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Chronic obstructive pulmonary disease in patients with coronary heart disease worsens long-term prognosis after percutaneous coronary interventions

Aim To evaluate the incidence rate of major adverse cardiovascular events (MACVE) in the long-term following percutaneous coronary interventions (PCCI) in patients with acute and chronic ischemic heart disease (IHD) and the contribution of concurrent chronic obstructive pulmonary disease (COPD) to the long-term prediction.

Material and methods This prospective cohort study included 254 patients with IHD and concurrent COPD and 392 patients with IHD without COPD. PCCI was performed in all patients: for acute coronary syndrome in 295 patients and for chronic IHD in 351 patients. The follow-up period lasted for up to 36 months. The outcome was a composite endpoint, MACVE, that included cardiovascular death, myocardial infarction, stroke, repeated unscheduled myocardial revascularization (MR), and the time to the event.

Results The age-standardized incidence of MACVE in patients with IHD and COPD was 31.5 vs. 23.2% in patients with IHD without concurrent COPD (p=0.025), primarily due to an increased frequency of repeated unscheduled MR (20.5 vs. 14.0%, p=0.041), which was associated with earlier occurrence of adverse events (p<0.001). Repeated unscheduled MR was more frequently performed in patients with moderate COPD; the frequency of MR decreased with increasing severity of COPD, whereas the total incidence of cardiovascular death, myocardial infarction, and stroke was the highest in patients with severe and very severe COPD.

Conclusion The presence of concurrent COPD increases the relative risk of MACVE 1.36 times (95% confidence interval: 1.05–1.75) and facilitates their earlier development. Repeated unscheduled MR makes the major contribution to the increase in the total risk (relative risk, 1.46; 95% confidence interval: 1.03–2.06). The increase in severity of COPD is associated with the increase in total frequency of MACVE (p=0.005).

Keywords Percutaneous coronary interventions; chronic obstructive pulmonary disease; ischemic heart disease; major adverse cardiovascular events

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Prognosis following percutaneous coronary intervention (PCI) is affected by the presence of concomitant diseases, which can confound cardiovascular prognosis, as well as traditional atherosclerosis risk factors and technical issues associated with the intervention. Thus, the role of diabetes mellitus (DM) and chronic kidney disease (CKD) has been carefully studied in numerous clinical studies [1–3]. Such studies are largely based on the fact that laboratory indicators, which are used as diagnostic criteria for DM and CKD, are routinely assessed in all hospitalized patients. This provides the opportunity to analyze larger electronic medical databases, including retrospectively. However, the diagnosis of chronic obstructive pulmonary disease

(COPD), which is as prevalent as DM and CKD, requires spirometry and broncholytic tests, which are not included in coronary protocols. Thus, in most clinical studies of the effect of COPD on the prognosis in patients with coronary artery disease (CAD), the diagnosis of COPD was neither reliably verified nor characterized in detail. This is supported by the latest editions of Global Strategy for the Diagnosis, Management, and Prevention of COPD (GOLD) [4–7]. Evidence from registries and retrospective studies is far less reliable for COPD than for DM and CKD, since most COPD cases remain undiagnosed and end up in the control groups producing biases. There are much fewer prospective studies evaluating the contribution of COPD in long-term cardiovascular prognosis following PCI than those that assess the impact of COPD on immediate and long-term outcomes of coronary artery bypass grafting (CABG). However, the social and medical significance of COPD has grown in recent years: it is now among the top three diseases causing the highest number of deaths worldwide and ahead of DM. The prevalence of these two diseases in the general population is comparable, especially in patients over the age of 40 years [7]. Therefore, it appears relevant to assess the impact of COPD on the long-term prognosis for patients with acute and chronic CAD after PCI.

Objective

To assess the rate of severe cardiovascular adverse events (AEs) in the long-term period after PCI in patients with acute and chronic CAD and the contribution of concomitant COPD to long-term prognosis.

Material and Methods

The prospective parallel-group cohort study included 646 patients who met the inclusion and exclusion criteria: 295 patients after PCI for acute coronary syndrome (ACS) and 351 patients after PCI for chronic CAD. Inclusion criteria: signed informed consent; age ≥40 years; stress test confirmed stable exertional angina or myocardial infarction (MI) or unstable angina; PCI with stenting performed during the index hospital treatment; active smoking status at inclusion or smoking cessation within less than 12 months before the inclusion and total smoking history ≥10 pack-years. All patients underwent spirometry (and inhalation test with salbutamol 400 μg if bronchial obstruction was detected). Patients were grouped for the follow-up depending on the presence of COPD as shown by spirometry. The severity of airflow limitation was determined by the spirometric criteria according to GOLD [7]. A total of 254 patients were included in the COPD group. Most of the patients were admitted to the Research Institute Krasnodar Regional Clinical Hospital No. 1. The control group included 392 patients who were randomized from 822 patients who met the inclusion criteria but did not have COPD. Exclusion criteria: history of myocardial revascularization (MR); valvular heart defects with indications for surgical treatment; left ventricular ejection fraction (LVEF) <35% one week after MR; glomerular filtration rate (GFR) < 30 mL/min/m^2 (CKD-EPI); resistant arterial hypertension; lung diseases other than COPD; cancer; diffuse connective tissue diseases; PCI complications.

As well as spirometry, all patients underwent electrocardiography and echocardiography according to the guidelines of the American Society of Echocardiography, as well as coronary angiography (CAG). Patients with chronic CAD were subjected to stress ECG tests or technetium-99m tests to verify myocardial ischemia. Laboratory tests included all routine measurements for CAD patients under the local protocol and current guidelines (glucose, creatinine, lipid profile, coagulation profile, troponin I, etc.). Statins, antiplatelet drugs (acetylsalicylic acid + clopidogrel), renin-angiotensin system inhibitors, and beta-blockers (if indicated) were ordered for all patients.

The outcome was a composite endpoint that included any of the following events: cardiovascular death, MI, stroke, repeated emergency MR. Time to endpoint was registered in months. Scheduled repeat visits to the hospital were made within 12 months and at the end of the study (up to 36 months); in case of any outcome within the composite endpoint, emergency visits were made. Phone calls were made every three months to inquire about the patient's vital status, treatment, and preliminary evaluation of the clinical outcomes of interest. The study protocol was approved by the local ethics committee.

Statistical analysis was performed using the STATISTICA 10.0 software suite (StatSoft Inc., USA). The tabular data are provided as the mean and standard deviation (M±SD) for near-normal distributions and the median and interquartile range (Me (Q1; Q3) for non-normal distributions. The relative rates are presented as percentages. Clinically significant effects are expressed as odds ratio (OR) and 95% confidence interval (CI). The Mann–Whitney test was used to compare the two independent groups; the χ^2 test was used to compare the relative proportions. The Kaplan–Mayer test was used to compare time to outcomes of interest, and the curves were compared using the Cox test. The critical significance level was p=0.05.

Results

As can be seen from the baseline patient characteristics presented in Table 1, there are no statistically significant differences between the groups other than small differences in age and the SYNTAX scores. The mean age was 1.7 years higher; the median SYNTAX score was 3 points higher in the COPD group than in the control group, respectively (however, patients of both groups were referred to the same risk category: SYNTAX ≤ 22).

110 (43.3%), 79 (31.1%), 47 (18.5%) and 18 (7.1%) patients had mild, moderate, severe and very severe COPD, respectively. Twelve months after PCI, there were no differences between the groups in terms of adherence to the main types of drugs affecting the cardiovascular prognosis (Figure 1). The high adherence to drug treatment may have been due to routine phone contacts with patients, during which they were interviewed about the drugs administered and outcomes of interest.

While the median follow-up was 21 months, the maximum was 36 months. The rates of the composite endpoint and its elements – i.e., cardiovascular events reported during the follow-up periods (cardiovascular death, MI, stroke, repeat emergency MR) – are presented in Table 2 for the entire sample; for ACS following PCI – in Table 3; for chronic CAD following PCI – in Table 4.

The distribution of the rates of cardiovascular events in patients with concomitant COPD was similar to that observed both in patients after PCI with ACS and those with chronic CAD, although there was a clear predominance of all adverse events other than stroke in patients with COPD. Statistical significance of differences was achieved only in the rates of the composite endpoint and repeat MR (including repeat PCI). Three patients in both the COPD and control groups underwent two repeat emergency MR procedures during the follow-up period. Due to the mean age of patients being relatively higher in the COPD group than in the control group, a statistical correction for age was added using direct standardization, having possible consequences for the incidence of adverse outcomes. Afterward, the significance of differences in the composite endpoint was preserved, although somewhat decreased (Table 5). The higher rates of repeat emergency MR contributed the most to those differences.

In patients with COPD and CAD, the odds ratio (OR) was 1.36 (95% CI: 1.05–1.75) for all severe cardiovascular AEs, 1.46 (95% CI: 1.03–2.06) for repeat emergency MR, and 1.62 (95% CI: 1.03–2.32) for repeat PCI. The incidence of severe cardiovascular AEs 31.5% and 23.2% in the COPD group and the control group, respectively (p=0.025). Both in the COPD and control groups, repeated episodes of CAD were most often stable exertional angina, for which repeat emergency MR was carried out; in both groups, the rates of PCI clearly prevailed over CABG.

Table 1. Baseline clinical characteristics of the patient groups

Indicator		CAD and COPD (n=254)	CAD (n=392)	p
Age, years (M ± SD)		59.4 ± 7.7	57.7 ± 8.2	0.007
Male, n (%)		242 (95.3)	370 (94.4)	0.75
Smokers, n (%)		219 (86.2)	355 (90.6)	0.09
History of smoking (stopped < 1 year ago), n (%)		35 (13.8)	37 (9.4)	–
Time of smoking cessation, months (Me [Q1; Q3])		8 [4; 10]	7 [3; 10]	0.41
Smoking experience, pack-years (Me [Q1; Q3])		40 [29; 47]	35 [28; 44]	0.012
ACS admission, n (%)		119 (46.9)	176 (44.9)	0.63
STE-ACS, n (% of patients with ACS)		77 (64.7)	124 (70.5)	0.30
AH, n (%)		186 (73.2)	293 (74.7)	0.74
History of MI, n (%)		99 (40.0)	137 (34.9)	0.34
DM, n (%)		46 (18.1)	66 (16.8)	0.76
LVEF	> 50%, n (%)	168 (66.1)	277 (70.7)	0.23
	36–50%, n (%)	86 (33.9)	115 (29.3)	–
SYNTAX score (Me [Q1; Q3])		12 [9; 16]	9 [6; 13]	< 0.001
Number of stents implanted, n (Me [Q1; Q3])		1 [1; 2]	1 [1; 1]	0.021
GFR (CKD EPI) < 60 mL/min/1.73 m^2, n (%)		47 (18.5)	69 (17.6)	0.66
Total cholesterol, mmol/L (M ± SD)		5.2 ± 1.5	5.4 ± 1.5	0.098
LDL-C, mmol/L (M ± SD)		3.44 ± 1.14	3.57 ± 1.22	0.18
HDL-C, mmol/L (M ± SD)		1.07 ± 0.25	1.08 ± 0.26	0.63

COPD – chronic obstructive pulmonary disease; CAD – coronary artery disease; ACS – acute coronary syndrome;
AH – arterial hypertension; MI – myocardial infarction; DM – diabetes mellitus; GFR – glomerular filtration rate; TC – total cholesterol;
LDL-C – low-density lipoprotein cholesterol; HDL-C – high-density lipoprotein cholesterol; LVEF – left ventricular ejection fraction.

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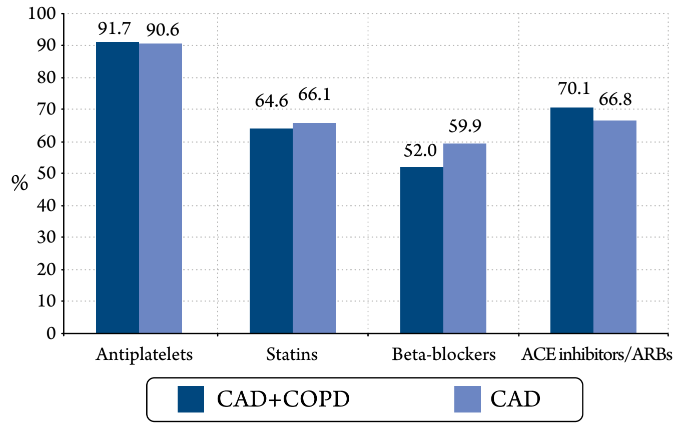
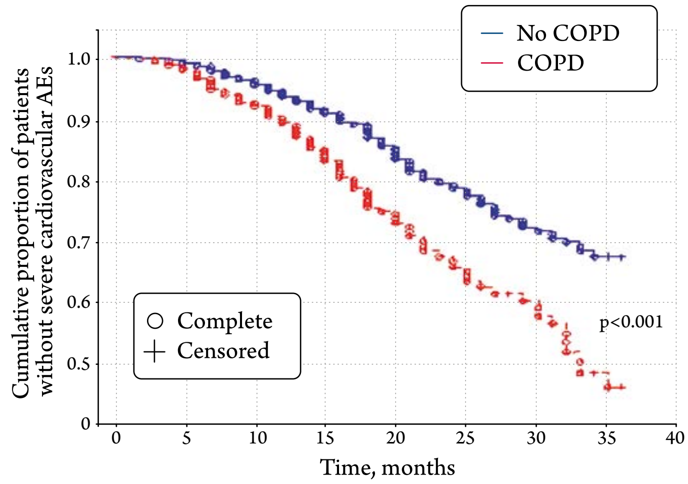
Figure 1. Adherence to drug treatment 12 months after PCI



ACE – angiotensin-converting enzyme;
ARB – angiotensin II receptor blocker;
CAD – coronary artery disease;
COPD – chronic obstructive pulmonary disease;
PCI – percutaneous coronary intervention.

Figure 2. Time to the first severe cardiovascular AE depending on the presence of COPD



AE – adverse event;
COPD – chronic obstructive pulmonary disease.

The Kaplan–Mayer curves showed earlier onset of severe cardiovascular AEs and repeat emergency MR in patients with CAD and COPD during the three-year follow-up (Figure 2 and Figure 3).

COPD severity was associated with the incidence of severe cardiovascular AEs: the incidence of severe cardiovascular AEs increased as COPD became more severe; p=0.005 (Figure 4).

However, a separate analysis of the incidence of cardiovascular events showed that the cumulative rates of cardiovascular death, MI, and stroke were significantly higher in patients with COPD grade III and IV ($p < 0.001$). No such correlation was reported for the repeated emergency MR procedures: repeat MR was performed less frequently in patients with most severe COPD (Figure 5 and Figure 6).

Discussion

A similar distribution of the incidence of severe cardiovascular AES among patients following scheduled and emergency PCIs performed allowed them to be included in a single group. The combination into a single group was also also justified according to the diagnosis of a patient experiencing acute MI with post-infarction cardiosclerosis in 28 days, i.e., he/she then has chronic CAD [8]. However, most of the patients who underwent scheduled PCI had a history of MI. In our study, COPD showed in CAD patients a clear negative effect on the

Table 2. Long-term incidence of cardiovascular events after PCI depending on the presence of COPD

Endpoints	CAD with COPD, n (%) (n=254)	CAD without COPD, n (%) (n=392)	p
Cardiovascular death	13 (5.1)	14 (3.6)	0.45
MI	20 (7.9)	20 (5.1)	0.21
Stroke	8 (3.2)	12 (3.1)	0.87
CABG	15 (5.9)	18 (4.6)	0.58
Repeat PCI	42 (16.5)	40 (10.2)	0.025
Repeat MR	54 (21.3)	55 (14.0)	0.022
Composite endpoint	85 (33.5)	91 (23.2)	0.006

PCI – percutaneous coronary intervention;
COPD – chronic obstructive pulmonary disease;
CAD – coronary artery disease; MI – myocardial infarction;
CABG – coronary artery bypass grafting;
MR – myocardial revascularization.

Table 3. Long-term incidence of cardiovascular events after PCI for ACS depending on the presence of COPD

Endpoints	ACS with COPD, n (%) (n=119)	ACS without COPD, n (%) (n=176)	p
Cardiovascular death	5 (4.2)	5 (2.8)	0.53
MI	10 (8.4)	7 (4.0)	0.13
Stroke	5 (4.2)	4 (2.3)	0.49
CABG	6 (5.0)	8 (4.6)	1.00
Repeat PCI	18 (15.1)	18 (10.2)	0.21
Repeat MR	23 (19.3)	26 (14.8)	0.34
Composite endpoint	40 (33.6)	38 (21.6)	0.023

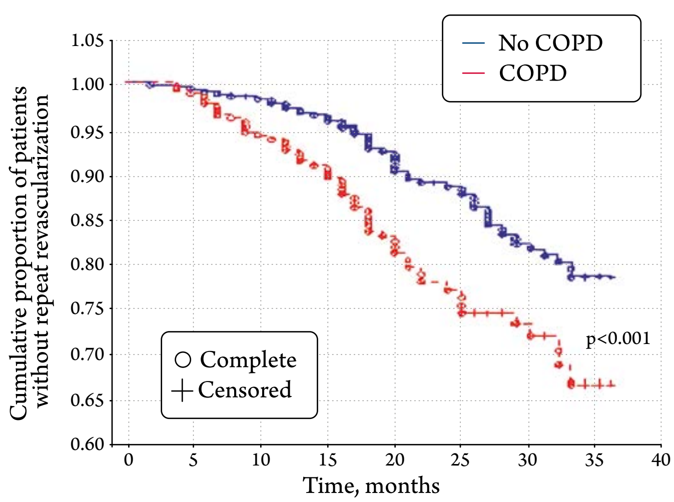
PCI – percutaneous coronary intervention;
ACS – acute coronary syndrome; COPD – chronic obstructive pulmonary disease; MI – myocardial infarction; CABG – coronary artery bypass grafting; MR – myocardial revascularization.

Table 4. Long-term incidence of cardiovascular events after PCI for chronic CAD depending on the presence of COPD

Endpoints	Chronic CAD with COPD, n (%) (n=135)	Chronic CAD without COPD, n (%) (n=216)	p
Cardiovascular death	8 (5.9)	9 (4.2)	0.46
MI	10 (7.4)	13 (6.0)	0.66
Stroke	3 (2.2)	8 (3.7)	0.65
CABG	9 (6.7)	10 (4.6)	0.47
Repeat PCI	24 (17.8)	22 (10.2)	0.051
Repeat MR	31 (23.0)	29 (13.4)	0.028
Composite endpoint	45 (33.3)	53 (24.5)	0.087

PCI – percutaneous coronary intervention; CAD – coronary artery disease; COPD – chronic obstructive pulmonary disease; MI – myocardial infarction; CABG – coronary artery bypass grafting; MR – myocardial revascularization.

Figure 3. Time to the repeat emergency MR depending on the presence of COPD



MR – myocardial revascularization; COPD – chronic obstructive pulmonary disease.

cardiovascular prognosis, resulting in a 1.36-fold increase in the incidence of severe cardiovascular AEs within the follow-up period of up to 36 months. All patients included in our sample were either smokers or had stopped smoking not more than 12 months before their inclusion in the study. The prevalence of DM, arterial hypertension, reduced GFR, and total cholesterol and LDL cholesterol levels were also comparable in both groups. Therefore, the differences between the groups in the incidence of severe cardiovascular AEs following age adjustment were considered to be the consequences of COPD. The fact that statistically significant differences were not found for all the outcomes reported in the study may be due both to the absence of differences and

Table 5. Age-adjusted long-term incidence of cardiovascular events after PCI depending on the presence of COPD

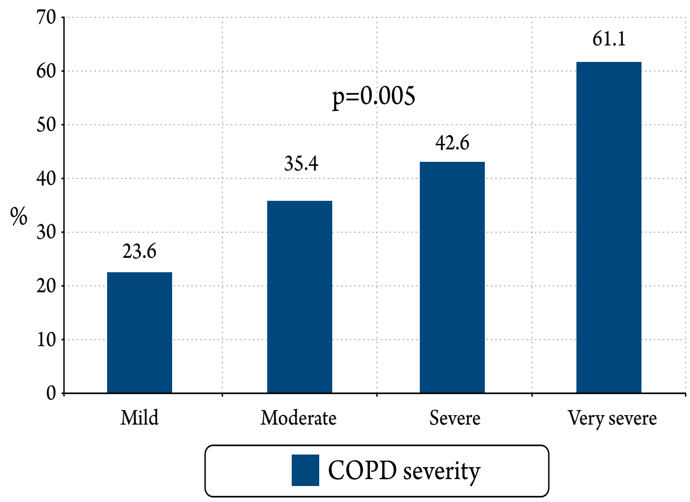
Endpoints	CAD with COPD, n (%) (n=254)	CAD without COPD, n (%) (n=392)	p
Cardiovascular death	11 (4.3)	14 (3.6)	0.78
MI	20 (7.9)	20 (5.1)	0.21
Stroke	7 (2.8)	12 (3.1)	0.99
CABG	15 (5.9)	18 (4.6)	0.58
Repeat PCI	40 (16.5)	40 (10.2)	0.049
Repeat MR	52 (20.5)	55 (14.0)	0.041
Composite endpoint	80 (31.5)	91 (23.2)	0.025

COPD – chronic obstructive pulmonary disease; MR – myocardial revascularization; MI – myocardial infarction; CABG – coronary artery bypass grafting; PCI – percutaneous coronary intervention.

the relatively short duration of follow-up given the sizes of the groups.

Progression of atherosclerosis or stent thrombosis/restenosis is the first reason given for repeat emergency PCI or CABG and newly onset MI. Our findings confirm that atherosclerosis worsens faster in patients with COPD: the appearance or recurrence of exertional angina is associated with the growth of a plaque in the vessel lumen or stent restenosis, while the development of MI is linked to local thrombosis occuring near the plaque. There is also evidence of an increased risk of stent thrombosis in COPD patients [9]. We previously established that repeat MR is rather due to coronary stenosis caused by atherosclerosis in the non-stented areas than stent restenosis [10].

Figure 4. Incidence of severe cardiovascular Es depending on the severity of COPD (comparison of incidence distribution using the χ2 test)



AE – adverse event; COPD – chronic obstructive pulmonary disease.

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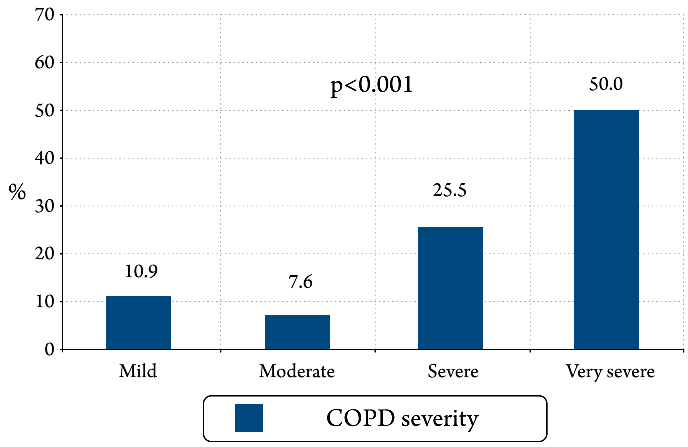
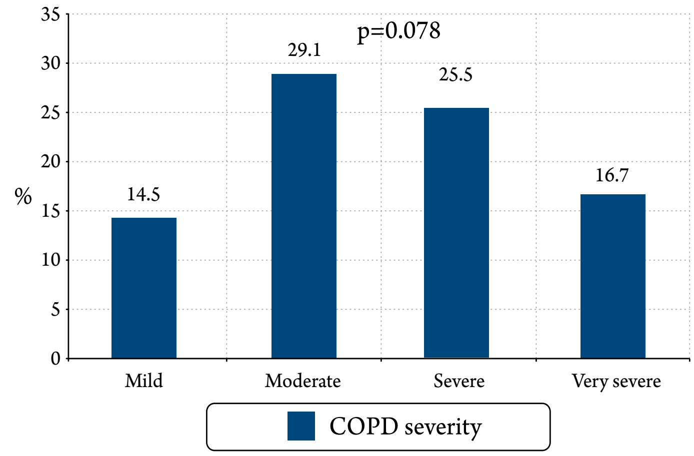
Figure 5. Cumulative prevalence of cardiovascular death, MI, and stroke depending on COPD severity (comparison of incidence distribution using the χ2 test)



MI – myocardial infarction;
COPD – chronic obstructive pulmonary disease.

Figure 6. Incidence of repeat emergency MR depending on the severity of COPD (comparison of incidence distribution using the χ2 test)



MR – myocardial revascularization;
COPD – chronic obstructive pulmonary disease.

Although not even a slight increase in stroke incidence was reported in the COPD group of our sample, such an association was found in other studies, e.g., the risk of ischemic stroke increased 1.39-fold (95% CI: 1.30–1.47) within 12 months after COPD exacerbation [11]. As well as the atherothrombotic mechanism, which has not been studied separately, higher prevalence of atrial fibrillation (AF) may increase the incidence of stroke rates in COPD patients: AF is known to increase the risk of thromboembolic stroke.

Cardiovascular death was more common in COPD patients; however, the statistically insignificant difference between the groups can probably be attributed to the relatively small number of patients and relatively low incidence of this outcome. Progression of atherosclerosis with the involvement of a coronary plaque can lead to repeat MI, which may be fatal. Nevertheless, COPD patients are known to have stent thrombosis more often, the most likely outcome of which is MI, which can lead to death [9]. Another possible and relatively frequent cause of cardiovascular death is ventricular arrhythmias. LV scarring can cause fatal ventricular rhythm disorders in patients with a history of MI. A history of MI was relatively more frequent in the COPD group (40.0 vs. 34.9), although the differences were statistically insignificant. While MI with low LVEF has the most significantly negative effect on long-term prognosis, such patients were not included in the study to avoid the biasing effect of this factor in evaluating the prognostic role of COPD.

Pathological systemic inflammatory response, oxidative stress, endothelial dysfunction, hypoxemia, myocardial remodeling, and hemostasis disorders that increase the risk of thrombotic events are usually considered as pathogenetic factors for additional deterioration of cardiovascular prognosis in patients with CAD and concomitant COPD [12–15]. The roles of these pathophysiological processes may vary at different stages of COPD. Smoking is a leading cause of the emergence and maintenance of systemic inflammation [16, 17]; the process involves not only lung parenchyma but also vascular endothelium [18–20].

The systemic effect of persistent chronic inflammation is caused by numerous immune mediators, including proinflammatory cytokines (interleukin-1β, interleukin-6, interleukin-8, tumor necrosis factor-α), C-reactive protein, fibrinogen, etc., which affect the cardiovascular system. Other mechanisms that increase the risk of atherothrombotic events in patients with concomitant COPD include higher platelet reactivity and prothrombotic changes in the plasma factor of hemostasis [21, 22]. Longer double antiplatelet therapy following PCI may be justified in such patients; however, there is as yet no clinical trial data on which basis such a recommendation could be made.

Conclusion

The presence of concomitant chronic obstructive pulmonary disease increases the odds ratio of severe cardiovascular adverse events (cardiovascular death, myocardial infarction, stroke, repeat emergency myocardial revascularization) by 1.36 times (95% CI: 1.05–1.75) as well as contributing to their earlier development. Repeat emergency myocardial revascularization contributes significantly to increased total risk, OR 1.46 (95% CI: 1.03–2.06), mainly due to the increased

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frequency repeat PCI, OR 1.62 (95% CI: 1.03–2.32). The increasing severity of COPD is associated with a higher total frequency of severe cardiovascular adverse events (p=0.005), but this association is not one-way in relation to the composite endpoint of severe cardiovascular adverse events: repeat emergency myocardial revascularization performed more often in patients with moderate COPD became less frequent as COPD severity decreased, while the total frequency of cardiovascular death, myocardial infarction, and stroke was the highest in patients with severe and very severe COPD.

No conflict of interest is reported.

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