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Original Article

Physiology Section

Heart Rate Variability in Young Adults with Bronchial Asthma with and without Previous COVID-19 Infection: A Cross-sectional Study

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ABSTRACT

Introduction: Heart Rate Variability (HRV) is a validated, non invasive measure of Autonomic Nervous System (ANS) function. Both bronchial asthma and post Coronavirus Disease-2019 (COVID-19) syndrome are independently associated with autonomic dysregulation. However, the combined impact of asthma and prior COVID-19 infection on HRV in young adults remains underexplored.

Aim: To compare HRV parameters between young adult bronchial asthma patients with and without a history of COVID-19, and assess the extent of autonomic dysfunction attributable to COVID-19.

Materials and Methods: This cross-sectional study included 100 bronchial asthma patients aged 20-40 years, equally divided into two groups based on COVID-19 history. Standardised HRV recordings were obtained in the supine position following a 15-minute rest period. HRV was assessed using time domain indices- Standard Deviation of NN intervals (SDNN) and Root Mean Square of Successive Differences (RMSSD)- and frequency domain indices- Low Frequency (LF), High Frequency (HF), and LF to HF ratio (LF/HF ratio)- using Kubios HRV software. Haemodynamic parameters including Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), and Pulse Rate (PR) were also compared. Statistical analysis was performed using One-way Analysis of Variance (ANOVA) and independent t-tests, with a p-value of less than 0.05 considered statistically significant.

Results: Asthma patients with prior COVID-19 infection demonstrated significantly higher DBP (84.48±7.38 vs. 79.12±6.22 mmHg; $p<0.001$) and PR (88.38±10.48 vs. 79.88±9.74 bpm; $p<0.001$). HRV analysis revealed significantly lower SDNN (30.18±5.18 vs. 33.54±6.27 ms; $p=0.002$), RMSSD (23.80±4.72 vs. 28.00±5.54 ms; $p<0.001$), and HF power (92.54±11.67 vs. 115.48±17.53 ms^2; $p<0.001$) in the post COVID group, along with elevated LF power (232.76±24.62 vs. 193.32±20.28 ms^2; $p<0.001$) and LF/HF ratio (2.52±0.32 vs. 1.67±0.23; $p<0.001$), suggesting heightened sympathetic activity and reduced vagal modulation.

Conclusion: Asthma patients with prior COVID-19 infection exhibit significant autonomic dysfunction marked by reduced parasympathetic modulation and heightened sympathetic dominance. These findings highlight the importance of cardiovascular autonomic assessment in asthma patients recovering from COVID-19, particularly for early identification of dysautonomia and cardiovascular risk stratification.

Keywords: Autonomic nervous system diseases, Cardiovascular physiological phenomena, Coronavirus disease 2019, Sympathetic nervous system

INTRODUCTION

The ANS plays a crucial role in maintaining cardiovascular homeostasis by regulating HRV, which reflects the dynamic balance between sympathetic and parasympathetic nervous system activity. HRV is recognised as a reliable, non invasive biomarker of cardiac autonomic regulation and overall cardiovascular health [1,2].

Bronchial asthma is a chronic inflammatory airway disorder characterised by episodic bronchoconstriction, airway hyperresponsiveness, and mucus hypersecretion. Its pathophysiology involves immune-mediated inflammation, but accumulating evidence also implicates autonomic dysregulation-particularly increased parasympathetic activity- as a contributor to bronchial hyperreactivity and disease severity [3,4]. Asthma-related autonomic dysfunction has also been associated with increased cardiovascular morbidity and elevated risk of arrhythmias, even in young and otherwise healthy individuals [5]. Studies employing HRV in asthma patients have reported increased HF power and decreased LF/HF ratios, indicating heightened vagal tone and impaired sympathovagal balance [6,7].

Since the onset of the COVID-19 pandemic, caused by the novel SARS-CoV-2 virus, autonomic involvement has emerged as a common post viral sequela. SARS-CoV-2 can invade the central and peripheral nervous systems via ACE2 receptors, triggering neuroinflammation, dysautonomia, and altered autonomic responses [8,9]. Post Acute Sequelae of COVID-19 (PASC), or "long COVID," frequently manifest as autonomic symptoms including tachycardia, palpitations, orthostatic intolerance, and reductions in HRV metrics indicative of sympathetic overactivity and parasympathetic withdrawal [10,11].

While both asthma and COVID-19 have independently demonstrated an association with autonomic dysfunction, very few studies have evaluated their combined impact, particularly in young adults [12,13]. Most existing research has focused on either asthma or COVID-19 in isolation [13,14]. There is a distinct lack of literature examining how these two conditions may synergistically affect autonomic tone. This represents a critical knowledge gap; especially as autonomic imbalance is a known precursor to cardiovascular morbidity in both conditions. This is clinically relevant, as the synergistic effect may exacerbate autonomic dysregulation and predispose patients

to cardiovascular morbidity. HRV analysis in such populations may provide an early indicator of altered autonomic tone and cardiovascular risk [15,16].

Given the potential long-term effects of COVID-19 on autonomic function and the pre-existing dysautonomia observed in bronchial asthma, this study addresses an important knowledge gap by systematically assessing HRV in young adult asthma patients with and without prior COVID-19 infection.

The primary objective of this study was to compare HRV parameters between two groups of bronchial asthma patients- those with and without a prior history of COVID-19 infection, in order to identify differences in autonomic function. The study specifically aimed to assess whether a previous COVID-19 infection contributes to greater autonomic imbalance, as reflected in both time domain and frequency domain HRV indices. By evaluating these parameters, the study seeks to explore the impact of COVID-19 on autonomic regulation in asthma and its potential implications for cardiovascular risk stratification and long-term monitoring in this population.

MATERIALS AND METHODS

The present cross-sectional study was conducted at SRM Medical College Hospital and Research Centre, Kattankulathur, Tamil Nadu, India from September 2024 to April 2025. Institutional Ethics Committee (IEC) approval was obtained prior to the commencement of the study (Reg No: EC/NEW/INST/2022/2933; Ethics Clearance Number: SRMIEC-ST0724-1458). Informed consent was obtained from the participants before the start of the study.

Inclusion and Exclusion criteria: Bronchial asthma patients aged 20 to 40 years were recruited from both outpatient and inpatient departments. Participants were divided into two groups based on COVID-19 history:

- **Group 1 (Post COVID Asthma Group):** Patients with a confirmed history of COVID-19 infection, based on a positive RT-PCR test and/or documented symptomatic illness consistent with COVID-19 (such as fever, cough, anosmia, or dyspnoea) occurring at least three months prior to recruitment.
- **Group 2 (Non COVID Asthma Group):** Patients with no known or documented history of COVID-19 infection based on clinical records and self-report, and no symptoms suggestive of prior COVID-19. However, the possibility of undiagnosed asymptomatic or subclinical infections in this group could not be entirely excluded.

Exclusion criteria included:

- Acute asthma exacerbation within the past two weeks;
- Current COVID-19 positivity (confirmed via RT-PCR or clinical suspicion);
- Presence of comorbid conditions such as diabetes mellitus or cardiovascular disease;
- Use of medications that may influence autonomic function within the past two weeks (e.g., beta-blockers, corticosteroids);
- Age below 20 years or above 40 years.

All participants provided written informed consent prior to enrolment.

Sample size calculation: The sample size was calculated based on previously published research and meta-analytic evidence indicating that moderate effect sizes are commonly observed in HRV studies. According to Quintana DS [17], the median effect size in HRV case-control studies is approximately Cohen's d=0.51, with the 75th percentile approaching d=0.88. Based on this, a moderate effect size (Cohen's d=0.5) was used for estimation. Using a two-tailed test, with a significance level (α) of 0.05 and power (1-β) of 0.80, the required sample size was calculated to be 45 participants per group to detect a statistically significant between-group difference in HRV parameters. To account for a potential 10% dropout or attrition, the final sample size was increased to 50 participants per group, resulting in a total of 100 participants.

Study Procedure

Participants were evaluated under standardised conditions after 15 minutes of supine rest in a quiet, temperature-controlled room. A five-minute Lead II ECG recording was obtained using the Multichannel Physiopac PP-8 system. All participants abstained from caffeine, alcohol, tobacco, and strenuous activity for at least eight hours before testing.

HRV parameters were assessed using Kubios HRV software (version 2.0) and included:

- **Time domain indices:**
  - i) Standard deviation of NN intervals (SDNN);
  - ii) Root Mean Square of Successive Differences (RMSSD);
  - iii) Percentage of successive RR intervals differing by more than 50 ms (pNN50).
- **Frequency domain indices:**
  - i) Low Frequency (LF);
  - ii) High Frequency (HF);
  - iii) LF/HF ratio.

Haemodynamic parameters including SBP, DBP, and PR were also recorded. To minimise bias, all ECG recordings were performed by a single trained operator blinded to group status. Strict procedural protocols and exclusion criteria were maintained to control for potential confounders.

STATISTICAL ANALYSIS

Statistical analysis was conducted using Statistical Package for Social Sciences (SPSS) version 20. Continuous variables were reported as mean±standard deviation. Normality was assessed using the Shapiro-Wilk test. Between-group comparisons were made using the independent t-test for normally distributed variables and Mann-Whitney U test for non-parametric data. Categorical data were analysed using the Chi-square test. A p-value <0.05 was considered statistically significant. Only complete cases were analysed; no imputation or subgroup analysis was performed.

RESULTS

A total of 100 participants were recruited into the study, comprising two equal groups: bronchial asthma patients with a documented past history of COVID-19 (n=50) and bronchial asthma patients without such history (n=50). All individuals assessed for eligibility met the inclusion criteria and provided informed consent. There were no exclusions following enrollment, and all participants completed the study procedures. Therefore, there were no instances of loss to follow-up or missing data, and the full dataset was analysed.

The demographic and clinical characteristics of the participants were comparable between the groups [Table/Fig-1]. The mean age was 31.04±3.88 years in the post COVID asthma group and 29.32±4.17 years in the non COVID asthma group (p=0.390). The proportion of female participants was slightly higher in the non COVID group (60%) compared to the post COVID group (56%), but this difference was not statistically significant (p=0.689). The average Body Mass Index

Parameters	Asthma with COVID-19	Asthma without COVID-19	p-value
Sample size (n)	50	50	-
Age (years)	31.04±3.88	29.32±4.17	0.390
Female (%)	28 (56%)	30 (60%)	0.689
BMI (kg/m^2)	26.00±4.27	25.38±3.82	0.246

[Table/Fig-1]: Demographic and clinical characteristics.
Data are presented as mean±standard deviation for continuous variables and number (percentage) for categorical variables. Independent t-test was used for continuous variables; Chi-square test was used for categorical variables.

(BMI) was marginally higher in the post COVID group (26.00±4.27 kg/m²) than in the non COVID group (25.38±3.82 kg/m²), although this difference was also not statistically significant (p=0.246).

Haemodynamic parameters showed notable differences between the two groups [Table/Fig-2]. DBP and PR were significantly higher in the post COVID asthma group compared to the non COVID asthma group (p<0.001 for both). Although SBP was also elevated in the post COVID group (127.08±9.46 mmHg) relative to the non COVID group (122.72±7.64 mmHg), the difference did not meet the predefined statistical significance threshold of p<0.001 (p=0.002).

Variables	Asthma with COVID-19	Asthma without COVID-19	p-value
SBP (mmHg)	127.08±9.46	122.72±7.64	0.002
DBP (mmHg)	84.48±7.38	79.12±6.82	<0.001
Pulse Rate (PR) (bpm)	88.38±10.48	79.88±9.74	<0.001

[Table/Fig-2]: Comparison of mean haemodynamic parameters between groups. Data are presented as mean±standard deviation. Independent t-test was used to compare continuous variables between groups.

Analysis of HRV parameters revealed significant autonomic differences between the two groups. In time domain analysis [Table/Fig-3], SDNN was markedly lower in the post COVID asthma group (30.18±5.18 ms) compared to the non COVID group (33.54±6.27 ms; p=0.002). RMSSD, a marker of short-term parasympathetic activity, was also reduced in the post COVID group (23.80±4.72 ms) relative to the non COVID group (28.00±5.54 ms; p<0.001). Additionally, pNN50, representing the percentage of successive RR intervals differing by more than 50 ms, was significantly lower in the post COVID group (3.82±2.77%) than in the non COVID group (7.46±3.39%; p<0.001), suggesting attenuated vagal modulation and parasympathetic tone.

Variables	Asthma with COVID-19 (n = 50)	Asthma without COVID-19 (n = 50)	p-value
SDNN (ms)	30.18±5.18	33.54±6.27	0.002
RMSSD (ms)	23.80±4.72	28.00±5.54	<0.001
pNN50 (%)	3.82±2.77	7.46±3.39	<0.001

[Table/Fig-3]: Comparison of time domain HRV parameters. Data are presented as mean±standard deviation. Independent t-test was used to compare continuous variables between groups

Frequency domain HRV indices [Table/Fig-4] also demonstrated a clear shift toward sympathetic predominance in the post COVID asthma group. HF power, indicative of vagal tone, was significantly lower in the post COVID group (92.54±11.67 ms²) compared to the non COVID group (115.48±17.53 ms²; p<0.001). In contrast, LF power was higher in the post COVID group (232.76±24.62 ms²) relative to the non COVID group (193.32±20.28 ms²; p<0.001). The LF/HF ratio, representing sympathovagal balance, was significantly elevated in the post COVID group (2.52±0.32) compared to the non COVID group (1.67±0.23; p<0.001), suggesting enhanced sympathetic dominance.

Parameters	Asthma with COVID-19	Asthma without COVID-19	p- value
HF Power (ms²)	92.54±11.67	115.48±17.53	<0.001
LF Power (ms²)	232.76±24.62	193.32±20.28	<0.001
LF/HF Ratio	2.52±0.32	1.67±0.23	<0.001

[Table/Fig-4]: Comparison of frequency domain HRV parameters. Data are presented as mean ± standard deviation. Independent t-test was used to compare continuous variables between groups.

These findings supported the study hypothesis, indicating that asthma patients with a prior history of COVID-19 exhibit greater autonomic dysfunction, characterised by elevated sympathetic activity and reduced parasympathetic tone. This highlights the importance of post COVID autonomic monitoring in chronic respiratory disease populations.

DISCUSSION

The present study assessed autonomic function in young adult bronchial asthma patients with and without a prior history of COVID-19 infection using HRV as a marker. The results demonstrated that asthma patients who had recovered from COVID-19 exhibited significantly reduced time domain indices (SDNN and RMSSD), along with lower HF power and elevated LF/HF ratios in frequency domain analysis. These findings reflected a shift toward sympathetic dominance and parasympathetic withdrawal, supporting the hypothesis that COVID-19 may aggravate underlying autonomic dysregulation in asthma.

The primary objective comparing autonomic profiles between the two groups was thus clearly met, with consistent and statistically significant differences across several key HRV parameters. However, it is worth noting that while the difference in SBP was statistically significant (p=0.002), the effect size was relatively small and should be interpreted with caution in terms of clinical relevance. These results are in agreement with previous findings highlighting autonomic alterations in both conditions [18]. In asthma, increased parasympathetic tone has been shown to contribute to airway hyperreactivity via cholinergic pathways, leading to exaggerated vagal responses [3,4].

Meanwhile, COVID-19 has been independently associated with autonomic dysfunction, often manifesting as sympathetic overactivity and HRV suppression [18]. The findings of this study suggested that in asthma patients with prior COVID-19, this typical parasympathetic dominance is attenuated or reversed, suggesting compounded dysautonomia. This trend aligns with observations by Mooren FC et al., who documented elevated LF/HF ratios in post COVID patients, indicating a persistent autonomic imbalance [19].

The pathophysiological basis for post COVID dysautonomia likely includes a combination of direct viral neuroinvasion through ACE2 receptors, cytokine-mediated neuroinflammation, microvascular and endothelial dysfunction, and impaired vagal signaling [20,21]. These mechanisms can disrupt the ANS's ability to maintain homeostasis, especially in patients with pre-existing conditions such as asthma. Autonomic symptoms such as orthostatic intolerance, palpitations, fatigue, and reduced HRV have been frequently reported among long COVID patients [10,11]. The observed increase in LF power and LF/HF ratio in the post COVID asthma group is consistent with findings by Borchini R et al., who demonstrated sustained sympathetic overactivity several months following SARS-CoV-2 infection [22]. In the context of asthma, such a sympathetic shift may not only elevate cardiovascular strain but also worsen asthma control through enhanced airway reactivity [4].

This interaction between COVID-19 and asthma may synergistically heighten the risk of cardiovascular morbidity including arrhythmias even in young adults without overt co-morbidities. A recent meta-analysis reported a 30% increased risk of cardiovascular disease and elevated cardiovascular mortality in individuals with asthma [23]. Additionally, persistent asthma has been associated with a 1.5 fold higher incidence of atrial fibrillation, even after adjusting for confounding variables [5].

Limitation(s)

While the study yields important insights, several limitations must be acknowledged. First, its cross-sectional design precludes causal inference. Although significant autonomic differences were detected between groups, it cannot be definitively concluded that COVID-19 caused these changes, nor whether these changes are reversible. Secondly, HRV was recorded at a single resting time point, which may not capture circadian variation or dynamic responses to stressors such as physical activity, orthostatic challenge, or mental workload [15]. Third, although participants were screened for confounding conditions, residual confounders such as anxiety, sleep disturbances, medication compliance, and subclinical inflammation

may still influence HRV [16]. Additionally, this study was restricted to a narrow age band (20-40 years) and excluded participants with known comorbidities, which may limit the generalisability of findings to older individuals or those with more severe disease phenotypes. Nevertheless, internal validity was strengthened by standardised recruitment and uniform measurement protocols. These results may be extrapolated to young, ambulatory asthma patients recovering from COVID-19 in similar urban and semi-urban Indian healthcare settings. The sample size, although sufficient for primary comparisons, limits subgroup analysis by sex or asthma severity. Lastly, the study was conducted in a single tertiary care center, potentially introducing selection bias and limiting external validity.

CONCLUSION(S)

This study demonstrates that bronchial asthma patients with a prior history of COVID-19 infection exhibit significantly greater autonomic dysfunction compared to their non COVID counterparts, as evidenced by reduced HRV indices and a marked shift toward sympathetic dominance. The alterations in both time domain and frequency domain parameters suggest compounded impairments in autonomic regulation due to the combined effects of chronic airway inflammation and post viral dysautonomia. These findings underscore the importance of routine cardiovascular autonomic monitoring in post COVID asthma patients, particularly in younger adults who may otherwise appear clinically stable. To better understand the clinical implications of these findings, future multi-center, longitudinal studies are recommended to evaluate the persistence of HRV alterations and their association with cardiovascular outcomes and therapeutic responses.

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