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TO STUDY THE PREVALENCE OF METABOLIC SYNDROME AND ITS COMPONENTS IN TYPE 2 DIABETES MELLITUS PATIENTS.

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Abstract

Background: Type 2 Diabetes Mellitus (T2DM) rarely presents as an isolated metabolic finding but frequently clusters with atherogenic dyslipidemia, central obesity, and systemic hypertension—collectively known as Metabolic Syndrome. Evaluating the co-prevalence of Metabolic Syndrome within a diabetic cohort is crucial for establishing comprehensive cardiovascular risk mitigation strategies. ***Objective:*** To study the prevalence of Metabolic Syndrome and evaluate its individual components among patients with Type 2 Diabetes Mellitus attending a tertiary care hospital. ***Materials and Methods:*** A cross-sectional observational study was conducted involving 200 randomly sampled adult participants with established T2DM (defined by HbA1c > 6.5%) at a tertiary care facility in Mathura, India. Metabolic Syndrome was diagnosed using the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (NCEP ATP III) guidelines, requiring at least three out of five clinical thresholds. Anthropometric indices, blood pressure, fasting plasma glucose, and fasting lipid profiles were recorded. Statistical analyses were executed using SPSS version 26. ***Results:*** The cohort comprised 100 males and 100 females with a mean age of 49.8 years. The overall prevalence of Metabolic Syndrome within this diabetic population was found to be 70.0% (n = 140). A distinct gender disparity was noted, with a significantly higher prevalence observed in females (83.0%) compared to males (57.0%) ($p < 0.05$). The frequencies of individual Metabolic Syndrome components were: Hyperglycemia (>FBS 100mg/dL) at 100% (n = 200); Systemic Hypertension (>130/85mmHg) at 83.0% (n = 166); Abdominal Obesity (WC > 102cms in males, > 88cms in females) at 71.0% (n = 142); Hypertriglyceridemia (TG>150mg/dL) at 53.0% (n = 106) ; and Low HDL Cholesterol (< 40mg/dL in males, < 50mg/dL in females) at 47.0% (n = 94). Notably, when applying Asian-specific modified criteria for abdominal obesity (WC >90cms in Males, >80cms in Females), central adiposity prevalence rose sharply to 85.5%. ***Conclusion:*** There is a high burden of clustered metabolic risk factors among T2DM patients, with hypertension and abdominal obesity representing near-universal comorbidities. The findings strongly validate the "Asian Indian Phenotype" characterized by severe central adiposity despite relatively normal Body Mass Index (BMI) values. Managing this dual epidemic necessitates a paradigm shift from glucose-centric workflows toward aggressive, multi-factorial "cardio-metabolic" care.

Keywords: Type 2 Diabetes Mellitus, Metabolic Syndrome, NCEP ATP III, Asian Indian Phenotype, Atherogenic Dyslipidemia, Hypertension.

INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) represents one of the defining global public health crises of the twenty-first century. Projections indicate that the global diabetic population will rise from 537 million in 2021 to an estimated 783 million by the year 2045. While persistent hyperglycemia characterizes the disease, clinical mortality is primarily dictated by its systemic macrovascular complications. T2DM heavily intersects with Metabolic Syndrome —a clinical constellation featuring visceral adiposity, atherogenic dyslipidemia, and arterial hypertension.

The pathophysiological link connecting T2DM and Metabolic Syndrome is best contextualized via Michael Stern's "Common Soil" hypothesis, which posits that both micro- and macrovascular diseases spring from a shared set of genetic and environmental antecedents driven by insulin resistance. Visceral fat deposition acts as a highly active, pro-inflammatory endocrine organ that releases an excess flux of free fatty acids (FFAs) and inflammatory cytokines (e.g.,IL-6, TNF-alpha). This microenvironment initiates a vicious cycle where chronic, low-grade systemic inflammation further disrupts peripheral insulin signaling cascades, driving the concomitant progression of hyperglycemia, endothelial dysfunction, and accelerated atherogenesis.

Within South Asian populations, this interaction is further compounded by the distinct "Asian Indian Phenotype". Individuals of this demographic demonstrate a "thin-fat" body composition, displaying higher visceral adiposity, severe insulin resistance, and elevated cardiovascular mortality profiles at significantly lower Body Mass Index (BMI) values compared to Western populations. Given these phenotypic unique features, monitoring simple glucose levels is insufficient to capture residual cardiovascular risks. Thus, this study seeks to systematically quantify the absolute prevalence and clinical characteristics of Metabolic Syndrome and its individual criteria within a dedicated North Indian diabetic cohort.

MATERIALS AND METHODS

This cross-sectional, hospital-based observational study was conducted at the Department of General Medicine, K.D. Medical College, Hospital & Research Center, Mathura, U.P., India. Data collection spanned from June 2024 to December 2025 following institutional ethical clearance. A total of 200 patients were selected using random sampling techniques.

- **Inclusion Criteria:** Patients aged >18 years with documented Type 2 Diabetes Mellitus (HbA1c > 6.5%) who provided valid, written informed consent.
- **Exclusion Criteria:** Patients with Type 1 Diabetes, pregnancy, or severe advanced chronic diseases (e.g., end-stage renal failure, decompensated liver disease) that could independently distort basic metabolic metrics.

RESULTS

Demographics and Baseline Characteristics

The sample size consisted of 200 patients with T2DM, precisely balanced with 100 males (50%) and 100 females (50%). The mean age of the entire cohort was 49.8 years.

Prevalence of Metabolic Syndrome and Gender Variance

The crude overall prevalence of Metabolic Syndrome within the study group was 70.0% (n = 140 out of 200). A statistically significant gender disparity was noted, with females exhibiting an absolute prevalence rate of 83.0% (n = 83) versus a rate of 57.0% (n = 57) among male counterparts. Females thus accounted for 59.3% of the total confirmed cases of Metabolic Syndrome within this trial.

Gender Profile	Total Patients evaluated (n)	Metabolic Syndrome Confirmed (n)	Metabolic Syndrome Absent (n)	Prevalence Rate within Gender (%)
Female	100	83	17	83.0%
Male	100	57	43	57.0%
Total Cohort	200	140	60	70.0%

Source: Dissertation Table 6

Distribution by Age Group Decade

Metabolic Syndrome prevalence remained consistently high across all age decadal brackets evaluated. The highest absolute concentration of cases was identified within the 50–59 age bracket (n = 40 cases, 67.8% group prevalence), closely followed by the 40–49 age bracket (n = 38 cases, 66.7% group prevalence). Together, these two middle-aged decades comprised 55.7% of the total Metabolic Syndrome cases. Alarmingly, 69.2% of the youngest diabetic cohort (aged 20–29) already met the complete clinical criteria for Metabolic Syndrome diagnosis.

Age Brackets (Years)	Total Patients in Group (n)	Metabolic Syndrome Confirmed Cases (n)	Group-Specific Prevalence (%)
20–29	13	9	69.2%
30–39	26	19	73.1%
40–49	57	38	66.7%
50–59	59	40	67.8%
60–69	29	22	75.9%
70–79	16	12	75.0%

Source: Dissertation Table 5

Prevalence of Individual Metabolic Syndrome Components

The relative distribution and statistical significance of each discrete NCEP ATP III component within the total sample size (N = 200) were assessed:

Metabolic Syndrome Criteria Component	Clinical Parameters Applied	Prevalence (n)	Overall Prevalence Percentage	Statistical Significance (p-value)
Hyperglycemia	FBS >100 mg/dL or on drugs	200	100%	0.2337 (Non-sig)
Systemic Hypertension	BP >130/85 mmHg or on drugs	166	83%	0.0001 (Highly Sig)
Abdominal Obesity	WC > 102 cms (M) / > 88 cms (F)	142	71%	< 0.0001 (Highly Sig)
Hypertriglyceridemia	TG>150mg/Dl	106	53%	0.0039 (Highly Sig)
Low HDL Cholesterol	HDL<40mg/dL (M)/ < 50 mg/dL (F)	94	47%	0.0815 (Significant)

Source: Dissertation Table 4

Detailed Component Evaluation

1. Anthropometric Patterns and the BMI Paradox

Analyzing the distribution of diagnosed Metabolic Syndrome cases across conventional Body Mass Index classifications revealed a striking paradox: 42.9% (n = 60) of all confirmed Metabolic Syndrome patients possessed a "Normal Weight" BMI score (18.5 - 24.9kg/m^2). In contrast, overweight (25.0 - 29.9kg/m^2) and obese (> 30.0kg/m^2) patients constituted 34.3% and 21.4% of the Metabolic Syndrome subset, respectively.

A comparison of mean waist circumference demonstrated extreme statistical divergence ($p = 7.90 * 10^{-9}$): Metabolic Syndrome patients exhibited a mean WC of 114.72 ± 17.52 cms compared to 98.58 ± 16.92 cms in the non-Metabolic Syndrome diabetic controls. Stratification by size revealed that 47.8% of all Metabolic Syndrome cases gathered inside the extreme ≥ 120 cms waist decade bracket, containing 54% of all female Metabolic Syndrome patients.

2. Systemic Hypertension Clustering

Hypertension was heavily clustered in patients matching the complete criteria for MetS. Among the 140 confirmed Metabolic Syndrome cases, 89.3% (n = 125) presented with concurrent hypertension. Within gender cohorts, 92.98% of male Metabolic Syndrome patients and 86.75% of female Metabolic Syndrome patients were hypertensive. The mean blood pressure parameters were significantly higher in the Metabolic Syndrome group than in non-Metabolic Syndrome cohorts:

- Mean Systolic Blood Pressure: 142.83 ± 24.25 mmHg vs. 128.45 ± 23.71 mmHg ($p = 0.0001$).
- Mean Diastolic Blood Pressure: 89.71 ± 11.94 mmHg vs. 84.30 ± 10.42 mmHg ($p = 0.0026$).

3. Lipid Profile Heterogeneity

The presence of Metabolic Syndrome was robustly tied to atherogenic lipid configurations ($p = 0.0040$). Hypertriglyceridemia (>150mg/dL) was present in 60.0% (n = 84) of the Metabolic Syndrome group compared to 36.7% (n = 22) of the non-Metabolic Syndrome group. The dominant subcategory among dyslipidemic patients was a high elevation range of 200 - 499mg/dL. Similarly, the prevalence of low gender-specific HDL-C was significantly higher in Metabolic Syndrome patients than in non-Metabolic Syndrome controls (55.71% vs. 26.71%; $p = 0.0003$).

DISCUSSION

The observed 70.0% overall prevalence of Metabolic Syndrome within this T2DM cohort highlights a high cardiorenal comorbidity burden that matches recent Indian literature describing ranges from 57% to over 80%. This cross-sectional profile reveals an extreme metabolic risk spike when compared against the baseline general South Indian urban adult prevalence of 25.8%, illustrating that established diabetes operates as both a component and a prominent accelerator of wider systemic metabolic breakdown.

The Primacy of Central Adiposity and the BMI Paradox

A key finding of this research is the dissociation between total body mass and visceral adipose accumulation. While the mean BMI profiles remained relatively uniform across the clinical groups, waist circumference showed severe statistical

divergence (114.72 cms vs. 98.58cms, $p < 0.0001$). This provides clear evidence for the "Asian Indian Phenotype" and the associated "Normal BMI Paradox". In this study, 74.5% of individuals with a technically normal BMI still met the full metabolic criteria for Metabolic Syndrome. This clinical finding underscores that standard BMI cut-offs fail to detect ectopic fat deposition within visceral, hepatic, and pancreatic beds in South Asians.

Visceral fat is uniquely dangerous because it has high baseline lipolytic activity and drains straight into the portal vein. This structural path directly floods the hepatic architecture with excess free fatty acids (FFAs), accelerating hepatic steatosis, causing systemic insulin resistance, and driving the assembly of highly atherogenic lipid configurations. Consequently, waist circumference must be prioritized as a mandatory clinical metric over standalone BMI tracking in South Asian healthcare settings.

Pathophysiological Mechanisms of Dyslipidemia and Hypertension

The statistical links connecting atherogenic lipid patterns to Metabolic syndrome cases reflect clear cellular changes driven by insulin resistance. Hypertriglyceridemia (60.0% prevalence in Metabolic Syndrome) combined with reduced HDL-C (55.7%) forms the foundational substrate for the "Atherogenic Lipid Triad". In states of insulin resistance, increased portal FFA flux stimulates hepatic overproduction of large, triglyceride-rich Very Low-Density Lipoprotein (VLDL) particles. Cholesteryl Ester Transfer Protein (CETP) then drives a bidirectional exchange, enriching both LDL and HDL structures with triglycerides in place of core cholesteryl esters. These triglyceride-heavy HDL particles undergo accelerated clear out by hepatic lipase enzymes, leaving patients with depleted protective functional systemic HDL pools and a high concentration of small, dense LDL particles (sdLDL) that easily penetrate arterial walls to accelerate plaque formation.

Similarly, the high co-prevalence of systemic hypertension (89.3% inside the Metabolic Syndrome cohort) illustrates a shared pathobiological foundation. Compensatory hyperinsulinemia activates local visceral Renin-Angiotensin-Aldosterone Systems (RAAS) and drives Sympathetic Nervous System (SNS) hyper-reactivity. This state forces increased renal tubular sodium reabsorption and chronic vasoconstriction, while systemic low-grade inflammation reduces nitric oxide bioavailability. The result is a cycle of progressive arterial stiffening and fixed systemic hypertension.

Glycemic Dissociation and Clinical Management Insights

A critical clinical finding is the near-total independence of raw Fasting Blood Sugar values from both anthropometric parameters ($r = 0.05$ with WC) and hemodynamic profiles ($r = -0.12$ with SBP). Mean fasting sugar levels were similarly high in both hypertensive and normotensive blocks. Furthermore, mean FBS values were actually higher in the non-Metabolic Syndrome group (262.2mg/dL) than in the Metabolic Syndrome group (247.0 mg/dl), despite the Metabolic Syndrome cohort displaying significantly worse dyslipidemia and higher baseline risk parameters.

This dissociation emphasizes that raw fasting hyperglycemia is largely driven by progressive pancreatic beta-cell exhaustion rather than acute adiposity metrics alone in patients with established diabetes. Clinically, this means that tracking or reducing HbA1c and sugar levels alone will not automatically normalize blood pressure or lipid profiles. Relying solely on a glucose-centric treatment plan leaves behind substantial residual macrovascular risk driven by the atherogenic lipid triad and high blood pressure.

To successfully reduce long-term mortality, healthcare providers must look beyond basic blood sugar control. Management strategies must pivot toward a multi-factorial cardio-metabolic approach, as validated by the landmark STENO-2 trial. This framework requires the early adoption of modern therapies with proven systemic benefits—such as SGLT2 inhibitors and GLP-1 receptor agonists—alongside strict blood pressure control (<130/80mmHg) and targeted lipid therapies to actively treat the entire metabolic cluster.

CONCLUSION

This research confirms a high 70.0% prevalence of Metabolic Syndrome within an established Type 2 Diabetes Mellitus cohort, driven by a strong clustering of visceral obesity and systemic hypertension. The data clearly illustrate the "Asian Indian Phenotype," where profound metabolic derangements and high cardiorenal risks manifest despite normal baseline BMI parameters.

The clear independence observed between fasting blood sugar levels and cardio-metabolic risk factors shows that glucose-focused treatment models are insufficient for comprehensive care. Type 2 diabetes should not be managed as an isolated issue of carbohydrate metabolism, but rather as part of a broader systemic collapse driven by insulin resistance and chronic low-grade inflammation.

To mitigate the rising burden of cardiovascular mortality, clinicians must transition to an integrated, patient-centric strategy. This requires early screening of waist circumference and blood pressure in every diabetic patient, combined with

aggressive, multi-factorial management targeting blood pressure, the triglyceride-HDL axis, and central obesity simultaneously.

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