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RESEARCH ARTICLE

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# Long COVID syndrome after SARS-CoV-2 survival in patients with pulmonary arterial hypertension and chronic thromboembolic pulmonary hypertension

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## Abstract

Pulmonary arterial hypertension (PAH) and chronic thromboembolic pulmonary hypertension (CTEPH) patients have a more severe COVID-19 course than the general population. Many patients report different persistent symptoms after SARS-CoV-2 infection. The aim of our study is to analyze the prevalence of long COVID-19 symptoms and assess if COVID-19 affects pulmonary hypertension (PH) prognosis. PAH/CTEPH patients who survived COVID-19 for at least 3 months before visiting the PH centers were included in the study. The patients were assessed for symptoms in acute phase of SARS-CoV-2 infection and persisting in follow-up visit, WHO functional class, 6-min walk distance, NT-proBNP concentration. The COMPERA 2.0 model was used to calculate 1-year risk of death due to PH at baseline and at follow-up. Sixty-nine patients—54 (77.3%) with PAH and 15 (21.7%) with CTEPH, 68% women, with a median age of 47.5 years (IQR 37–68)—were enrolled in the study. About 17.1% of patients were hospitalized due to COVID-19 but none in an ICU. At follow-up (median: 155 days after onset of SARS-CoV-2 symptoms), 62% of patients reported at least 1 COVID-19-related symptom and 20% at least 5 symptoms. The most frequently reported symptoms were: fatigue (30%), joint pain (23%), muscle pain (17%), nasal congestion (17%), anosmia (13%), insomnia (13%), and dyspnea (12%). Seventy-two percent of PH patients had a low or intermediate-low risk of 1-year death due to PH at baseline, and 68% after COVID-19 at follow-up. Over 60% of PAH/CTEPH patients who survived COVID-19 suffered from long COVID-19 syndrome, but the calculated 1-year risk of death due to PH did not change significantly after surviving mild or moderate COVID-19.

KEYWORDS
chronic thromboembolic pulmonary hypertension, COVID-19, long COVID syndrome, pulmonary arterial hypertension, risk of death

## INTRODUCTION

Precapillary pulmonary hypertension (PH) is a pathophysiological disorder that may involve multiple clinical conditions characterized by a mean pulmonary arterial pressure (mPAP) >20 mmHg at rest.[1] In addition to elevated mPAP, precapillary PH is characterized by elevated pulmonary vascular resistance (PVR) (>2 Wood units) and normal mean pulmonary artery wedge pressure (PAWP) (≤15 mmHg) on right heart catheterization.[1] PH is a progressive disease; if left untreated, it may lead to right heart failure, low output and death. Pulmonary arterial hypertension (PAH) is the group 1 of clinical PH classification. It is characterized by precapillary PH on right heart catheterization and similar histopathological changes in the pulmonary arteries, and can be successfully treated by targeted PH therapy.[1–3] Chronic thromboembolic pulmonary hypertension (CTEPH) belongs to group 4 of clinical PH classification and is characterized by chronic pulmonary artery obstructions by thromboembolic material.[1,4,5] Following the announcement of SARS-CoV-2 infection as a pandemic on March 11, 2020 by the World Health Organization (WHO),[6] different PH centers in Europe and the United States reported higher hospitalization and mortality rates due to COVID-19 among PAH/CTEPH patients than in the general populations.[7–13] This may be due to the underlying medical conditions—namely chronic lung diseases and/or coronary heart diseases—and/or the reduced visits to PH centers during the pandemic, which delayed PH diagnosis and early detection of disease progression.[8,12,14] Vaccination against COVID-19 should be prioritized in advanced PAH/CTEPH patients and PH patients waiting for lung transplant.[15] Survivors of SARS-CoV-2 infection may complain of persistent symptoms defined as long COVID or post-COVID syndrome. The most common definition of long COVID syndrome describes one or more symptoms that persist for at least 3 months after the onset of symptoms of acute SARS-CoV-2 infection.[16–19] The most frequent symptoms of long COVID in the general population are fatigue and dyspnea.[16,17] Early dyspnea could be a predictor of long-Covid syndrome.[18] There is no data about long COVID symptoms and their consequences for PAH/CTEPH patients. The aim of the study is to analyze the prevalence of long COVID symptoms among PAH/CTEPH patients and compare characteristics of PAH/CTEPH patients and calculated 1-year risk of death due to PH before acute COVID-19 and 3–6 months after surviving acute COVID-19.

## METHODS

The cohort observational prospective study was started in three PH centers in Poland during the COVID-19 pandemic. Patients who survived COVID-19 and for whom follow-up was possible in the first 2 years of pandemic, between March 11, 2020 and March 11, 2022 were included to the study. All study participants were adult, had PAH or CTEPH according to diagnosis based on right heart catheterization and additional exams described in current European Society of Cardiology (ESC) and European Respiratory Society guidelines,[1] and were COVID-19 survivors. COVID-19 diagnosis was confirmed by the RT-PCR method. Patients who suffered from COVID-19 before the diagnosis of PAH or CTEPH, and were unable to perform the 6-min walk test (6MWT) were excluded from the study. All participants signed an informed consent to participate in the study. The study protocol was approved by the local Bioethics Committee in accordance with the Declaration of Helsinki. All the PAH/CTEPH patients who had survived SARS-CoV-2 infection on the following visit for at least 3 months after contracting COVID-19 were asked to fill in questionnaire about persistent symptoms associated with the viral infection. The questionnaire included questions about the most common symptoms of the acute phase of SARS-CoV-2 infection and the persistence of symptoms at the follow-up visit. The list of possible long COVID symptoms has been created based on the results of observational studies in the general population.[20–24]

All the participants underwent the 6MWT, according to the American Thoracic Society guidelines,[25] and blood sampling for NT-pro-B-type Natriuretic Peptide (NT-proBNP) level, and were assessed to establish their WHO functional classes. Baseline patient characteristics, current treatment, history of concomitant disease and SARS CoV-2 vaccination status, distance in 6MWT, NT-proBNP level, and WHO functional class before COVID-19 were obtained from the medical records for each patient. A four-stratum model for predicting the 1-year risk of death for PH patients was used, based on WHO functional class, distance in 6MWT and NT-proBNP, and recommended by the ESC/ERS 2022 guidelines.[1] The

model is based on three parametric approaches (WHO functional class, distance in 6MWT, and NT-proBNP), variables with the highest predictive value in PAH. Patients are classified as low, intermediate-low, intermediate-high, or high risk of death.[1] The four-stratum model is superior than the three-stratum model because it better assesses the group of PAH patients with intermediate risk of death (60%–70% of patients) (1). The model was proposed by Humbert and colleagues and it has been validated on the beginning in the COMPERA register population as COMPERA 2.0 model,[26] and in medically treated CTEPH patients.[27] It may be useful tools for guiding balloon pulmonary angioplasty.[28] Baseline for long COVID was onset of symptoms of acute SARS-CoV-2 infection, other parameters (6MWT distance, NTproBNP level, baseline NYHA functional class) were taken from the last visit before acute COVID-19.

Statistical analysis was performed using IBM SPSS Statistics software (IBM Corporation), version 28.0.1.0. Data distribution was tested using the Shapiro–Wilk test. Categorical variables were presented as numbers and percentages, while continuous variables were presented as medians and interquartile ranges or as means and standard deviations. For group comparisons, $\chi^2$, Fisher's, paired *t* test or the Wilcoxon test was used as appropriate. The relationship differences were considered significant for $p \leq 0.05$.

## RESULTS

Seven out of 85 disposed PAH/CTEPH patients died due to acute COVID-19 and could not be included in the study. All of them had severe COVID-19 pneumonia and needed intensive care unit. Description of the entire group of 85 patients suffering from COVID-19 is performed in another paper.[12] Mortality rate due to acute COVID-19 was 8.2% and it was higher than in the general population.[12] In the following months after COVID-19 another four patients died from another reason and did not receive 3–6 months of follow-up: two patients died due to exacerbation of right heart failure, and two due to sudden cardiac death. All-cause mortality at a median follow-up of 155 days after COVID-19 among PAH/CTEPH patients was 12.9%. Flowchart showing the number of patients with PAH and CTEPH disposed and included in the study is presented in Figure 1.

A total of 69 PAH/CTEPH patients who had survived COVID-19 were included in the study. The study participants' mean age was 47.5 years (IQR 37–68). Most of these participants were female (68.1%). Fifty-four patients (78.3%) suffered from PAH, while 15 (21.7%) suffered from CTEPH. Baseline clinical characteristics of the study group is presented in Tables 1 and 2.

We do not have data on the type of SARS-CoV-2 specification of each patient. From the national COVID-19 register of the Polish Ministry of Heath we know that no patient from the study group had the delta variant or the Omicron variant of the SARS CoV-2 virus. During the acute phase of SARS-CoV-2 infection, 12 (17%) PAH/CTEPH patients were hospitalized while 57 (83%) were treated at home. Thirteen patients (18.8%) suffered from COVID-19-related pneumonia, 14 (20%) patients required new oxygen therapy or increase in the oxygen dosage being used, none of them required high-flow nasal oxygen therapy. Six patients (8.7%) had aggravated right heart failure due to COVID-19, four of them due to COVID-19 pneumonia, one due to supraventricular arrythmia, one due to excessive fluid intake. During the acute period of the COVID-19 illness, the most common symptoms reported by PAH/CTEPH patients were: fatigue (77%), fever (59%), myalgia (59%), arthralgia (47%), headache (44%), runny nose (43%), ageusia (41%), cough (39%), anosmia (39%), and dyspnea defined as new onset dyspnea or aggravation of existing dyspnea (32%).

The median time from the onset of symptoms to the follow-up visit was 155 days (IQR 116–183 days). In the following 3 months after COVID-19, four (5.8%) patients needed change of targeted PH therapy because they didn't meet therapeutic goals, one (1.5%) suffered from COVID-19-related myocarditis, and eight (11.5%) patients had persistent radiological changes in the lungs. The long COVID syndrome describes one or more symptoms persisted for at least 3 months after the onset of COVID-19. At follow-up, 38% of patients had no COVID-19 related symptoms. Sixty-two percent of patients reported at least one symptom, and 20% of patients reported at least five long COVID symptoms. The most frequent symptom presented at follow-up was fatigue (30%). Only eight (12%) patients reported dyspnea, new post-COVID dyspnea or worsening dyspnea compared to pre-COVID dyspnea scale, as a long COVID symptom at follow-up. The number of long COVID-related symptoms reported by PAH/CTEPH patients is presented in Figure 2. The prevalence of long COVID symptoms in PAH/CTEPH patients is presented in Figure 3.

Most of the PAH/CTEPH patients had low or intermediate-low risk of 1-year death at baseline, using four classification strata. The functional WHO class, the 6-min walk distance, and the NT-pro-BNP level did not change significantly 3 months after COVID-19 compared to the baseline (Table 3). The 1-year risk of mortality did not change at follow-up. Results are presented in Table 3 and Figure 4. Similar results were obtained when only

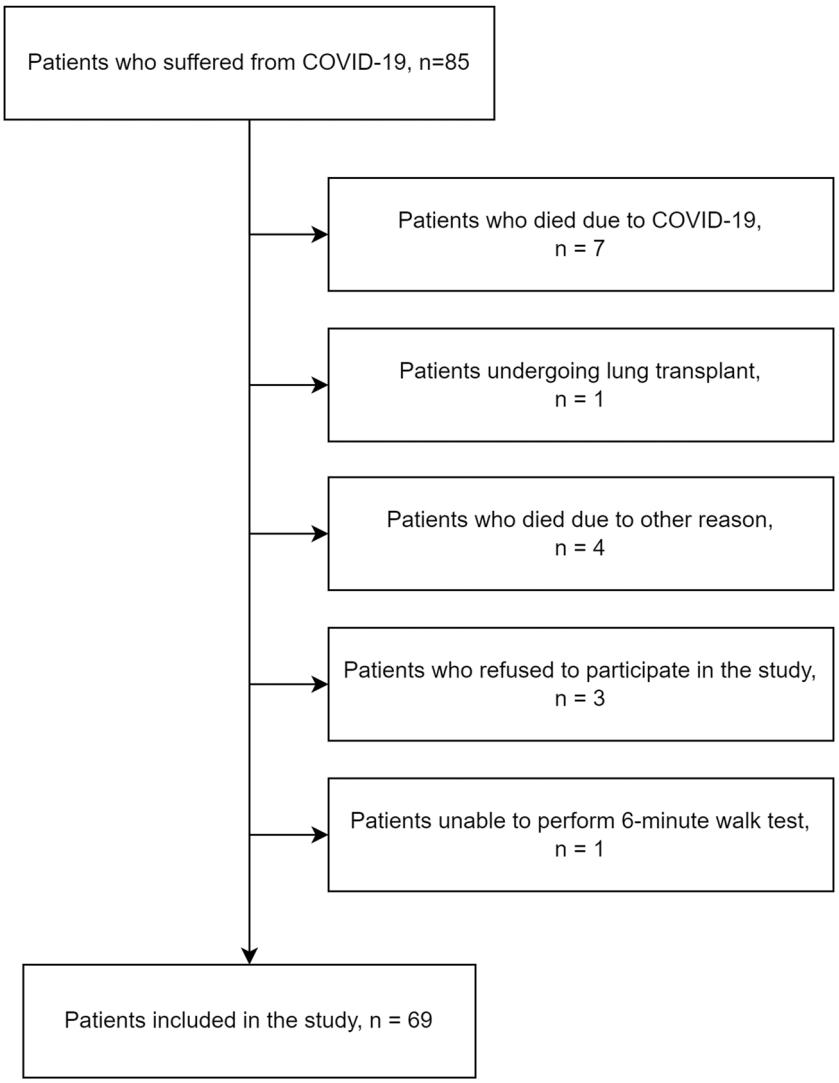


**FIGURE 1** Patients' disposition participate in the study.

the subgroups of patients with idiopathic PAH, hereditary PAH, PAH associated with connective tissue disease, and PAH (porto-pulmonary) were analyzed (Table 4).

## DISCUSSION

The COVID-19 pandemic has impacted the lives of people all over the world. The risk of death due to COVID-19 among PAH/CTEPH patients is much higher compared to the general population. In the first months of the pandemic, March–May 2020, the mortality rate due to COVID-19 among PAH/CTEPH patients was reported to be around 45%[13,29]; in subsequent months the rate was 8%–22%.[8–12] According to various authors, 30%–81% of PAH/CTEPH patients required hospitalization due to COVID-19.[8–13,29] Risk factors for adverse outcomes in PH patients who contracted COVID-19 in the acute phase were: older age, comorbidities, and more severe PH.[8,12]

There are no data on the chronic consequences of SARS-CoV-2 infection for PAH/CTEPH patients who survived COVID-19. Long COVID is defined by the persistence of one or more symptoms at least 3 months from the onset of the disease.[18,19] Several studies reported an incidence of long COVID syndrome of 30%–90% in general population.[18,19] Three potential pathophysiological mechanism are responsible for long COVID syndrome: pathological chronic inflammation, immunological aberration and virus-specific long-term tissue damage.[18,19] In our study group, 62% had long COVID syndrome. Sixty-two percent of PAH/CTEPH patients reported at least one symptom related to COVID-19, 20% at least five symptoms persisting more than 3 months after acute SARS-CoV-2 infection. This is in concordance with the general population, where 39%–76% of people who had survived COVID-19 reported ≥1 symptom lasting 103–186 days after the onset of symptoms.[22–24,30–32] The most frequent symptoms of long COVID syndrome in our study group were fatigue (30%), arthralgia (23%), myalgia (17%), and

**TABLE 1** Clinical characteristics of the study group, $n = 69$.

| | Total study group *n* (%) or median (IQR) |
|---|---|
| Number of patients | 69 (100%) |
| Age, years | 47.5 (37–68) |
| Sex, female | 47 (68.1) |
| Duration of disease, years | 9.0 (3.0–15.0) |
| PAH | 54 (77.3) |
| Idiopathic PAH | 25 (36.2) |
| PAH associated with CHD | 19 (27.5) |
| PAH associated with CTD | 9 (13.0) |
| PAH porto-pulmonary | 1 (1.5) |
| CTEPH | 15 (21.7) |
| PDE5/sCG | 57 (82.6) |
| ERA | 45 (65.2) |
| Prostacyclin/IP receptor agonist | 18 (26.0) |
| One drug | 26 (37.7) |
| Two drugs | 23 (33.3) |
| Three drugs | 16 (23.2) |
| Oral anticoagulation | 28 (40.6) |
| Oxygen | 10 (14.5) |
| Vaccination against COVID-19 | 25 (36.2) |
| Concomitant disease | 35 (49.3) |
| Arterial hypertension | 23 (31.9) |
| Diabetes | 4 (5.8) |
| COPD | 4 (5.8) |
| Coronary artery disease | 8 (11.6) |
| Neoplasm | 3 (4.3) |
| Obesity, BMI ≥30 kg/m$^2$ | 13 (18.8) |

Abbreviations: BMI, body mass index; COPD, chronic obstructive pulmonary disease; CTD, pulmonary arterial hypertension associated with connective tissue disease; ERA, endothelin receptor agonists; IP, prostacyclin receptor; IPAH, idiopathic pulmonary arterial hypertension; PAH, pulmonary arterial hypertension; PAH-CHD, pulmonary arterial hypertension related to congenital heart disease; PAH-PAH-porto-pulmonary, pulmonary arterial hypertension associated with portal hypertension; PDE5, phosphodiesterase 5-inhibitors; PH, pulmonary hypertension; sGC, soluble guanylate cycle stimulator; WHO, World Health Organization.

**TABLE 2** Baseline characteristics of the study group.

| | Total study group *n* (%) or median (IQR) |
|---|---|
| WHO functional class | |
| I | 6 (8.7) |
| II | 31 (44.9) |
| III | 31 (44.9) |
| IV | 1 (1.5) |
| NT-proBNP, ng/L | 268 (93.5–667.5) |
| 6MWD, m | 423.3 ± 128.2 |
| MPAP, mmHg | 39 (30–60.8) |
| PVR, wood units | 5.4 (3.7–8.3) |
| CI, L/min | 2.8 (2.3–3.2) |
| MVSat, % | 73 (70–77) |
| sPAP, mmHg | 72.2 ± 26.2 |
| TAPSE, mm | 20.6 ± 4.5 |
| RAA, cm$^2$ | 20.5 (17–24.3) |
| Presence of fluid in pericardium, yes/no | 3 (4.5) |

Abbreviations: 6MWD, six-min walk distance; 6MWT, six-min walk test; CI, cardiac index; MPAP, mean pulmonary arterial pressure; NT-proBNP, NT-proB-type Natriuretic Peptide; PVR, pulmonary vascular resistance; sPAP, systolic pulmonary arterial pressure; TAPSE, tricuspid annular plane systolic excursion; RAA, right atrium area; WHO, World Health Organization.

nasal congestion (17%). Dyspnea (new onset dyspnea or persistent aggravation of previous symptom) was reported only by 12% of PAH/CTEPH patients. This is different from the general population, where the most frequently reported symptoms of long COVID syndrome were fatigue (29%–63%) and dyspnea (15%–61%).[21,22,24,30,32] This may

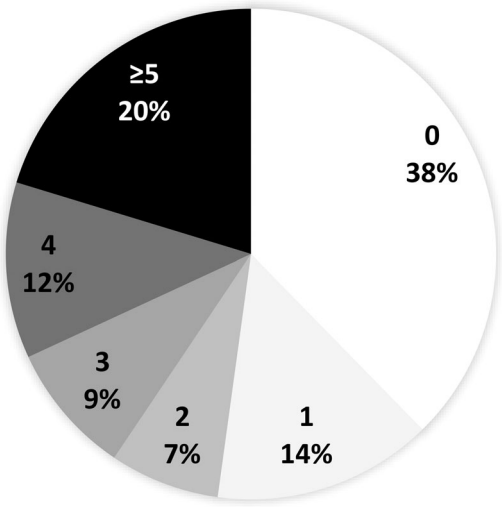


**FIGURE 2** The number of long COVID symptoms reported by PAH/CTEPH patients (number of symptoms and percent of patients). CTEPH, chronic thromboembolic pulmonary hypertension; PAH, pulmonary arterial hypertension.

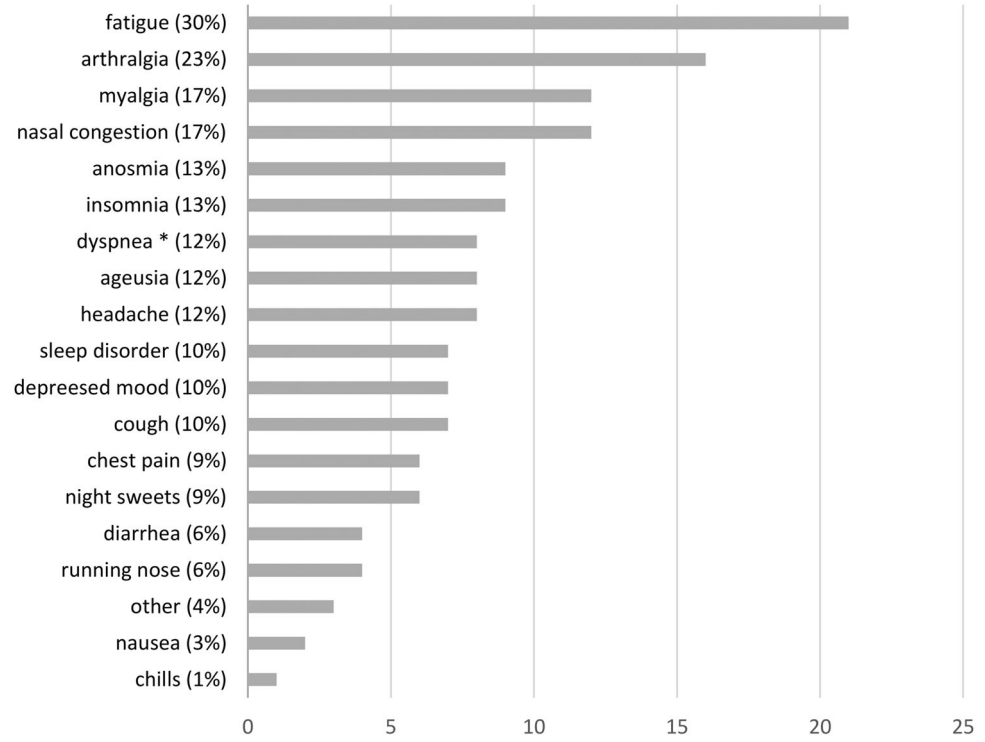


**FIGURE 3** Prevalence of symptoms of long COVID in PAH and CTEPH patients at least 3 months after the onset of SARS-CoV-2 infection, * new onset dyspnea or worsening of existing dyspnea. CTEPH, chronic thromboembolic pulmonary hypertension; PAH, pulmonary arterial hypertension.

**TABLE 3** Functional parameters and 1-year risk of death at baseline and follow-up after recovery from COVID-19 in the whole study group.

| Variables | Study group median (IQR) at baseline $n = 69$ | Study group median (IQR) at follow-up $n = 69$ | *P* baseline versus follow-up |
|---|---|---|---|
| WHO functional class | | | 0.81 |
| I | 6 (8.7) | 4 (5.8) | |
| II | 31 (44.9) | 38 (55.1) | |
| III | 32 (46.4) | 25 (36.2) | |
| IV | 2 (2.9) | 4 (5.8) | |
| 6MWD, m | $423.3 \pm 128.2$ | $416 \pm 133.5$ | 0.45 |
| NT-proBNP, ng/L | 268 (93.5-667.5) | 225 (83–754.5) | 0.27 |
| One-year risk of death, points | 1.7 (1.0–2.6) | 1.5 (1.0–2.9) | 0.76 |
| Low, *n* (%) | 30 (43.5) | 29 (42.0) | |
| Intermediate-low, *n* (%) | 20 (29.0) | 18 (26.1) | |
| Intermediate-high, *n* (%) | 18 (26) | 19 (27.5) | |
| High, *n* (%) | 1 (1.5) | 3 (4.4) | |

Abbreviations: 6MWD, six-min walk distance; NT-proBNP, NT-proB-type Natriuretic Peptide; PH, pulmonary hypertension; WHO FC, World Health Organization functional class.

be due to the fact that dyspnea is a typical common symptom of pulmonary hypertension that patients experience on a daily basis. For this reason, the small difference of shortness of breath patients may not be associated with a history of SARS-CoV-2 infection but with PH. In contrast to the general population in the study group the question about dyspnea as the symptom of long COVID syndrome meant new post-COVID dyspnea or worsening

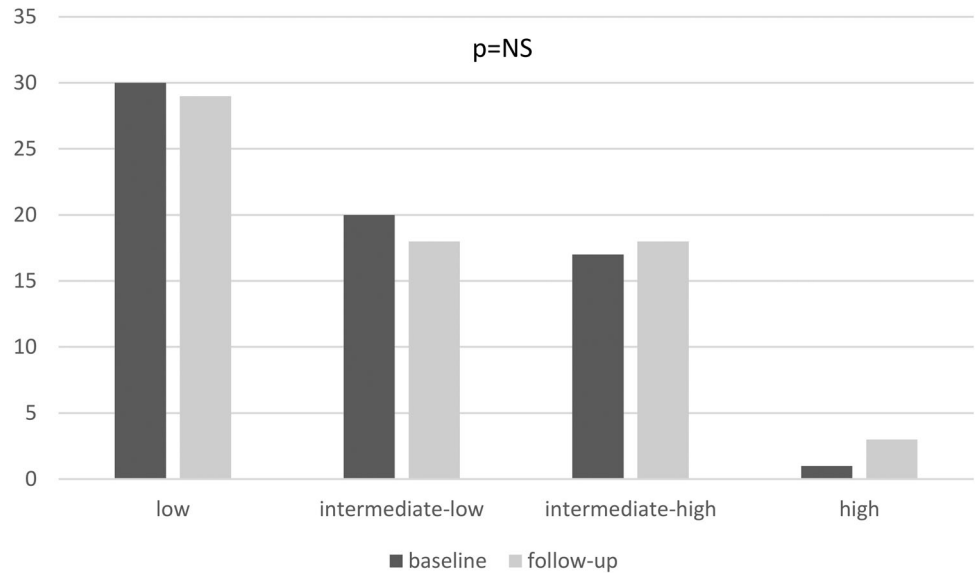


**FIGURE 4** Number of patients with calculated 1-year risk of death in four-strata COMPERA 2.0 model at baseline and 3–6 months after COVID-19 in PAH/CTEPH patients; CTEPH, chronic thromboembolic pulmonary hypertension; PAH, pulmonary arterial hypertension.

**TABLE 4** Functional parameters and 1-year risk of death at baseline and during follow-up after COVID-19 in study subgroup (IPAH, HPAH, DPAH, PAH-CTD, PAH-PoP).

| Variables | Study group median (IQR) or n (%) at baseline $n = 32$ | Study Group median (IQR) or $n$ (%) at follow-up $n = 32$ | $P$ baseline versus follow-up |
|---|---|---|---|
| WHO functional class | | | 0.48 |
| I | 1 (3.1) | 1 (3.1) | |
| II | 13 (40.6) | 17 (53.1) | |
| III | 17 (53.1) | 11 (34.4) | |
| IV | 1 (3.1) | 3 (9.4) | |
| 6MWD, m | 409.3 ± 144 | 406.6 ± 152.9 | 0.94 |
| NT-proBNP, ng/L | 318 (74.3–738) | 272 (89.8–949.8) | 0.86 |
| Risk of 1-year risk of death due to PH, points | 1.7 (1.3–2.5) | 1.5 (1.0–3.0) | 0.89 |
| Low, $n$ (%) | 11 (34.4) | 15 (46.8) | |
| Intermediate-low, $n$ (%) | 12 (37.5) | 5 (15.6) | |
| Intermediate-high, $n$ (%) | 8 (21.8) | 18 (25.0) | |
| High, $n$ (%) | 1 (3.1) | 3 (9.4) | |

Abbreviations: 6MWD, distance in six-min walk test; NT-proBNP, NT-proB-type Natriuretic Peptide;PAH, pulmonary arterial hypertension, idiopathic PAH, hereditary PAH, drug-induced PAH, PAH with connective tissue disease, PAH porto-pulmonary; PH, pulmonary hypertension; WHO FC, World Health Organization functional class.

dyspnea compared to pre-COVID dyspnea scale and no dyspnea at all. The reported rates of other symptoms, such as myalgia, arthralgia, and smell dysfunction, were similar to those in the general population.[22,23,30,31] Old age (over 70 years), female sex, presence of comorbidities, and more of the five symptoms during the first week of SARS-CoV-2 infection were identified as predictors of long COVID syndrome in the general population.[33] In another study, female sex and history of anxiety or depression or antidepressant therapy were identified as risk factors of long COVID syndrome.[34] Hospitalization or necessary to ICU due to COVID-19 had no impact on long COVID.[18] In our study, 68% of PAH/CTEPH patients were female, 49% had comorbidities, 13% were ≥70 years old, 16% have been

diagnosed of depression, and 20% had at least five acute COVID-19 symptoms. Only 17% of our study group were hospitalized, but not in an ICU. In the 2022 ESC/ERS guidelines for the diagnosis and treatment of PH, a four-stratum model for the prediction of 1-year risk of death of PH patients at follow-up is recommended to better assess the intermediate group, which constitutes 60%–70% of PAH patients.[1] The model is based on three parametric approaches (WHO functional class, distance in 6MWT, and NT-proBNP), variables with the highest predictive value. Patients are classified as low, intermediate-low, intermediate-high, or high risk of death.[1] The observed 1-year mortality rates in the four risk strata were 0%–3%, 2%–7%, 9%–19%, and ≥20%, respectively.[1] Two recent registry studies have evaluated a four-stratum risk assessment tool based on refined cut-off levels for WHO functional class, distance in 6MWT, and level of NT-proBNP.[1,26,35] The four-stratum model predicted survival in patients with idiopathic PAH, hereditary PAH, drug-induced PAH, PAH associated with connective tissue disease, and porto-pulmonary PAH in a COMPERA 2.0 registry and a French registry.[1,26,35] Moreover, COMPERA 2.0 model predicted survival in medically treated CTEPH patients[27] and was useful to assess the risk of mortality due to balloon pulmonary angioplasty in CTEPH.[28] In current ESC/ERS guidelines, a risk-based, goal-orientated treatment approach in PAH patients, where achieving and/or maintaining a low-risk status, is recommended.[1] In our study 1-year risk of death didn't change significantly among mild and moderate COVID-19 survivors. Seventy-two percent of PAH/CTEPH patients had low or moderate-low risk of 1-year death at baseline, and 68% at follow-up. At follow-up, none of the crucial parameters (WHO functional class, distance in 6MWT, NT-proBNP) had changed significantly. When we analyzed only subgroups for whom the four-stratum model had been validated in registers (idiopathic PAH, hereditary PAH, drug-induced PAH, PAH associated with connective tissue disease, and porto-pulmonary PAH), the risk of 1-year death did not changed significantly after surviving COVID-19. In the following 3 months after COVID-19 only four (5.8%) patients needed change of targeted PH therapy. According to the current ESC guidelines for the diagnosis and treatment of PH panel of data derived from clinical assessment, exercise tests, biochemical markers, echocardiography, and hemodynamic evaluations is recommended to evaluate PH severity.[1] Achieving and maintaining a low-risk profile on optimized medical therapy is recommended as a treatment goal in PAH patients.[1] For this reason, four (5.8%) patients in the study group after COVID-19 had escalated targeted therapy, not because of proven PH progression. Our study has some limitations. The study group was relatively small. Patients who did not sign informed consent, who were unable to complete the 6MWT, or who had a lung transplant were excluded from the study, which may have influenced study results. Data on the course of the acute phase of COVID-19 were obtained from discharge cards, information from a family doctor or from the COVID-19 register of the Polish Ministry of Health. Some information, such as the type and dosage of anticoagulants in the acute phase of COVID-19, were incomplete and could potentially influence the results. We limited our study to assessing the symptoms of long COVID, WHO functional class, distance in 6MWT, NT-proBNP level and calculated 1-year risk of death due to PH. Other exams were performed only on patients whose clinical condition suggested worsening or new abnormalities during physical examination, so asymptomatic organ damage caused by COVID-19 could be omitted. We asked patients about the onset of new dyspnea or worsening of existing dyspnea in acute COVID-19 and in long COVID, not about dyspnea at all. This seemed more appropriate to us as exertional dyspnea is a typical symptom of PAH/CTEPH, but this may have resulted in a discrepancy in results compared to patients in the general population. Due to the small study group, the COMPERA 2.0 model to assess 1-year risk of death related to PH was used together for patients with PAH and CTEPH, although this model was validated for selected group of patients with PAH or CTEPH.

In our study, more than 60% of PAH/CTEPH patients who had survived COVID-19 reported symptoms of long COVID. The most frequently reported symptoms of long COVID were fatigue, arthralgia and myalgia. The COVID-19 is high mortality rate condition, but the calculated 1-year risk of death due to PH stayed unchanged in PAH/CTEPH patients surviving mild or moderate COVID-19.

## AUTHOR CONTRIBUTIONS

**Maria Wieteska-Miłek**: Conceptualization; methodology; software; validation; formal analysis; investigation; resources; data curation; writing—original draft preparation; writing—review and editing; visualization; project administration. **Piotr Zieliński**: Software; validation. **Sebastian Szmit**: Investigation; data curation; writing—review and editing. **Michał Florczyk**: Investigation; data curation. **Katarzyna Betkier-Lipińska**: Investigation; data curation. **Beata Kuśmierczyk-Droszcz**: Investigation; data curation. **Michał Florczyk**: Investigation; data curation; writing—review and editing. **Marcin Kurzyna**: Writing—review and editing; visualization; supervision; funding acquisition. **Piotr Hoffman**: Writing—review and editing. **Paweł Krzesiński**: Writing—review and editing.

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## CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

## ETHICS STATEMENT

The study was conducted according to the guidelines of the Declaration of Helsinki and approved by the Bioethics Committee of the Centre of Postgraduate Medical Education (protocol number KBE 22/2021 and date of approval: April 14, 2021). Informed consent was obtained from all participants involved in the study.

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