Section: General Medicine

Original Research Article

COMPARATIVE STUDY OF CLINICAL AND BIOCHEMICAL PROFILE IN TYPE 2 DIABETES MELLITUS PATIENTS OF DIFFERENT AGE GROUPS: A HOSPITAL-BASED CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a heterogeneous disease whose clinical and biochemical expression varies considerably with age. Comparative age-group data from Indian hospital settings remain limited. This study aimed to compare demographic, anthropometric, clinical, biochemical and complication profiles of T2DM patients across three age groups. **Materials and Methods:** This hospital-based, observational cross-sectional study enrolled 300 patients with T2DM diagnosed as per American Diabetes Association criteria at the Department of General Medicine, L.N. Medical College & Research Centre and J.K. Hospital, Bhopal, and stratified them into three equal age groups: Group 1 (18–40 years, n=100), Group 2 (41–65 years, n=100) and Group 3 (>65 years, n=100). Demographic, anthropometric, clinical, biochemical, complication and medication data were compared using ANOVA/Kruskal–Wallis, chi-square, and correlation analyses, with $p<0.05$ considered significant. **Result:** BMI was significantly higher in younger patients (27.6±4.5 kg/m²) and declined with age (24.8±3.6 kg/m², $p<0.001$), whereas waist–hip ratio showed the opposite trend, rising from 0.93±0.07 to 0.99±0.06 ($p<0.001$); a "normal-weight central obesity" phenotype was seen in 45% of elderly versus 5% of younger patients. Younger patients had poorer glycaemic control (HbA1c 9.1±2.0% vs 8.0±1.5%, $p=0.001$) and a more atherogenic lipid profile (TG:HDL ratio 4.93±2.07 vs 3.79±1.60, $p<0.001$). Microvascular complications (retinopathy, neuropathy, nephropathy), macrovascular disease (CAD, stroke), hypertension and renal impairment all increased significantly with age ($p<0.001$), as did insulin requirement and polypharmacy. Age correlated positively with disease duration and all microvascular complications, and negatively with HbA1c, BMI and TG:HDL ratio. **Conclusion:** T2DM demonstrates markedly age-dependent clinical and biochemical phenotypes. Younger patients carry a disproportionate metabolic and atherogenic risk burden despite shorter disease duration, while elderly patients show central (sarcopenic) obesity, progressive organ complications and greater polypharmacy. BMI alone is inadequate to capture metabolic risk in elderly diabetics, and age-specific management strategies are warranted.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterised by hyperglycaemia arising from a combination of insulin resistance and relative insulin deficiency, and accounts for nearly 90–95% of all diabetes cases worldwide.[1,2] Its global prevalence has risen sharply owing to urbanisation, lifestyle change, an ageing population and unhealthy dietary patterns, with an estimated 463 million adults affected in 2019, projected to reach 700 million by 2045.[3,4]

India, often termed the "diabetes capital of the world," harbours more than 77 million patients with diabetes, a figure expected to rise substantially in the coming decades.[4] While T2DM was traditionally a disease of older adults, its epidemiological profile has shifted markedly, with a growing burden now

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observed among younger and developing-world populations.[5,6] Age is a key determinant of disease phenotype: younger-onset T2DM is associated with aggressive metabolic dysfunction and prolonged lifetime exposure to hyperglycaemia, whereas older patients accumulate comorbidities and age-related physiological decline that shape a distinct clinical picture.[7,8]

Clinical presentation, anthropometric indices, glycaemic and lipid parameters, and the burden of microvascular and macrovascular complications may all vary substantially across age groups, yet comparative Indian hospital-based data addressing this variation remain limited.[9,10] Imbalances in biochemical parameters — glycaemic indices, lipid profile, and renal function — underlie both the diagnosis and the long-term complication burden of T2DM, and dyslipidaemia in particular is a key driver of atherosclerotic cardiovascular disease.[10,11] Diabetic kidney disease affects up to 25–40% of patients over their lifetime, and early biochemical detection of renal and cardiovascular risk is therefore of considerable clinical importance.[8,12]

Given the paucity of Indian data directly comparing demographic, clinical and biochemical parameters of T2DM across the adult age spectrum, the present study was undertaken to characterise this variation and its implications for age-specific diabetes management.

Aim and Objective

To study the variation in clinical and biochemical profile among patients with type 2 diabetes mellitus across different age groups.

MATERIALS AND METHODS

Study Design and Setting: This was a single-centre, hospital-based, observational cross-sectional study conducted in the Department of General Medicine, L.N. Medical College & Research Centre and J.K. Hospital, Bhopal, Madhya Pradesh, a tertiary care teaching hospital, over a period of 18 months (encompassing a 3-month planning phase, 12-month recruitment and data collection phase, and 3-month analysis and reporting phase). Ethical clearance was obtained from the Institutional Ethics Committee prior to initiation, and the study was conducted in accordance with the principles of the Declaration of Helsinki.

Study Participants: A total of 300 patients diagnosed with T2DM as per American Diabetes Association criteria (fasting plasma glucose ≥126 mg/dL, or 2-hour post-glucose ≥200 mg/dL on more than one occasion, with symptoms of diabetes) were recruited from the Medicine outpatient and inpatient departments using purposive sampling. Sample size was calculated (Z=1.96, estimated prevalence 10%, margin of error 4%) to yield 225 patients; allowing for 30% dropout, a final sample of 300 was recruited. Participants were equally stratified into three age groups: Group 1 (18–40 years, n=100), Group 2 (41–65 years, n=100) and Group 3 (>65 years, n=100). Patients with gestational diabetes, altered sensorium, acute myocardial infarction, renal failure, liver disease, tuberculosis, malignancy or severe infection were excluded. Written informed consent was obtained from all participants.

Clinical Assessment and Investigations: Each participant underwent a structured clinical history and examination (demographics, diabetes history, current medications, general and systemic examination including anthropometry, and assessment for diabetic complications — fundus examination, peripheral neuropathy testing, and urine protein screening). A fasting venous blood sample was analysed for fasting and postprandial blood glucose, HbA1c, complete haemogram, renal function tests, lipid profile and thyroid profile using standardised, fully automated laboratory analysers; urine albumin-creatinine ratio and 12-lead electrocardiography were also performed. Special investigations (C-peptide, anti-GAD65 antibodies, MODY panel) were undertaken where clinically indicated.

Statistical Analysis: Data were recorded on structured case-record forms, entered into Microsoft Excel 2021, and analysed using R version 4.5.2. Continuous variables were expressed as mean ± standard deviation and compared across age groups using one-way ANOVA or the Kruskal–Wallis test, with post-hoc analysis as appropriate; categorical variables were expressed as frequencies and percentages and compared using the chi-square or Fisher's exact test. Spearman's or Pearson's correlation was used to assess the relationship between age and clinical/biochemical parameters. A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

A total of 300 patients with type 2 diabetes mellitus were enrolled and categorised into three equal age groups (n=100 each).

Demographic and Anthropometric Profile

Table 1: Sociodemographic characteristics of study participants across age groups

Parameter	Group 1 (18–40 y) n=100	Group 2 (41–65 y) n=100	Group 3 (>65 y) n=100	p-value
Age (years)	33.8 ± 6.1	50.7 ± 5.9	72.9 ± 6.3	—
Male : Female ratio	62:38	59:41	55:45	0.842
BMI (kg/m²)	27.6 ± 4.5	26.1 ± 3.9	24.8 ± 3.6	<0.001
Duration of diabetes (years)	3.4 ± 2.3	7.5 ± 4.8	12.1 ± 6.5	<0.001
Urban residence, n (%)	62 (62%)	55 (55%)	48 (48%)	—

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BMI was significantly higher in younger diabetic patients and declined progressively with age (p<0.001), while disease duration increased progressively with age (p<0.001).

Table 2: Waist–hip ratio across age groups

Parameter	Group 1	Group 2	Group 3	p-value
Waist circumference (cm)	94.7 ± 9.8	92.1 ± 10.3	88.5 ± 10.4	<0.001
Hip circumference (cm)	100.3 ± 8.7	96.5 ± 8.5	89.7 ± 9.3	<0.001
Waist–hip ratio (WHR)	0.93 ± 0.07	0.96 ± 0.08	0.99 ± 0.06	<0.001
Abnormal WHR*, n (%)	67 (67%)	77 (77%)	83 (83%)	0.021

*Abnormal WHR defined as ≥0.90 in males and ≥0.85 in females (WHO criteria). Although absolute waist and hip circumferences declined with age, WHR increased progressively, indicating a shift toward central adiposity in older patients despite lower overall body mass.

Table 3: BMI versus WHR — obesity phenotype distribution

Parameter	Group 1	Group 2	Group 3	p-value
Generalised obesity (BMI ≥25), n (%)	69 (69%)	52 (52%)	38 (38%)	<0.001
Central obesity (abnormal WHR), n (%)	67 (67%)	77 (77%)	83 (83%)	0.021
Both BMI ≥25 and abnormal WHR, n (%)	62 (62%)	52 (52%)	38 (38%)	0.002
Normal BMI but abnormal WHR*, n (%)	5 (5%)	25 (25%)	45 (45%)	<0.001

*"Normal-weight central obesity" / sarcopenic obesity phenotype. This discordance indicates that BMI alone underestimates metabolically harmful adiposity in elderly diabetics, for whom WHR is a more sensitive marker of insulin-resistance-related body-fat distribution.

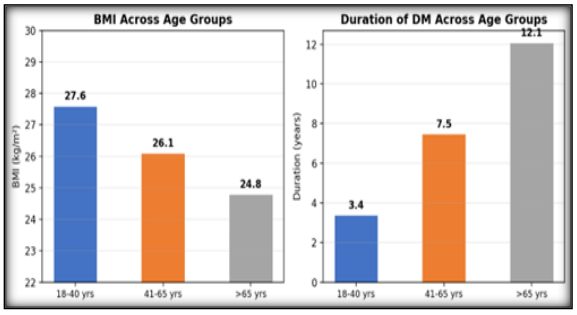

Figure 1. BMI and duration of diabetes across age groups.

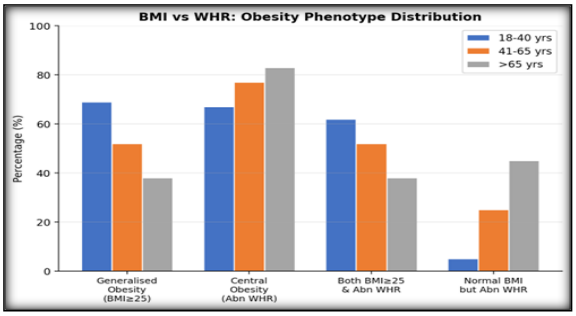

Figure 2: BMI versus WHR — obesity phenotype distribution across age groups.

Clinical Presentation

Table 4: Presenting symptoms and clinical features across age groups

Parameter	Group 1	Group 2	Group 3	p-value
Polyuria, n (%)	69 (69%)	63 (63%)	57 (57%)	0.118
Polydipsia, n (%)	65 (65%)	60 (60%)	52 (52%)	0.141
Polyphagia, n (%)	41 (41%)	34 (34%)	26 (26%)	0.064
Weight loss, n (%)	22 (22%)	30 (30%)	37 (37%)	0.047
Tingling and numbness, n (%)	13 (13%)	29 (29%)	36 (36%)	<0.001
Chest pain, n (%)	8 (8%)	22 (22%)	34 (34%)	<0.001
Obesity (BMI ≥25), n (%)	69 (69%)	52 (52%)	38 (38%)	<0.001
Acanthosis nigricans, n (%)	38 (38%)	27 (27%)	16 (16%)	<0.001

Classical osmotic symptoms declined with age (not statistically significant), whereas neuropathic symptoms and chest pain increased significantly with age. Markers of insulin resistance (obesity, acanthosis nigricans) were significantly more common in younger patients.

Biochemical Profile

Table 5: Glycaemic parameters across age groups

Parameter	Group 1	Group 2	Group 3	p-value
Fasting blood glucose (mg/dL)	169.7 ± 51.8	156.3 ± 47.6	143.5 ± 43.9	0.001
Postprandial blood glucose (mg/dL)	267.2 ± 70.4	245.6 ± 64.9	231.3 ± 59.1	<0.001
HbA1c (%)	9.1 ± 2.0	8.5 ± 1.8	8.0 ± 1.5	0.001

All glycaemic parameters were significantly higher in younger patients despite their shorter disease duration, indicating poorer glycaemic control at the extremes of youth relative to age.

Table 6: Lipid profile and TG:HDL ratio across age groups

Parameter	Group 1	Group 2	Group 3	p-value
Total cholesterol (mg/dL)	201.6 ± 43.1	206.8 ± 46.2	196.3 ± 39.5	0.094
Triglycerides (mg/dL)	186.7 ± 68.2	179.4 ± 63.7	165.2 ± 58.4	0.048
HDL cholesterol (mg/dL)	37.9 ± 8.7	40.8 ± 9.4	43.6 ± 10.1	<0.001

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LDL cholesterol (mg/dL)	121.6 ± 35.9	133.8 ± 37.5	130.6 ± 32.8	0.054
VLDL cholesterol (mg/dL)	37.7 ± 13.6	35.8 ± 12.9	32.8 ± 11.7	0.038
TG:HDL ratio	4.93 ± 2.07	4.40 ± 1.83	3.79 ± 1.60	<0.001
TG:HDL >3, n (%)	75 (75%)	68 (68%)	53 (53%)	0.002
TG:HDL >4, n (%)	59 (59%)	47 (47%)	32 (32%)	<0.001

Younger patients showed a more atherogenic lipid profile — higher triglycerides, lower HDL, and a markedly elevated TG:HDL ratio — despite a shorter duration of diabetes, indicating disproportionate long-term cardiovascular risk in this group.

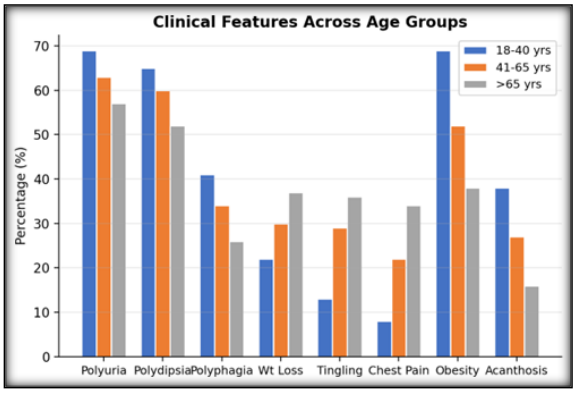

Figure 3: Clinical features across age groups.

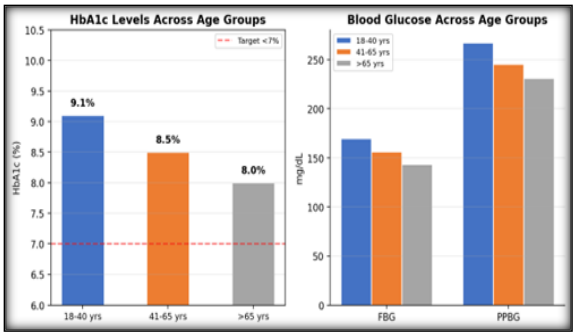

Figure 4: Glycaemic parameters across age groups.

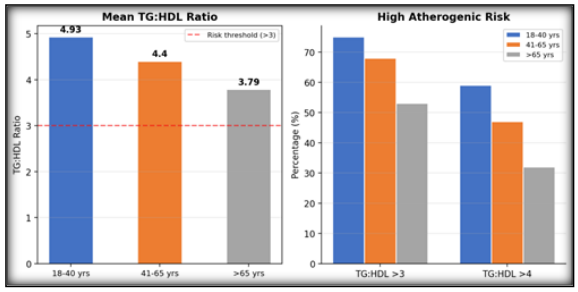

Figure 5: TG:HDL ratio and atherogenic risk across age groups.

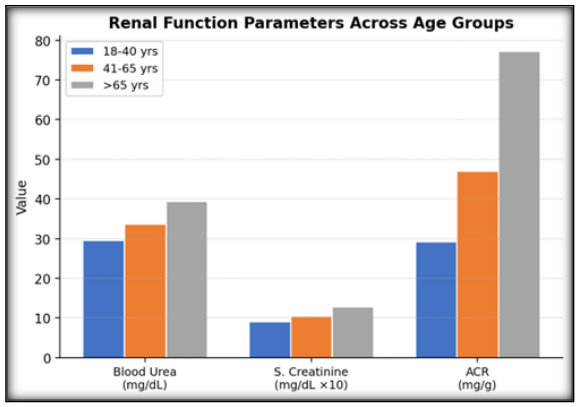

Figure 6: Renal function parameters across age groups.

Table 7: Renal function parameters across age groups

Parameter	Group 1	Group 2	Group 3	p-value
Blood urea (mg/dL)	29.6 ± 9.2	33.7 ± 10.8	39.4 ± 12.6	<0.001
Serum creatinine (mg/dL)	0.91 ± 0.26	1.05 ± 0.34	1.28 ± 0.48	<0.001
Urine ACR (mg/g)	29.3 ± 18.5	47.1 ± 30.2	77.3 ± 52.6	<0.001

Renal function showed significant age-related deterioration across all three parameters, consistent with progressive diabetic nephropathy with advancing age and disease duration.

Table 8: Haemoglobin and thyroid profile across age groups

Parameter	Group 1	Group 2	Group 3	p-value
Haemoglobin (g/dL)	13.9 ± 1.4	13.1 ± 1.6	12.3 ± 1.9	<0.001
TSH (μIU/mL)	3.1 ± 1.9	3.4 ± 2.1	3.8 ± 2.3	0.210
Thyroid dysfunction, n (%)	7 (7%)	12 (12%)	16 (16%)	0.048

Haemoglobin declined significantly with age, while thyroid dysfunction showed a significant increasing trend with age.

Diabetic Complications

Table 9: Microvascular and macrovascular complications across age groups

Parameter	Group 1	Group 2	Group 3	p-value
Retinopathy, n (%)	15 (15%)	24 (24%)	38 (38%)	<0.001
Elevated urine ACR (nephropathy), n (%)	11 (11%)	23 (23%)	38 (38%)	<0.001
Peripheral neuropathy, n (%)	13 (13%)	29 (29%)	40 (40%)	<0.001
Diabetic foot, n (%)	2 (2%)	7 (7%)	13 (13%)	<0.001
Coronary artery disease, n (%)	5 (5%)	16 (16%)	24 (24%)	<0.001
Stroke, n (%)	2 (2%)	8 (8%)	13 (13%)	<0.001

All microvascular and macrovascular complications increased significantly with age, with proliferative diabetic retinopathy observed exclusively in the elderly group.

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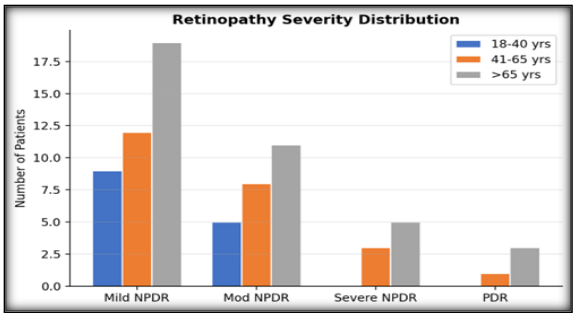

Figure 7: Microvascular complications across age groups.

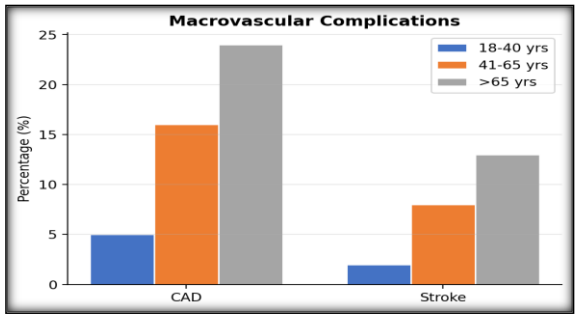

Figure 8: Macrovascular complications across age groups.

Table 10: Cardiovascular parameters across age groups

Parameter	Group 1	Group 2	Group 3	p-value
Systolic BP (mmHg)	124.7 ± 12.3	133.9 ± 14.1	142.8 ± 16.8	<0.001
Diastolic BP (mmHg)	81.6 ± 8.6	86.1 ± 9.8	88.2 ± 11.3	<0.001
Hypertension, n (%)	16 (16%)	39 (39%)	63 (63%)	<0.001
ECG abnormalities, n (%)	7 (7%)	24 (24%)	44 (44%)	<0.001

Blood pressure, hypertension prevalence and ECG abnormalities all increased significantly with age, with ischaemic ST-T changes the commonest abnormality in the elderly.

Medication Pattern

Table 11: Diabetes medication usage across age groups

Parameter	Group 1	Group 2	Group 3	p-value
Metformin, n (%)	91 (91%)	94 (94%)	90 (90%)	0.039
Sulfonylureas, n (%)	37 (37%)	45 (45%)	40 (40%)	0.008
DPP4 inhibitors, n (%)	33 (33%)	46 (46%)	52 (52%)	0.021
SGLT2 inhibitors, n (%)	22 (22%)	32 (32%)	30 (30%)	0.097
Insulin, n (%)	12 (12%)	23 (23%)	35 (35%)	<0.001
Multiple drug therapy (≥3 drugs), n (%)	22 (22%)	43 (43%)	50 (50%)	<0.001

Insulin requirement and polypharmacy increased significantly with age, reflecting progressive beta-cell decline and accumulating comorbidity in older patients.

Correlation Analysis

Table 12: Correlation between age and clinical/biochemical parameters

Parameter	Correlation coefficient (r)	p-value
Duration of diabetes	+0.742	<0.001
Fasting blood glucose	−0.248	<0.001
HbA1c	−0.312	<0.001
BMI	−0.468	<0.001
Waist–hip ratio	+0.324	<0.001
Triglycerides	−0.184	0.002
HDL cholesterol	+0.268	<0.001
TG:HDL ratio	−0.282	<0.001
Serum creatinine	+0.486	<0.001
Retinopathy	+0.528	<0.001
Neuropathy	+0.562	<0.001
Nephropathy	+0.498	<0.001

Age correlated strongly and positively with disease duration and all microvascular complications, and negatively with BMI, HbA1c and TG:HDL ratio — confirming a paradoxically more adverse glycaemic and atherogenic profile in younger patients alongside a rising organ-complication burden in older patients.

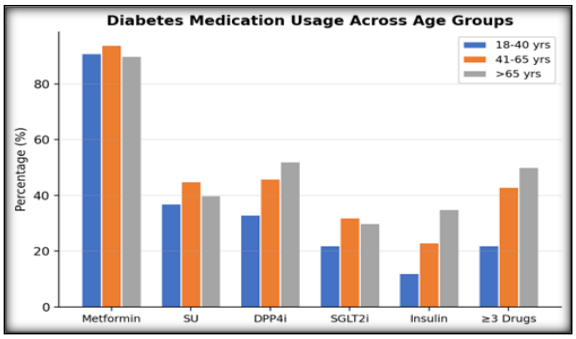

Figure 9: Diabetes medication usage across age groups.

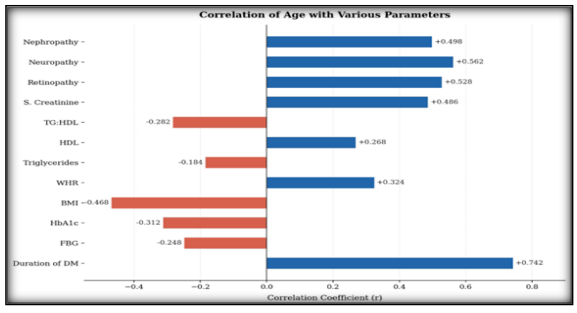

Figure 10: Correlation of age with clinical and biochemical parameters.

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DISCUSSION

This hospital-based cross-sectional study compared the clinical and biochemical profile of 300 T2DM patients across three age groups and demonstrated marked age-dependent heterogeneity in disease phenotype, spanning anthropometry, glycaemic and lipid control, organ complications, and treatment intensity.

Demographic and Anthropometric Findings: The male predominance observed across all age groups is consistent with other large Indian cohorts. Ashraf et al., in a cross-sectional study of 5,142 North Indian T2DM patients, similarly reported that younger patients (<40 years) had higher BMI and more severe dyslipidaemia than older patients.[13] The progressive rise in disease duration with age was expected given the chronic, progressive natural history of T2DM, and Chen et al., in a population-based cohort of 652,290 patients, likewise found higher HbA1c in early-onset compared with late-onset disease (median 9.3% vs 7.8%), reflecting a more aggressive metabolic phenotype in youth.[14]

BMI declined while WHR rose with advancing age, and the "normal-weight central obesity" phenotype increased from 5% to 45% between the youngest and oldest groups — indicating that BMI alone underestimates metabolically harmful adiposity in elderly diabetics. This is consistent with the well-documented Asian Indian phenotype in which insulin resistance and central adiposity, rather than generalised obesity, are the dominant drivers of metabolic risk, and with the concept of sarcopenic obesity increasingly recognised in older diabetic populations.

Clinical Presentation: Classical osmotic symptoms (polyuria, polydipsia, polyphagia) were more frequent, though not significantly so, in younger patients, while neuropathic symptoms and chest pain were significantly more common in the elderly — consistent with the established pattern of overt hyperglycaemic presentation in youth versus atypical, complication-related presentation in older age. Acanthosis nigricans, a marker of hyperinsulinaemia, was significantly more prevalent in younger patients, corroborating the dominant insulin-resistant phenotype of young-onset T2DM.

Glycaemic and Lipid Parameters: A key finding of this study was the paradox of poorer glycaemic control in younger patients despite their shorter disease duration — higher fasting and postprandial glucose and higher HbA1c compared with older patients. This mirrors the findings of Chen et al,[14] and the TODAY study,[15] which reported accelerated beta-cell decline and high rates of treatment failure in youth-onset T2DM. Possible explanations include more aggressive underlying disease biology, poorer medication adherence, greater insulin resistance, and diagnostic delay due to the assumption that diabetes predominantly affects older adults.

Younger patients also showed a more atherogenic lipid profile, with higher triglycerides, lower HDL, and a markedly elevated TG:HDL ratio — a simple, reproducible surrogate marker of insulin resistance and atherogenic risk. Three-quarters of younger patients had a TG:HDL ratio >3, compared with just over half of elderly patients, underscoring a disproportionate long-term cardiovascular risk in younger-onset T2DM despite shorter disease exposure.

Renal, Thyroid and Complication Burden: Renal function parameters (creatinine, urea, urine ACR) showed significant, progressive age-related deterioration, in keeping with the known natural history of diabetic kidney disease progressing from normoalbuminuria to overt proteinuria with cumulative hyperglycaemic exposure. Thyroid dysfunction also increased significantly with age, consistent with the recognised co-occurrence of T2DM and thyroid disorders via shared autoimmune and inflammatory pathways.

Microvascular complications — retinopathy, nephropathy and neuropathy — and macrovascular disease (coronary artery disease and stroke) all rose sharply with age, in line with large Indian screening studies such as SMART India[16] and with the TODAY study's,[15] observation that a substantial proportion of youth-onset patients nonetheless develop complications early in the disease course. Hypertension prevalence, blood pressure, and ECG abnormalities likewise increased progressively with age, reflecting cumulative vascular and structural cardiac changes.

Medication Pattern and Correlation Analysis: Insulin requirement and polypharmacy rose significantly with age, reflecting progressive beta-cell failure and accumulating comorbidity, while SGLT2 inhibitor use was relatively higher in younger patients, reflecting both their cardio-renal benefit profile and caution in prescribing to older patients with reduced renal function. Correlation analysis reinforced these patterns: age correlated strongly with disease duration and all microvascular complications, but negatively with HbA1c, BMI and TG:HDL ratio — collectively confirming a distinct "younger, metabolically aggressive" versus "older, complication-laden" dichotomy in T2DM phenotype.

CONCLUSION

- Type 2 diabetes mellitus demonstrates markedly different clinical and biochemical profiles across age groups, underscoring the heterogeneity of the disease.
- Younger patients (18–40 years) present with generalised obesity, more severe insulin-resistance markers, poorer glycaemic control, and a higher TG:HDL ratio, placing them at disproportionately elevated long-term cardiovascular risk despite shorter disease duration.

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- Elderly patients (>65 years) exhibit central (sarcopenic) obesity with normal BMI but abnormal WHR, progressive renal impairment, higher microvascular and macrovascular complication rates, and increased polypharmacy.
- BMI alone is an inadequate anthropometric measure in elderly diabetic patients; WHR should be routinely assessed to capture central adiposity and metabolic risk.
- The TG:HDL ratio is a simple, useful surrogate marker of atherogenic dyslipidaemia that is significantly elevated in younger diabetic patients and may aid early cardiovascular risk stratification.
- Age-specific management strategies are warranted: aggressive glycaemic and lipid control to prevent long-term complications in younger patients, and individualised targets emphasising safety, hypoglycaemia avoidance and comorbidity management in elderly patients.
- These findings support early screening, lifestyle intervention and risk-factor control in young-onset T2DM to mitigate the cumulative burden of complications over a longer lifetime.

Limitations

- This was a single-centre study conducted at one tertiary care hospital, which may limit generalisability.
- The cross-sectional design precludes causal inference; prospective longitudinal studies are needed to confirm these findings.
- Hospital-based sampling may introduce selection bias relative to the broader community diabetic population.
- Surrogate markers of insulin resistance (WHR, acanthosis nigricans, TG:HDL ratio) were used in place of HOMA-IR, fasting insulin or C-peptide.
- A single HbA1c measurement may not fully capture long-term glycaemic variability, and age- and sex-matched non-diabetic controls were not included.
- Potential confounders such as diet, physical activity, occupational exposure and medication adherence were not extensively evaluated, and advanced cardiac and autonomic investigations were not performed.

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