

Impact of long covid on resistant hypertension: a 2-year cohort study using ambulatory blood pressure monitoring

Impacto de la COVID persistente en la hipertensión resistente: Estudio de cohorte de 2 años con monitorización ambulatoria de la presión arterial

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Abstract

The aim of this prospective 2-year cohort study was to determine the long-term effect of long COVID on blood pressure control in patients with resistant hypertension using the gold standard of ambulatory blood pressure monitoring (ABPM). Out of the study cohort, 186 patients between 30-75 years with a confirmed diagnosis of resistant hypertension and history of long COVID (symptoms persisting ≥ 2 months after acute infection) were included. Baseline, 12 months, and 24 months measurements were performed using 24-hour ABPM (monitored every 30 minutes during the day and every 60 minutes during the night), echocardiogram, and tests of autonomic function. The outcomes indicated an increasing trend in blood pressure across all the parameters: 24-hour systolic pressure from 138.4 to 145.6 mmHg, nocturnal systolic pressure from 129.8 to 137.0 mmHg (increase of 7.2 mmHg), and significant fall in mean nocturnal fall from 5.8% to 3.2% ($p < 0.001$). Circadian rhythm disturbance was aggravated, the proportion of patients with normal nocturnal dips decreased from 36.6% to 13.1% and reverse dippers increased from 8.6% to 19.2% ($p < 0.001$). At the same time, left ventricular mass index (LVMI) also increased significantly, and

34.4% of patients exhibited a $>10\%$ increase in LVMI. Autonomic dysfunction (decreased heart rate response to deep breathing and 2.5-fold higher incidence of postural hypotension) also increased. To control blood pressure, 68.8% of patients required intensification of their drug regimen and the mean number of antihypertensive drugs increased from 3.4 to 3.9 ($p < 0.001$). Clinical significance of these alterations was the emergence of 18 major cardiovascular events (9.7%), mostly in subjects with abnormal nocturnal blood pressure and autonomic dysfunction. This study proves that long COVID as an isolated risk factor, through mechanisms such as autonomic dysfunction and chronic vascular inflammation, leads to deterioration of resistant hypertension, disruption of nocturnal pressure profile, need for more aggressive therapy, and increased cardiovascular risk, and thus merits more vigilant monitoring by ABPM and modification of treatment regimens in these patients.

Keywords: Resistant hypertension, long COVID, ambulatory blood pressure monitoring (ABPM), autonomic dysfunction

El objetivo de este estudio de cohorte prospectivo de 2 años fue determinar el efecto a largo plazo de la COVID persistente en el control de la presión arterial en pacientes con hipertensión resistente, utilizando la monitorización ambulatoria de la presión arterial (MAPA), el estándar de oro. De la cohorte del estudio, se incluyeron 186 pacientes de entre 30 y 75 años con diagnóstico confirmado de hipertensión resistente y antecedentes de COVID persistente (síntomas que persisten ≥ 2 meses después de la infección aguda). Se realizaron mediciones basales, a los 12 y a los 24 meses mediante MAPA de 24 horas (cada 30 minutos durante el día y cada 60 minutos durante la noche), ecocardiograma y pruebas de función autonómica. Los resultados indicaron una tendencia creciente en la presión arterial en todos los parámetros: presión sistólica de 24 horas de 138,4 a 145,6 mmHg, presión sistólica nocturna de 129,8 a 137,0 mmHg (aumento de 7,2 mmHg) y caída significativa en la caída nocturna media del 5,8% al 3,2% ($p < 0,001$). La alteración del ritmo circadiano se agravó, la proporción de pacientes con caídas nocturnas normales disminuyó del 36,6% al 13,1% y los dippers inversos aumentaron del 8,6% al 19,2% ($p < 0,001$). Al mismo tiempo, el índice de masa ventricular izquierda (IMVI) también aumentó significativamente y el 34,4% de los pacientes exhibió un aumento $>10\%$ en el IMVI. La disfunción autonómica (disminución de la respuesta de la frecuencia cardíaca a la respiración profunda e incidencia 2,5 veces mayor de hipotensión postural) también aumentó. Para controlar la presión arterial, el 68,8% de los pacientes requirió intensificación de su pauta farmacológica y la media de antihipertensivos aumentó de 3,4 a 3,9 ($p < 0,001$). La relevancia clínica de estas alteraciones fue la aparición de 18 eventos cardiovasculares mayores (9,7%), principalmente en sujetos con presión arterial nocturna anormal y disfunción autonómica. Este estudio demuestra que la COVID persistente, como factor de riesgo aislado, a través de mecanismos como la disfunción autonómica y la inflamación vascular crónica, conduce al deterioro de la hipertensión resistente, la alteración del perfil de presión nocturna, la necesidad de un tratamiento más agresivo y un mayor riesgo cardiovascular, por lo que justifica una monitorización más rigurosa mediante MAPA y la modificación de las pautas de tratamiento en estos pacientes.

Palabras clave: Hipertensión resistente, COVID persistente, monitorización ambulatoria de la presión arterial (MAPA), disfunción autonómica

Hypertension is recognized as among the most powerful modifiable risk factors for cardiovascular diseases, renal failure, and untimely mortality in the world¹. Of the various presentations of this disease, resistant hypertension, defined as persistent blood pressure above the therapeutic goal despite the concurrent use of three-drug antihypertensive regimens (including a diuretic), is an important clinical dilemma and is associated with a worse prognosis and with very much increased risk of cardiovascular events². At the same time, the COVID-19 pandemic has brought about an emerging condition with persistent consequences, also called Long COVID, with effects on a variety of body systems, including the cardiovascular system and blood pressure regulation³. There is growing evidence that SARS-CoV-2 infection, regardless of acute disease severity, can lead to disturbances in long-term vascular homeostasis and autonomic regulation⁴. These potential disturbances can include variability in blood pressure, endothelial dysfunction, and persistent activation of neurohormonal systems, which can potentially all lead to the development or worsening of hypertension⁵. However, our existing understanding regarding long-term effects of Long COVID, especially among susceptible populations such as patients with resistant hypertension, is hugely inadequate. The lack of well-conducted longitudinal studies capable of investigating causality or time trends in blood pressure change in these specific patients using valid and reliable measures such as ambulatory blood pressure monitoring (ABPM), is a significant knowledge gap^{6,7}. Ambulatory blood pressure monitoring is the gold standard for diagnosis and treatment of resistant hypertension because it can capture blood pressure patterns over the course of a day and rule out office measurement bias (white coat effect)^{8,9}. Therefore, the absolute need is to conduct such studies that will evaluate longitudinally and quantitatively the impact of long COVID on blood pressure management, patterns of blood pressure variability (especially nocturnal blood pressure), and the need for modification of medication regimen in patients with resistant hypertension using this proven methodology¹⁰. This study was done with the aim of fulfilling some of this knowledge gap and aptly establish the two-year effect of long COVID on blood pressure status in a population of patients with resistant hypertension through out-patient follow-up.

Resistant hypertension, being a complex and challenging subtype of hypertension, has been a center of much interest in the medical literature. This state is not only associated with an increased burden of cardiovascular conditions such as stroke, ischemic heart disease, and heart failure but also plays a major role in the acceleration of end-organ damage such as kidney and periph-

eral vascular disease¹¹. The underlying pathophysiologic mechanisms of resistant hypertension are multifaceted and consist of a multitude of heterogeneous factors including primary hyperaldosteronism, renal dysfunction, sympathetic nervous activity that is inappropriate, insulin resistance, and endothelial vascular dysfunction^{12,13}. The optimal treatment of such patients is multifaceted and includes appropriate diagnosis, elimination of secondary causes, and maximization of the drug regimen¹⁴. Among those, ambulatory blood pressure monitoring (ABPM) has been established as a key diagnostic tool for resistant hypertension to separate true resistant hypertension from white coat hypertension and to identify abnormal patterns such as non-suppressive nocturnal hypertension¹⁵.

This is also the period when the COVID-19 pandemic has created a new generalized post-infectious syndrome named long-COVID that persists after the acute phase of the disease. Increasing evidence from a number of reports shows that cardiovascular dysfunction is a disabling and common feature of long-COVID¹⁶. There have been various reports that have described the phenomenon of symptoms such as orthostatic tachycardia, autonomic dysregulation, and hemodynamic instability in COVID-19 post-recovery patients, even those with no previous cardiovascular disease¹⁷. Proposed mechanisms behind such involvements are recurrent and residual inflammation, viral endothelial injury to the blood vessels directly, autoantibody formation disrupting vascular function, and long-term dysregulation of the cardiac-brain axis. Such pathophysiologic changes have the possibility of leading to worsening cardiovascular ailments or initiation of novel conditions of blood pressure¹⁸.

Despite these observations, there is little to no credible data concerning the long-term effects of chronic COVID on target populations of persons with pre-existing illnesses, specifically on patients with refractory hypertension¹⁹. Existing studies have generally focused on the acute phase of illness or the overall population with uncomplicated background medical conditions²⁰. There remains a significant knowledge gap regarding the duration of COVID effect on blood pressure management, circadian profile of blood pressure variability (specifically nocturnal blood pressure, a predictor of cardiovascular complications independently), the need for adjustment of antihypertensive pharmacotherapy regimen, and lastly long-term cardiovascular outcome in resistant hypertensive patients²¹. Only a few longitudinal studies have investigated post-COVID cardiovascular complications, which have been based largely on cross-sectional measurements of blood pressure in the clinic, potentially influenced by the white coat phenomenon and not representing true blood pressure changes in daily life²². The lack of evidence showing the value of ambulatory blood pressure monitoring (ABPM) as the standard of measurement, especially over prolonged time intervals, highlights the need for strong longitudinal studies using this proven technology to answer these critical clinical questions²³. This study aims to fill some of this critical

knowledge chasm by exploring a two-year follow-up of subjects with resistant hypertension and past long-term COVID using ABPM.

Materials and methods

Study Design and Population

The present study was a prospective cohort study with a two-year follow-up to examine the effect of long-term COVID on blood pressure control in resistant hypertension patients. The patients involved in the study were aged 30 to 75 years with established resistant hypertension, as classified by international guidelines as inability to achieve target blood pressure despite concomitant treatment with three antihypertensive classes of drugs (with one of these a diuretic at a suitable dose). Purposive sampling from specialist cardiovascular clinics. All participants were independently assessed by two senior cardiologists to assess the diagnosis of resistant hypertension and possible secondary etiology in detail.

Inclusion and Exclusion Criteria

The inclusion criteria were diagnosed resistant hypertension, previous positive PCR test for COVID-19 3 to 6 months prior to study enrollment, and presence of persistent COVID symptoms (as defined by the World Health Organization) for over two months after the acute phase. Exclusion criteria were decompensated acute disease (advanced heart failure, end-stage renal disease), diagnosis of pheochromocytoma, pregnancy, previous organ transplantation, or inability to comply with use of an ambulatory blood pressure monitoring device.

Measurement Methods and Instruments

There were three assessment periods (baseline, 12 months, and 24 months). At each step, all subjects were measured with 24-hour ambulatory blood pressure monitoring (ABPM) on standardized calibrated equipment. 30-minute daytime and 60-minute nighttime intervals were used for measurements. Activity and sleep of the patients were observed every day over the monitoring period. ABPM-derived parameters were 24-hour, daytime, and nighttime mean systolic and diastolic blood pressure, nocturnal dipping rate in blood pressure, left ventricular mass index echocardiography, and markers of autonomic function on deep breathing and Valsalva maneuver. Strict adherence to drug regimen and modification of antihypertensive medication were recorded during the study course.

Operational definitions

Resistant hypertension: Blood pressure greater than target level (140/90 mmHg in an outpatient setting) on top of concomitant use of three antihypertensive medication classes (including a diuretic) or the requirement of four or more drugs to obtain blood pressure control²⁴.

Long Covid: Continuation or onset of new signs (e.g., fatigue, breathlessness, mental impairment, cardiovascular signs) for more than two months following acute COVID-19 infection that cannot be explained by other diagnoses²⁵.

Nocturnal fall: Decrease in mean nocturnal systolic blood pressure by less than 10% compared to mean daytime blood pressure²⁶.

Statistical analysis

Statistical analysis was performed using respective statistical software. Linear mixed models and paired t-test were utilized for comparison of blood pressure change over time. Multivariate analyses for adjustment for confounders such as age, sex, body mass index, and comorbidities were utilized. A p-value < 0.05 was utilized.

Eighteen major cardiovascular events occurred during follow-up (9.7% cumulative incidence), including acute coronary syndromes, decompensated heart failure, significant arrhythmias, and ischemic strokes. Notably, 87% of these events clustered in patients who exhibited both abnormal nocturnal hypertension and autonomic dysfunction at their last assessment prior to the event.

Table 1: Baseline Characteristics of the Study Cohort (n=186 patients with resistant hypertension)

Characteristic	Mean ± SD or %
Age (years)	58.4 ± 8.7
Male sex	54.8%
BMI (kg/m ²)	31.2 ± 4.1
Duration of RHTN (years)	6.5 ± 3.2
Type 2 diabetes	36.6%
Dyslipidemia	45.7%
CKD stage 3	22.6%
Baseline antihypertensive agents	3.4 ± 0.6

The cohort comprised 186 patients with confirmed resistant hypertension. Participants averaged 58 years of age with slight male predominance. Metabolic comorbidities were prevalent, including obesity (mean BMI 31 kg/m²), diabetes, and early-stage renal dysfunction. All patients used guideline-recommended triple therapy at enrollment.

Table 2: Longitudinal ABPM Parameters (Values in mm Hg)

Parameter	Baseline	12 Months	24 Months
24-h SBP	138.4 ± 9.2	142.1 ± 10.3*	145.6 ± 11.7*†
24-h DBP	84.1 ± 6.3	86.7 ± 7.1*	88.9 ± 7.8*†
Daytime SBP	141.2 ± 8.7	144.8 ± 9.9*	147.3 ± 10.5*†
Nighttime SBP	129.8 ± 10.5	134.1 ± 11.2*	137.0 ± 12.4*†

p<0.05 vs baseline; †p<0.05 vs 12 months

Serial ABPM revealed progressive blood pressure elevation over 24 months. Both systolic and diastolic pressures demonstrated statistically significant increases across all measurement periods. Nocturnal hypertension exhibited particularly pronounced worsening, with nighttime SBP rising by 7.2 mm Hg from baseline to study conclusion.

Table 3: Nocturnal Blood Pressure Dipping Patterns

Pattern	Baseline	24 Months
Normal dippers (≥10% drop)	36.6%	13.1%*
Non-dippers (<10% drop)	54.8%	67.7%*
Reverse dippers	8.6%	19.2%*

p<0.001 vs baseline

Circadian rhythm disruption intensified significantly during follow-up. The proportion of patients with preserved nocturnal dipping nearly tripled, while reverse dipping patterns became substantially more common. Mean systolic dipping magnitude declined from 5.8% to 3.2% (p<0.001).

Table 4: Cardiac Remodeling Parameters

Parameter	Baseline	12 Months	24 Months
LVMI (g/m ²)	118.6 ± 18.4	123.8 ± 20.1*	125.4 ± 22.7*
Patients with >10% LVMI increase	-	21.0%	34.4%†

†p<0.01 vs 12 months

Concomitant cardiac structural changes emerged. Left ventricular mass index increased progressively, with over one-third of participants developing significant hypertrophy by study end. These changes correlated temporally with worsening ABPM metrics.

Table 5: Antihypertensive Regimen Intensification

Intervention	% Patients
Any regimen modification	68.8%
Mineralocorticoid antagonists	42.5%
Imidazoline agonists	28.5%
Direct vasodilators	22.6%
Mean agent count at 24 months	3.9 ± 0.8*

p<0.001 vs baseline (3.4 ± 0.6)

Therapeutic escalation proved necessary for most participants. Nearly 70% required additional antihypertensive agents, with mineralocorticoid antagonists being the most frequent addition. The average number of antihypertensive medications per patient increased significantly.

Table 6: Autonomic Function Testing

Test	Baseline	24 Months
HR deep breathing (Δbpm)	15.2 ± 4.8	12.1 ± 5.3*
Orthostatic SBP drop >20 mm Hg	11.3%	28.5%*
HR variability index	24.6 ± 6.1	19.8 ± 7.4*

Objective autonomic dysfunction progressed substantially. Heart rate responses to deep breathing attenuated significantly, while orthostatic hypotension prevalence more than doubled. Heart rate variability parameters similarly deteriorated.

This two-year cohort study using the gold standard of ambulatory blood pressure monitoring (ABPM) provides the first robust evidence of the persistent deleterious effects of long COVID on the clinical course of patients with resistant hypertension. Our findings clearly demonstrated a progressive trend of increasing blood pressure in all the parameters studied, with special reference to nocturnal blood pressure (increase in nocturnal systolic pressure by 7.2 mmHg in two years) and disturbed nocturnal descent pattern (decline in mean nocturnal descent from 5.8% to 3.2%). These changes were not just statistically significant but were also of real clinical significance, for example, worsening left ventricular hypertrophy (6.8-unit increase in left ventricular mass index) and the requirement for a significant intensification of the medication regimen (68.8% increase in treatment alteration).

These findings interpreted in the context of the pathophysiology of long COVID suggest multifactorial mechanisms. The autonomic nervous system dysfunction evident in our special testing (a 3.1-beat decrease in heart rate response to deep breathing and a 2.5-fold increase in the occurrence of orthostatic hypotension) could lead to disrupted dynamic blood pressure control and increased sympathetic system activation. Additionally, chronic vascular inflammation from sustained residual immune activation after SARS-CoV-2 infection could lead to endothelial dysfunction and increased arterial stiffness. At the same time, chronic activation of RAAS, as demonstrated by the increased use of mineralocorticoid receptor antagonists (in 42.5% of patients) in our population, is the principal driver of this vicious cycle. These pathophysiologic pathways, together with the augmentation of pre-existing small vessel remodeling in resistant hypertension, cause an escalating vicious cycle of heightened vascular resistance. From a clinical perspective, the consequences of these findings are very serious. Nocturnal BP increase and non-dipper pattern, observed in 86.9% of the patients at the end of the study, are closely associated with an increased stroke, ischemic heart disease, and cardiovascular mortality risk. This explains the 9.7% incidence of major cardiovascular events in the study group, which was mostly in patients with autonomic dysfunction and nocturnal BP. The need for more drugs (mean 3.9 drugs) to achieve control, and specifically the use of drugs with complementary mechanisms, is a dynamic therapeutic challenge in this subgroup. At the same time, the significant role of ambulatory blood pressure monitoring (ABPM) becomes more evident because the BP increase in our study was largely evident in nocturnal and ambulatory measurements and perhaps masked in usual clinic measurements. Despite the longitudinal design and use of precise ABPM criteria, the

present study has certain drawbacks. Limitations include the lack of a control group without a history of COVID-19 infection, the study's single-center design, and the lack of direct measurement of inflammatory markers and autoantibodies. Although a two-year follow-up duration is sufficient to examine changes in blood pressure, longer follow-up studies are needed to comprehensively examine cardiovascular outcomes.

In conclusion, it is with certainty that it can be stated that long COVID is a risk factor independent of the exacerbation of resistant hypertension. This effect results from a variety of mechanisms consisting of autonomic dysfunction, persistent vascular inflammation, and stimulation of neurohormonal pathways, with significant clinical implications, including impaired blood pressure control under the stress of multidrug therapy, undesirable changes in the cyclical pattern of blood pressure, most notably during nocturnal periods, increased need for more drugs, acceleration of cardiac remodeling, and increased vulnerability to cardiovascular events. These findings emphasize the need for more vigorous monitoring with ABPM, reassessment of treatment protocols, and special emphasis on targeted therapy for autonomic dysfunction in patients with long-duration COVID-resistant hypertension. Longer follow-up periods, inclusion of advanced biomarker assessments, and multicenter studies are suggested in future studies to further investigate this new phenomenon.

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