Research Article

Evaluation Of Fasting And Postprandial Dysglycemia, Hyperinsulinemia, And Blood Pressure Combination In An Adult Health Check-Up Cohort: A Cross-Sectional Observational Study

Dr.N.Murugesan[1], Dr. Venkatram Murugesan[2], Dr.S.C.Rajan[3], Dr.Sumathy Masanam Kasi[4]

[1]Chief Nephrologist East coast hospitals Puducherry ORCID: 0009 0008 6178 6920 Email: nm@eastcoasthospitals.com
[2]Chief Nephrologist East coast hospitals Puducherry ORCID: 0000 0002 3513 2279 Email: drvenkatram@gmail.com
[3]Consultant Family Medicine East Coast Hospitals Puducherry ORCID:0009 0000 9348 1365 Email:docrajan@gmail.com
[4]Research Coordinator East coast hospitals Puducherry ORCID: 0000-0003-3907-0420 Email: clinicalresearch@eastcoasthospitals.com

Abstract

Introduction

Hyperinsulinemia and dysglycemia represent key precursors to type 2 diabetes mellitus and cardiovascular disease. However, the simultaneous coupling of glycemic, insulinemic, and hemodynamic metrics in real-world clinical screening settings remains insufficiently characterized.

Objectives

To evaluate baseline demographic, glycemic, hyperinsulinemic, and blood pressure profiles in an adult clinical cohort, investigating intra-domain physiological coupling, postprandial metabolic reactivity, and potential sex-based variances.

Methods

A retrospective descriptive cross-sectional study was conducted at a tertiary care hospital in Pondicherry, India, following institutional review board approval. From an eligible registry of 356 health check-up records (participants aged 30–75 years), a sample size of N = 61 (male n = 37, female n = 24) was systematically selected using a fixed sampling interval (k = 6). Standardized clinical evaluations included mean resting blood pressure from three seated readings. Biochemical assays following a 10–12 hour fast measured fasting blood sugar (FBS) and fasting insulin; postprandial blood sugar (PPBS) and postprandial insulin were evaluated 2 hours after a 75g glucose load or standardized meal. Systemic insulin resistance was estimated via HOMA-IR. Data were analyzed using IBM SPSS version 26, employing non-parametric inferential tests including Spearman rank correlation (r_s), Wilcoxon signed-rank test (Z) , and Mann-Whitney U test (U),to assess physiological coupling, postprandial reactivity, and sex-based differences ($p < 0.05$).

Results

The cohort (mean age 56.89 ± 11.96 years) demonstrated high baseline burdens of dysglycemia (fasting blood glucose 132.07 ± 49.55mg/dL; postprandial blood glucose 212.77 ±114.91 mg/dL), hyperinsulinemia (fasting insulin 43.13 ± 48.24IU/mL; postprandial insulin 74.65 ± 75.94IU/mL), and systemic hypertension (mean systolic blood pressure 150.59 ± 13.89 mmHg; mean diastolic blood pressure 88.67 ±8.57 mmHg). Spearman rank correlation revealed strong intra-domain coupling between fasting and postprandial glucose ($r_s = 0.877$, $p < 0.001$) as well as fasting and postprandial insulin ($r_s = 0.900$, $p < 0.001$). Cross-domain correlations between glycemic, insulinemic, and hemodynamic variables were statistically non-significant ($p > 0.05$). Wilcoxon signed-rank tests confirmed significant postprandial increases in glucose ($Z = -6.727$, $p < 0.001$) and insulin ($Z = -6.360$, $p < 0.001$), with 96.7% (n = 59) of participants showing positive rank increases. Mann-Whitney U tests showed no significant sex-based differences across glycemic, postprandial insulinemic, or blood pressure profiles ($p > 0.05$).

Conclusion

The findings highlight pronounced compensatory hyperinsulinemia and severe postprandial metabolic excursions, reflecting blunted tissue sensitivity and near-exhaustion of pancreatic β-cell reserve. The physiological independence between metabolic and hemodynamic axes suggests that while these risk factors co-occur in metabolic syndrome, their individual severity follows distinct pathophysiological trajectories. The absence of sex-based variance underscores a shared cardiometabolic disease burden across both sexes in this clinical setting, supporting the routine inclusion of hyperinsulinemia screening alongside standard glycemic and blood pressure metrics.

Keywords: Hyperinsulinemia, Insulin Resistance, Glucose Intolerance, Metabolic Syndrome ,Hypertension, Postprandial Period, Cross-Sectional Studies

1. Introduction

Insulin is a 51-amino acid peptide hormone synthesized in the endoplasmic reticulum and Golgi apparatus of pancreatic β-cells within the islets of Langerhans. Under normal physiological conditions, basal blood glucose concentrations between 2.0 and 4.0 mM trigger insulin biosynthesis, whereas postprandial glucose elevations within the range of 4.4 to 6.6 mM stimulate its biphasic exocytosis (1,2). Cleaved from proinsulin alongside C-peptide in equimolar amounts, insulin undergoes rapid hepatic clearance (50–60% first-pass extraction), yielding a systemic half-life of approximately 4 minutes (3). In contrast, C-peptide is cleared renally with a longer plasma half-life, serving as a stable biomarker for endogenous β-cell reserve (3,4).

When peripheral tissues develop blunted sensitivity to circulating insulin- a hallmark of insulin resistance, the pancreatic β-cells undergo compensatory hypersecretion to maintain euglycemia, culminating in hyperinsulinemia (5). Globally, acquired hyperinsulinemia driven by metabolic dysfunction affects an estimated 40% to 50% of the adult population (6). In India, where metabolic syndrome and early-onset type 2 diabetes mellitus present at lower body mass index thresholds, hyperinsulinemia is a critical precursor state. National epidemiological data indicate that the prevalence of elevated fasting or postprandial insulin levels ranges from 30.0% to 47.2% across at-risk adult populations (7,8).

Chronic hyperinsulinemia is not merely a benign compensatory response; it acts as an independent driver of systemic disease across multiple organ systems. Persistent hyperinsulinemia promotes renal sodium retention, enhances sympathetic nervous system activity, and induces vascular smooth muscle proliferation, directly contributing to essential hypertension and accelerated arterial stiffness (9–11). Furthermore, sustained hyperinsulinemia is tightly linked to mitochondrial dysfunction, chronic kidney disease progression, microvascular damage, and subclinical atherosclerosis even among normoglycemic individuals (12–15).

Despite its central role in cardiometabolic disease pathogenesis, cross-sectional profiles evaluating the simultaneous coupling of fasting and stimulated glycemic, insulinemic, and hemodynamic metrics in real-world clinical screening settings remain insufficiently characterized. Therefore, this study evaluated baseline demographic, glycemic, hyperinsulinemic, and blood pressure profiles among an adult cohort (N=61) selected systematically from a master health check-up registry in India, investigating intra-domain physiological coupling, postprandial metabolic reactivity, and potential sex-based variances.

2.Materials and Methods

Study Design and Ethical Considerations

This descriptive, retrospective cross-sectional study was designed to evaluate cardiovascular and metabolic risk stratification using standardized laboratory and physical indices. The study was conducted at a tertiary care hospital in Pondicherry, India, in strict adherence to the ethical principles outlined in the Declaration of Helsinki. Ethical clearance was obtained from the institutional review board prior to data retrieval. To safeguard participant confidentiality, all patient identities were completely anonymized at all stages of data entry and analysis.

Study Population and Sampling Strategy

The study population comprised male and female participants aged 30 to 75 years who attended the screening outpatient department (OPD) for a general master health check-up between January and June 2026. The lower age boundary of 30 years was selected due to the premature onset of cardiometabolic risks in South Asian populations, while the upper boundary was restricted to 75 years to minimize the confounding effects of advanced senescent comorbidities. Furthermore, this middle-to-elderly age bracket reflects regional health-seeking behaviors, as individuals in this demographic actively utilize free institutional health check-ups and state-sponsored healthcare schemes to offset out-of-pocket expenditures (16,17).

From the baseline master health check-up registry, a total pool of 356 participants met the specific inclusion criteria for age and complete record availability. Based on this finite sub-population, the final required sample size was calculated using the finite population correction formula. Assuming a 95% confidence level, a 10% margin of error, and a conservative response variability of 50%, the minimum statistically representative sample size was determined to be 61 participants. To achieve this target while minimizing selection bias, a systematic sampling frame was utilized to select every 6th patient record (k = N/n = 356/61 approx 5.84) chronologically from the eligible registry frame until the required sample size of 61 was reached.

Clinical and Biophysical Assessments

Hemodynamic and anthropometric assessments were performed by trained clinical staff using standardized protocols.

- Resting Blood Pressure (BP): Measured using a validated digital sphygmomanometer. Participants rested quietly in a seated position for at least 5 minutes prior to measurement. To ensure accuracy, three consecutive readings were taken with a 2-minute interval between draws; the mean value of these readings was calculated and utilized for formal analysis.
- Anthropometric Indices: Standard procedures were used to record participant demographic data, age, gender, and baseline physical parameters during the OPD screening.

Biochemical Analysis and Laboratory Assays

Venous blood samples were collected through venipuncture by certified laboratory technicians under strict aseptic conditions.

The biochemical evaluation was divided into two distinct functional phases:

1. Fasting State: Participants were on mandatory overnight fast of 10 to 12 hours prior to the initial blood draw. This baseline specimen was analyzed for Fasting Blood Sugar (FBS), Fasting Insulin, Total Cholesterol, High-Density Lipoprotein Cholesterol (HDL-C), and Triglycerides. Low-Density Lipoprotein Cholesterol (LDL-C) and Very Low-Density Lipoprotein Cholesterol (VLDL-C) concentrations were derived using standard automated formulas.

2. Postprandial State: Following the fasting draw, participants consumed a standardized 75g glucose load or a standardized metabolic meal. A second blood draw was executed exactly 2 hours post-ingestion to determine Postprandial Blood Sugar (PPBS) and Postprandial Insulin concentrations.

All biochemical assays were processed immediately using automated laboratory analyzers according to standard institutional quality control guidelines. Systemic insulin resistance was mathematically estimated using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) index, calculated using the standard formula.

Statistical analysis All the data was tabulated and analysed by using mean, standard deviation, proportion and chi square with Rates correction. Statistical analyses were carried out using the SPSS (IBM) software, version 26. Clinical variables were presented as means for continuous variables and frequencies for categorical variables. basal blood glucose concentrations between 2.0 and 4.0 mM

3.Results:

Table 1 presents the demographic presentation of the sample population (N = 61). The mean age of the participants was 56.89 ± 11.96 years, spanning a wide age range from a minimum of 18 years to a maximum of 75 years. This distribution reflects an adult to middle-aged study cohort, providing a representative demographic baseline across young, middle-aged, and older adult age brackets.

Table 1 Presents the Gender presentation of the study population

Gender		**Frequency**	**Percent**
Valid	Male	37	60.7
	Female	24	39.3
	Total N	61	100.0

Table 2 Baseline Metabolic, Glycemic, and Hemodynamic Characteristics of the Study Participants (N = 61)

Descriptive Statistics					
	N	Minimum	Maximum	Mean	Std. Deviation
FBS	61	76	359	132.07	49.54
PPBS	61	97	579	212.77	114.91
FI	61	5.50	200.00	43.1272	48.244
PPI	61	6.53	458.50	74.6497	75.93
SBP	61	120	174	150.59	13.89
DBP	61	70	110	88.67	8.57

Table 2 presents the metabolic, glycemic, and hemodynamic characteristics of the study participants (N = 61), detailing the minimum to maximum ranges, means, and standard deviations across clinical parameters.

- **Glycemic Profile:** The mean fasting blood sugar (FBS) was 132.07 ± 49.55 mg/dL (range: 76–359 mg/dL), while the postprandial blood sugar (PPBS) showed a marked elevation at 212.77 ± 114.91 mg/dL (range: 97–579 mg/dL). Both parameters demonstrate substantial glycemic variability and elevated group averages above normal reference limits.
- **Insulin Kinetics:** Fasting insulin (FI) averaged 43.13 ± 48.24 µIU/mL (range: 5.50–200.0 µIU/mL), and postprandial insulin (PPI) rose to 74.65 ± 75.94 µIU/mL (range: 6.53–458.5 µIU/mL). The wide standard deviations relative to the means reflect pronounced heterogeneity in metabolic and beta-cell response among participants.
- **Hemodynamic Profile:** Mean systolic blood pressure (SBP) was 150.59 ± 13.89 mmHg (range: 120–174 mmHg), and mean diastolic blood pressure (DBP) was 88.67 ± 8.57 mmHg (range: 70–110 mmHg), indicating a cohort with baseline stage-1 to stage-2 systemic hypertension.

Table 3: Spearman's Rank Correlation Matrix Across Metabolic and Hemodynamic Parameters (N = 61)

Clinical Parameter	**FBS**	**PPBS**	**FI**	**PPI**	**SBP**	**DBP**
1. Fasting Blood Sugar (FBS)	1.000	**0.877****	-0.120	-0.151	-0.140	0.064

2. Postprandial Blood Sugar (PPBS)	—	1.000	-0.208	-0.220	-0.131	0.027
3. Fasting Insulin (FI)	—	—	1.000	**0.900****	-0.007	-0.101
4. Postprandial Insulin (PPI)	—	—	—	1.000	-0.047	-0.112
5. Systolic Blood Pressure (SBP)	—	—	—	—	1.000	**0.582****
6. Diastolic Blood Pressure (DBP)	—	—	—	—	—	1.000

* Correlation is significant at the p < 0.01 level (2-tailed).

Due to the non-parametric distribution of baseline variables, Spearman's rank correlation coefficient (r_s) was performed to evaluate monotonic relationships across glycemic, insulinemic, and hemodynamic parameters (N = 61).

- **Glycemic Coupling:** A strong, statistically significant positive correlation was identified between Fasting Blood Sugar (FBS) and Postprandial Blood Sugar (PPBS) ($r_s = 0.877$, $p < 0.001$).
- **Insulin Kinetics:** Similarly, Fasting Insulin (FI) demonstrated a strong, statistically significant positive correlation with Postprandial Insulin (PPI) ($r_s = 0.900$, $p < 0.001$).
- **Hemodynamic Profile:** A moderate, statistically significant positive correlation was observed between Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP) ($r_s = 0.582$, $p < 0.001$).
- **Cross-Domain Correlations:** No statistically significant correlations were observed between glycemic markers (FBS, PPBS) and insulinemic levels (FI, PPI) or blood pressure parameters (SBP, DBP) ($p > 0.05$ across all cross-domain pairwise evaluations), indicating distinct physiological variance across glucose, insulin, and pressure dynamics within this cohort.

Table 4: Wilcoxon Signed-Ranks Test Comparing Fasting and Postprandial Glycemic and Insulinemic Responses(N = 61)

Paired Comparison	Rank Direction	n	Mean Rank	Sum of Ranks	Test Statistic (Z)	p-value (2-tailed)
PPBS – FBS	Negative Ranks (PPBS} < FBS)	2	4.50	9.00	**-6.727**	**< 0.001**
	Positive Ranks (PPBS > FBS)	59	31.90	1882.00		
	Ties (PPBS= FBS)	0	—	—		
PPI – FI	Negative Ranks (PPI < FI)	2	30.00	60.00	**-6.360**	**< 0.001**
	Positive Ranks (PPI > FI)	59	31.03	1831.00		
	Ties (PPI} = FI)	0	—	—		

*Based on negative ranks; significance set at $p < 0.05$.

A Wilcoxon signed-ranks test was conducted to evaluate significant paired changes from fasting to postprandial states for glucose and insulin dynamics across the cohort (N = 61).

- **Glycemic Excursion (PPBS vs. FBS):** Postprandial blood sugar levels were significantly higher than fasting blood sugar levels ($Z = -6.727$, $p < 0.001$). A total of 59 out of 61 participants (96.7%) demonstrated a positive rank response (PPBS > FBS), with only 2 participants showing lower postprandial values.
- **Insulinemic Response (PPI vs. FI):** Similarly, postprandial insulin levels exhibited a statistically significant elevation compared to fasting insulin levels ($Z = -6.360$, $p < 0.001$). Positively ranked responses (PPI > FI) were observed in 59 participants (96.7%), confirming a robust endogenous insulin release following metabolic challenge.

Table 5: Mann-Whitney U Test Comparing Metabolic and Hemodynamic Parameters Across Sex (N = 61)

Clinical Parameter	Sex	n	Mean Rank	Sum of Ranks	Mann-Whitney U	Z-score	p-value(2-tailed)
Fasting Blood Sugar (FBS)	Male (1)	37	32.62	1207.00	384.000	-0.886	0.376
	Female (2)	24	28.50	684.00			
Postprandial Blood Sugar (PPBS)	Male (1)	37	31.00	1147.00	444.000	0.000	1.000
	Female (2)	24	31.00	744.00			
Fasting Insulin (FI)	Male (1)	37	34.59	1280.00	311.000	-1.964	0.050

	Female (2)	24	25.46	611.00			
Postprandial Insulin (PPI)	Male (1)	37	33.24	1230.00	361.000	-1.225	0.220
	Female (2)	24	27.54	661.00			
Systolic Blood Pressure (SBP)	Male (1)	37	32.16	1190.00	401.000	-0.636	0.525
	Female (2)	24	29.21	701.00			
Diastolic Blood Pressure (DBP)	Male (1)	37	33.23	1229.50	361.500	-1.270	0.204
	Female (2)	24	27.56	661.50			

*Grouping variable: Sex (1 = Male, 2 = Female); significance set at $p < 0.05$.

A finally Mann-Whitney U test was performed to evaluate sex-based differences across metabolic, glycemic, and hemodynamic parameters between male (n = 37) and female (n = 24) participants.

- **Glycemic Parameters:** No statistically significant differences were observed between males and females for Fasting Blood Sugar (U = 384.000, Z = -0.886, p = 0.376) or Postprandial Blood Sugar (U = 444.000, Z = 0.000, p = 1.000), with both groups displaying identical mean ranks for PPBS (31.00).
- **Insulin Kinetics:** Fasting Insulin demonstrated a borderline trend toward higher levels in males (Mean Rank = 34.59) compared to females (Mean Rank = 25.46), though it did not cross the threshold for statistical significance (U = 311.000, Z = -1.964, p = 0.050). Postprandial Insulin levels similarly showed no significant sex-based variation (U = 361.000, Z = -1.225, p = 0.220).
- **Hemodynamic Parameters:** Systemic blood pressure parameters showed no significant variation between sexes for Systolic Blood Pressure (U = 401.000, Z = -0.636, p = 0.525) or Diastolic Blood Pressure (U = 361.500, Z = -1.270, p = 0.204).

Overall, baseline metabolic and hemodynamic distributions remained comparable across sex categories, indicating that baseline glycemic and cardiovascular risk factors in this cohort were independent of sex.

Inferential statistical evaluations confirmed significant intra-domain physiological coupling alongside robust postprandial metabolic excursions across the study cohort (N = 61). Non-parametric correlation analysis using Spearman's rank test demonstrated strong, statistically significant positive relationships between fasting and postprandial glucose ($r_s = 0.877$, $p < 0.001$), fasting and postprandial insulin ($r_s = 0.900$, $p < 0.001$), and systolic and diastolic blood pressure ($r_s = 0.582$, $p < 0.001$), whereas cross-domain correlations between glycemic, insulinemic, and pressure parameters remained non-significant ($p > 0.05$). Wilcoxon signed-ranks testing demonstrated significant postprandial elevations over baseline fasting levels for both blood glucose (Z = -6.727, $p < 0.001$) and serum insulin (Z = -6.360, $p < 0.001$), with 96.7% of participants (n = 59) exhibiting positive rank responses. Finally, subgroup comparisons via Mann-Whitney U testing revealed no statistically significant sex-based differences across glycemic, postprandial insulinemic, or blood pressure parameters ($p > 0.05$), with only fasting insulin showing a borderline trend toward higher levels in males (U = 311.000, Z = -1.964, p = 0.050).

4. Discussion

This study evaluated baseline demographic, glycemic, insulinemic, and hemodynamic characteristics among an adult cohort (N=61) systematically selected from a master health check-up registry. Overall, the findings reveal a high burden of metabolic dysfunction characterized by baseline dysglycemia, hyperinsulinemia, and systemic hypertension. These observations align with those of Kumar and Singh (7), who highlighted a high prevalence of hyperinsulinemia (>39%) and metabolic syndrome clustering among young Indian adults, often occurring independently of elevated body weight or overt fasting hyperglycemia. Untreated hyperinsulinemia and metabolic syndrome serve as critical precursors to type 2 diabetes mellitus and cardiovascular disease (7,8). Inferential non-parametric testing demonstrated strong intra-domain physiological combination, marked postprandial metabolic excursions, and an absence of gender -based divergence across primary metabolic axes. Conversely, Alshehri (18) observed significant sex disparities in epidemiological evaluations, and Alnozhah et al.(27) reported an overall age-adjusted metabolic syndrome prevalence of 39.3% in Saudi Arabia with higher rates in females (42.0% vs. 37.2%). This divergence may stem from their community-based sampling strategy and larger sample population.

The cohort's demographic profile (mean age 56.89 ± 11.96 years) represents a middle-aged population at heightened cardiometabolic risk. Baseline mean fasting blood glucose (132.07±49.55 mg/dL) and postprandial blood glucose (212.77±114.91 mg/dL) exceed standard diagnostic cut-offs for overt diabetes mellitus, indicating severe peripheral insulin resistance and compromised β-cell compensatory mechanics. Baseline fasting insulin (43.13±48.24 µIU/mL) and postprandial insulin (74.65±75.94 µIU/mL) reflect hyper insulinemic compensation, wherein pancreatic β-cells hyper secrete insulin to maintain glycemic control despite blunted tissue sensitivity. The wide standard deviations reflect marked inter-individual heterogeneity typical of real-world primary screening cohorts. Existing literature (19–21) indicates that islet cell mass dynamically adapts to

metabolic demands to maintain normoglycemia. However, while obesity induces up to a 30-fold increase in β-cell mass in murine models (22), human compensatory expansion is limited to approximately 30% (23,24).

Spearman rank correlation analyses identified distinct functional clusters within the cohort. A strong intra-domain correlation between fasting and postprandial glucose (r_s=0.877, p<0.001) confirms that fasting dysglycemia is a primary determinant of postprandial glycemic peaks. Similarly, fasting insulin strongly predicted postprandial insulin output (r_s=0.900, p<0.001), demonstrating preserved basal-to-stimulated β-cell secretory dynamics. As established in pathophysiological models of insulin resistance and β-cell fate (19,20), β-cell dysfunction compounded by insulin resistance is the central driver of type 2 diabetes pathogenesis, forming a complex bidirectional interplay where acute hyperglycemia exacerbates both defects.

Hemodynamically, a moderate correlation between systolic and diastolic blood pressure (r_s =0.582, p<0.001) reflects expected arterial stiffness and elevated peripheral vascular resistance characteristic of an essential hypertension phenotype (mean SBP 150.59±13.89 mmHg; mean DBP 88.67±8.57 mmHg). Cross-domain correlations between glycemic, insulinemic, and blood pressure variables were statistically non-significant (p > 0.05). This physiological independence suggests that although these cardiometabolic risk factors co-occur within the metabolic syndrome continuum, their individual severity in cross-sectional evaluation follows distinct pathophysiological trajectories. While clustering of abdominal obesity, hypertension, impaired glucose tolerance, insulin resistance, and dyslipidemia produces synergistic cardiovascular risk (25–28), cross-sectional severity markers across these axes may vary independently.

Paired Wilcoxon signed-rank tests demonstrated significant physiological reactivity to metabolic challenge. Both postprandial glucose (Z=−6.727, p<0.001) and postprandial insulin (Z=−6.360, p<0.001) increased markedly over baseline fasting levels, with 96.7% (n=59) of participants exhibiting positive rank increases. This postprandial excursion underscores that even in individuals with elevated fasting baselines, meal-stimulated glycemic loads exert acute stress on endogenous insulin reserves.

Finally, Mann-Whitney U tests confirmed that baseline glycemic, postprandial insulinemic, and blood pressure profiles were comparable between male (n=37) and female (n=24) participants (p>0.05). Although fasting insulin demonstrated a borderline trend toward higher levels in males (U = 311.000, Z = −1.964, p = 0.050), the overall absence of significant gender -based variance indicates that cardiometabolic risk represents a shared burden across both genders in this clinical cohort.

Limitations

Several limitations warrant consideration. First, the cross-sectional design restricts inferences regarding causal progression between hyperinsulinemia, dysglycemia, and systemic hypertension. Second, while the sample size of n = 61 met the statistical requirement derived from the finite population correction formula (N = 356, 10 % margin of error), larger multi-center cohorts are needed to confirm the borderline trend observed in male fasting insulin levels.

Declarations

Conflict of Interest The authors declare that there is no conflict of interest regarding the publication of this paper. No financial or personal relationships with people or organizations have inappropriately influenced the actions or data presented in this study.

Funding Statement This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors. The study was conducted using existing institutional resources and clinical records from the East Coast Hospitals Medical Records Department.

Ethical Statement and Informed Consent

Patient Anonymity and Confidentiality: The authors declare that the patient's identity has been strictly anonymized throughout this report. All identifying information, including name, specific date of birth, and hospital identification numbers, has been removed or de-identified to ensure complete privacy in accordance with the ethical standards of the Institutional Ethics Committee and the Declaration of Helsinki. (Protocol No.EC/NEW/INSG/2024/4589/EIMS/2026/37),

Informed Consent: Written informed consent was obtained from the patient for the publication of this case report and any accompanying images or clinical data. The patient was briefed on the educational and research-driven purpose of this documentation. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon request.

Use of Data: The clinical data and diagnostic findings presented herein are utilized exclusively for research and educational purposes to advancement of medical science.

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