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Obesity Phenotypes and Their Relationship with Visceral Adiposity and Adiponectin in an Indian Outpatient Cohort

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ABSTRACT

Background: Obesity is a heterogeneous condition with diverse metabolic profiles and fat distribution patterns. Body mass index (BMI) alone may not adequately reflect adiposity and cardiometabolic risk. Different obesity phenotypes metabolically healthy obese (MHO), metabolically unhealthy obese (MUO), metabolically obese normal weight (MONW), normal weight obese (NWO), and lipodystrophy may have varying metabolic risks. This study aimed to characterize obesity phenotypes in a North Indian cohort and assess their anthropometric, metabolic, and biochemical profiles along with associated comorbidities.

Methods: A cross-sectional study was conducted in the Department of Medicine at a tertiary care hospital between January and October 2024. A total of 100 adults aged 18–70 years with BMI ≥23 kg/m² were included. Anthropometric measurements and body composition parameters were assessed using bioimpedance analysis. Biochemical investigations included fasting glucose, insulin, lipid profile, liver and thyroid function tests, and serum adiponectin levels. Participants were classified into five phenotypes: MUO, MHO, MONW, NWO, and lipodystrophy. Statistical analysis was performed using SPSS version 25.

Results: MUO was the most prevalent phenotype (46%), followed by MHO (29%), NWO (12%), MONW (8%), and lipodystrophy (5%). MUO individuals had significantly higher visceral fat, insulin resistance, adverse lipid profiles, and the lowest adiponectin levels ($p < 0.001$). Metabolically unhealthy phenotypes showed higher prevalence of diabetes/prediabetes, hypertension, coronary artery disease, and sarcopenia ($p < 0.05$).

Conclusion: Obesity phenotypes show significant metabolic heterogeneity not captured by BMI alone. Phenotype-based assessment may improve cardiometabolic risk stratification and guide targeted management in South Asian populations.

Keywords: Obesity phenotypes, Adiponectin, Metabolic Syndrome, Visceral fat, Bio impedance analysis

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INTRODUCTION

Obesity is increasingly recognized as a condition of persistent low-grade inflammation driven by excess body fat. It has become a major global health challenge, contributing to higher rates of chronic illness and premature death.[1] Total fat in the human body is distributed into central (mainly around the trunk and upper body) and peripheral (mainly around the hips and lower body) regions.[2] Central adiposity includes both visceral fat (the fat that surrounds the intraabdominal organs) and abdominal subcutaneous fat.[2] In clinical practice, obesity is often classified using body mass index (BMI), a simple ratio of weight to height. However, BMI alone does not adequately reflect variations in fat distribution, metabolic status, or clinical outcomes. For South Asian populations, lower BMI action points are recommended, as metabolic risks occur at comparatively lower BMI levels. The WHO Expert Consultation suggests increased risk at BMI $\geq$23 kg/m² and high risk at $\geq$27.5 kg/m² in Asian populations.[3]

Recent research emphasizes that obesity is not a uniform disorder but rather a collection of distinct phenotypes. These phenotypes are shaped by metabolic health indicators such as insulin resistance and lipid abnormalities, patterns of fat deposition (visceral versus subcutaneous), inflammatory markers, and genetic influences.[4,5] Identifying these clusters provides a more precise understanding of how obesity contributes to diseases such as cardiovascular disorders, diabetes, fatty liver disease, certain cancers, musculoskeletal disorders.[6]

This study was designed to explore obesity phenotypes in a North Indian cohort, focusing on their metabolic characteristics and associated comorbidities. By examining visceral fat and adiponectin levels alongside BMI, the research aims to highlight the value of a phenotype-based approach in assessing metabolic risk.

MATERIALS AND METHODS

Study Design: This cross-sectional study was carried out in a tertiary care hospital's Department of Medicine. Data collection spanned over seven months, between January and October 2024. Ethical clearance was obtained prior to recruitment, and all participants were provided with informed consent.

Participants: A total of 100 consecutive patients were enrolled from the outpatient department. Eligibility criteria included adults aged 18-70 years with a body mass index (BMI) of at least 23 kg/m², consistent with South Asian definitions of obesity.[3] Individuals who were critically ill, in shock, septic, or on mechanical ventilation were excluded.

Study Outcomes: The primary objectives of this study were to classify obese patients according to their anthropometric, clinical, and metabolic characteristics; to determine the prevalence of distinct obesity phenotype clusters within the study population; and to explore how these phenotypes were linked to the occurrence of additional health problems, particularly cardiometabolic complications.

Sample Size: We included 100 sequential obese patients attending the Medicine Outpatient Department (OPD) in this study.

Based on the inclusion criteria, patients were enrolled in the study after obtaining informed consent. A detailed history of existing comorbidities was recorded.

Height (cm), weight (kg), waist and hip circumference (cm), waist-to-hip ratio, and skinfold thickness (mid-arm and mid-thigh) were measured using standardized protocols. BMI was computed as weight (kg)/height² (m²).

Fasting blood samples were collected for various analyses. Fasting glucose (2 Ml blood) was measured using the glucose oxidase-peroxidase method. For HbA1c, 3 mL of blood was collected in an ethylenediaminetetraacetic acid (EDTA) vacutainer and analyzed by High-Performance Liquid Chromatography. Another 3 mL of fasting blood in a red vacutainer was used for liver (LFT) and kidney function tests (KFT), lipid profile, serum fasting insulin, and thyroid function tests (TFT). For adiponectin estimation, 250 µL of serum was stored at -80°C and analyzed using a two-step sandwich ELISA with the Adiponectin ELISA kit (LOT: S3241, REF: CAN-APN-5000-10L, Expiry: 26/09/2025) from Diagnostics Biochem Canada (DBC).

Body composition assessment including Total fat mass, total skeletal muscle mass, subcutaneous fat, and visceral fat mass was measured using a bioimpedance analyzer. Participants were instructed to stand barefoot on the device while holding the handles/electrodes with their arms extended at a 90° angle to their body. Age, sex, and height were recorded before proceeding with body composition analysis, following the device's manual instructions (Omron HBF 702T Body Composition Monitor).[8] Although, Bioimpedance analyser shows reduced precision versus the reference methods, being influenced by hydration status, body geometry, and device algorithms, previous studies have shown that it may be a reliable and cheaper alternative that the gold standard investigations- CT, MRI, DEXA Scan.[8] (HOMA -IR) formula was used to calculate Insulin resistance.

Patients were then categorised into five phenotypes based on body composition, BMI and metabolic syndrome.

Definition of Obesity Phenotypes:[4]

Metabolically Unhealthy Obese (MUO) phenotype represents obese individuals with metabolic syndrome, insulin resistance, high level of proinflammatory cytokines.

Lipodystrophy Phenotype includes obese individuals with metabolic syndrome, high visceral fat and low subcutaneous fat.[4]

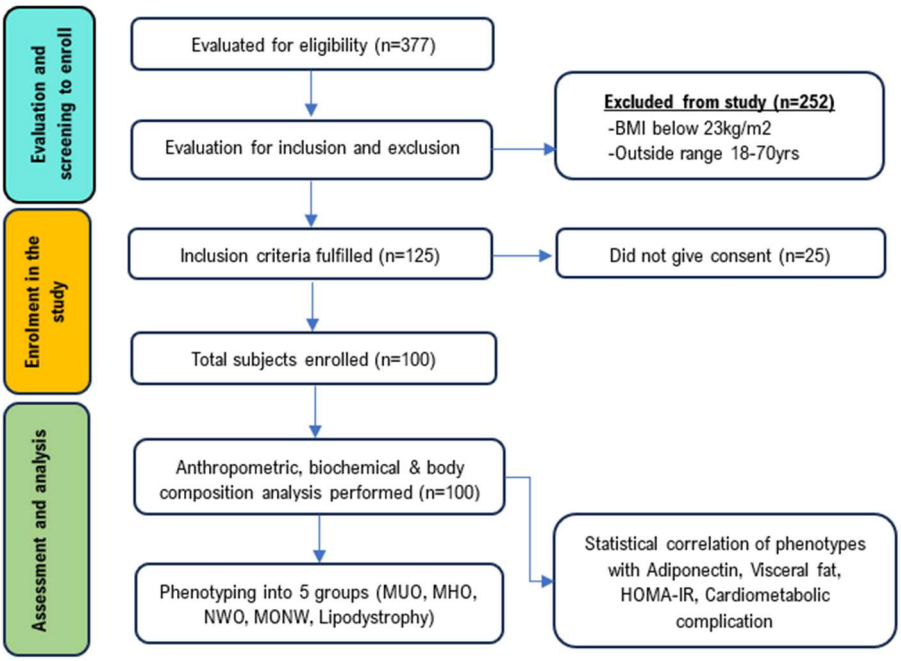
Metabolically Healthy Obese (MHO) phenotype refers to a subset of obese individuals with a healthy metabolic

profile, characterized by high insulin sensitivity, favourable lipid profile and low pro-inflammatory cytokine levels in plasma.

Metabolically Obese Normal Weight (MONW) phenotype, represent a subgroup of individuals who have normal weight with metabolic syndrome; having higher percentage of visceral adipose tissue, hyperinsulinemia and lower insulin sensitivity, dyslipidaemia and higher plasma level of pro-inflammatory cytokines and increased CVD risk.

Normal weight obese (NWO) individuals are characterized by a high fat mass despite normal BMI, lack of metabolic syndrome abnormalities. NWO individuals have shown to be in a pro-inflammatory condition with a level of cytokines intermediate to those of the non-obese and pre-obese individuals.[9]

These phenotypes were also compared in terms of clinical, metabolic, anthropometric profile, body composition and biochemical characteristics. Additionally, various comorbidities associated with obesity phenotypes were analyzed and correlated, as shown in figure 1.



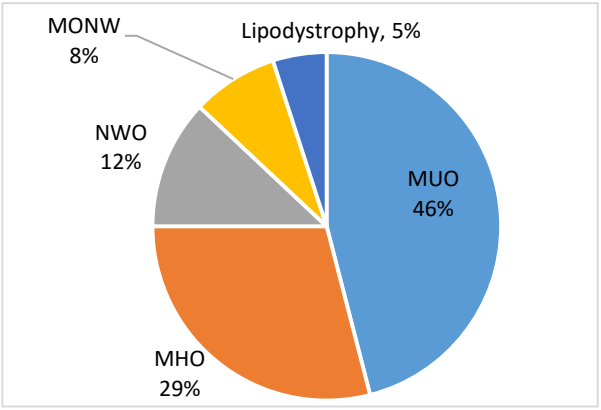
MHO: Metabolically Healthy and Obese; MONW: Metabolically Obese with Normal Weight; MUO: Metabolically Unhealthy and Obese; NWO: Normal Weight and Obesity

Figure 1: Flow diagram of study design

Statistical Analysis: Data analysis was conducted using SPSS version 25. Distribution of continuous variables was examined with Kolmogorov-Smirnov and Shapiro-Wilk tests. Parametric comparisons employed Student's t-test, while non-parametric data were analyzed using Mann-Whitney U and Kruskal-Wallis tests. Chi-square or Fisher's exact test, were used for Categorical variables. Statistical significance was set at $p < 0.05$.

RESULTS

This study cohort comprised 22% (n= 22) males and 78% (n=78) females. 51% (n=51) of the study participants were aged between 41 and 50 years, 22% (n=22) were within the 51 to 60-year age range. A further 20% (n=20) of participants were aged between 31 and 40 years, while only 7% (n=7) were in the 21 to 30-year age group. The mean±SD BMI of the study population was 28.34 ± 3.11 Kg/m². According to the South Asian BMI classification, 20% (n = 20) of participants were categorized as overweight (BMI: 23-24.9 kg/m²), while the remaining 80% (n = 80) were classified as obese (BMI ≥25 kg/m²).[3] As per the Adult Treatment Panel (ATP) III criteria, 59.0% (n=59) of the participants in this study had Metabolic Syndrome, while the remaining 41% (n=41) did not meet the criteria for Metabolic Syndrome.[10]



MHO: Metabolically Healthy and Obese; MONW: Metabolically Obese with Normal Weight; MUO: Metabolically Unhealthy and Obese; NWO: Normal Weight and Obesity

Figure 2: Prevalence of obesity phenotypes in the study population

In this study, 46.0% (n=46) of the participants were found to be MUO phenotype. 29.0% (n=29) of the participants were of MHO phenotype. 12.0%(n=12) of the participants had NWO phenotype, 8.0% (n=8) of the participants had MONW phenotype & 5.0% (n=5) of the participants were classified as Lipodystrophic as in Figure 2.

Anthropometric Parameters and body composition parameters [i.e. Total Fat Mass, Total Skeletal Muscle Mass, Subcutaneous Fat, Visceral Fat] of the five obesity phenotypic clusters have been consolidated in table 1.

Sex specific analysis of Body composition is shown in table 2.

Table 1: Anthropometric, body composition parameters of the five obesity phenotypes

	MUO (n = 46)	MHO (n = 29)	NWO (n = 12)	MONW (n = 8)	LP (n = 5)	p-value (Kruskal Wallis test)
Body composition						
TFM (%)	36.18 ± 2.71	33.27 ± 4.19	31.54 ± 5.03	32.29 ± 2.91	32.50 ± 2.93	<0.001
TSMM (%)	24.20 ± 2.56	27.17 ± 4.50	29.71 ± 5.07	24.25 ± 3.95	24.68 ± 3.14	0.001
SC Fat (%)	31.13 ± 4.73	27.53 ± 6.32	25.72 ± 5.85	22.35 ± 6.24	18.98 ± 2.52	<0.001
Visceral Fat (%)	13.92 ± 2.40	11.88 ± 2.79	10.05 ± 3.72	11.38 ± 3.28	14.16 ± 4.35	0.001
Anthropometry						
Height (Meter)	1.54 ± 0.04	1.55 ± 0.06	1.60 ± 0.08	1.51 ± 0.04	1.54 ± 0.06	0.015
Weight (Kgs)	71.37 ± 6.83	68.30 ± 5.99	62.69 ± 6.86	55.11 ± 3.25	65.82 ± 6.53	<0.001
BMI (Kg/m²)	30.13 ± 2.83	28.44 ± 1.84	24.31 ± 0.47	24.24 ± 0.62	27.60 ± 2.20	<0.001
Waist Circumference (cm)	103.84 ± 6.37	101.00 ± 3.88	93.68 ± 8.60	95.05 ± 7.62	98.48 ± 4.98	<0.001
Hip Circumference (cm)	110.05 ± 6.19	104.84 ± 6.04	97.33 ± 5.60	99.19 ± 10.53	103.04 ± 5.17	<0.001
WHR	0.94 ± 0.05	0.97 ± 0.04	0.96 ± 0.08	0.96 ± 0.07	0.96 ± 0.04	0.360
Skin Fold Thickness mid arm (mm)	29.21 ± 2.92	26.56 ± 4.76	22.86 ± 5.08	22.82 ± 5.51	21.80 ± 5.04	<0.001
Skin Fold Thickness Mid-Thigh (mm)	30.27 ± 3.38	27.42 ± 4.09	23.19 ± 5.33	24.40 ± 5.32	21.72 ± 5.88	<0.001

Abbreviation: LP: Lipodystrophy; MHO: Metabolically Healthy and Obese; MONW: Metabolically Obese with Normal Weight; MUO: Metabolically Unhealthy and Obese; NWO: Normal Weight and Obese; SC fat: Subcutaneous Fat; TFM: Total Fat Mass; TSMM: Total Skeletal Muscle Mass; WHR: Waist-to-Hip Ratio

Table 2: sex specific differences in body composition parameters

Parameters	Gender		p value
	Male (n = 22)	Female (n = 78)	
Total Fat Mass (%)	33.12 ± 3.54	34.62 ± 3.99	0.003
Total Skeletal Muscle Mass (%)	30.33 ± 4.82	24.45 ± 2.77	<0.001[1]
Subcutaneous Fat (%)	26.18 ± 5.51	28.68 ± 6.48	0.005[1]
Visceral Fat (%)	11.23 ± 3.09	13.08 ± 3.05	0.004[1]

P value in Chi-Squared Test

Table 3: Biochemical profile of the five obesity phenotypes

Biochemical and metabolic profile	MUO (n = 46)	MHO (n = 29)	NWO (n = 12)	MONW (n = 8)	LP (n = 5)	p-value (Kruskal Wallis test)
FBG (mg/dL)	91.87 ± 14.96	75.59 ± 9.26	77.67 ± 10.33	89.88 ± 9.52	98.00 ± 7.07	<0.001
S. Fasting Insulin (µIU/mL)	13.79 ± 4.15	10.35 ± 2.70	8.80 ± 3.47	11.10 ± 2.88	14.29 ± 2.54	<0.001
Urea (mg/dL)	32.95 ± 13.71	25.70 ± 7.68	22.84 ± 5.34	29.64 ± 9.07	30.90 ± 9.88	0.076
Creatinine (mg/dL)	0.96 ± 1.08	0.60 ± 0.30	0.57 ± 0.17	0.62 ± 0.23	0.91 ± 0.14	0.035
TSH (µIU/mL)	5.20 ± 2.36	4.27 ± 1.98	4.48 ± 1.50	3.58 ± 1.29	4.81 ± 2.13	0.038
HbA1c (%)	6.38 ± 1.14	5.12 ± 0.41	5.12 ± 0.46	7.11 ± 1.64	7.00 ± 1.26	<0.001
Total Cholesterol (mg/dL)	177.03 ± 37.79	131.30 ± 19.72	150.63 ± 26.02	143.54 ± 20.48	175.54 ± 38.95	<0.001
TG (mg/dL)	191.95 ± 52.42	136.48 ± 24.13	142.29 ± 19.31	183.42 ± 108.04	170.20 ± 12.93	<0.001
LDL (mg/dL)	91.18 ± 20.42	70.23 ± 8.67	77.62 ± 22.12	75.67 ± 16.91	103.59 ± 29.65	<0.001
HDL (mg/dL)	35.45 ± 6.81	50.74 ± 9.91	50.02 ± 5.42	38.89 ± 6.67	40.22 ± 3.65	<0.001
AST (mg/dL)	33.59 ± 14.12	27.00 ± 9.96	26.81 ± 10.45	30.28 ± 21.20	53.64 ± 31.30	0.094
ALT (mg/dL)	40.37 ± 17.34	30.48 ± 13.92	27.09 ± 8.06	45.38 ± 37.39	43.96 ± 24.88	0.060
ALP (mg/dL)	124.04 ± 35.19	107.33 ± 24.58	103.00 ± 18.60	109.84 ± 31.92	145.09 ± 39.51	0.087
Serum Adiponectin (ng/mL)	1.45 (0.87)	13.85 (1.63)	15.64 (0.96)	10.34 (1.03)	3.98 (2.41)	<0.001

Abbreviation: ALP: Alkaline Phosphatase; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; FBG: Fasting Blood Glucose; HDL: High-Density Lipoprotein; LDL: Low-Density Lipoprotein; LP: Lipodystrophy; MHO: Metabolically Healthy and Obese; MONW: Metabolically Obese with Normal Weight; MUO: Metabolically Unhealthy and Obese; NWO: Normal Weight and Obese; TG: Triglyceride Level

Table 4: Prevalence of Comorbidities in the five obesity phenotypic clusters

Parameters	Phenotype					p-value
	MUO (n = 46)	MHO (n = 29)	NWO (n = 12)	MONW (n = 8)	Lipodystrophy (n = 5)	
Diabetes/Pre-Diabetes (Present)	31 (67.4%)	2 (6.9%)	2 (16.7%)	7 (87.5%)	5 (100.0%)	<0.001[1]
Hypertension (present)	24 (52.2%)	6 (20.7%)	1 (8.3%)	6 (75.0%)	2 (40.0%)	0.001[1]
Hypothyroid (present)	18 (39.1%)	8 (27.6%)	4 (33.3%)	2 (25.0%)	2 (40.0%)	0.845[1]
Prediagnosed coronary artery disease (present)	8 (17.4%)	0 (0.0%)	0 (0.0%)	3 (37.5%)	1 (20.0%)	0.007[1]
Sarcopenia (Present)	41 (89.1%)	13 (44.8%)	6 (50.0%)	6 (75.0%)	4 (80.0%)	<0.001[1]

MHO: Metabolically healthy obese; MONW: metabolically obese normal weight; MUO: Metabolically unhealthy obese; NWO: normal weight obese.
[1]p-value was measured by Fisher's Exact Test.

Biochemical and metabolic parameters [serum fasting blood glucose, serum fasting insulin level, HBA1c, serum urea and creatinine, Thyroid stimulating hormone, lipid profile, liver function tests and serum adiponectin level] have been consolidated in table 3.

The prevalence of comorbidities including diabetes/prediabetes, hypertension, hypothyroidism, coronary artery disease (CAD) and sarcopenia varied across different obesity phenotypes in this study as shown in table 4.

DISCUSSION

This study provides a comprehensive phenotypic characterization of obesity in a North Indian outpatient cohort, highlighting the heterogeneity of metabolic risk that is not captured by body mass index alone. The predominance of the metabolically unhealthy obese (MUO) phenotype (46%) underscores the high cardiometabolic burden in this population and aligns with prior Indian and Asian data demonstrating a disproportionate clustering of insulin resistance, dyslipidemia, and central adiposity at comparatively lower BMI thresholds than in Western populations. [7]

The MUO phenotype in the present cohort exhibited the most adverse anthropometric and biochemical profile, including significantly higher visceral fat, insulin resistance, atherogenic lipid parameters, and the lowest serum adiponectin concentrations. These findings reinforce the concept that visceral adiposity, rather than generalized obesity, is the principal driver of metabolic dysfunction. Després and Lemieux previously demonstrated that excess intra-abdominal fat is strongly associated with impaired glucose metabolism, elevated triglycerides, and heightened cardiovascular risk independent of BMI.[11] Similarly, imaging-based studies have shown that visceral fat burden predicts metabolic syndrome across the entire BMI spectrum, supporting the notion that body composition assessment adds incremental risk stratification beyond traditional anthropometry.[12]

The lipodystrophy phenotype, though less prevalent, displayed the most pronounced visceral fat accumulation with relative subcutaneous fat deficiency and severe metabolic derangements. This pattern is consistent with the “adipose tissue expandability” hypothesis, which proposes that limited subcutaneous fat storage capacity leads to ectopic lipid deposition in visceral and non-adipose tissues, thereby exacerbating insulin resistance and lipotoxicity.[13] Clinical and mechanistic studies in both congenital and acquired lipodystrophy syndromes demonstrate extreme metabolic vulnerability, characterized by profound insulin resistance, hypertriglyceridemia, and accelerated cardiovascular disease, despite the absence of generalized obesity.[14,15]

The metabolically obese normal-weight (MONW) phenotype in this study exhibited significant metabolic impairment despite BMI values in the overweight range, emphasizing the limitations of BMI-based risk assessment. MONW individuals showed elevated fasting glucose, insulin resistance, dyslipidemia, and a higher prevalence of coronary artery disease and hypertension, corroborating earlier observations in Asian populations that cardiometabolic risk can be substantial even in individuals who do not meet conventional obesity criteria. [3,7]

In contrast, the metabolically healthy obese (MHO) phenotype demonstrated relatively preserved metabolic and lipid profiles, lower visceral fat burden, and higher adiponectin levels, despite elevated BMI and total fat mass. These findings are consistent with longitudinal data suggesting that MHO status is characterized by favorable adipose tissue function, lower inflammatory burden, and greater insulin sensitivity, that can persist for long time in some individual [16,17] Emerging evidence indicates that MHO may represent a transient state, with a substantial proportion of individuals progressing to MUO over time, particularly in the presence of increasing visceral adiposity and declining adiponectin concentrations.[18]

The normal-weight obese (NWO) phenotype in the present study exhibited the most favorable metabolic profile and the highest serum adiponectin levels, despite increased body fat percentage. This observation highlights the protective role of adiponectin in modulating insulin sensitivity, lipid metabolism, and vascular function. Experimental and clinical studies have consistently demonstrated an inverse relationship between circulating adiponectin and insulin resistance, triglyceride levels, and cardiovascular risk, independent of BMI. [19,20]

The strong association between metabolically unhealthy phenotypes (MUO, MONW, and lipodystrophy) and deranged liver enzymes and lipid profiles aligns with the

evolving concept of metabolic dysfunction-associated steatotic liver disease (MASLD).[21] Visceral adiposity and insulin resistance are central to hepatic lipid accumulation and progression to steatohepatitis, even in individuals with normal or near-normal BMI, a phenomenon increasingly recognized as “lean MASLD”.[21]

Sex-specific differences in body composition observed in this cohort, with females demonstrating higher total and visceral fat and lower skeletal muscle mass, are consistent with prior reports indicating that sex hormones, fat distribution patterns, and muscle mass contribute to differential metabolic risk profiles between men and women. [6]

From a clinical perspective, the results of this study support a paradigm shift from BMI-centered obesity assessment toward a phenotype-driven approach incorporating visceral fat estimation, metabolic profiling, and adipokine assessment. Imaging-based modalities such as CT and MRI remain the gold standard for visceral fat quantification; however, the use of bioelectrical impedance analysis in this study reflects a pragmatic and scalable approach for resource-limited settings. Prior validation studies have demonstrated acceptable reliability of commercial bioimpedance devices for population-level body composition assessment.[8]

Several limitations merit consideration. The relatively small sample size and cross-sectional design limit causal inference and generalizability. Additionally, reliance on bioimpedance rather than imaging-based modalities may introduce measurement variability. Nonetheless, the consistency of the observed associations with established mechanistic and epidemiological literature strengthens the validity of the findings.

CONCLUSION

In conclusion, this study highlights the substantial heterogeneity of metabolic risk across obesity phenotypes in an Indian outpatient population. Visceral adiposity and low serum adiponectin emerged as key determinants of cardiometabolic and hepatic risk, independent of BMI. Early identification of high-risk phenotypes such as MUO, MONW, and lipodystrophy through routine body composition and metabolic assessment may enable targeted preventive and therapeutic strategies. Future large-scale, longitudinal studies are warranted to determine whether phenotype-guided interventions can improve cardiometabolic outcomes in South Asian populations.

Individual Author’s Contribution: MM contributed to study conception, design, data collection, data analysis and interpretation, and manuscript preparation. **SG** and **AC** contributed to conception, design, data analysis and interpretation, and manuscript preparation. **SA** contributed to conception, design, and manuscript preparation. **MG** and **PA** contributed to study design, data collection, data analysis and interpretation, and manuscript preparation. **RG** contributed to data collection, data analysis and interpretation, and manuscript preparation. All authors approved the final manuscript.

Availability of data: The data that support the findings of this study are available from the corresponding author on reasonable request.

Declaration of Non-use of generative AI Tools: This article was prepared without the use of generative AI tools for content creation, analysis, or data generation. All findings and interpretations are based solely on the authors' independent work and expertise.

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