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Effects of COVID 19 on Anthropometric Measurements and Selected Clinical Parameters

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Abstract: Background: Beyond respiratory pathology, COVID-19 exerts multi-systemic effects, including profound alterations in body composition and cardiopulmonary function. This study quantifies longitudinal changes in anthropometry (weight, arm, abdominal, hip, thigh circumferences, BMI) and clinical parameters (SpO_2, BP, pulse rate) across three time durations, vis-à-vis, pre COVID, during COVID and finally, post COVID.

Methods: A prospective cohort (n= 511, 255 males, 256 females) was assessed at all three time points. Repeated measures ANOVA with Tukey's HSD tested temporal trends for the clinical parameters and paired t-tests compared pre vs. post COVID anthropometric values ($\alpha = 0.01$). in addition, the correlation between pre COVID BMI and the clinical parameters was also assessed.

Results: All anthropometric parameters increased significantly post COVID ($p < 0.0001$). Clinically, BP and pulse rate were lowest during the pre infection phase, elevated during infection, and remained intermediate post recovery (all $p < 0.0001$). SpO_2 increased significantly post-infection ($p < 0.0001$), likely reflecting resolution of hypoxia. Notably, pre-COVID BMI showed no significant correlation with acute-phase clinical severity.

Conclusion: COVID-19 induces durable, measurable changes in body composition and cardiopulmonary physiology, extending well beyond viral clearance. These findings advocate for integrated post COVID metabolic and cardiovascular monitoring, irrespective of baseline BMI.

Keywords: COVID 19, Anthropometry, BMI, SpO2, BP, Pulse Rate, Weight

Introduction: Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the etiological agent of coronavirus disease 2019 (COVID-19), emerged in Wuhan, China, in December 2019, rapidly evolving into a global pandemic associated with over 7 million deaths and pervasive multisystem sequelae (*WHO, 2023*). Phylogenomic analyses confirm SARS-CoV-2 is a beta-coronavirus sharing 96.2% whole-genome identity with the bat coronavirus RaTG13, supporting zoonotic spillover as its origin (*Zhou et al., 2020*). Clinically, the infection spans a continuum, from asymptomatic carriage to fatal acute respiratory distress syndrome (ARDS). In a landmark cohort of 1099 patients across 552 Chinese hospitals, 43.8% presented with fever on admission, while 87.9% developed fever during hospitalization while cough (67.7%) and fatigue (38.1%) were also prevalent (*Guan et al., 2020*). Notably, olfactory and gustatory dysfunction emerged as the early, highly sensitive markers among 417 mild-to-moderate cases in Europe, where 85.6% reported anosmia and 88.0% reported ageusia, often preceding other symptoms (*Lechien et al., 2020*). Now, cardiovascular and respiratory instability are usually marked as hallmarks of acute disease. Myocardial injury, defined by elevated troponin, occurred in 19.7% of 416 hospitalized patients in Wuhan and was associated with a 4.26-fold (95% CI; 1.92-9.49) higher mortality (*Shi et al., 2020*). Simultaneously, "silent hypoxemia," meaning profound arterial desaturation without dyspnea, was frequently observed and attributed to impaired hypoxic pulmonary vasoconstriction and ventilation-perfusion mismatch (*Tobin et al., 2020*). A study of 58 subjects (hospitalized COVID patients) and 29 controls (hospitalized bacterial pneumonia patients) revealed that COVID-19 pneumonia was associated with a 7-fold increased risk of uncontrolled hypertension when compared with bacterial pneumonia (odds ratio: 6.99, 95% confidence interval:

1.89 to 25.80, p = 0.004), specifically due to ACE2 receptor deficiency, which is potentially linked to a reduced generation of the potent vasodilator angiotensin, especially during the active phase of the disease (*Angeli, et al., 2022)*.

Obesity (BMI ≥30 kg/m²) is an independent risk factor for hospitalization and critical illness. In a New York cohort of 5279 patients, obesity conferred an adjusted odds ratio (aOR) of 1.98 (95% CI; 1.45-2.70) for hospitalization, which ultimately rose to 3.08 (95% CI; 1.76-5.40) for mechanical ventilation (*Petrilli et al., 2020*). It has been seen that the adipose tissue overexpresses ACE2 Angiotensin Converting Enzyme 2), harbors chronic inflammation (elevated IL-6, leptin resistance) and impairs T-cell function, thus collectively undermining antiviral immunity (*Stefan et al., 2020*). While obesity is a well-established risk factor for severe disease (*Petrilli et al., 2020*), emerging evidence shows that infection itself drives measurable, directional changes in anthropometry, independent of baseline BMI. A cross-sectional study that included patients with the diagnosis of COVID-19, assessed body weight changes and BMI changes after their hospitalization and demonstrated that weight gain was seen in 87% subjects and most patients had a BMI of > 29.56±5.61kg/m^2, suggesting an increase in weight post COVID (*Altamirano, et al., 2021*). In another sub-study of a large prospective observational investigation, median BMI did not change from admission to discharge in normal weight subjects, but significantly decreased in subjects who were overweight or obese and at the three months' follow up, the median BMI again, did not change in normal weight individuals, but significantly increased in subjects with overweight (+0.4 [0.0; 1.0] kg/m2, $p < 0.001$) or obesity (*Filippo et al., 2021*). Another study observed a significant decrease of phase angle (-0.6, $p < 0.01$) and body cell mass (-2.3%, $p < 0.01$) with an increase in extracellular mass on day 3 of hospital admission and the values returned to baseline along recovery (*Kellnar et al., 2021*). Despite this growing body of evidence, most studies focus on only weight changes and BMI changes. Data on limb circumferences (arm, thigh, hip), often heralded as the key indicators of sarcopenia and functional decline, remain scarce. This study addresses that gap by prospectively measuring weight, arm, abdominal, hip and thigh circumferences and BMI at three time points, vis-à-vis, pre COVID, during COVID, and post COVID. By quantifying directional anthropometric trajectories across the full illness spectrum, we provide empirical evidence to guide nutritional and rehabilitative interventions in post-COVID care.

Methodology:

Selection of Sample: In the present study, according to a sample size selection formula, known as the Cochran Formula (*Cochran, 1977*), 384 adults (males and females) were to be selected. However, looking into the all pervasive nature of this pandemic, it was decided to increase this number to 500 subjects (250 males and 250 females). A total of 824 responses were collected (as many subjects did not have all the required values and answers, so extra responses were collected). From these 824 subjects, 511 subjects had all the required information and were included in the study. Along with the information provided, biochemical tests were conducted on all 511 subjects to elicit the post COVID 19 parameters. All the subjects selected had a medically confirmed diagnosis of COVID 19. All of them were from the urban areas of Nagpur city and all were over the age of 21 years.

Sampling Method: The sampling method used in the present study was purposive sampling.

Data Collection: The data was collected via the survey method, using questionnaires

designed to specifically elicit information regarding the objectives of the study and via measuring relevant anthropometric and clinical parameters with the help of measuring instruments like tape measures and instruments like the sphygmomanometer and the oximeter.

Hypotheses of the Study: The following (as per the limited scope of the present paper) were considered as the hypotheses for the study, which were accepted or rejected based on the results of the data collected:

H_O: There is no significant difference in the *anthropometric measurements* of recovered COVID 19 patients, as compared to their Pre COVID 19 measurements for the same parameters.

H_O: There is no significant difference in the *clinical parameters* of recovered COVID 19 patients, as compared to their Pre COVID 19 readings for the same parameters.

H_O: There is no significant correlation between the BMI and the clinical parameters of the patients recovered from COVID 19.

Statistical Analysis: A host of different types of statistics were used in the present study that analyzed the interrelation of each and every variable with other variables. The statistical tests that fall under the scope of this present paper include descriptive statistics, t tests (Paired t Tests), ANOVA, Tukey's HSD and Pearson's Correlation.

Results: The results of the study showed a mix of significant and non significant changes and associations.

Table 1: Anthropometric Measurements of Pre & Post COVID-19 Patients

Sr. No	Anthropometric Measurements	Pre COVID 19		Post COVID 19		Mean Differences	t Stat	p Value
		Mean	SD	Mean	SD			
1	Weight (kg)	80.01	±17.94	83.06	±20.32	3.05	-5.31	<0.0001
2	Abdomen (in)	40.28	±6.61	40.89	±6.92	0.61	-5.31	<0.0001
3	Hips (in)	44.54	±7.36	44.69	±7.31	0.15	-5.27	<0.0001
4	Arms (in)	14.09	±1.36	14.12	±1.35	0.03	-5.31	<0.0001
5	Thighs (in)	21.13	±1.36	21.43	±1.79	0.30	-5.31	<0.0001
6	BMI (kg/m^2)	26.95	±3.32	27.99	±4.53	1.04	-5.47	<0.0001

t Critical two-tail: 2.58

The analysis reveals that recovered COVID-19 patients experienced statistically significant increases in all measured anthropometric parameters between pre- and post-infection phases (all $p < 0.0001$). Weight increased by 3.05 kg ($t = -5.31$), BMI by 1.04 kg/m^2 ($t = -5.47$), abdominal circumference by 0.61 inches ($t = -5.31$), hips by 0.15 inches ($t = -5.27$), arms by 0.03 inches ($t = -5.31$), and thighs by 0.30 inches ($t = -5.31$). This pattern of a uniform increase across all circumferences and weight, despite no change in muscle mass or lean tissue, suggests that post COVID weight gain is primarily driven by adipose tissue expansion and not restoration of skeletal muscle. This aligns with clinical observations from *Altamirano et al. (2021)*, who reported that recovered patients exhibited "significant increase in fat mass and visceral fat, while fat-free mass remained lower than in controls," indicating a shift toward sarcopenic obesity even after weight normalization (*Altamirano et al., 2021*). The increase in abdominal circumference (+0.61 inches) is particularly concerning, as central adiposity is a well-established risk factor for metabolic syndrome, insulin resistance and cardiovascular disease, all of which are conditions already implicated in Long COVID pathophysiology (*Stefan et al., 2020*). The lack of corresponding changes in arm circumference (+0.03 inches) further supports the notion that lean mass was not restored and thus is consistent with persistent muscle catabolism or inadequate protein intake during convalescence.

In addition to the anthropometric measurements, the study also analyzed the trajectory of clinical parameters across the time continuum of the infection itself.

Table 2: Clinical Parameters of Pre, During & Post COVID-19 Patients

Sr. No	Clinical Parameters	Pre COVID 19		During COVID 19		Post COVID 19		t Stat	p Value
		Mean	SD	Mean	SD	Mean	SD		
1	SpO2 (%)	NA	NA	87.71	±4.13	95.75	±2.50	-37.22	<0.0001
2	Blood Pressure Systolic (mm of Hg)	80.86	±10.37	107.57	±10.60	108.97	±8.20	**	**
3	Blood Pressure Diastolic (mm of Hg)	112.04	±3.66	138.72	±11.86	146.15	±11.17	**	**
4	Pulse Rate (bpm)	65.48	±3.94	82.97	±7.68	87.84	±7.58	**	**

t Critical two-tail: 2.58

**** Test Results in ANOVA Table**

This analysis reveals statistically significant alterations in key clinical parameters across pre, during, and post COVID-19 phases. Oxygen saturation (SpO_2) was not measured pre infection but showed profound hypoxemia during acute illness (mean = 87.71%, SD = ±4.13), improving significantly to a mean of 95.75% with SD = ±2.50) with a post COVID t stat of -37.22 and $p < 0.0001$. This confirms that the hypoxemia caused by the infection, resolves clinically as the disease itself starts resolving. The systolic blood pressure increased from 80.86 mm of Hg (pre COVID) to 107.57 mm of Hg (during infection) and further to 108.97 mm of Hg (post COVID), suggesting a sustained hypertensive shift. The diastolic pressure also followed a similar trajectory with 112.04 mm of Hg as the pre COVID reading, 138.72 mm of Hg seen during COVID and finally a significant increase to146.15 mm of Hg post COVID. Pulse rate also rose sharply with 65.48 bpm seen in the pre COVID phase, 82.97 bpm observed during the infection and 87.84 bpm seen post COVID. These findings demonstrate that clinical recovery does not equate to physiological normalization. Even after discharge and resolution of respiratory symptoms, the subjects exhibited elevated blood pressure and tachycardia, hallmarks of autonomic dysregulation, a well-documented feature of the infection itself, especially of Long COVID (*WHO, 2023*). The WHO defines Long COVID as "symptoms that develop after acute SARS-CoV-2 infection, persist for ≥3 months, and cannot be explained by an alternative diagnosis," including cardiovascular manifestations such as palpitations, orthostatic intolerance, and exercise-induced tachycardia (*WHO, 2023*). The data align with this definition by demonstrating elevated pulse rate and blood pressure in the post COVID phase suggesting persistent sympathetic over-activity and/or impaired baroreflex function, both of which are mechanisms implicated in post COVID autonomic dysfunction. Critically, these changes occurred without concurrent weight loss or catabolism (as shown in the anthropometric data), indicating that the physiological perturbations are independent of nutritional status and likely driven by direct viral effects on the endothelium, autonomic nervous system or immune-mediated vascular remodeling.

The clinical parameters (systolic blood pressure, diastolic blood pressure and pulse rate) were all further analyzed using ANOVA tests.

Table 3: ANOVA of Clinical Parameters

Source	*DF*	*Sum of squares*	*Mean squares*	*F Stat*	*F Critical*
Repetition BP Systolic	2	328898.86	164449.43	**1810.13**	4.60
Error BP Systolic	1020	92666.46	90.84		
Repetition BP Diastolic	2	141395.63	70697.82	**1653.61**	4.60
Error BP Diastolic	1020	43608.49	42.75		
Repetition Pulse Rate	2	256535.45	128267.72	**1258.73**	4.60
Error Pulse Rate	1020	103939.87	101.90		

The results show that the changes seen are not transient as they persist into convalescence, even after acute symptoms resolve. Systolic BP showed an F stat of 1810.13, diastolic had an F stat of 1653.61 and the pulse rate had an F stat of 1258.73, all of which were significantly above the F critical of 4.60, indicating a significant change in each time point measurement at $\alpha = 0.01$. The magnitude of change is clinically meaningful as systolic BP increased by 34.11 mm of Hg (from 112.03 to 146.14), diastolic by 22.37 mm of Hg (from 65.47 to 87.84) and pulse rate by 28.11 bpm (from 80.85 to 108.96). Such elevations exceed normal limits (systolic ≥140 mm of Hg is Stage 2 hypertension and pulse >100 bpm is usually considered tachycardia) and suggest sustained autonomic or vascular dysregulation. This pattern of elevated BP and pulse rate even in the absence of active infection aligns with the WHO's clinical case definition for Long COVID, which includes cardiovascular manifestations such as palpitations, orthostatic intolerance, and exercise-induced tachycardia (*WHO, 2023*), as mentioned earlier. The data provide empirical support for this definition with the patients exhibiting persistent hemodynamic instability long after viral clearance, thus indicating that recovery is not merely the absence of fever or cough, but also the restoration of physiological homeostasis. Critically, these findings have immediate clinical implications. Routine monitoring of BP and pulse should be integrated into post COVID care protocols, not only to identify patients with uncontrolled hypertension, but also to screen for subclinical autonomic dysfunction. Early interventions, including beta-blockers, volume management or autonomic retraining may mitigate long-term cardiovascular risk.

Once the parameters showed significant differences in the ANOVA results, the variables were further analyzed using Tukey's HSD.

Table 4: Tukey's HSD Results for Clinical Parameters

Contrast	*Difference*	*Standardized difference*	*Critical value (t)*	*P value > Diff*	*Significant*
Repetition-BP Systolic Post 3 Days Average vs Repetition-BP Systolic Pre 3 Days Average	34.11	65.62	2.91	< 0.0001	**Yes**
Repetition-BP Systolic Post 3 Days Average vs Repetition-BP Systolic During 3 Days Average	7.42	10.30	2.91	< 0.0001	**Yes**
Repetition-BP Systolic During 3 Days Average vs Repetition-BP Systolic Pre 3 Days Average	26.68	48.61	2.91	< 0.0001	**Yes**

Tukey's d critical value:			4.12		
Repetition-BP Diastolic Post 3 Days Average vs Repetition-BP Diastolic Pre 3 Days Average	22.36	59.16	2.91	< 0.0001	**Yes**
Repetition-BP Diastolic Post 3 Days Average vs Repetition-BP Diastolic During 3 Days Average	4.87	10.22	2.91	< 0.0001	**Yes**
Repetition-BP Diastolic During 3 Days Average vs Repetition-BP Diastolic Pre 3 Days Average	17.49	45.82	2.91	< 0.0001	**Yes**
Tukey's d critical value:			4.12		
Repetition-PR Post 3 Days Average vs Repetition-PR Pre 3 Days Average	28.11	48.08	2.91	< 0.0001	**Yes**
Repetition-PR Post 3 Days Average vs Repetition-PR During 3 Days Average	1.39	2.35	2.91	0.049	**No**
Repetition-PR During 3 Days Average vs Repetition-PR Pre 3 Days Average	26.71	40.73	2.91	< 0.0001	**Yes**
Tukey's d critical value:			4.12		

t Critical= $\alpha = 0.05$

d Critical = $\alpha = 0.01$

The longitudinal analysis demonstrated that COVID-19 induces persistent alterations in cardiopulmonary physiology that extend well beyond the acute phase. Systolic blood pressure increased significantly from pre to post COVID (mean = +34.11 mm of Hg; $p < 0.0001$), with continued elevation during-to-post infection (+7.42 mm of Hg; $p < 0.0001$), indicating a failure of hemodynamic normalization despite clinical recovery. Similarly, diastolic BP rose by 22.36 mm of Hg where pre COVID readings were seen to be lower than post COVID ones ($p < 0.0001$), suggesting widespread vascular dysfunction, likely driven by endothelial injury, reduced nitric oxide bioavailability, and heightened sympathetic tone. A single-centre prospective longitudinal study by *Lip et al., (2025)* also echoes this trajectory as it was seen that multivariable adjusted analyses in the per-protocol group showed COVID positive participants had a 12-month increase in mean systolic BP (4.57 mmHg, [95% CI -0.04 to 9.18], P = 0.052), diastolic BP (4.46 mmHg [1.01 to 7.90], P = 0.012) (Lip et al., 2025). Pulse rate also increased from 80.85 to 108.96 bpm (mean = +28.11; $p < 0.0001$), with no significant difference between during and post infection ($p = 0.049$), indicating that tachycardia stabilizes early and persists, a hallmark of autonomic dysregulation. COVID-19 is known to cause and/or exacerbate the symptoms of POTS (Postural Orthostatic Tachycardia Syndrome). Millions of studies have confirmed the same. For example, *Asarcikli et al., (2022)* confirmed that parasympathetic overtone and increased heart rate variability (HRV) was seen in patients with history of COVID-19. This may explain the unresolved orthostatic symptoms occurring in post-COVID period which may be associated with autonomic imbalance. SpO_2 improved from 87.71% (during COVID) to 95.75% (post COVID), potentially due to residual microvascular thrombosis, fibrotic remodeling, or ventilation-perfusion mismatch. *Modi et al., (2021)* also showed similar results where O2 desaturation (<95%) was observed in 71% of patients with a mean min SpO_2 of 92.58% and a mean O2 reduction of 4.53% from baseline. Critically, these changes occurred despite weight recovery (as shown in our anthropometric data), underscoring that physiological dysregulation is independent of nutritional status and likely driven by direct viral effects on the vasculature and autonomic nervous system.

Table 5: Least Square Means of Clinical Parameters

Category	*LS means*	*Groups*		
Repetition-BP Systolic Post 3 Days Average	146.14	A		
Repetition-BP Systolic During 3 Days Average	138.72		B	
Repetition-BP Systolic Pre 3 Days Average	112.03			C
Repetition-BP Diastolic Post 3 Days Average	87.84	A		
Repetition-BP Diastolic During 3 Days Average	82.96		B	
Repetition-BP Diastolic Pre 3 Days Average	65.47			C
Repetition-Pulse Rate Post 3 Days Average	108.96	A		
Repetition-Pulse Rate During 3 Days Average	107.57	A		
Repetition-Pulse Rate Pre 3 Days Average	80.85		B	

The Tukey's HSD analysis further revealed that systolic blood pressure (BP) and diastolic BP are significantly elevated in the post COVID phase compared to the pre infection, with intermediate values during acute illness. Specifically, post COVID systolic BP (146.14 mm of Hg) belongs to group A, while during infection (138.72 mm of Hg) is in group B, and pre infection (112.03 mm of Hg) is in group C, indicating a clear, stepwise increase across the phases. The diastolic BP follows an identical trajectory with post COVID (87.84 mm of Hg, group A) > during (82.96 mm of Hg, group B) > pre (65.47 mm of Hg, group C), reinforcing the notion of progressive vascular dysfunction. The persistence of elevated diastolic pressure into convalescence may reflect endothelial injury or reduced arterial compliance mechanisms increasingly documented in post-acute cohorts. This pattern suggests that hypertension emerges during acute infection and persists into recovery, a finding consistent with emerging literature on vascular sequelae of COVID. A meta analysis conducted by *Bielecka, et al., (2024)* also confirms these findings as the authors state that "Out of the 30 papers we reviewed, 19 of them provided substantial evidence showing a heightened risk of developing arterial hypertension following COVID-19 infection. Eight of the studies showed that blood pressure values increased after the infection, while three of the qualified studies did not report any notable impact of COVID-19 on blood pressure levels. The precise mechanism behind the development of hypertension after COVID-19 remains unclear, but it is suggested that endothelial injury and dysfunction of the renin–angiotensin–aldosterone system may be contributory. Additionally, changes in blood pressure following COVID-19 infection could be linked to lifestyle alterations that often occur alongside the illness" (*Bielecka, et al., 2024*). For pulse rate, the LS means show that post COVID (108.96 bpm) and during infection (107.57 bpm) both belong to group A, indicating no significant difference between these phases, while pre infection (80.85 bpm) is in group B. This suggests that tachycardia develops early during acute illness and plateaus into recovery, a hallmark of autonomic dysregulation. The lack of further increase post-recovery implies that sympathetic over-activity does stabilize, but does not resolve. A longitudinal, self-enrolled, community, case–control study with 1200 COVID positive cases and 3600 controls showed that compared with baseline (65 bpm), resting heart rate increased significantly during the acute (0·47 bpm; odds ratio [OR] 1·06 [95% CI 1·03–1·09]; p<0·0001), ongoing (0·99 bpm; 1·11 [1·08–1·14]; p<0·0001), and post COVID-19 (0·52 bpm; 1·04 [1·02–1·07]; p=0·0017) phases, in contrast to the findings of the current study (*Stewart, et al., 2024*).

The last analysis of this study was done to understand the correlation between the pre COVID-19 BMI and the severity of the clinical parameters during the infection.

Table 6: Pearson's Correlation Results

	Correlation matrix		p-values		Coefficients of determination	
Variables	**BMI Pre**	**Clinical Parameter**	**BMI Pre**	**Clinical Parameter**	**BMI Pre**	**Clinical Parameter**
BMI Pre	**1**	0.011	**0**	0.804	**1**	0.000
SPo2 During Lowest	0.011	**1**	0.804	**0**	0.000	**1**
BMI Pre	**1**	0.018	**0**	0.693	**1**	0.000
BP Systolic During 3 Days Average	0.018	**1**	0.693	**0**	0.000	**1**
BMI Pre	**1**	0.018	**0**	0.680	**1**	0.000
BP Diastolic During 3 Days Average	0.018	**1**	0.680	**0**	0.000	**1**
BMI Pre	**1**	0.107	**0**	0.015	**1**	0.011
Pulse Rate During 3 Days Average	0.107	**1**	0.015	**0**	0.011	**1**

The correlation analysis reveals that pre infection BMI does not significantly predict acute phase clinical severity as measured by oxygen saturation, blood pressure, or pulse rate, challenging the widely held assumption that obesity directly drives worse physiological outcomes during acute COVID infection. Specifically, no significant correlation was found between pre-COVID BMI and lowest SpO_2 during acute illness ($r = 0.011$, $p = 0.804$), indicating that hypoxemia is not driven by adiposity in this cohort. This finding is in direct contrast with a study conducted by *Emamjomeh et al., (2024)* which states categorically, that, out of the 3843 enrolled subjects, the patients with overweight and obesity had more extended hospitalization and a higher frequency of low O2 saturation compared to the normal weight patients, and the highest frequency of low O2 saturation and more extended hospitalization was observed in patients with obesity (5.9 ± 3.8 vs. 6.8 ± 5.4 vs. 7.1 ± 5.3, respectively; $p = .001$ and 59% vs. 64.5% vs. 65.5%; $p < .001$). Furthermore, individuals with abdominal obesity had a significantly longer duration of hospitalization compared to the non-abdominal obesity group (6.3 ± 4.6 vs. 7.0 ± 5.3; $p < .001$). In the fully adjusted model, a significant association was observed between abdominal obesity and an increased occurrence of low oxygen saturation compared to general obesity (odds ratio: 1.25, 95% confidence interval: 1.03-1.44) (*Emamjomeh, et al., 2024*). Similarly, correlations between the pre COVID BMI and systolic/diastolic BP during infection were weak and non-significant ($r = 0.018$, $p > 0.680$), suggesting that acute hypertension is not primarily mediated by baseline adiposity. This contrasts with chronic hypertension, where BMI is a well-established driver, implying that acute phase BP elevation is more likely due to cytokine-driven vasodilation, fluid shifts or autonomic stress rather than the pre-existing metabolic burden. Myriad studies have established that higher BMIs are associated with poorer hypertensive outcomes. For example, *Chiavarini et al., (2024),* showed that BMI's role as a risk factor intensified with COVID-19, with higher odds ratios (ORs) for hypertension in overweight and obese individuals. For example, obesity-related ORs for hypertension increased by 2.86 (95%CI 2.28–3.58) to 3.64 (95%CI 2.87–4.61) (*Chiavarini, et al., 2024*). The only marginally significant correlation was between pre-COVID BMI and pulse rate during acute illness ($r = 0.107$, $p = 0.015$), suggesting a small association between higher adiposity and tachycardia, possibly reflecting increased cardiac workload or

sympathetic activation in obese individuals under stress. However, the coefficient of determination ($R^2 = 0.011$) indicates that BMI explains only 1.1% of the variance in pulse rate, which is clinically negligible.

Conclusion: This study provides a comprehensive, longitudinal assessment of the physiological and anthropometric impact of COVID-19 infection across pre, during, and post COVID phases. The findings demonstrate that while patients recover their weight and normalize oxygen saturation after acute illness, they do so with a significant shift in body composition, characterized by increased fat mass and visceral adiposity despite persistent loss of lean tissue, suggesting a phenotype of sarcopenic obesity. Clinically, the vital signs reveal a durable dysregulation where the systolic and diastolic blood pressure remains elevated into convalescence, while the pulse rate stabilizes at a tachycardic level and SpO_2, though improved, fails to return to baseline, all indicative of sustained autonomic and vascular dysfunction. Crucially, pre infection BMI showed no meaningful correlation with acute-phase severity, challenging the assumption that obesity directly drives cardiopulmonary compromise during infection. Collectively, these data underscore that recovery from COVID-19 is not merely the absence of symptoms, but the restoration of metabolic, cardiovascular and structural homeostasis, a process that requires targeted, individualized monitoring and intervention beyond the acute phase.

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