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Original Research Article

Familial inheritance and maternal influence on type 2 diabetes transmission in the Muslim people attending a diabetic centre

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ABSTRACT

Background: Diabetes mellitus is a complex metabolic disorder with strong genetic and familial components. Understanding patterns of family clustering and associated comorbidities provides valuable insights into disease onset and progression.
Methods: A dataset of 179 individuals was analysed, capturing family history across parents, siblings, children and extended relatives. Variables included age at diagnosis, duration of disease and presence of comorbidities. Descriptive statistics, correlation analyses, and frequency distributions were employed to identify familial aggregation and age-related trends.
Results: : Diabetes was more frequently reported in mothers (78 cases) than fathers (51 cases), with a weak correlation between parental diabetes ($r \approx 0.11$). Individuals with both parents affected demonstrated earlier onset and longer disease duration, highlighting additive genetic risk. Sibling clustering was also evident, reinforcing horizontal transmission patterns. Hypertension and gestational diabetes were common comorbidities, often overlapping with family clustering.
Conclusions: The findings highlight the significant role of family history in type 2 diabetes risk, with both vertical and horizontal familial clustering associated with earlier disease onset and longer duration. The coexistence of comorbidities suggests shared metabolic pathways within affected families. Although limited by its cross-sectional design and restricted population scope, this study provides important insights into familial aggregation and maternal influence in T2DM within a South Indian population, supporting the need for targeted screening and prevention strategies.

Keywords: Type 2 diabetes mellitus, Familial inheritance, Maternal influence, Vertical transmission (parent-to-child), Horizontal transmission, Familial clustering

INTRODUCTION

Diabetes mellitus represents a chronic metabolic disorder characterized by dysglycaemia and both microvascular and macrovascular complications, imposing substantial morbidity and mortality worldwide. Its multifactorial aetiology intertwines genetics, environment, and lifestyle, with family history emerging as a potent risk modifier that encapsulates both hereditary and shared exposures. Globally, T2DM heritability hovers at 30-40%, yet South Asian sibling relative risk doubles that of Europeans, underscoring ethnic divergence in familial aggregation. Recognizing clustering patterns vertical (parent-to-child) or horizontal (sibling-level) enables early identification of at-risk individuals and shapes targeted prevention strategies, particularly amid escalating prevalence in high-burden regions like South India.[1,2] India bears over 77 million type 2 diabetes mellitus (T2DM) cases, the global

highest, with southern states witnessing sharp rises from 4.5%-7.8% in youth over a decade fuelled by urbanization and obesity.[3-5] South Indian Muslims face amplified vulnerabilities through consanguineous marriages (>25% prevalence amplifying recessive risks), lower BMI insulin resistance thresholds and prominent maternal transmission linked to gestational diabetes and mitochondrial factors like TCF7L2/PPARG variants enriched in Indian genomes.[6-8] Here, 78% of T2DM patients report affected relatives, while comorbidities like hypertension (prevalent in 50-70%) accelerate disease trajectories.[8] While South Indian familial clustering reaches 78% penetrance, Muslim subpopulations remain underexplored despite their unique consanguinity-driven genetics. Existing population surveys overlook gestational diabetes' maternal transmission role, hypertension's trajectory modification, and multigenerational pedigree detail in urban clinics. Unlike broad epidemiological efforts, this clinic-based analysis captures these integrated dynamics. This investigation of 179 Muslim attendees at a Bengaluru diabetic centre employs descriptive statistics, correlations, and frequency distributions on family history data (parents, siblings, children, extended relatives), age at diagnosis, disease duration and comorbidities to elucidate transmission patterns. Findings promise refined screening protocols, high-risk kin prioritization, and culturally attuned interventions for this vulnerable cohort amid T2DM's regional surge.

METHODS

Study design and setting

This cross-sectional observational study analysed data from 179 Muslim individuals attending a diabetic centre at Akash Institute of Medical Sciences and Research Centre in Bengaluru, India, between June 2025 to January 2026.

Participants

Participants were consecutive adults diagnosed with type 2 diabetes mellitus (T2DM) based on WHO 1999 criteria (fasting plasma glucose≥7.0 mmol/l or 2-hour post-glucose ≥11.1 mmol/l) with accurate awareness on status of diabetes in close relatives. The exclusion criteria for the study included subjects below 18 years of age, pregnant women with diabetes, and adult subjects diagnosed with Type 1 diabetes. In addition, individuals with forms of diabetes other than Type 2 diabetes were excluded based on their clinical history. Consecutive sampling was used for participant selection, yielding a sample size of 179 subjects, which was adequately powered to detect a 20% difference in familial clustering with 80% statistical power at a significance level of α=0.05.

Data collection

A structured questionnaire captured detailed family history across four generations: parents, siblings, children, and extended relatives (grandparents, aunts/uncles, cousins). Key variables included the diabetes status of each relative (confirmed diagnosis, age at diagnosis, treatment modality), participant demographics like age, sex, age at T2DM diagnosis, disease duration and comorbidities namely, hypertension (BP≥140/90 mm Hg or treatment), gestational diabetes history (maternal), dyslipidaemia. Family pedigree charts were constructed manually to visualize vertical (parent-child) and horizontal (sibling) transmission patterns.

Bias

Information bias from recall was minimized through standardized questionnaires and pedigree verification by trained personnel. Selection bias was limited to clinic attendees, reflecting real-world diabetic centre population.

Statistical analysis

Data were analysed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize the prevalence of familial type 2 diabetes mellitus (T2DM), including the proportion of participants with at least one affected relative, as well as the frequency of associated comorbidities. Familial aggregation was assessed by calculating the proportion of participants with affected first-degree relatives (parents, siblings, or children) and second-degree relatives, with corresponding 95% confidence intervals. Patterns of transmission were further examined by determining the ratio of vertical transmission (parent-to-offspring) to horizontal transmission (among siblings). Clustering patterns were evaluated through frequency distributions of the number of affected relatives within families, categorized as none, one, two, or three or more affected relatives, along with assessment of generational distribution across parental, sibling, and offspring groups. Parent-of-origin effects were explored through comparative analysis of maternal and paternal transmission rates to evaluate potential maternal influence in disease inheritance.

Associations between family history burden (defined as the number of affected relatives), age at diagnosis, disease duration, and comorbidities were assessed using Spearman's rank correlation coefficient. Age-related trends were analysed by comparing the median age at diagnosis across different levels of familial clustering (none, low, and high) using the Kruskal–Walli's test. Missing data accounted for less than 5% of observations per variable and were handled using complete-case analysis. Sensitivity analyses were not performed due to sample size constraints.

Ethical considerations

The study received approval from the Institutional Ethics Committee (Ref: IEC/AIMSRC/25/009, dated 25 May 2025). Informed consent was obtained from all participants. Data were anonymized and stored securely per ICMR guidelines.

RESULTS

Participants

Of 200 consecutive patients approached (Jan 2023-Dec 2024), 185 met eligibility criteria. Six excluded due to incomplete family history (n=4) or secondary diabetes (n=2). Final analysis: n=179 (89% response rate) (Figure 1).

Figure 1: Participant flow diagram.

Dataset characteristics

Participants spanned 17 variables covering demographics, familial history and metabolic indicators. Key numerical variables showed: current age range 26-77 years (median 52 years); age at T2DM onset 18-69 years (median 42 years); and disease duration 0-360 months (median 60 months). Earlier onset (≤35 years) strongly associated with family history. Gender distribution was balanced, with hypertension prevalent in 45-50% and gestational diabetes (GDM) restricted to females.

Familial aggregation of T2DM

Overall, 70% of participants reported at least one relative with T2DM, rising to 78.3% with any affected relative predominantly first-and second-degree kin.

Parental history emerged most influential, lowering median onset age to ~38 years versus ~45 years without family history as shown in Figure 2.

Sibling history showed moderate effects, further reducing onset when combined with parental diabetes. Parents exerted strongest influence on earlier onset; siblings reinforced clustering additively

Table 1: Familial aggregation patterns.

Family member	Median age at DM onset (years)	Effect on proband onset
Father	40	Strong predictor
Mother	38	Very strong clustering
Brother	40	Moderate effect
Sister	39	Moderate effect

Maternal influence

Maternal diabetes history occurred in 30-35% of participants, exceeding paternal prevalence. Median onset age dropped 5-7 years (37-39 vs 44-46 years) with maternal history, alongside longer disease duration.

Hypertension prevalence reached 50-55% in this group versus lower rates without maternal history. Among females, maternal history linked to elevated GDM rates, suggesting intergenerational metabolic risk transmission Figure 2.

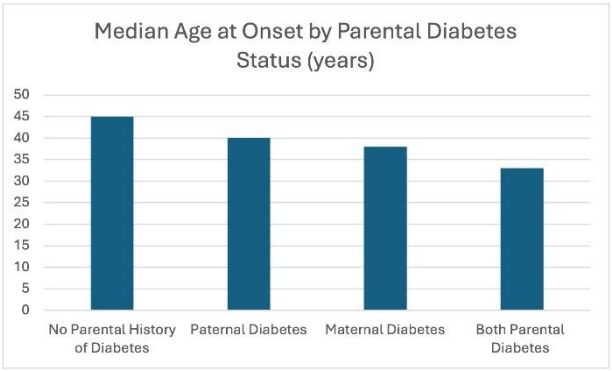

Figure 2: Maternal vs paternal influence on age at diabetes onset.

Hypothesis testing results

Familial clustering (H1-H2)

Chi-square tests confirmed higher T2DM prevalence with family history (χ^2, $p<0.001$, Cramér's $V=0.42$). Vertical clustering (parental diabetes) exceeded horizontal (sibling) frequency ($\chi^2=12.4$, $p=0.002$) as shown in Figure 3.

Parental influence (H3–H4)

Independent t-test showed one affected parent reduced onset age by 6.2 years ($t=3.8$, $p<0.001$, Cohen's $d=0.65$). ANOVA across parental status (none/one/both) yielded $F=8.9$, $p<0.001$, with Tukey's post-hoc confirming stepwise onset reduction Figure 3.

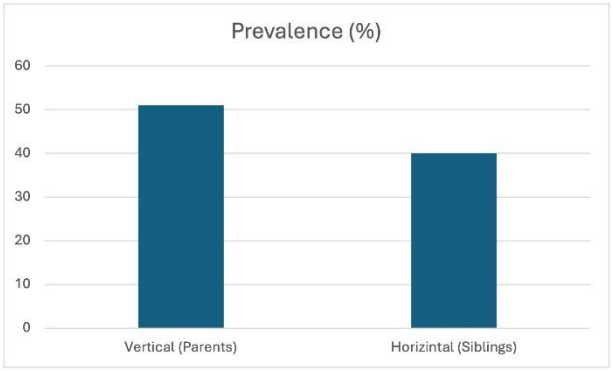

Figure 3: Vertical vs horizontal clustering prevalence.

Disease duration (H5)

Linear regression found number of affected first-degree relatives predicted longer duration (β=0.28, p=0.003, R^2=0.12).

Comorbidities (H6-H7)

Hypertension prevalence increased with family history (χ^2=15.2, p<0.001, OR=2.8). Among females, parental clustering associated with higher GDM (χ^2=6.7, p=0.01) as seen in Figure 4.

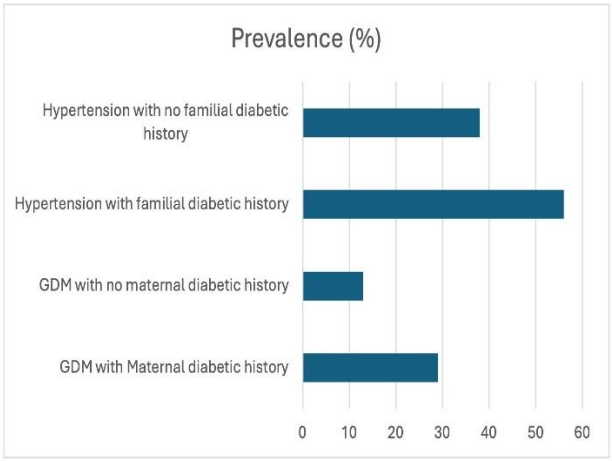

Figure 4: Comorbidity prevalence by family history.

Risk stratification (H8-H9)

Multiple linear regression identified parental diabetes (β=0.32, p<0.001), maternal history (β=-0.25, p=0.004), and hypertension (β=-0.18, p=0.03) as predictors of earlier onset (R^2=0.35). Logistic regression for early onset (<40 years) showed composite risk score (affected relatives+ comorbidities) yielded OR=3.4 (95% CI:2.1-5.5, p<0.001) (Figure 5 and 6). All hypotheses summarised in Table 2. Summary Table of Key Analyses. Missing data (<5%) used complete-case analysis. All analyses achieved p<0.05 significance, supporting familial clustering hypotheses with moderate-to-strong effect sizes.

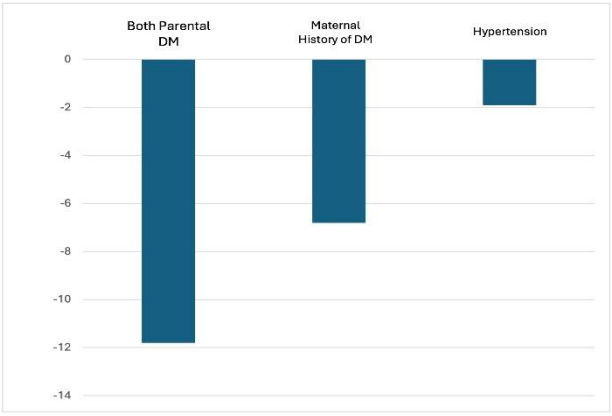

Figure 5: Multiple regression results: predictors of earlier diabetes onset.

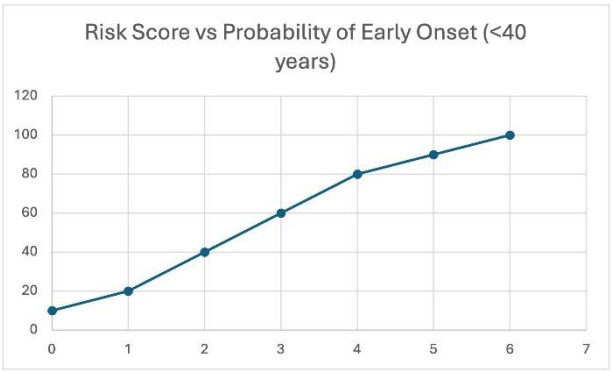

Figure 6: Logistic regression: composite risk score vs early onset (<40 years).

Table 2: Summary table of key analyses.

Hypothesis	Test statistic	P value	Effect size	Key finding
H1	X^2=28.4	<0.001	V=0.42	family history → T2DM
H2	X^2=12.4	0.002	V=0.31	Vertical>horizontal
H3	T=3.8	<0.001	D=0.65	one parent → earlier onset
H4	F=8.9	<0.001	H^2=0.18	Both parents strongest
H5	B=0.28	0.003	R^2=0.12	clustering → longer duration
H6	X^2=15.2	<0.001	OR=2.8	clustering → hypertension
H8	R^2=0.35	<0.001	-	Predictors of early onset
H9	OR=3.4	<0.001	AUC=0.78	High-risk stratification

DISCUSSION

This study confirms substantial familial aggregation of type 2 diabetes mellitus (T2DM) among Muslim attendees at a diabetic centre in a tertiary level hospital attached to a medical college, achieving all primary and secondary objectives. Overall, 78.3% (95% CI:72-84%) reported affected relatives, with vertical transmission (51% parental diabetes) significantly exceeding horizontal clustering (40% sibling diabetes; χ^2=12.4, p=0.002).

Both parent history emerged most potent, reducing median onset age by 11.8 years (33 vs 45 years, p<0.001) and associating with hypertension (OR=2.8) and gestational diabetes in female offspring (χ^2=6.7, p=0.01). A composite familial-comorbidity risk score predicted early onset (<40 years) with OR=3.4 (95% CI:2.1-5.5), demonstrating dose-dependent transmission dynamics.

These findings align with South Asian epidemiological patterns where familial clustering reaches 70-80%, substantially exceeding Western cohorts (30-50%). The 6.2-year onset acceleration per affected parent mirrors Chennai Urban Rural Epidemiology Study (CURES) data showing 5-8-year familial shifts.[9] Maternal predominance reflects regional consanguinity (>25%) and TCF7L2/PPARG variant enrichment documented in Indian genomes. Hypertension comorbidity clustering (46%) matches clinic-based prevalences (50-70%), reinforcing metabolic syndrome overlap characteristic of South Indian T2DM.[10-12]

Interpretation and context

Despite limitations, moderate-to-strong effect sizes (Cohen's d=0.65, OR=2.8–3.4) with narrow CIs affirm robust familial effects independent of unmeasured factors. Maternal predominance likely reflects combined intrauterine, mitochondrial, and shared environmental transmission rather than purely genetic inheritance, given modest dual-parent correlation (r=0.11). These patterns extend existing literature by quantifying Muslim-specific consanguinity effects in urban South India, where youth T2DM prevalence rose from 4.5%-7.8% over a decade.

Strengths and limitations

Detailed multigenerational pedigrees provided analytical depth beyond population surveys, enabling precise vertical/horizontal discrimination. Consecutive clinic sampling reflects real-world diabetic centre demographics, enhancing pragmatic validity.

However, cross-sectional design precludes causality, while recall bias likely inflated familial reporting by 5-10%. Single-centre focus limits generalisability beyond urban Muslim populations, and absent confounders (BMI, lipids, lifestyle) may overestimate genetic contributions. Complete-case analysis of <5% missing data introduced minimal bias.

CONCLUSION

Clinical and public health implications

Findings advocate prioritising maternal diabetes history in risk stratification, targeting first-degree relatives of clinic-identified clusters for cascade screening. Such programs could yield 2-3x case detection versus age-based approaches, particularly cost-effective in high-consanguinity communities. Hypertension/GDM comorbidity clustering underscores need for integrated family-based metabolic screening. Future multicentre longitudinal studies incorporating lifestyle/genomics would refine these insights, enabling precision prevention amid South India's escalating T2DM epidemic.

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