

Original article

## Dynamics of Clinical Parameters in Arterial Hypertension Patients after COVID-19: Impact of Obstructive Airway Disease (OAD)

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**Abstract:** *Objective* — To compare the longitudinal dynamics of clinical parameters in patients with arterial hypertension (AH) according to the presence of obstructive airway disease (OAD) – encompassing asthma, chronic obstructive pulmonary disease (COPD), and pre-COPD – during the year following COVID-19-related lung damage.

*Methods* — 127 patients with AH were examined during hospitalization for COVID-19-related lung damage and re-examined at 3 and 12 months after discharge. The OAD group included 38 patients, while the non-OAD group included 89 patients; the groups were matched for age, gender, BMI, severity of AH, and comorbidities.

*Results* — Non-OAD patients had a higher incidence of stage 3 hypertension. One year after discharge, left-ventricular (LV) relaxation disorders were more frequently detected in the non-OAD group. Right-heart structural and functional parameters also worsened: the right atrial volume index increased (15.0 [12.0; 17.8] mm/m<sup>2</sup> vs. 16.7 [14.2; 19.5] mm/m<sup>2</sup>, p=0.004); basal right-ventricular width rose (32.2±4.7 mm vs. 33.5±4.7 mm, p=0.007); tricuspid annulus displacement amplitude fell (23.4±2.7 mm vs. 22.4±2.2 mm, p=0.009); and its early diastolic velocity decreased (13.0 [11.0; 15.0] cm/s vs. 12.0 [11.0; 14.0] cm/s, p=0.047).

*Conclusion* — In AH patients, the hospital course of COVID-19-related lung damage did not differ between those with and without OAD. One year after discharge, OAD patients showed a significantly reduced frequency of heart failure with preserved ejection fraction (HFpEF) incidence compared to three months after COVID-19. In non-OAD patients, BMI and the prevalence of stage 3 arterial hypertension increased, LV relaxation disorders were more frequent, and right-heart structural and functional parameters were worse compared with OAD patients.

**Keywords:** Arterial hypertension, obstructive airway disease, right heart, ventricular systolic and diastolic function.

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### Introduction

Any viral infection can destabilize the cardiovascular system. After clearance of SARS-CoV-2, a substantial proportion of patients with arterial hypertension experience decompensation of their somatic status, manifesting as unstable blood pressure – particularly those who had severe COVID-19 [1].

*Hypothesis:* The primary target of COVID-19 is the bronchopulmonary system; patients who already have AH and a pre-existing obstructive airway disease (OAD) may endure a more severe disease course and a slower recovery of cardiovascular health parameters.

*Objective:* To compare the longitudinal dynamics of clinical parameters in AH patients, stratified by the presence or absence of OAD, during the year following COVID-19-related lung injury.

### Material and Methods

A cohort observational study was conducted using data from the database registry “Prospective registry of persons who have

had COVID-19-associated pneumonia,” which comprised 350 patients [2]. The study protocol received approval from the Biomedical Ethics Committee of the Tyumen Cardiology Research Center (Protocol No. 159, July 23, 2020) and was registered in the International Clinical Trials Registry (ClinicalTrials.gov, Identifier No.: NCT04501822). All participants provided written informed consent before inclusion.

A total of 38 patients with AH and OAD were selected from the database registry: 12 with asthma, 8 with chronic obstructive pulmonary disease (COPD), and 12 with pre-COPD (defined by a smoking history of ≥10 pack-years, “definite smokers,” with a smoking index >160). In the non-OAD group, 89 non-smoking AH patients were selected using stratified randomization; strata factors included AH stage and risk, gender, age, and BMI.

Exclusion criteria comprised coronary artery disease (CAD), oncological diseases diagnosed during the observation period, refusal to participate, suboptimal echocardiographic (EchoCG) imaging, hemodynamically significant valvular regurgitation, and atrial fibrillation. Data from the single-infection hospital

examination were assessed using hospital discharge summaries. Clinical, laboratory, and instrumental data for both groups were collected at 3 and 12 months post-discharge follow-up visits (first and second visits, respectively). Concomitant disease diagnoses were based on anamnesis, medical records, and clinical, instrumental, and laboratory examinations, in accordance with current clinical guidelines.

Heart failure with preserved ejection fraction (HFpEF) was diagnosed using the HFA-PEFF algorithm: a score of 0-1 indicated low probability (no further testing required); a score of 2-4 indicated probable HFpEF (horizontal bicycle ergometry was performed to confirm); and a score  $\geq 5$  indicated established HFpEF. Chest computed tomography (CT) was performed with a Toshiba Aquilion 64 system (Japan). EchoCG was performed on a GE Healthcare Vivid S70 ultrasound machine; images were saved in DICOM format and processed on an IntelliSpace Cardiovascular workstation using the TOMTEC program (Philips, USA). Echocardiography adhered to current guidelines, including assessment of longitudinal strain [3]. Myocardial diastolic function was assessed in accordance with guidelines [3-5]. Variable distributions were evaluated using the Shapiro-Wilk and Kolmogorov-Smirnov tests, with Liliefors significance correction applied. Between-group comparisons of quantitative parameters employed the Student's *t*-test or the Mann-Whitney *U* test. Within-group changes over time were analyzed with the paired Student's *t*-test or the Wilcoxon signed-rank test; for comparisons across three time points, the Friedman test was used, and Bonferroni correction was applied to the resulting *p*-values. Qualitative variables were compared using the chi-square test or Fisher's exact test, and temporal changes were assessed with the

McNemar test. Statistical significance was set at two-sided  $p < 0.05$ . A generalized linear model was used for multivariate analysis.

### Results

Patients in the OAD group did not differ in age across the specific OAD subtype: those with COPD had an age of  $55.1 \pm 5.6$  years, those with pre-COPD  $54.8 \pm 10.0$  years, and those with asthma  $54.7 \pm 5.2$  years ( $p = 0.996$ ). The only significant difference observed during hospitalization was the more frequent use of carbapenems in the OAD group (Table 1), likely reflecting a higher risk of infection with multidrug-resistant flora in COPD patients. The increased LV volume (Table 2) is attributed to the higher infusion load administered during hospitalization.

After discharge (Table 2) at the second visit in the non-OAD group, a significant increase in BMI and in the frequency of stage 3 AH was observed, driven by a rise in the number of patients with chronic kidney disease (CKD) stages 2 and 3A according to the glomerular filtration rate (GFR). Newly diagnosed CAD also appeared: it was first identified in five non-OAD patients and in one OAD patient.

In the absence of baseline differences in HFpEF, the OAD group showed a dynamic decrease in the number of patients with probable HFpEF (candidates for diastolic stress testing). At the first visit, normalization of chest-CT data in the OAD group was detected less frequently, but at the second visit, the frequency of residual lung changes was low and did not differ between the groups. The mean levels of laboratory parameters during outpatient observation were within normal limits, except for elevated total cholesterol and low-density lipoproteins in both groups, with no inter-group differences (Table 2).

**Table 1. Clinical characteristics of hypertensive patients hospitalized for COVID-19-related lung injury, stratified by obstructive airway disease (OAD)**

Hospitalization data		<b>OAD patients (n=38)</b>	<b>Non-OAD patients (n=89)</b>	<b>p-value</b>
Male gender (n %)		18 (47.4)	35 (39.3)	0.436
Age (years)		55 [50; 59]	54 [48; 62]	0.920
Duration of hospitalization (days)		15.5±6.9	13.4±4.1	0.217
Severity of lung damage assessed by chest CT (degree, n %)	1	6 (6.7)	4 (10.5)	0.824
	2	28 (31.2)	14 (36.8)	0.871
	3	41 (46.1)	12 (31.6)	0.708
	4	14 (15.7)	8 (21.1)	0.831
ICU admission (n %)		8 (21.1)	10 (11.2)	0.146
Biological therapy (tocilizumab/sarilumab) (n %)		2 (5.3)	6 (6.7)	0.754
Hormonal therapy (prednisolone/dexamethasone) (n %)		15 (39.5)	38 (42.7)	0.736
Protected penicillins (n %)		19 (50.0)	40 (44.9)	0.601
Cephalosporins (n %)		19 (50.0)	55 (31.8)	0.217
Fluoroquinolones (n %)		12 (31.6)	28 (31.5)	0.990
Macrolides (n %)		35 (92.1)	80 (89.9)	0.890
Carbapenems (n %)		13 (35.1)	12 (13.5)	0.006
Lincosamides (n %)		1 (2.6)	2 (22.2)	0.896
Aminoglycosides (n %)		38 (100)	89 (100)	1.000
Antifungal agents (n %)		4 (10.5)	5 (5.6)	0.324
Antiviral therapy (n %)	Umifenovir + interferon	11 (12.4)	3 (7.9)	0.552
	Hydroxychloroquine + azithromycin	80 (89.9)	32 (84.2)	0.379
	Kaletra (lopinavir + ritonavir)	10 (11.2)	7 (18.4)	0.272
	Favipiravir	11 (12.4)	5 (13.2)	0.901
Heparin (n %)		21 (55.3)	61 (68.5)	0.512
Enoxaparin (n %)		19 (50.0)	35 (39.3)	0.265
Thromboembolic complications (n %)		0	1	0.660

Data presented from this point forward are expressed as median (Q1-Q3) for continuous variables and n (%) for categorical variables. Statistically significant differences ( $p < 0.05$ ) are highlighted in bold. OAD, obstructive airway disease; ICU, intensive care unit.

**Table 2. Longitudinal clinical and laboratory comparisons in hypertensive patients with post-COVID-19 lung injury, stratified by OAD**

Parameter		OAD patients (n=38)	Non-OAD patients (n=89)	p-value	
Body mass index (kg/m <sup>2</sup> )	Visit 1	34.2±7.3	33.1±4.6	0.705	
	Visit 2	34.3±6.9	34.1±5.2	0.847	
	p-value	0.549	<0.001		
Obesity (n %)	Visit 1	30 (78.9)	69 (77.5)	0.860	
	Visit 2	30 (78.9)	71 (79.8)	0.916	
	p-value	1.000	0.727		
Blood pressure (mm Hg)	Systolic	Visit 1	135±12	134±15	0.576
		Visit 2	134±13	135±18	0.901
		p-value	0.986	0.735	
	Diastolic	Visit 1	90±8	88±12	0.205
		Visit 2	90±12	91±14	0.956
		p-value	0.872	0.079	
Stages of arterial hypertension (n %)	Stage 1	Visit 1	16 (42.1)	25 (28.1)	0.122
		Visit 2	13 (35.1)	20 (22.7)	0.189
		p-value	0.250	0.063	
	Stage 2	Visit 1	20 (52.6)	60 (67.4)	0.114
		Visit 2	18 (48.6)	58 (65.9)	0.084
		p-value	0.727	0.754	
	Stage 3	Visit 1	2 (5.3)	4 (4.5)	0.852
		Visit 2	6 (16.2)	10 (11.4)	0.561
		p-value	0.125	0.031	
Cardiovascular risk SCORE (n %)	1st visit:	1 (low)	1 (2.6)	0 (0)	0.477
		2 (medium)	5 (13.2)	10 (11.2)	
		3 (high)	21 (55.3)	52 (58.4)	
		4 (very high)	11 (28.9)	27 (30.3)	
	2nd visit:	1 (low)	1 (2.6)	0 (0)	0.486
		2 (medium)	2 (5.3)	6 (6.7)	
		3 (high)	23 (60.5)	55 (61.8)	
		4 (very high)	12 (31.6)	28 (31.5)	
p-value	0.135	0.239			
History of AH experience at 1st visit (years)		6 [1; 13]	7 [3; 10]	0.374	
First detected CAD during observation (n %)		1 (2.7)	5 (5.6)	0.668	
Type 2 diabetes (n %)	Visit 1	9 (23.7)	14 (15.7)	0.287	
	Visit 2	10 (26.3)	15 (16.9)	0.219	
	p-value	1.000	1.000		
Impaired fasting glucose (n %)	Visit 1	0	1 (1.1)	0.512	
	Visit 2	1 (2.6)	4 (4.5)	0.621	
	p-value	1.000	0.375		
Total cholesterol (mmol L <sup>-1</sup> )	Visit 1	5.7 [5.3; 6.4]	5.9 [4.7; 6.9]	0.748	
	Visit 2	5.3 [4.6; 6.2]	5.2 [4.2; 6.6]	0.934	
	p-value	0.129	0.008		
Low-density lipoprotein cholesterol (mmol L <sup>-1</sup> )	Visit 1	3.7 [2.9; 4.1]	3.7 [2.7; 4.6]	0.671	
	Visit 2	3.1 [2.5; 4.0]	3.1 [2.4; 4.3]	0.865	
	p-value	0.095	0.004		
Points of HFA-PEFF (n %)	0-1 (no HFpEF)	Visit 1	10 (26.3)	24 (27.0)	0.935
		Visit 2	17 (44.7)	24 (27.0)	0.051
		p-value	0.065	1.000	
	2-4 (probable HFpEF)	Visit 1	26 (68.4)	57 (64.0)	0.633
		Visit 2	18 (47.4)	52 (58.4)	0.254
		p-value	0.039	0.458	
≥5 (HFpEF)	Visit 1	2 (5.3)	8 (9.0)	0.479	
	Visit 2	3 (7.9)	13 (14.6)	0.297	
	p-value	1.000	0.332		
Concomitant diseases (1st visit, n %)	Cerebrovascular insufficiency	0	5 (5.6)	0.136	
	Osteoarthritis	0	2 (2.2)	0.352	
	Sleep apnea syndrome	1 (2.6)	5 (5.6)	0.668	
	Hypothyroidism (incl. subclinical)	6 (15.8)	7 (7.9)	0.177	
Chest computed tomography data (n %)	Normalization	Visit 1	5 (13.2%)	26 (29.9%)	0.046
		Visit 2	12 (32.4%)	33 (37.1%)	0.620
		p-value	0.016	0.118	
Chest computed tomography data (n %)	Residual effects	Visit 1	17 (44.7%)	33 (37.9%)	0.475
		Visit 2	17 (45.9%)	39 (43.8%)	0.827
		p-value	1.000	0.267	
	Lung damage	Visit 1	16 (42.1%)	28 (32.2%)	0.285
		Visit 2	8 (21.6%)	17 (19.1%)	0.747
		p-value	0.008	0.002	

Parameter		OAD patients (n=38)	Non-OAD patients (n=89)	p-value
CKD stage (GFR) (n %)	1st visit:			
	Stage 1	21 (56.8)	55 (63.2)	0.607
	Stage 2	16 (43.2)	31 (35.6)	
	Stage 3A	0	1 (1.1)	
	2nd visit:			
	Stage 1	21 (56.8)	41 (50.6)	0.452
Stage 2	16 (43.2)	37 (45.7)		
Stage 3A	0	3 (3.7)		
	p-value	1.000	0.010	
Vaccination against COVID-19 between visits 1 & 2 (n %)	Total	23 (60.5)	41 (46.1)	0.136
	EpiVacCorona	3 (14.3)	3 (7.5)	0.232
	Sputnik V	12 (57.1)	29 (72.5)	0.089
	CoviVac	0	2 (5.0)	0.161
	Sputnik Light	6 (28.6)	6 (15.0)	0.074
Recovered from COVID-19 again (n %)		1 (2.6)	6 (6.7)	0.673

AH, arterial hypertension; CAD, coronary artery disease; HFA PEFF, algorithm for diagnosing heart failure with preserved ejection fraction; HFpEF, heart failure with preserved ejection fraction; CKD, chronic kidney disease; GFR, glomerular filtration rate. Significant differences ( $p < 0.05$ ) are highlighted in bold.

**Table 3.** Comparative dynamics of echocardiographic parameters in patients with AH 3 and 12 months after COVID-19-related lung injury, with OAD (n=38) versus without OAD (n=89)

Parameter (unit)	Group / p between groups	During hospitalization	3 months	12 months	p dynamics between visits
Left ventricle end-diastolic volume (LV EDV, ml)	OAD cohort	119.2±24.5	92.5±23.3	92.2±23.5	$p_{0-3} < 0.001$
	non-OAD cohort	113.4±23.4	90.2±24.6	91.2±19.3	$p_{0-3} < 0.001$
	p (between groups)	0.223	0.225	0.948	
Left ventricle myocardial mass (M-mode, g)	OAD cohort	201 [181; 220]	176 [153; 201]	188 [159; 207]	$p_{0-3} = 0.038$
	non-OAD cohort	200 [175; 234]	175 [153; 201]	182 [153; 207]	$p_{0-3} < 0.001$
	p (between groups)	0.271	0.827	0.663	
Left ventricle ejection fraction (LVEF, %)	OAD cohort	68.7±6.2	69.0±4.3	68.9±4.7	0.712
	non-OAD cohort	68.9±4.7	67.0±4.5	67.3±3.3	$p_{0-3} = 0.020$
	p (between groups)	0.833	0.040	0.088	
Left atrium volume (ml)	OAD cohort	56 [48; 62]	56 [45; 62]	52 [46; 62]	0.712
	non-OAD cohort	52 [48; 58]	47 [40; 62]	50 [44; 60]	$p_{0-3} < 0.001$ $p_{3-12} = 0.006$
	p (between groups)	0.243	0.113	0.415	
Right atrium volume (ml)	OAD cohort	50 [42; 57]	30 [24; 44]	35 [30; 40]	$p_{0-3} < 0.001$
	non-OAD cohort	45 [39; 52]	30 [22; 36]	34 [28; 42]	$p_{0-3} < 0.001$ $p_{3-12} = 0.001$
	p (between groups)	0.035	0.345	0.397	
Right ventricle anteroposterior dimension (mm)	OAD cohort	27.8±3.6	26.2±2.8	26.2±2.5	$p_{0-12} = 0.048$
	non-OAD cohort	27.5±3.2	26.0±2.3	26.2±2.5	$p_{0-3} < 0.001$
	p (between groups)	0.606	0.286	0.815	

According to echocardiography, left-ventricular ejection fraction (LVEF) remained within the normal range for all patients throughout the study (Table 3). At the first visit, LVEF was lower in the non-OAD group than in the OAD group, but this difference disappeared by the second visit. A more sensitive marker of systolic function, global longitudinal strain (LV GLS), showed a trend toward decline in the non-OAD group at the second visit (Table 4). The proportion of patients with reduced LV GLS increased, although not significantly; at the second visit, 35 % of the non-OAD group and 25% of the OAD group exhibited reduced LV GLS, yet the difference between groups was not statistically significant.

LV isovolumic relaxation time (IVRT) and the proportion of IVRT > 100 ms increased in the non-OAD cohort, indicating an elevated level of initial manifestations of left-ventricular diastolic dysfunction. The ratio of early to late diastolic filling velocities ( $E/A$ ) < 0.7 showed an upward trend only in the non-OAD cohort. Consequently, although LV relaxation disturbances were evident at the first visit in both groups, progression was observed only in the non-OAD cohort. As a result, intergroup differences emerged in the  $E/e'$  ratio – the age-independent measure of early LV diastolic filling velocity relative to early diastolic mitral annulus velocity. The  $E/e'$  value was higher in the non-OAD cohort, and the proportion

of patients with  $E/e' \geq 9$  – an indicator of impaired LV relaxation – was greater in the non-OAD cohort than in the OAD cohort.

During hospitalization, the only parameter that significantly differentiated the groups was right atrial volume, which was larger in the OAD cohort (Table 2). This likely reflects the increased load on the right atrium due to concomitant OAD. Assessment of right-heart chamber dimensions (Tables 2 and 5) showed that, although within normal limits throughout the study, these dimensions decreased in both groups by the first visit. However, by the second visit, the non-OAD cohort exhibited a significant increase in right-ventricular (RV) volume and basal width, accompanied by a significant decline in RV systolic function as measured by tricuspid annular plane systolic excursion (TAPSE) and early diastolic velocity. The number of patients with reduced RV GLS at the second visit decreased not significantly and did not differ between groups.

During the outpatient phase of the study, all participants were monitored under close supervision of a cardiologist, which led to expanded medication regimens (Table 6). The spectrum of antihypertensive agents used broadened in both groups, with greater utilization of angiotensin II receptor blockers, statins, and diuretics (Table 7). Consequently, adherence to antihypertensive therapy among study participants varied from 64.3 % to 100 % (Figure 1). Adherence was highest for  $\beta$ -blockers and angiotensin II

receptor antagonists and lowest for statins in both groups. Multivariate analysis revealed no significant associations between the prescribed medications and the presence of OAD with the structural and functional parameters of the right heart ([Table 8](#)).

At the first visit, only four of the eight patients with COPD received bronchodilator therapy. Among these, one patient was placed on monotherapy with the long-acting anticholinergic tiotropium bromide; another received combined tiotropium bromide therapy with situational administration of short-acting  $\beta_2$ -agonists (SABA) (ipratropium bromide + fenoterol); a third was treated with a combination of inhaled glucocorticosteroids (IGC

and long-acting  $\beta_2$ -agonists (LABA) (budesonide/formoterol); and the fourth used SABA on demand (ipratropium bromide + fenoterol). All patients with COPD and pre-COPD were advised to cease smoking, and all COPD patients received training on the correct inhaler technique. The eight patients were prescribed short-acting bronchodilators for as-needed use; two patients with severe COPD were recommended a fixed combination of LABA and long-acting anticholinergic (LAMA) to achieve greater bronchodilation, while six patients received LAMA monotherapy.

**Table 4. Comparative dynamics of left-heart echocardiographic parameters in hypertensive patients 3 and 12 months after COVID-19-related lung injury, stratified by OAD status (OAD cohort n=38; non-OAD cohort n=89)**

Parameter	Group/between-group comparison	3-Month value	12-Month value	p-Value for within-group change
Left ventricle end-diastolic volume index (LV EDVi, ml/m <sup>2</sup> )	OAD cohort	44 [36; 55]	42 [38; 49]	0.777
	non-OAD cohort	44 [37; 51]	43 [38; 50]	0.767
	p (between groups)	0.850	0.643	
LV myocardial mass index (M-mode, g/m <sup>2</sup> )	OAD cohort	87 [77; 98]	90 [78; 97]	0.945
	non-OAD cohort	88 [79; 100]	89 [78; 101]	0.139
	p (between groups)	0.270	0.137	
LV hypertrophy according to body mass index (n %)	OAD cohort	14 (36.8)	13 (34.2)	1.000
	non-OAD cohort	29 (32.6)	34 (38.2)	0.383
	p (between groups)	0.647	0.669	
LV normal geometry (n %)	OAD cohort	21 (55.3)	18 (47.4)	0.581
	non-OAD cohort	44 (49.9)	44 (49.4)	1.000
	p (between groups)	0.577	0.836	
LV concentric remodeling (n %)	OAD cohort	10 (26.3)	12 (31.6)	0.724
	non-OAD cohort	30 (33.7)	26 (29.2)	0.608
	p (between groups)	0.411	0.787	
LV concentric hypertrophy (n %)	OAD cohort	3 (7.9)	4 (10.5)	1.000
	non-OAD cohort	10 (11.2)	9 (10.1)	1.000
	p (between groups)	0.574	0.946	
LV eccentric hypertrophy (n %)	OAD cohort	4 (10.5)	4 (10.5)	1.000
	non-OAD cohort	5 (5.6)	10 (11.2)	0.227
	p (between groups)	0.324	0.908	
LV outflow tract deceleration time (LVOT DT, ms)	OAD cohort	203±32	205±34	0.137
	non-OAD cohort	205±32	212±33	0.151
	p (between groups)	0.113	0.886	
LV outflow tract velocity time integral (LVOT VTI, cm)	OAD cohort	20.3 [17.1; 24.9]	20.7 [18.7; 22.7]	0.983
	non-OAD cohort	21.1 [18.6; 23.6]	20.7 [18.0; 23.4]	0.260
	p (between groups)	0.335	0.956	
LV isovolumic relaxation time (IVRT, ms)	OAD cohort	100 [88; 115]	105 [90; 119]	0.695
	non-OAD cohort	98 [88; 115]	110 [94; 122]	0.003
	p (between groups)	0.545	0.370	
IVRT>100 ms (n %)	OAD cohort	18 (47.4)	23 (60.5)	0.332
	non-OAD cohort	38 (42.7)	60 (67.4)	0.001
	p (between groups)	0.627	0.455	
Percentage of patients whose early-to-late diastolic filling velocity ratio (E/A) is less than 0.7 (n %).	OAD cohort	5 (13.2)	4 (10.5)	1.000
	non-OAD cohort	13 (14.6)	21 (23.6)	0.057
	p (between groups)	0.830	0.090	
Early diastolic mitral annulus velocity (e', cm/s)	OAD cohort	8.7±2.2	8.8±1.8	0.613
	non-OAD cohort	8.4±2.3	8.1±2.1	0.288
	p (between groups)	0.514	0.066	
Percentage of patients with an E/e' ratio (early diastolic filling velocity divided by early diastolic mitral annulus velocity) (n %).	OAD cohort	8.0 [6.5; 9.1]	7.4 [6.7; 8.7]	0.432
	non-OAD cohort	8.0 [7.0; 9.6]	8.7 [7.1; 9.6]	0.166
	p (between groups)	0.410	0.021	
E/e'≥9 (n %)	OAD cohort	11 (28.9)	8 (21.1)	0.549
	non-OAD cohort	30 (33.7)	37 (41.6)	0.265
	p (between groups)	0.839	0.037	
LV global longitudinal strain (LV GLS, %)	OAD cohort	-19.4±2.4	-19.9±2.5	0.338
	non-OAD cohort	-19.4±2.2	-19.0±2.5	0.089
	p (between groups)	0.965	0.166	
Reduced LV GLS (≥-18 %) (n %)	OAD cohort	3 (18.8%)	4 (25.0%)	1.000
	non-OAD cohort	16 (25.4%)	22 (34.9%)	0.238
	p (between groups)	0.579	0.451	

**Table 5.** Comparative dynamics of right-heart echocardiographic parameters in hypertensive patients 3 and 12 months after COVID-19-related lung injury, stratified by OAD status (OAD cohort n=38; non-OAD cohort n=89)

Parameter	Group/between-group comparison	3-Month value	12-Month value	p-Value for within-group change
Right atrium volume index (ml/m <sup>2</sup> )	OAD cohort	15.3 [12.4; 20.2]	16.7 [15.3; 19.2]	0.428
	non-OAD cohort	15.0 [12.0; 17.8]	16.7 [14.2; 19.5]	0.004
	p (between groups)	0.464	0.547	
Right ventricle anterior-posterior dimension index (mm/m <sup>2</sup> )	OAD cohort	13.0 [12.0; 13.6]	12.8 [12.0; 13.6]	0.210
	non-OAD cohort	12.9 [12.1; 13.7]	13.0 [12.0; 13.7]	0.389
	p (between groups)	0.660	0.490	
Right ventricle basal dimension (4-chamber position, mm)	OAD cohort	32.1±5.8	33.5±4.8	0.155
	non-OAD cohort	32.2±4.7	33.5±4.7	0.007
	p (between groups)	0.609	0.885	
Right ventricle diastolic area (cm <sup>2</sup> )	OAD cohort	15.8 [14.2; 19.0]	14.8 [13.0; 17.6]	0.108
	non-OAD cohort	16.5 [13.7; 18.6]	15.0 [13.0; 17.8]	0.001
	p (between groups)	0.941	0.821	
Right ventricle diastolic area index (cm <sup>2</sup> /m <sup>2</sup> )	OAD cohort	8.0±1.9	7.6±1.6	0.096
	non-OAD cohort	8.2±1.5	7.5±1.2	<0.001
	p (between groups)	0.482	0.811	
Right ventricle fractional area change (FAC, %)	OAD cohort	51.7±9.4	54.9±8.0	0.076
	non-OAD cohort	51.1±8.7	53.8±8.4	0.025
	p (between groups)	0.800	0.384	
FAC<35% (n %)	OAD cohort	1 (2.6)	0	1.000
	non-OAD cohort	4 (4.5)	1 (1.1)	0.375
	p (between groups)	0.614	0.516	
Tricuspid annular plane systolic excursion (TAPSE, mm)	OAD cohort	23.0±2.9	22.7±2.2	0.678
	non-OAD cohort	23.4±2.7	22.4±2.2	0.009
	p (between groups)	0.783	0.389	
Tricuspid annulus systolic velocity (S') of the pulmonary artery (pStv, cm/s)	OAD cohort	10.0 [9.0; 11.0]	9.5 [8.0; 12.0]	0.307
	non-OAD cohort	9.0 [7.0; 11.0]	9.0 [7.0; 10.0]	0.111
	p (between groups)	0.220	0.091	
pStv<9.5 cm/s (n %)	OAD cohort	15 (39.5)	19 (50.0)	0.454
	non-OAD cohort	50 (56.2)	60 (67.4)	0.121
	p (between groups)	0.085	0.064	
RV global longitudinal strain (RV GLS, %)	OAD cohort	-20.6±4.1	-21.6±4.5	0.130
	non-OAD cohort	-21.4±3.5	-22.2±3.4	0.194
	p (between groups)	0.444	0.837	
Reduced RV global longitudinal strain (RV GLS≥-20 %) (n %)	OAD cohort	8 (47.1%)	5 (29.4%)	0.375
	non-OAD cohort	16 (35.6%)	10 (22.2%)	0.146
	p (between groups)	0.407	0.555	
Tricuspid annular early diastolic velocity (pEtv, cm/s)	OAD cohort	13.3±2.9	13.0±3.0	0.206
	non-OAD cohort	13.1±2.7	12.5±2.2	0.047
	p (between groups)	0.622	0.867	

All eighteen asthmatic patients received baseline therapy at the first visit: three were on inhaled glucocorticosteroids (IGC) monotherapy (two with budesonide, one with beclomethasone), and three combined IGC monotherapy with a short acting combination of ipratropium bromide + fenoterol as needed. Nine asthmatic patients were prescribed a fixed combination of IGC/LABA (budesonide/formoterol) both as basic therapy and for symptom relief. Three asthmatic patients did not receive anti-inflammatory therapy but used short-acting IGC/SABA on an as-needed basis, followed by monitoring of therapeutic effectiveness.

A multivariate analysis demonstrated no significant influence of the medications taken or OAD on the structural and functional parameters.

## Discussion

The small size of the OAD group is a consequence of the study's reliance on data from the database registry "Prospective registry of persons who have had COVID-19-associated pneumonia" [2] and is limited by the total sample size of 350 patients at baseline. According to the 2025 consensus statement of the European Society of Cardiology and the European

Association of Cardiovascular Imaging, clinical cardiologists and researchers are encouraged to use registry data, as registries provide large, unselected, consecutive cohorts for collecting real-world prospective data in a standardized manner, whereas randomized clinical trial samples are limited. Registries are therefore key research tools for assessing the real-world consequences of the phenomenon under study.

Patients with COPD, pre-COPD, and bronchial asthma were combined into the OAD group based on the commonality of echocardiographic manifestations – the ability of OAD to cause right-ventricular overload.

It has been previously shown that in patients with AH, RV diastolic dysfunction accompanies LV diastolic dysfunction regardless of the presence or absence of pulmonary arterial hypertension [6]. Moreover, diastolic dysfunction of the LV and RV develops asynchronously in 36% of AH patients, and disturbances in RV diastolic function are accompanied by RV enlargement [7]. This was observed in our cohort: in the non-OAD group, a decrease in early diastolic tricuspid annular velocity was accompanied by an increase in basal RV dimension.

**Table 6. Comparative characteristics of prescribed therapy at inclusion (3 months post-COVID-19 lung injury) and at 9 months post-inclusion (12 months post-COVID-19 lung injury) according to OAD status**

Group of medicine (n %)	Visit / p within groups in dynamics	OAD patients (n=38)	Non-OAD patients (n=89)	p between groups
β-blockers	Visit 1	10 (26.3%)	53 (59.6%)	0.001
	Visit 2	10 (26.3%)	59 (66.3%)	<0.001
	p	1.000	0.180	
Calcium channel blockers	Visit 1	12 (31.6%)	24 (27.0%)	0.597
	Visit 2	17 (44.7%)	32 (36.0%)	0.352
	p	0.267	0.057	
Adenosine-converting enzyme inhibitors	Visit 1	7 (18.4%)	36 (40.4%)	0.016
	Visit 2	6 (15.8%)	34 (38.2%)	0.013
	p	1.000	0.774	
Mineralocorticoid receptor antagonists	Visit 1	1 (2.6%)	1 (1.1%)	0.532
	Visit 2	4 (10.5%)	6 (6.7%)	0.485
	p	0.250	0.125	
Angiotensin II receptor blockers	Visit 1	30 (78.9%)	44 (49.4%)	0.002
	Visit 2	30 (78.9%)	53 (59.6%)	0.035
	p	1.000	0.012	
Centrally acting antihypertensive drugs	Visit 1	3 (7.9%)	13 (14.6%)	0.297
	Visit 2	5 (13.2%)	23 (25.8%)	0.114
	p	0.500	0.006	
Statins	Visit 1	23 (60.5%)	68 (76.4%)	0.069
	Visit 2	29 (76.3%)	74 (83.1%)	0.368
	p	0.109	0.070	
Diuretics	Visit 1	28 (73.7%)	64 (71.9%)	0.838
	Visit 2	25 (65.8%)	68 (76.4%)	0.216
	p	0.453	0.219	

Percentages are calculated relative to the total number of patients in each cohort (OAD=38, non-OAD=89).

**Table 7. Comparative characteristics of medication taken**

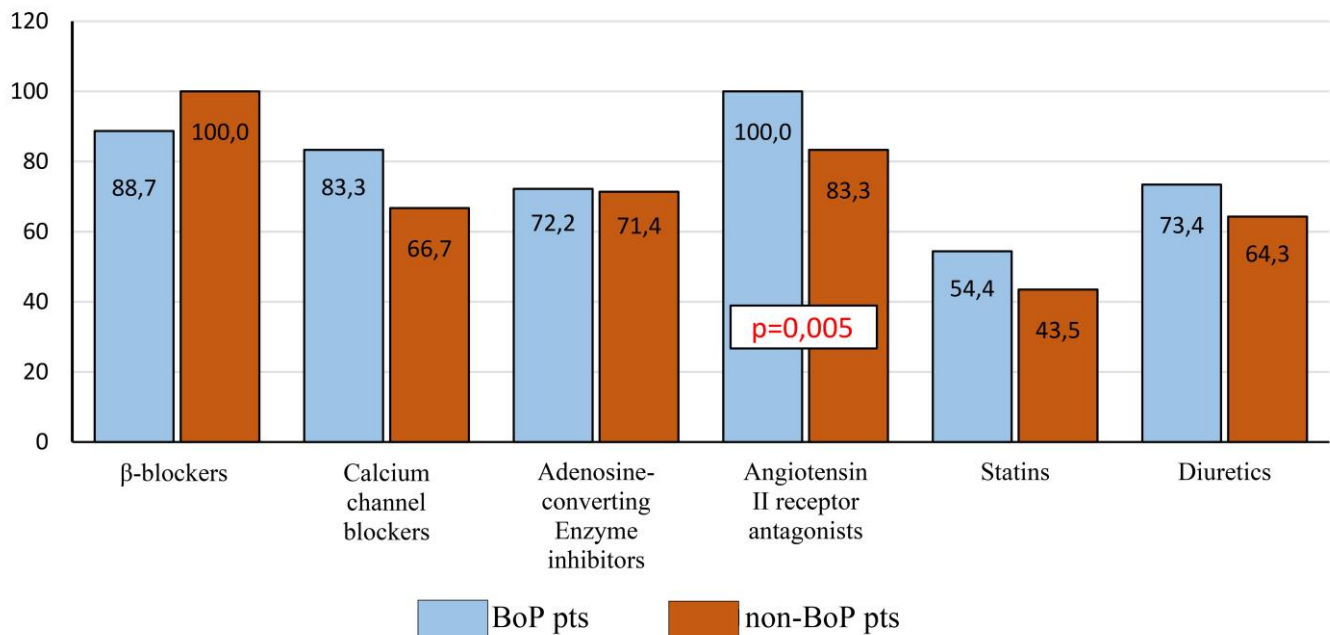
Group of medicine (n %)	Visit / p within groups in dynamics	OAD patients (n=38)	Non-OAD patients (n=89)	p between groups
β-blockers	Visit 1	8 (21.1%)	31 (34.8%)	0.123
	Visit 2	10 (26.3%)	47 (52.8%)	0.006
	p	0.687	0.004	
Calcium channel blockers	Visit 1	6 (15.8%)	14 (15.7%)	0.993
	Visit 2	8 (21.1%)	20 (22.5%)	0.860
	p	0.625	0.109	
Adenosine-converting enzyme inhibitors	Visit 1	6 (15.8%)	25 (28.1%)	0.139
	Visit 2	5 (13.2%)	26 (29.2%)	0.054
	p	1.000	1.000	
Mineralocorticoid receptor antagonists	Visit 1	1 (2.6%)	-	0.124
	Visit 2	1 (2.6%)	3 (3.4%)	0.827
	p	1.000	1.000	
Angiotensin II receptor blockers	Visit 1	18 (47.4%)	33 (37.1%)	0.279
	Visit 2	25 (65.8%)	44 (49.4%)	0.090
	p	0.016	0.013	
Centrally acting antihypertensive drugs	Visit 1	3 (7.9%)	3 (3.4%)	0.271
	Visit 2	3 (7.9%)	11 (12.4%)	0.552
	p	1.000	0.008	
Statins	Visit 1	4 (10.5%)	20 (22.5%)	0.115
	Visit 2	10 (26.3%)	37 (41.6%)	0.103
	p	0.031	<0.001	
Diuretics	Visit 1	10 (26.3%)	29 (32.6%)	0.483
	Visit 2	18 (47.4%)	47 (52.8%)	0.574
	p	0.021	0.001	

Patients at inclusion (3 months after COVID-19 lung injury) and at 9 months after inclusion (12 months after COVID-19 lung injury) according to OAD status. Percentages are calculated relative to the total number of patients in each cohort (OAD=38, non-OAD=89).

**Table 8. Multivariate analysis of medication effects on right heart parameters**

Medication (or OAD)	Tricuspid annular plane systolic excursion (TAPSE)		Right atrium volume index (RAVI)		E/e12≥9	
	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
OAD	1.414 (0.584-3.421)	0.442	3.167 (0.070-142.84)	0.553	0.396 (0.154-1.019)	0.055
β-blockers	0.929 (0.418-2.063)	0.856	0.476 (0.015-14.781)	0.672	1.152 (0.521-2.547)	0.727
Adenosine-converting enzyme inhibitors	1.39 (0.451-4.286)	0.567	3.884 (0.030-496.57)	0.583	1.783 (0.581-5.468)	0.312
Angiotensin II receptor blockers	1.17 (0.443-3.089)	0.752	1.678 (0.026-109.99)	0.808	1.319 (0.485-3.588)	0.587

Influence of medications taken and OAD on structural and functional right-heart indices.



**Figure 1. Adherence to Antihypertensive Therapy.** Adherence of hypertensive patients at the second visit to treatment prescribed at the first visit. ACE, adenosine-converting enzyme.

Subclinical LV systolic dysfunction is characteristic of AH patients with LV hypertrophy, a finding that aligns with our results: the frequency of reduced LV global longitudinal strain at the end of the study was relatively high – 35% in the non-OAD group and 25% in the OAD group (for comparison, the prevalence of LV hypertrophy was 38% in the non-OAD group and 34% in the OAD group). LV damage with systolic and diastolic dysfunction after pulmonary injury associated with COVID-19 has also been shown to be associated with levels of immune-inflammation markers [8]. No data were found regarding the incidence of subclinical RV systolic dysfunction in AH patients one year after COVID-19. In our study, its frequency was 22% in the non-OAD group and 29% in the OAD group.

Thus, the hospitalization course for patients with AH was essentially the same regardless of their OAD status. During outpatient follow-up, OAD patients had a lower incidence of HFpEF, whereas non-OAD patients exhibited more than a twofold increase in the frequency of stage 3 hypertension, primarily attributable to CKD stages 2 and 3A as defined by GFR. One year after discharge, non-OAD patients had a higher incidence of LV relaxation abnormalities and displayed worsening right-heart parameters – both structural (increased right-atrial volume and basal right-ventricular dimension) and functional (decreased TAPSE and early diastolic tricuspid annular velocity). A trend toward reduced tricuspid annular systolic velocity further suggests impaired RV systolic function in non-OAD patients. These findings imply a comparatively favorable recovery trajectory from COVID-19 in the OAD group.

Our results align with those reported by other investigators. A retrospective observational study of COVID-19 outpatients, with and without asthma, found better outcomes in asthmatic patients, including lower rates of hospitalization, intensive care unit admission, and shorter hospital stays [9]. A single-center study confirmed a relatively benign course of COVID-19 in asthmatic individuals, noting that asthma does not predispose to severe

disease and that its treatment may positively influence the disease course. However, the coexistence of asthma with AH, obesity, and diabetes is recognized as worsening the prognosis of COVID-19 [10].

Consequently, the initial hypothesis was not supported; OAD patients proved more resistant to the factors that drive pathological (structural and functional) cardiac remodeling in AH patients during the late phase of pulmonary injury in COVID-19.

The adaptation of the myocardium in OAD patients to the factors responsible for pathological remodeling during the late recovery phase of COVID-19 can be explained by myocardial preconditioning – a metabolic adaptation to ischemia that arises after repeated episodes of reduced oxygen delivery to the myocardium and confers increased resistance to subsequent prolonged hypoxia. Accordingly, OAD may act as a chronic hypoxic stressor.

Another plausible explanation involves the protective effect of basic therapy in OAD patients. Of the 38 OAD patients, 22 did not receive basic therapy during hospitalization or at the first follow-up visit. Nevertheless, drawing conclusions about the role of basic therapy in preventing RV remodeling would require a larger OAD cohort.

In contrast, non-OAD patients were prescribed β-blockers and adenosine-converting enzyme inhibitors significantly more frequently at both visits, a factor that could have improved structural and functional myocardial parameters in this group. However, such improvement was not observed; echocardiography parameters in the non-OAD group worsened. The results could have been influenced by the administration of β-blockers in patients without OAD at the second visit; however, any observed relationship was inverse (Table 8). Therefore, the negative trends in echocardiographic parameters among non-OAD patients provide grounds to exclude the impact of differential drug therapy on the study outcomes.

### Limitations

1. A small cohort of patients with OAD.
2. Absence of pre-COVID-19 data.
3. Lack of spirometry data with bronchodilator in pre-COPD patients.

### Conclusion

In patients with AH, the hospital period of lung injury during acute COVID-19 does not differ between those with and without OAD. One year after discharge, OAD patients show a significantly lower likelihood of HFpEF compared with assessments at three months post-COVID-19. In non-OAD patients, one year after COVID-19, BMI, and the prevalence of stage 3 AH increase, LV relaxation abnormalities are more frequently detected, and structural and functional parameters of the right heart are worse compared with OAD patients.

### Conflict of Interest

The authors declare no conflict of interest.

### Ethical Approval

All procedures involving human participants complied with the ethical standards of the institutional and/or national research committee, the 1964 Declaration of Helsinki, and its subsequent amendments. Written informed consent was obtained from all individual participants included in the study.

### AI Disclosure Statement

During the preparation of this work, the authors did not use any generative AI or AI-assisted tools for the generation of content, data analysis, or the production of figures.

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