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ASSOCIATION OF GLYCEMIC VARIABILITY WITH DISEASE SEVERITY IN PATIENTS WITH PULMONARY HYPERTENSION: A HOSPITAL-BASED CROSS-SECTIONAL STUDY

Internal Medicine

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ABSTRACT

Background Pulmonary hypertension (PH) is a progressive cardiopulmonary disorder characterized by elevated pulmonary arterial pressure, progressive pulmonary vascular remodeling, right ventricular dysfunction, and premature mortality. Increasing evidence suggests that metabolic disorders, particularly diabetes mellitus and impaired glucose homeostasis, contribute to pulmonary vascular dysfunction through endothelial injury, oxidative stress, chronic inflammation, mitochondrial dysfunction, and insulin resistance. Although glycated hemoglobin (HbA1c) has been widely investigated as an indicator of long-term glycemic control, the relationship between glycemic variability and pulmonary hypertension severity remains inadequately explored. **Aim** To evaluate the association between glycemic variability, HbA1c, and the severity of pulmonary hypertension in patients attending a tertiary care teaching hospital. **Materials and Methods** A hospital-based cross-sectional observational study was conducted in patients diagnosed with pulmonary hypertension at Akash Institute of Medical Sciences and Research Centre, Bengaluru. After obtaining institutional ethical approval and informed consent, eligible patients underwent detailed clinical evaluation, anthropometric assessment, biochemical investigations, HbA1c estimation, and echocardiographic assessment of pulmonary artery systolic pressure (PASP). Glycemic variability parameters were calculated and correlated with PASP and pulmonary hypertension severity using appropriate statistical methods. **Results** The study demonstrated that a considerable proportion of patients with pulmonary hypertension exhibited impaired glycemic control. Increasing HbA1c levels were associated with progressively higher pulmonary artery systolic pressures. Similarly, patients demonstrating greater glycemic variability had more severe pulmonary hypertension. Significant correlations were observed between glycemic parameters and pulmonary hypertension severity, indicating that chronic hyperglycemia and glucose fluctuations may contribute to pulmonary vascular disease progression. **Conclusion** Glycemic abnormalities, particularly increased HbA1c and glycemic variability, are associated with increased pulmonary hypertension severity. Assessment of glycemic parameters may provide additional prognostic information in patients with pulmonary hypertension. Early identification and correction of dysglycemia may improve risk stratification and contribute to better long-term clinical outcomes.

KEYWORDS

Pulmonary Hypertension; Glycemic Variability; Hba1c; Pulmonary Artery Systolic Pressure; Diabetes Mellitus; Echocardiography.

INTRODUCTION

Pulmonary hypertension (PH) is a progressive cardiopulmonary disorder characterized by elevated pulmonary arterial pressure and increased pulmonary vascular resistance, leading to right ventricular dysfunction, heart failure, and premature mortality. According to the 2022 European Society of Cardiology/European Respiratory Society (ESC/ERS) guidelines, PH is defined as a mean pulmonary arterial pressure (mPAP) >20 mmHg measured by right heart catheterization. Based on etiology and pathophysiology, PH is classified into five major clinical groups: pulmonary arterial hypertension, PH due to left heart disease, PH associated with lung disease or hypoxia, chronic thromboembolic PH, and PH with multifactorial or unclear mechanisms. Despite advances in diagnosis and targeted therapies, PH remains associated with significant morbidity and mortality due to delayed diagnosis and progressive pulmonary vascular remodeling.

Increasing evidence suggests that metabolic disorders, particularly diabetes mellitus, play an important role in the pathogenesis and progression of pulmonary hypertension. Chronic hyperglycemia promotes endothelial dysfunction, oxidative stress, inflammation, insulin resistance, and vascular remodeling, all of which contribute to pulmonary vascular injury and elevated pulmonary vascular resistance. These mechanisms may accelerate disease progression and worsen clinical outcomes in patients with PH.

Glycated hemoglobin (HbA1c) is the standard marker of long-term glycemic control and reflects average blood glucose levels over the preceding 2–3 months. However, HbA1c does not capture short-term fluctuations in glucose concentrations. Glycemic variability (GV), which represents oscillations in blood glucose levels, has emerged as an independent marker of oxidative stress, endothelial dysfunction, and cardiovascular risk. Experimental and clinical studies have demonstrated that glucose fluctuations may induce greater vascular injury than sustained hyperglycemia alone, suggesting that GV may provide additional prognostic information beyond HbA1c.

Recent studies have reported a high prevalence of impaired glucose metabolism in patients with pulmonary hypertension, even in the absence of overt diabetes mellitus. Elevated HbA1c and increased glycemic variability have been associated with reduced exercise capacity, worsening right ventricular function, increased pulmonary artery pressures, and poorer survival. However, data evaluating the relationship between glycemic variability and pulmonary hypertension severity remain limited, particularly in the Indian population. Therefore, the present study was undertaken to evaluate the association between HbA1c, glycemic variability, pulmonary artery systolic pressure (PASP), and the severity of pulmonary hypertension in patients attending a tertiary care teaching hospital. The findings may help identify simple, readily available metabolic markers that could improve risk stratification and clinical assessment of patients with pulmonary hypertension

Materials and Methods

Study Design

A hospital-based observational cross-sectional study was conducted to evaluate the association between glycemic variability and the severity of pulmonary hypertension among patients attending a tertiary care teaching hospital.

Study Setting

The study was conducted in the Department of General Medicine, Akash Institute of Medical Sciences and Research Centre, Devanahalli, Bengaluru, Karnataka, India, after obtaining Institutional Ethics Committee approval.

Study Period

The study was conducted during the approved postgraduate dissertation period.

Study Population

Patients diagnosed with pulmonary hypertension attending the outpatient department or admitted to the Department of General Medicine were screened for eligibility. Eligible patients fulfilling the inclusion criteria were consecutively recruited after obtaining written informed consent.

Sample Size

A total of 72 eligible patients diagnosed with pulmonary hypertension

who satisfied the predefined inclusion and exclusion criteria were included during the study period.

Inclusion Criteria
Patients satisfying all the following criteria were included:
- Age ≥18 years
- Pulmonary hypertension diagnosed by echocardiography
- Willingness to participate
- Written informed consent

Exclusion Criteria
Patients were excluded if they had:
- Acute critical illness requiring intensive care
- Conditions affecting glycemic assessment
- Incomplete laboratory investigations
- Refusal to provide informed consent

Study Procedure
After enrolment, all participants underwent standardized clinical evaluation including detailed history, demographic profile, duration of symptoms, comorbidities, medication history, smoking and alcohol history, and family history. Physical examination included measurement of height, weight, body mass index (BMI), pulse rate, blood pressure, respiratory rate, and oxygen saturation.

Laboratory Investigations
Routine investigations included complete blood count, renal and liver function tests, fasting blood glucose, HbA1c estimation, lipid profile, and other clinically indicated investigations. HbA1c was measured using standardized laboratory methods and interpreted according to accepted diagnostic criteria.

Assessment of Glycemic Variability
Glycemic variability was assessed using predefined indices described in the study protocol. HbA1c, fasting blood glucose, and glycemic variability parameters were evaluated for their association with pulmonary artery systolic pressure (PASP) and pulmonary hypertension severity.

Echocardiographic Assessment
Two-dimensional transthoracic Doppler echocardiography was performed by experienced cardiologists. PASP was estimated using peak tricuspid regurgitation velocity and the modified Bernoulli equation with estimation of right atrial pressure. Pulmonary hypertension was categorized as mild, moderate, or severe according to accepted guideline recommendations.

Variables Analysed
The variables analyzed included demographic characteristics (age and gender), clinical parameters (BMI, duration of diabetes mellitus, blood pressure, and associated comorbidities), laboratory variables (fasting blood glucose, HbA1c, and glycemic variability indices), and echocardiographic parameters (PASP and severity of pulmonary hypertension).

Outcome Measures
Primary Outcome
To determine the association between glycemic variability and pulmonary hypertension severity.

Secondary Outcomes
- Correlation between HbA1c and PASP
- Correlation between glycemic variability and PASP
- Association between glycemic control and pulmonary hypertension severity
- Relationship between demographic variables and disease severity

Statistical Analysis
Data were entered into Microsoft Excel and analyzed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. Comparisons were performed using the Chi-square test, Fisher's exact test, Student's *t*-test, or one-way analysis of variance (ANOVA), as appropriate. Pearson's correlation coefficient was used to assess the relationship between HbA1c, glycemic variability, and PASP. A p-value <0.05 was considered statistically significant.

Ethical Considerations
Institutional Ethics Committee approval was obtained before commencement of the study. Written informed consent was obtained from all participants, and the study was conducted in accordance with the Declaration of Helsinki while maintaining patient confidentiality.

Strengths of the Study
- Hospital-based prospective data collection
- Comprehensive clinical evaluation
- Standardized laboratory assessment
- Echocardiographic evaluation in all participants
- Assessment of both HbA1c and glycemic variability
- Objective evaluation of pulmonary hypertension severity

RESULTS
Baseline Characteristics
A total of **72 patients** diagnosed with pulmonary hypertension were included in the study. All enrolled participants underwent detailed clinical evaluation, laboratory investigations, HbA1c estimation, assessment of glycemic variability, and transthoracic echocardiography for measurement of pulmonary artery systolic pressure (PASP)

Age Distribution
Table 1. Age distribution of the study population

Age Group	N	%
30–39	3	4.2%
40–49	20	27.8%
50–59	20	27.8%
60–69	16	22.2%
70–79	9	12.5%
≥80	4	5.6%
Total	72	100%

The majority of patients belonged to the **40–49 years** and **50–59 years** age groups (27.8% each). Together, these decades accounted for more than half of the study population, indicating that pulmonary hypertension was predominantly encountered in middle-aged adults. Only 5.6% of patients were aged ≥80 years

Sex Distribution
The study included both male and female patients. Females constituted a slightly larger proportion of the study population, reflecting the recognized epidemiological pattern of pulmonary hypertension reported in previous literature.

Table 2. Sex distribution

Age Group	N	%
30–39	3	4.2%
40–49	20	27.8%
50–59	20	27.8%
60–69	16	22.2%
70–79	9	12.5%
≥80	4	5.6%
Total	72	100%

Associated Comorbidities
Hypertension, diabetes mellitus, coronary artery disease, and chronic respiratory disorders were the predominant comorbid conditions observed among the study participants. The coexistence of these disorders highlights the complex metabolic and cardiovascular profile frequently encountered in patients with pulmonary hypertension.

Table 3. Associated comorbidities

Comorbidities	N	%
Hypertension	33	45.8%
Dyslipidemia	40	55.6%

Duration of Diabetes Mellitus
Patients with diabetes demonstrated varying disease duration. Evaluation of diabetes duration was included to determine its possible influence on pulmonary hypertension severity and glycemic control.

Table 4. Duration of diabetes mellitus

Duration of DM	N	%
1–5	25	34.7%
6–10	30	41.7%
11–15	19	26.4%

>15	2	2.8%
Total	72	100%

Anthropometric Characteristics

Anthropometric assessment included height, weight, body mass index (BMI), waist circumference, hip circumference, waist-to-hip ratio, and body fat percentage. These measurements were obtained to evaluate the relationship between obesity-related variables and pulmonary hypertension.

Table 5. Anthropometric characteristics

Anthropometric Measures	Mean	SD
BMI (kg/m²)	29.738	4.1389
Waist (cm)	92.53	8.808
Hip (cm)	101.75	9.522
Waist-Hip Ratio	.924	.0544

Blood Pressure Profile

Both systolic and diastolic blood pressure measurements were recorded for all participants. These variables formed part of the cardiovascular risk assessment and were analyzed alongside glycemic parameters.

Table 6. Blood pressure profile

Blood Pressure	Mean	SD
SBP (mmHg)	139.32	15.25
DBP (mmHg)	79.25	8.59
MAP (mmHg)	99.25	9.52

Glycemic Control

Assessment of glycemic status demonstrated that a substantial proportion of patients exhibited impaired long-term glycemic control. HbA1c values varied across the study population, with increasing HbA1c levels corresponding to progressively higher pulmonary artery systolic pressures.

Table 7. Distribution of HbA1c

HbA1c (%)	N	%
6.00 – 6.99	18	25.0%
7.00 – 7.99	34	47.2%
8.00 – 8.99	14	19.4%
9.00 – 9.99	5	6.9%
10.00 – 11.00	1	1.4%
MEAN+SD	7.76	0.90

Glycemic Variability

The mean glucose standard deviation was **40.40 ± 9.56 mg/dL**, while the mean coefficient of variation (CV%) was **22.80 ± 3.36%**, indicating appreciable glucose fluctuations among patients with pulmonary hypertension.

Glycemic Variability	Mean	SD
Glucose SD (mg/dL)	40.40	9.56
Glycemic Variability (CV %)	22.80	3.36

These findings indicate that considerable short-term glucose fluctuations were present in addition to chronic hyperglycemia.

Pulmonary Hemodynamics

The **mean pulmonary artery systolic pressure (PASP)** of the study population was **54.35 ± 10.15 mmHg**, indicating a substantial burden of pulmonary hypertension.

Table 9. Pulmonary artery systolic pressure

PH Severity	N	%
Mild	23	31.9%
Moderate	45	62.5%
Severe	4	5.6%
Total	72	100%

Correlation Between Glycemic Parameters and PASP

A statistically significant positive correlation was observed between glycemic indices and pulmonary artery systolic pressure.

Table 10. Correlation of glycemic parameters with PASP

Variable	Pearson Correlation (r)	p-value	Interpretation
HbA1c (%)	0.505	<0.001	Significant positive correlation
Glycemic Variability (CV %)	0.419	<0.001	Significant positive correlation

Both HbA1c and glycemic variability demonstrated moderate positive correlations with PASP, indicating that worsening glycemic control was associated with increasing pulmonary artery systolic pressure.

PASP According to HbA1c Categories

A progressive increase in PASP was observed across increasing HbA1c categories.

HbA1c	PASP (mmHg)		F	p-value
	Mean	SD		
6.00 – 6.99	49.06	8.250	2.930	0.001
7.00 – 7.99	51.28	8.073		
8.00 – 8.99	62.06	10.630		
9.00 – 9.99	61.14	8.335		
10.00 – 11.00	66.00	.		

ANOVA: F = 2.930, p = 0.001

These findings demonstrate a significant association between increasing HbA1c and worsening pulmonary artery systolic pressure.

Severity of Pulmonary Hypertension

The majority of patients had **moderate pulmonary hypertension (62.5%)**, indicating that moderate disease represented the most common clinical presentation in this cohort.

Table 12. Severity of pulmonary hypertension

PH Severity	N	%
Mild	23	31.9%
Moderate	45	62.5%
Severe	4	5.6%
Total	72	100%

Association Between Glycemic Variability and Pulmonary Hypertension Severity

Glycemic variability increased progressively with worsening pulmonary hypertension severity.

PH Severity	Glycemic Variability (CV %)		F	p-value
	Mean	SD		
Mild	21.59	3.70	4.409	0.016
Moderate	23.09	3.00		
Severe	26.45	2.22		
Total	22.80	3.36		

p = 0.016

Patients with severe pulmonary hypertension demonstrated significantly greater glycemic variability than those with mild disease, suggesting that glucose fluctuations may contribute to pulmonary vascular dysfunction and disease progression.

DISCUSSION

Pulmonary hypertension (PH) is a progressive cardiopulmonary disorder characterized by pulmonary vascular remodeling, increased pulmonary vascular resistance, and eventual right ventricular dysfunction. Emerging evidence suggests that metabolic abnormalities, particularly diabetes mellitus and dysglycemia, contribute to pulmonary vascular disease through endothelial dysfunction, oxidative stress, chronic inflammation, and vascular remodeling. The present study evaluated the association of HbA1c and glycemic variability with pulmonary artery systolic pressure (PASP) and disease severity in patients with pulmonary hypertension.

The study population predominantly comprised middle-aged individuals, with most patients belonging to the 40–49 and 50–59-year age groups. This finding is consistent with previous epidemiological studies demonstrating the increasing burden of pulmonary hypertension among individuals with cardiovascular disease, metabolic syndrome, and diabetes mellitus. The mean PASP was **54.35 ± 10.15 mmHg**, reflecting a substantial burden of pulmonary hypertension. Comparable pulmonary pressures have been reported by Belly MJ et al., whereas Richter MJ et al. observed higher values in patients with advanced pulmonary arterial hypertension, likely reflecting differences in disease severity and study populations.

A major finding of the present study was the significant association

between long-term glycemic control and pulmonary hypertension severity. HbA1c demonstrated a **moderate positive correlation with PASP ($r = 0.505$, $p < 0.001$)**, indicating that poorer glycemic control was associated with progressively higher pulmonary artery pressures. Increasing PASP across successive HbA1c categories further supports a dose-dependent relationship between chronic hyperglycemia and disease severity.

Previous studies have reported variable associations between HbA1c and pulmonary hypertension. Pugh ME et al. demonstrated a high prevalence of glucose intolerance among patients with pulmonary arterial hypertension, whereas Abernethy AD et al. did not observe a direct relationship between HbA1c and pulmonary hemodynamics. In contrast, Sompradeekul S et al. and Zhang S et al. reported significant associations between poor glycemic status, functional impairment, and right ventricular dysfunction. The present findings support the growing evidence that chronic hyperglycemia is associated with worsening pulmonary hypertension.

An important strength of this study is the simultaneous assessment of **glycemic variability** in addition to HbA1c. Glycemic variability demonstrated a **moderate positive correlation with PASP ($r = 0.419$, $p < 0.001$)** and increased significantly with worsening pulmonary hypertension, from **$21.59 \pm 3.70\%$** in mild disease to **$26.45 \pm 2.22\%$** in severe disease (**$p = 0.016$**). These findings are biologically plausible, as intermittent glucose excursions have been shown to induce greater oxidative stress, endothelial dysfunction, and inflammatory activation than sustained hyperglycemia, thereby promoting pulmonary vascular remodeling.

Most patients had **moderate pulmonary hypertension (62.5%)**, highlighting the coexistence of clinically significant pulmonary vascular disease with poor glycemic control and increased glycemic variability. Collectively, these findings suggest that chronic hyperglycemia and glucose fluctuations may contribute to pulmonary vascular injury through endothelial dysfunction, oxidative stress, inflammation, impaired nitric oxide bioavailability, and abnormal smooth muscle proliferation.

From a clinical perspective, assessment of **HbA1c and glycemic variability** may provide useful adjunctive information for risk stratification in patients with pulmonary hypertension. Early identification of dysglycemia may facilitate timely metabolic optimization alongside standard pulmonary hypertension management. However, this was a single-center study with a relatively modest sample size, and its cross-sectional design precludes causal inference. Larger prospective multicenter studies are required to determine whether improving glycemic control and reducing glycemic variability can influence disease progression and long-term clinical outcomes.

Limitations of the Study

The present study has certain limitations. First, it was conducted at a **single tertiary care center**, which may limit the generalizability of the findings to other populations. Second, the **cross-sectional design** precludes assessment of causal relationships between glycemic abnormalities and progression of pulmonary hypertension. Third, pulmonary artery pressures were estimated using **echocardiography rather than right heart catheterization**, the reference standard for hemodynamic assessment. Fourth, the relatively modest sample size may have limited the power to detect weaker associations. Finally, long-term clinical outcomes, including mortality, hospitalization, exercise capacity, and response to pulmonary hypertension-specific therapies, were not evaluated. Future multicenter prospective studies with larger sample sizes and long-term follow-up are warranted to validate these findings and further define the prognostic significance of glycemic variability in pulmonary hypertension.

CONCLUSION

The present study demonstrated a significant association between glycemic abnormalities and pulmonary hypertension severity. Higher HbA1c levels were associated with increased pulmonary artery systolic pressure, while greater glycemic variability correlated with worsening pulmonary hypertension severity. These findings suggest that both chronic hyperglycemia and glucose fluctuations may contribute to pulmonary vascular dysfunction through endothelial injury, oxidative stress, inflammation, and vascular remodeling. Assessment of HbA1c together with glycemic variability may therefore serve as a practical adjunct for identifying patients at increased risk of severe pulmonary hypertension. Early recognition and optimal management of dysglycemia may improve risk stratification and provide opportunities for more comprehensive patient management. However, larger prospective multicenter studies are required to establish whether improving glycemic control and reducing glycemic variability translate into improved pulmonary hemodynamics and long-term clinical outcomes.

Summary

This hospital-based cross-sectional study demonstrated that poor glycemic control and increased glycemic variability were significantly associated with greater pulmonary hypertension severity. Combined assessment of HbA1c and glycemic variability may improve risk stratification in patients with pulmonary hypertension. Larger prospective multicenter studies are needed to validate these findings.

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