	10 (34)	0.601
Past	194 (42)	12 (41)	
Current	80 (17)	7 (24)	
Smoking (in pack-years)			
< 25	391 (85)	19 (65.5)	0.015
≥ 25	69 (15)	10 (34)	
Diabetes	36 (8)	1 (3)	0.714
Antihypertensive drug treatment	116 (25)	10 (34)	0.256
Systolic blood pressure (mmHg)	135 ± 17	138 ± 15	0.316
Diastolic blood pressure (mmHg)	81 ± 9	82 ± 7	0.384
Lipid-lowering drug treatment	196 (42)	20 (69)	0.005
Statins	114 (25)	14 (48)	
Fibrates	22 (5)	0 (0)	
Other lipid-lowering drugs	60 (13)	6 (21)	
Total cholesterol (mg/dL)	230 ± 50	210 ± 50	0.106
LDL-cholesterol (mg/dL)	140 ± 50	130 ± 40	0.226
HDL-cholesterol (mg/dL)	60 ± 20	60 ± 20	0.748
Triglycerides (mg/dL)	1.6 ± 1.6	1.2 ± 0.7	0.269
Body mass index (kg/m^2)	26 ± 4.6	24.9 ± 4.1	0.254
C-reactive protein (mg/L)	2.9 ± 4.6	3.2 ± 5.3	0.768
Family history of premature myocardial infarction[a]	27 (6)	2 (7)	0.685
Framingham CHD risk (hard event)[b]			
< 10%	247 (56)	9 (32)	0.1031
10—20%	116 (26)	4 (14)	
> 20%	44 (4)	5 (18)	
Personal history of cardiovascular disease[c]	37 (10)	10 (34)	< 0.001
Respiratory characteristic			
FEV_1 (L)[d]	3.2 ± 0.8	2.9 ± 0.9	0.021
FEV_1 (% predicted)	113 ± 17	95 ± 26	< 0.001
FEV_1/FVC[e]	80 ± 5	64 ± 7	< 0.001
Cough or sputum[f]	41 (9)	3 (10)	0.736
Dyspnoea (MRC scale)[g]			
Grade I	135 (46)	6 (38)	0.350
Grade II	127 (43)	7 (44)	
Grade III	19 (6)	1 (6)	
Grade IV	11 (4)	2 (12)	
Grade V	1 (1)	0 (0)	
EuroQol 5D scale			
No problem in mobility	383 (94)	20 (83)	0.014
No problem in self care	419 (99)	26 (100)	0.999
No problem in usual activity	391 (93)	25 (93)	0.719
No pain or discomfort	221 (54)	11 (44)	0.472
No anxiety or depression	210 (50)	12 (46)	0.880
Visual analogue scale[h]	73 ± 16	73 ± 18	0.865

Data are number (%) or mean ± SD. CHD: coronary heart disease; FEV_1/FVC: forced expiratory volume in 1 s/forced vital capacity; HDL: high-density lipoprotein; LDL: low-density lipoprotein.

[a] Family history of premature myocardial infarction, i.e. < 55 years for the father or < 65 years for the mother.

[b] Ten-year risk of hard CHD event according to the Framingham equation (chart using LDL-cholesterol categories) [25] in subjects without any history of cardiovascular disease.

[c] Ischaemic heart disease, history of cerebrovascular disease, atherosclerosis in other arteries such as aorta, renal or lower limb arteries.

[d] Forced expiratory volume in 1 s.

[e] Forced vital capacity.

[f] For ≥ 3 months during the past 2 consecutive years.

[g] Medical Research Council scale [21].

[h] The best health state you can imagine is marked 100 and the worst health state you can imagine is marked 0.

Table 3 Independent determinants of airflow limitation (AL).

	Odds ratio	[95% Confidence interval]	*P*
Women	1.00		
Men	3.55	[1.19—10.63]	0.023
Age (per 1-year increase)	1.01	[0.98—1.05]	0.559
Smoking < 25 pack-years	1.00		
Smoking ≥ 25 pack-years	2.15	[0.92—5.01]	0.076
Personal history of cardiovascular disease[a]			
No	1.00		
Yes	4.18	[1.72—10.15]	0.002

Variables initially introduced in the multivariable logistic regression model were gender, age, smoking, lipid-lowering-drug treatment, total cholesterol, Framingham coronary heart disease risk and personal history of cardiovascular disease.
[a] Ischaemic heart disease, history of cerebrovascular disease, atherosclerosis in other arteries such as aorta, renal or lower limb arteries.

In a study conducted by Soriano et al. in the Balearic Islands (Spain) [13], the prevalence of AL was 17.5% in middle-aged subjects without CVD, randomly selected from the general population; 19.2% in subjects with CVD; and 33.6% in a group of hospital patients with coronary artery disease. Overall, these prevalences were higher than in our study, which could probably be explained by the much lower proportion of never smokers among Spanish people (18% in the Spanish study vs 41% in our population). In the French working general population, the prevalence of AL was reported to range from 5.7 to 7.5% in two recent studies [28,29]. We found a prevalence of 5.9%, which is close to these results but lower than would have been expected for people with cardiovascular risk factors [28,29]. This relatively low prevalence could in part be explained by the low proportion of subjects exposed to occupational dusts and chemicals (0.8%, $n = 4$) or environmental tobacco smoke (passive exposure to cigarette smoke; 1%, $n = 5$) (data not shown). Maybe more importantly, the mean age of the sample (57.4 ± 11.1 years) was relatively low, whereas COPD is a disease preferentially developing among people older than 60 years [30]. Moreover, the level of cardiovascular risk of our population was actually relatively low, as shown by the Framingham 10-year risk of CHD [25], which was < 10% for 54% of the sample. Here again, the relatively young age of the population could be involved, as increasing age is a risk factor for CVD.

Surprisingly, smoking was not associated with AL in the multivariable analysis conducted in the whole population, whereas the relationship was significant before adjustment. A first explanation is a possible lack of power (the adjusted odds ratio was very close to the significance level: $P = 0.076$). Moreover, the effect of smoking on AL could have been confounded by the association between AL and CVD or gender. Indeed, on the one hand, smoking and CVD are frequently associated (in our sample, 74% of patients with CVD were smokers vs only 58% of patients without CVD [$P = 0.027$]). On the other hand, smoking and male gender are also frequently associated (in our sample, 70% of men were smokers vs only 44% of women [$P < 0.001$]). Adjusting for gender and CVD took partly into account the effect of smoking on AL. In a similar way, lipid-lowering drug treatment was significantly associated with AL in univariate analysis, but this association did not remain after adjustment (in particular after the introduction of CVD as an adjustment covariate). Given that all subjects presenting with both AL and CVD were treated with a lipid-lowering drug, CVD carried very similar information to that carried by lipid-lowering drugs in our database. Consequently, adjusting for CVD removed the significant statistical link observed between AL and lipid-lowering-drug treatment in univariate comparisons.

The combination of COPD with CVD is known to be associated with poorer prognosis [9—11]. Observational data have suggested that slowing down COPD progression might help to reduce cardiovascular morbidity and mortality, which are themselves associated with more severe pulmonary symptoms [31] and COPD exacerbation [32]. Therefore, there is a good rationale for screening COPD in order to manage the disease as soon as possible, prevent exacerbation, and thus, reduce the risk of cardiovascular events. The increased risk of AL among subjects with CVD and the low proportion of patients with respiratory symptoms among those with AL, emphasize the need for a systematic screening of AL in these patients, irrespective of their smoking status or respiratory symptoms. Among patients without CVD, such a screening could be proposed in people who have smoked 25 pack-years or more.Several mechanisms could explain the association between AL and CVD. First, the two diseases share similar risk factors such as male gender, age and smoking. A very recent study showed that COPD is not more frequent among hospital patients with ischaemic heart disease compared with 'control' hospital patients, when traditional cardiovascular risk factors are neutralized by matching cases and controls according to gender and age and adjusting for smoking and other traditional cardiovascular risk factors. The authors concluded that the higher prevalence of cardiovascular risk factors in COPD patients could explain the often-reported association between COPD and CVD [33]. COPD-associated systemic inflammation could be another explanation, as it is known that chronic inflammatory diseases are often associated with increased cardiovascular risk. For instance, this point has been underlined clearly by the recent recommendations on rheumatoid arthritis and atherosclerotic CVD [34]. Besides, COPD and CVD could both belong to the so-called 'chronic systemic inflammatory syndrome' [35]. Finally, COPD is associated with decreased daily life activity, even when airflow obstruction is only mildly to moderately impaired [36]. Interestingly, reduced physical activity is a risk factor for both COPD and CVD occurrence.

It is also known to impair the prognosis of the two diseases [37—39].

Limitations

A limitation of the study is the lack of postbronchodilator spirometry. Indeed, airflow obstruction was defined according to prebronchodilator spirometry results, in line with several recent epidemiological studies [28,29,40]. Given that salbutamol is associated with spontaneous arrhythmias, its use in patients at cardiovascular risk after a stress test was potentially dangerous [41]. Consequently, asthma could not be differentiated from COPD in patients with altered spirometry, and, for this reason, known asthmatic subjects were excluded from the analyses, which may have underestimated the prevalence of AL.

Conclusions

Among people attending a CVD prevention unit, we identified two groups of subjects with an increased probability of AL: patients with CVD and, in primary prevention, people smoking 25 pack-years or more. As COPD is frequently asymptomatic but may impair cardiovascular prognosis, these patients should be systematically screened for AL.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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