

A multicentre survey of liver fibrosis burden in type 2 diabetes in India: the DiaFib-Liver Study



Ashish Kumar,^{a,*} Jayant K. Panda,^b Prabhat Agrawal,^c Sunil Gupta,^d Dinesh Kumar,^e Mohammad S. Kuchay,^f Jothydev Kesavadev,^g Viswanathan Mohan,^h Aravinda Jagadeesha,ⁱ Bharat Saboo,^j Ajoy Tewari,^k Kunal Das,^l Kumar Prafull Chandra,^m Shriram V. Kulkarni,ⁿ Shubhashree Patil,^o Amit Gupta,^p Parikshit Goswami,^q Dhruvi Hasnani,^r Vipul Chavda,^s Mayur Agrawal,^t Gaurav Chhaya,^u Chakkarai Muralidharan,^v Aarathy Kannan,^w Parag Rana,^x Ashu Rastogi,^y Sangram Biradar,^z Khemeswar Agasti,^{aa} Prasanth Sankar,^{ab} L. Sreenivasa Murthy,^{ac} Deepak Suvarna,^{ad} Rohit Kapoor,^{ae} Kiran Shah,^{af} Monica Gupta,^{ag} Sandeep Suri,^{ah} Anubha Srivastava,^{ai} Debmalya Sanyal,^{aj} Seema Bagri,^{ak} Vitull K. Gupta,^{al} Alpna Sowani,^{am} Purvi Chawla,^{an} Manish H. S.,^{ao} Niles M. Banthia,^{ap} Faraz Farishta,^{aq} Shivam Kapoor,^{ar} Syed Haider M. Husaini,^{as} Praveen Sharma,^{at} Anil Arora,^{au} and Anoop Misra^{av}

^aSir Ganga Ram Hospital, Rajinder Nagar, New Delhi, India

^bWellbeing Clinic, Cuttack, Odisha, India

^cAgra Diabetes Research Center, Agra, Uttar Pradesh, India

^dSunil's Diabetes Care n' Research Centre, Nagpur, Maharashtra, India

^eHarsha Clinic and Diabetes Centre, Lucknow, Uttar Pradesh, India

^fMedanta the Medicity, Gurugram, Haryana, India

^gJothydev's Diabetes Research Centre, Thiruvananthapuram, Kerala, India

^hMadras Diabetes Research Foundation (ICMR- Collaborating Centre of Excellence) and Dr. Mohan's Diabetes Specialties Centre (IDF Centre of Excellence in Diabetes Care), Chennai, Tamil Nadu, India

ⁱDr Aravind's Diabetes Centre, Bengaluru, Karnataka, India

^jPrayas Diabetes Center, Indore, Madhya Pradesh, India

^kHind Institute of Medical Sciences, Ataria, Sitapur, Uttar Pradesh, India

^lYashoda Super Speciality Hospital Kaushambi, Ghaziabad, Uttar Pradesh, India

^mChandra Diabetes & Heart Clinic, Lucknow, Uttar Pradesh, India

ⁿM. G. Medical College, Panvel District Raigad, Maharashtra, India

^oDiabetes & Wellness Clinic, Mumbai, Maharashtra, India

^pCentre for Diabetes Care, Noida, Uttar Pradesh, India

^qAyush Diabetes Clinic, Himmatnagar, Gujarat, India

^rRudraksha Institute of Medical Sciences, Ahmedabad, Gujarat, India

^sHormone India Diabetes and Endocrine Centre, Bhopal, Madhya Pradesh, India

^tShivam Medicare Clinic, Ahmedabad, Gujarat, India

^uVijaya Clinic Diabetes Care Centre, Dindigul, Tamil Nadu, India

^vSundaram Arulrhaj Hospital, Thoothukudi, Tamil Nadu, India

^wDev Medical Hospital, Vadodara, Gujarat, India

^xPGIMER, Chandigarh, India

^ySB Medical Centre, Kalaburagi, Karnataka, India

^zSCB Medical College and Hospital, Cuttack, Odisha, India

^{aa}MGM Muthoot Hospitals, Pathanamthitta, Kerala, India

^{ab}Lifecare Hospital and Research Center, Bengaluru, Karnataka, India

^{ac}SS Hospital, Mysuru, Karnataka, India

^{ad}Carewell Heart and Super Speciality Hospital, Amritsar, Punjab, India

*Corresponding author. Institute of Liver, Gastroenterology and Pancreaticobiliary Sciences, Ganga Ram Postgraduate Institute of Medical Education and Research (GRIPMER), Sir Ganga Ram Hospital, Rajinder Nagar, New Delhi, 110 060, India.

E-mail addresses: ashishk10@yahoo.com (A. Kumar), drjayantpanda@gmail.com (J.K. Panda), prabhatagrawal321@gmail.com (P. Agrawal), drsunilgupta@gmail.com (S. Gupta), dineshharshaclinic@gmail.com (D. Kumar), drshafikuchay@gmail.com (M.S. Kuchay), jothydev@gmail.com (J. Kesavadev), drmohans@diabetes.ind.in (V. Mohan), arvi03@yahoo.com (A. Jagadeesha), drbharatsaboo@gmail.com (B. Saboo), docajoy@yahoo.co.in (A. Tewari), drkunaladas@yahoo.com (K. Das), drkpchandra@gmail.com (K.P. Chandra), dr.svk88@gmail.com (S.V. Kulkarni), drshubhashreepatil@gmail.com (S. Patil), dramitaol@gmail.com (A. Gupta), parixit.goswami@gmail.com (P. Goswami), dhruvi.hasnani@gmail.com (D. Hasnani), drvpchavda@gmail.com (V. Chavda), mayuragrawal2006@gmail.com (M. Agrawal), gaurav.chhaya2010@icloud.com (G. Chhaya), drcm60@gmail.com (C. Muralidharan), draarathykannan@gmail.com (A. Kannan), askdrparag@gmail.com (P. Rana), ashuendo@gmail.com (A. Rastogi), drsangramb@gmail.com (S. Biradar), windowsagasti@gmail.com (K. Agasti), muthootdiabcare@gmail.com (P. Sankar), drlsm@lrcr.in (L.S. Murthy), drdeepaksuvarna@gmail.com (D. Suvarna), rohit_kapoor_rohit@yahoo.co.in (R. Kapoor), theconsultingroom03@gmail.com (K. Shah), monicag4@yahoo.com (M. Gupta), drsandeepsuri@rediffmail.com (S. Suri), drvarma_anubha@yahoo.co.in (A. Srivastava), drdebmalysanyal@gmail.com (D. Sanyal), sbagri2000@yahoo.com (S. Bagri), vitullgupta2000@yahoo.com (V.K. Gupta), diabetespecialitycentre@gmail.com (A. Sowani), linadiabetes@gmail.com (P. Chawla), drmanishhs@gmail.com (M.H. S.), banthianilesh76@yahoo.co.in (N.M. Banthia), drfarazfarishta@gmail.com (F. Farishta), shivam.kapoor75@gmail.com (S. Kapoor), dr.haiderhusaini2k@gmail.com (S.H.M. Husaini), drpraveen_sharma@yahoo.com (P. Sharma), dranilarora50@gmail.com (A. Arora), anoopmisra@gmail.com (A. Misra).

^{ae}The Consulting Room, Mumbai, Maharashtra, India
^{af}Samvedna Hospital, Varanasi, Uttar Pradesh, India
^{ag}Ever Healthy Hospital Diabetes Care Education and Research Center, Hisar, Haryana, India
^{ah}MLN Medical College, Prayagraj, Uttar Pradesh, India
^{ai}KPC Medical College, Jadavpur, Kolkata, West Bengal, India
^{aj}Bagri Diabetes Care Centre, Mumbai, Maharashtra, India
^{ak}Kishori Ram Hospital and Diabetes Care Centre, Bathinda, Punjab, India
^{al}Diabetes Speciality Centre, Mumbai, Maharashtra, India
^{am}Lina Diabetes Care Center, Mumbai, Maharashtra, India
^{an}Vinay Pediatric and Multispecialty Hospital, Hassan, Karnataka, India
^{ao}Neo Clinic and Echo Centre, Panvel, Maharashtra, India
^{ap}F S Endocrinology & Diabetic Center, Hyderabad, Telangana, India
^{aq}JN Medical College, AMU, Aligarh, Uttar Pradesh, India
^{ar}Fortis C-DOC Centre of Excellence for Diabetes, Metabolic Diseases and Endocrinology, New Delhi, India

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Summary

Background Type 2 diabetes (T2D) is closely linked to metabolic dysfunction–associated steatotic liver disease (MASLD). However, the burden of clinically significant and advanced liver fibrosis, including cirrhosis, from community-based Indian populations remains poorly defined, with most prior studies limited by small size, referral bias, or single-centre design.

Methods The DiaFib-Liver Study was a cross-sectional investigation conducted between January and July 2024 across multiple centres in India. Consecutive adults with T2D, asymptomatic for liver disease, who had undergone vibration-controlled transient elastography (VCTE) within the preceding six months were enrolled by diabetologists and endocrinologists across India. Patients with competing liver diseases or hepatology referrals were excluded. Liver stiffness measurement (LSM) thresholds defined clinically significant fibrosis (≥ 8.0 kPa), advanced fibrosis (≥ 10.0 kPa), and probable cirrhosis (≥ 15.0 kPa). Hepatic steatosis was assessed using the controlled attenuation parameter (CAP), with CAP ≥ 248 dB/m used to define steatosis. Predictors of clinically significant fibrosis were assessed with multivariable logistic regression.

Findings A total of 9202 adults with T2D were included (mean age 53.3 years [SD 11.8]; 61% [5617/9202] male). Overall, 26% (2433/9202) had clinically significant fibrosis, 14% (1289/9202) advanced fibrosis, and 5% (491/9202) had LSM values consistent with probable cirrhosis (≥ 15.0 kPa). Among participants with CAP data, 65% (5289/8136) had CAP-defined hepatic steatosis (CAP ≥ 248 dB/m). Importantly, 13% (370/2847) of patients without steatosis (CAP < 248 dB/m) already had clinically significant fibrosis, including 4% (107/2847) with probable cirrhosis (LSM ≥ 15.0 kPa). Independent predictors of clinically significant fibrosis included obesity (OR 1.98, 95% CI 1.80–2.19), dyslipidaemia (OR 1.21, 95% CI 1.10–1.34), reduced eGFR (OR 1.23, 95% CI 1.02–1.49), and diabetes duration ≥ 10 years (OR 1.12, 95% CI 1.00–1.24). Regional variation was evident, with prevalence ranging from 21% (256/1241) in central to 30% (643/2126) in southern India. Among non-obese patients (BMI < 25), 19% (752/3996) had clinically significant fibrosis, with age as the only independent predictor.

Interpretation One in four adults with T2D in India has clinically significant liver fibrosis and one in twenty already has probable cirrhosis based on elastography thresholds, establishing advanced liver disease as a “fourth major complication” of diabetes. Fibrosis—not steatosis—should be the focus of systematic assessment in diabetes care. These findings highlight the urgent need to integrate fibrosis screening into national diabetes programs. Prospective outcome studies and cost-effectiveness analyses are warranted to inform strategies for MASLD in T2D.

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Keywords: Type 2 diabetes; MASLD (Metabolic dysfunction–associated steatotic liver disease); MAFLD (Metabolic dysfunction–associated fatty liver disease); NAFLD (Nonalcoholic fatty liver disease); MASH (Metabolic dysfunction–associated steatohepatitis); Liver fibrosis; Hepatic steatosis; Transient elastography (FibroScan®); VCTE

Research in context

Evidence before this study

We searched PubMed, Embase, and Google Scholar for studies published from Jan 1, 2000, to March 31, 2024, using the terms “non-alcoholic fatty liver disease”, “metabolic dysfunction-associated steatotic liver disease”, “type 2 diabetes”, “fibrosis”, “elastography”, “India”, and “Asia”, without language restrictions. We reviewed relevant meta-analyses, multicentre studies, and guideline documents. Multiple studies have established type 2 diabetes (T2D) as the strongest risk factor for progressive MASLD. However, most Indian data are derived from small, single-centre, hospital-based cohorts, often enriched for hepatology referrals, with reported prevalence of advanced fibrosis ranging from 20% to 40%. Large community-based datasets such as the MAP study provided insights into MASLD prevalence but were limited by retrospective design and referral bias. No prior nationally representative, community-level study has systematically evaluated the burden of liver fibrosis in Indian patients with T2D.

Added value of this study

The DiaFib-Liver Study is, to our knowledge, the largest real-world survey of liver fibrosis in T2D from any low- or middle-income country. More than 9000 adults with T2D were enrolled across diverse regions of India, with hepatology

referrals systematically excluded and quality control applied. We found that one in four patients had clinically significant fibrosis and one in twenty had probable cirrhosis based on elastography thresholds, most often undiagnosed in community practice. Importantly, clinically significant fibrosis was identified even in non-obese individuals and in participants without steatosis, consistent with the well-recognized phenomenon of declining hepatic fat content with advancing fibrosis and underscoring that hepatic steatosis and fibrotic burden may be dissociated in T2D. These findings establish advanced liver disease as a “fourth major complication” of diabetes in India.

Implications of all the available evidence

Together with existing international data, our findings highlight the urgent need to move beyond steatosis and systematically integrate fibrosis assessment into routine diabetes care, particularly in high-burden countries such as India. Non-invasive tools such as vibration-controlled transient elastography (VCTE) can be deployed in community practice to identify high-risk patients earlier. Prospective outcome studies and cost-effectiveness analyses are needed to inform screening strategies, refine cut-offs for South Asian populations, and evaluate therapeutic interventions.

Introduction

Type 2 diabetes mellitus (T2D) affects over 500 million adults worldwide and is projected to exceed 780 million by 2045, with the sharpest increases occurring in low- and middle-income countries (LMICs).¹ According to the ICMR–INDIAB study, India currently has 101 million people with diabetes and 136 million with prediabetes, and the number of diabetes cases alone is projected to surpass 150 million by mid-century, positioning the country as the epicentre of the global diabetes epidemic.^{2,3} The incidence of new-onset T2D is high, and diabetes also tends to occur at younger ages.⁴ While microvascular (retinopathy, nephropathy, neuropathy) and macrovascular complications remain central to diabetes management, chronic liver disease has emerged as a critical yet under-recognized comorbidity.

Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly termed non-alcoholic fatty liver disease (NAFLD),⁵ is now the most common cause of chronic liver disease worldwide, with T2D as its strongest risk factor.^{6–9} Obesity, a major driver of both T2D and MASLD,^{10,11} amplifies risk and, according to the revised Indian Obesity Commission framework, the presence of obesity-related comorbidities—including MASLD—qualifies individuals as having stage 2 obesity, a category for which intensified management is

recommended.¹⁰ Patients with T2D not only have a higher prevalence of MASLD but also experience faster progression to advanced fibrosis and cirrhosis—the key determinants of liver-related morbidity and mortality. Recent global meta-analyses estimate that 15–20% of patients with T2D harbour advanced fibrosis,^{12–14} with even higher prevalence in biopsy-based cohorts.¹⁵ However, liver fibrosis often evolves insidiously in individuals with T2D, driven by the interplay of metabolic dysfunction, adiposity, insulin resistance, and vascular injury, and frequently remains clinically silent until advanced stages—rendering it an under-recognized complication in routine diabetes care.

Despite the scale of India’s diabetes burden, population-level data on liver fibrosis in patients with T2D are limited. Prior studies from India have been small, hospital-based, or enriched for hepatology referrals, reporting fibrosis prevalence estimates ranging from 20% to 40%.^{16,17} Large datasets such as the MAP study¹⁸ have provided important insights into MASLD prevalence but are constrained by retrospective design and potential selection bias. Consequently, the true burden of advanced fibrosis in routine, community-based diabetes practice remains unknown.

We therefore conducted the DiaFib-Liver Study, an investigator-initiated, real-world, cross-sectional survey of adults with T2D across India. The study aimed to (i)

establish the prevalence of clinically significant and advanced fibrosis in Indian patients with T2D, (ii) identify clinical predictors of fibrosis, including within non-obese and comorbidity-free subgroups of individuals with T2D, and (iii) explore the relationship between hepatic steatosis and fibrosis in this population.

Methods

The DiaFib-Liver Study was led by the Diabetes and Liver Interest Group of Diabetes India and designed as a nationwide, cross-sectional study using routinely collected clinical data, with a mixed prospective and retrospective data capture approach. Consecutive adults with T2D were enrolled across community diabetes clinics between January and July 2024, with hepatology referrals systematically excluded.

Data was collected in real-world practice settings using a structured, anonymized Google Form. Treating physicians entered data either prospectively at the point of care or retrospectively from recent clinical records, provided that vibration-controlled transient elastography (VCTE) had been performed as part of routine clinical evaluation within the preceding six months.

VCTE was performed at the discretion of the treating physician as part of routine diabetes care and not for research purposes. Typical indications included long-standing T2D, obesity, metabolic syndrome, abnormal liver enzymes, or cardiometabolic risk stratification in diabetes clinics. Importantly, VCTE was not triggered by clinical suspicion of advanced liver disease or decompensation, and all included participants were asymptomatic for liver disease at the time of assessment. Referral to a gastroenterologist or hepatologist was not required for VCTE.

Between January and July 2024, a structured anonymized Google Form was disseminated to physicians, diabetologists, and endocrinologists across India through institutional and professional networks. Participating clinicians were instructed to enter data for consecutive adult patients (aged ≥ 18 years) with T2D, who were asymptomatic for liver disease and had undergone VCTE as part of routine clinical care within the preceding six months.

Patients were not directly approached or contacted for research purposes, and no additional investigations were performed specifically for the study. Clinical, laboratory, and elastography data were recorded by treating physicians either prospectively during routine clinic visits or retrospectively from recent medical records.

Exclusion criteria included competing aetiologies—significant alcohol intake (defined as >30 g/day for men and >20 g/day for women),⁵ chronic hepatitis B or C infection—as well as jaundice (bilirubin ≥ 5 mg/dL), pregnancy, heart failure, valvular heart disease,

maintenance haemodialysis, and known active malignancy involving the liver or metastatic liver disease.

Absence of competing liver disease aetiologies was determined by the treating physician using a combination of documented clinical diagnosis, medical history, and available laboratory data from routine care. Chronic viral hepatitis was excluded based on prior documented diagnosis and/or available serological testing in the medical record. Alcohol intake was assessed using routine clinical history obtained during standard outpatient care and quantified according to predefined thresholds. Use of known steatogenic medications (including systemic corticosteroids, amiodarone, tamoxifen, methotrexate, and valproate) was not systematically captured in this real-world dataset and therefore was not applied as a formal exclusion criterion. Contributors were specifically instructed not to submit data from patients meeting any exclusion criteria.

Both private practitioners and hospital-based clinicians were eligible to participate. To minimise referral and spectrum bias, data contributed from hepatologists, gastroenterologists, and physicians with a predominantly gastrointestinal practice were excluded, because patients referred for such practices are more likely to have suspected liver disease, abnormal liver tests, or advanced fibrosis, even when asymptomatic, which could inflate fibrosis prevalence estimates in a community diabetes cohort. In routine diabetes practice in India, VCTE is commonly performed in asymptomatic patients for risk stratification, including long-standing diabetes, obesity, metabolic syndrome, mildly abnormal liver enzymes, or as part of opportunistic metabolic screening. Importantly, elastography was not performed in response to clinical features of advanced liver disease, and patients evaluated in hepatology or gastroenterology practices were excluded.

All submitted records were initially included, and standardized thresholds were applied to identify potential quality concerns. Submissions with implausibly high rates of probable cirrhosis (e.g., $>20\%$ of patients with LSM ≥ 15 kPa) were flagged for review and adjudicated by the study team. This process ensured exclusion of data sources at risk of referral bias or quality concerns, yielding a robust final cohort.

The electronic case report form captured demographics (age, sex), BMI categories, diabetes duration categories, and major cardiometabolic comorbidities, including hypertension, dyslipidaemia, CAD, hypothyroidism, CKD (eGFR <60). LSM and CAP values, as measured by VCTE, were entered by contributors in accordance with standard procedures. Socioeconomic variables (such as education, income, occupation, or indices of social deprivation) were not captured in this real-world dataset.

Comorbid conditions were ascertained by the treating physician using a combination of documented

clinical diagnosis, current or prior use of disease-specific medications, and available laboratory or imaging data from routine clinical care. Hypertension and dyslipidaemia were defined by a recorded physician diagnosis and/or ongoing use of antihypertensive or lipid-lowering therapy. Coronary artery disease was defined by a prior documented diagnosis, history of myocardial infarction or coronary revascularization, or ongoing use of antiplatelet or anti-ischemic therapy. Chronic kidney disease was defined by a documented diagnosis and/or reduced eGFR (<60 mL/min per 1.73 m²) on available laboratory testing. Hypothyroidism was defined by a documented diagnosis and/or ongoing thyroid hormone replacement therapy.

No additional investigations were performed specifically for the study, and all comorbidities were recorded as part of routine clinical care.

Operational definitions are as follows:

- Fibrosis thresholds (LSM):
 - Clinically significant fibrosis: ≥ 8.0 kPa⁸
 - Advanced fibrosis: ≥ 10.0 kPa
 - Probable cirrhosis: ≥ 15.0 kPa
 - Exploratory thresholds: ≥ 20.0 kPa (proxy for clinically significant portal hypertension) and ≥ 25.0 kPa (proxy for high-risk varices), consistent with prior studies.¹⁹
- MASLD:

Defined according to the international Delphi consensus as the presence of hepatic steatosis detected by any validated modality (imaging, histology, or biomarkers) in conjunction with at least one cardiometabolic risk factor.⁵ In this study, CAP measurements were available only at the time of elastography and were used to describe CAP-defined steatosis severity and its relationship with fibrosis, but not to define MASLD status.
- Steatosis thresholds (CAP)²⁰:
 - S0: <248 dB/m
 - S1: 248 – 267 dB/m
 - S2: 268 – 279 dB/m
 - S3: ≥ 280 dB/m

CAP categories were used to describe CAP-defined steatosis severity and its relationship with fibrosis; CAP <248 dB/m was not interpreted as absence of MASLD, because hepatic fat content may be underestimated by CAP, particularly in individuals with advanced fibrosis or “burnt-out” steatohepatitis, and because steatosis may have been documented by ultrasound or other modalities not captured in the study dataset.
- Other definitions:
 - Overweight: BMI 23.0 – 24.9 kg/m² (Asian Indian-specific cut-offs)
 - Obesity: BMI ≥ 25 kg/m² (Asian Indian-specific cut-offs)
 - CKD: eGFR <60 mL/min/ 1.73 m²²¹

- Diabetes duration: <1 year, 1 – 5 years, 5 – 10 years, 10 – 20 years, >20 years

The planned study size was informed by a priori prevalence-based calculation to allow region-wise analyses. Assuming an expected prevalence of clinically significant liver fibrosis of approximately 15% among adults with T2D, a precision of 5%, and a 95% confidence level, a target sample size of approximately 9000 participants was estimated. To account for exclusions and incomplete data, enrolment was extended, and the final analytic cohort comprised 9202 participants.

Data integrity was maintained through built-in validation checks in the Google Form. All entries were de-identified at source, and no patient-identifiable information was collected.

Statistical analysis

Continuous variables were summarized as means (SD) or medians (IQR), as appropriate, and categorical variables as counts and percentages. Between-group comparisons were made using the independent-sample t test, Mann–Whitney U test, or chi-square test, as appropriate. The chi-square test for trend was used to evaluate linear associations across ordered categories.

Univariable analyses were first performed to identify factors associated with clinically significant fibrosis. Variables showing an association at $p < 0.10$ in univariable analysis were subsequently entered simultaneously into a multivariable logistic regression model using a forced-entry (enter) approach. No stepwise or automated variable selection procedures were used at the multivariable stage. Adjusted odds ratios (OR) with 95% confidence intervals (CI) were reported. Pre-specified subgroup analyses were performed to examine fibrosis prevalence and predictors across key clinical strata, including obesity status, comorbidity burden, CAP-defined steatosis categories, and geographic regions; formal interaction testing was not undertaken. Subgroup analyses in non-obese participants, metabolically healthy non-obese participants, and those without CAP-detected steatosis were undertaken as sensitivity analyses to assess robustness of findings. Analyses were conducted using complete-case data; no imputation was performed. Subgroup analyses involving CAP were restricted to participants with available CAP measurements. No sampling weights were applied, as this was a clinic-based, real-world cohort rather than a population-probability sample. Statistical significance was defined as a two-sided $p < 0.05$. Analyses were conducted using SPSS version 30.0 (IBM Corp., Armonk, NY, USA).

Ethics statement

The study protocol was reviewed and approved by the Royal Pune Independent Ethics Committee, Pune, India (approval number: RPIEC160424). Given the

exclusive use of anonymized, routinely collected clinical data, absence of direct patient contact, and non-interventional design, individual informed consent was not required. No patient underwent VCTE specifically for research purposes; only data from clinically indicated examinations were analysed. The study complied with the Declaration of Helsinki (2013 revision)²² and prespecified authorship and acknowledgment criteria. The full study protocol and the ethics committee approval letter are provided in the [Supplementary Appendix](#).

Role of the funding source

No funding was received for present work.

Results

Between January 29 and July 31, 2024, a total of 10,739 patient records were submitted. To minimise referral bias, entries from two gastroenterologists (n = 270 and 92) and two physicians (n = 1106 and 52) were excluded, as were 17 patients younger than 18 years. The final

analysis included 9202 adults with T2D ([Fig. 1](#)). Baseline characteristics of participants stratified by Fibrosis status are shown in [Table 1](#).

Overall, 26% (2433/9202) had clinically significant liver fibrosis (LSM ≥ 8 kPa), 14% (1289/9202) had advanced fibrosis (LSM ≥ 10 kPa), and 5% (491/9202) met thresholds consistent with probable cirrhosis (LSM ≥ 15.0 kPa) ([Figs. 2 and 3](#)).

Patients with clinically significant fibrosis, compared with those without, were slightly older (mean 54.1 [SD 11.4] vs 53.0 [SD 11.9] years, $p < 0.001$), had longer diabetes duration (≥ 10 years: 36% [886/2433] vs 31% [2081/6769], $p < 0.001$), and were more often obese (69% [1681/2433] vs 52% [3525/6769], $p < 0.001$) ([Table 1](#)). Comorbidities were also more prevalent, including hypertension (55% [1338/2433] vs 49% [3307/6769]), dyslipidaemia (55% [1349/2433] vs 47% [3208/6769]), coronary artery disease (9% [222/2433] vs 7% [456/6769]), and chronic kidney disease (8% [183/2433] vs 6% [378/6769]), whereas hypothyroidism did not differ (19% in both groups [452/2433 vs 1283/6769]). On univariable logistic regression, clinically significant

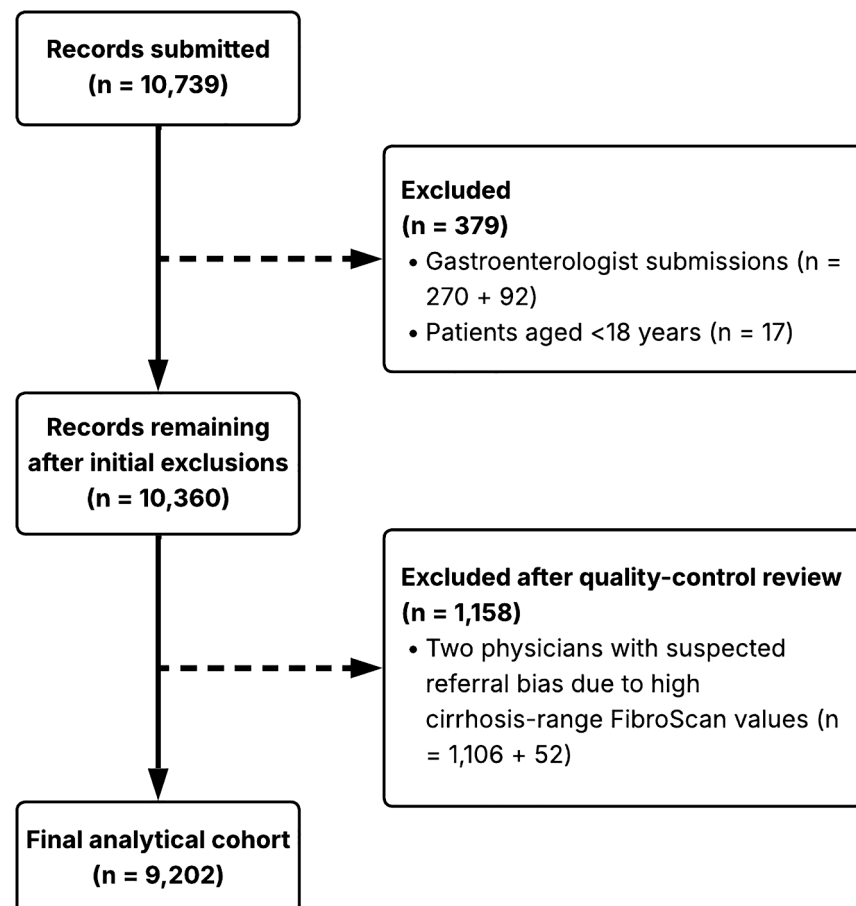


Fig. 1: Flow of participants through the DiaFib-Liver Study.

Variable	Overall (n = 9202)	LSM <8 kPa (n = 6769)	LSM ≥8 kPa (n = 2433)	p value
Mean age (SD), years	53.3 (SD 11.8)	53.0 (SD 11.9)	54.1 (SD 11.4)	<0.0001
Sex, n (%)				0.13
Male	5617 (61%)	4100 (61%)	1517 (62%)	
Female	3585 (39%)	2669 (39%)	916 (38%)	
Duration of diabetes, n (%)				<0.0001
Less than 1 year	1087 (12%)	850 (13%)	237 (10%)	
1 year–5 years	2314 (25%)	1742 (26%)	572 (24%)	
5 years–10 years	2834 (31%)	2096 (31%)	738 (30%)	
10 years–20 years	2241 (24%)	1599 (24%)	642 (26%)	
More than 20 years	726 (8%)	482 (7%)	244 (10%)	
Body mass index, n (%)				<0.0001
<18.5 kg/m ²	158 (2%)	134 (2%)	24 (1%)	
18.5–22.9 kg/m ²	1449 (16%)	1189 (18%)	260 (11%)	
23.0–24.9 kg/m ²	2389 (26%)	1921 (28%)	468 (19%)	
25.0–29.9 kg/m ²	3552 (39%)	2501 (37%)	1051 (43%)	
>30 kg/m ²	1654 (18%)	1024 (15%)	630 (26%)	
Hypertension, n (%)	4645 (50%)	3307 (49%)	1338 (55%)	<0.0001
Dyslipidemia, n (%)	4557 (50%)	3208 (47%)	1349 (55%)	<0.0001
Coronary artery disease, n (%)	678 (7%)	456 (7%)	222 (9%)	0.0001
eGFR <60, n (%)	561 (6%)	378 (6%)	183 (8%)	0.0006
Hypothyroidism, n (%)	1735 (19%)	1283 (19%)	452 (19%)	0.68
Median LSM (IQR), kPa	6.2 (4.9–8.1)	5.5 (4.6–6.5)	10.1 (8.8–13.6)	<0.0001
Median CAP (IQR), dB/m, (n = 8136)	269 (229–303)	260 (220–295)	291 (261–323)	<0.0001

Table 1: Baseline characteristics of the study population, overall and stratified by clinically significant liver fibrosis (LSM ≥8 kPa).

fibrosis was associated with age, diabetes duration of at least ten years, obesity, hypertension, dyslipidaemia, and coronary artery disease (all $p \leq 0.0001$), and with

reduced eGFR ($p = 0.0006$). Male sex ($p = 0.12$) and hypothyroidism ($p = 0.68$) were not significant. In multivariable analysis, independent predictors for

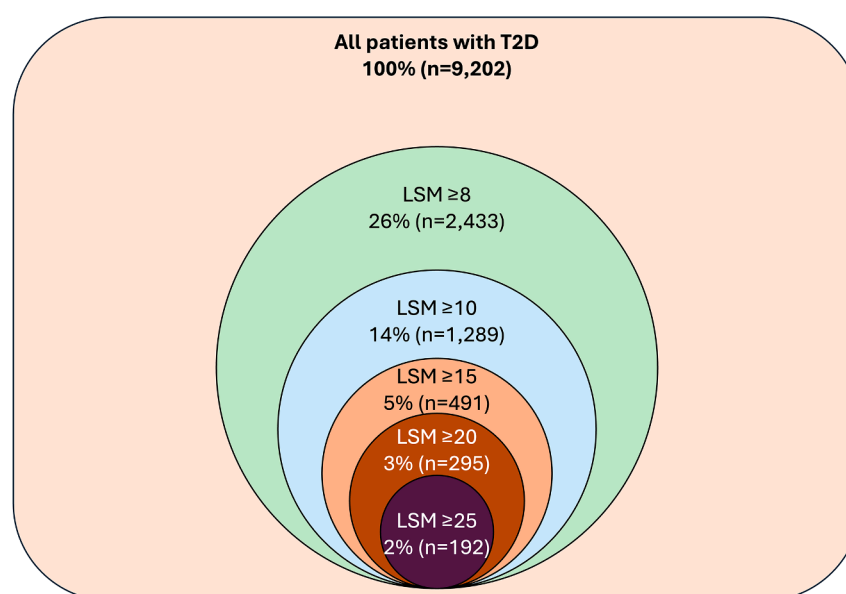


Fig. 2: Prevalence of liver fibrosis stages in patients with T2DM. Venn diagram illustrating the stepwise prevalence of clinically significant liver fibrosis (LSM ≥8 kPa), advanced fibrosis (LSM ≥10 kPa), probable cirrhosis (LSM ≥15 kPa), likely CSPH (LSM ≥20 kPa), and high-risk varices (LSM ≥25 kPa) in the cohort (n = 9202).

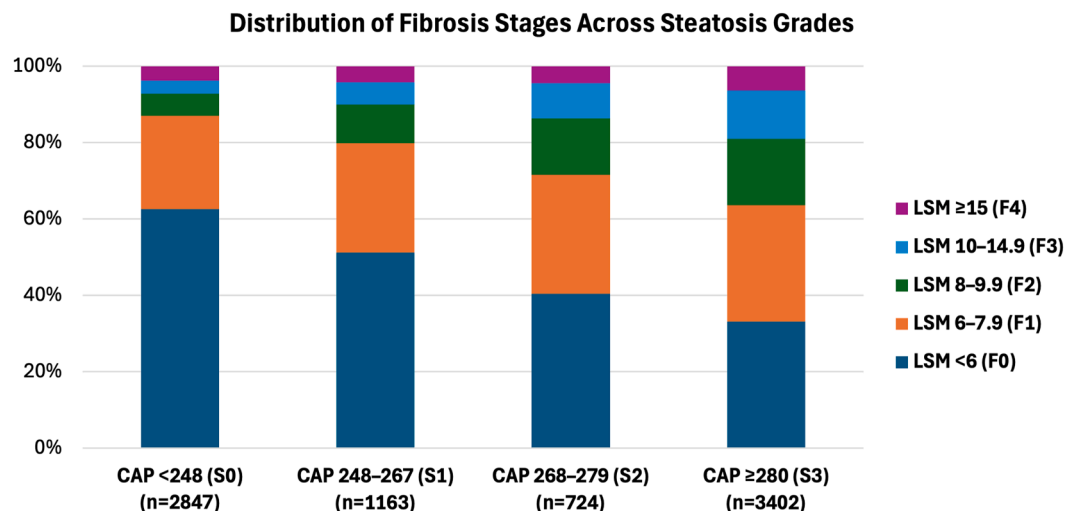


Fig. 3: Distribution of liver stiffness measurement (LSM)-defined fibrosis stages (F0–F4) across controlled attenuation parameter (CAP)-defined steatosis grades (S0–S3) in participants with available CAP data (n = 8136). CAP-defined steatosis categories were S0 <248 dB/m, S1 248–267 dB/m, S2 268–279 dB/m, and S3 ≥280 dB/m.

clinically significant fibrosis were diabetes duration of at least ten years (OR 1.115, 95% CI 1.001–1.243; $p = 0.048$), obesity (OR 1.984, 95% CI 1.796–2.191; $p < 0.0001$), dyslipidaemia (OR 1.212, 95% CI 1.098–1.338; $p < 0.0001$), and reduced eGFR (OR 1.233, 95% CI 1.022–1.489; $p = 0.029$), while age, sex, hypertension, hypothyroidism, and coronary artery disease were not independently associated. Full results are provided in Table 2.

Marked variation in prevalence of fibrosis was observed between centres located in different areas of India. Further details are included in supplementary folder (Supplementary Figure S1).

Further, fibrosis prevalence differed across clinical subgroups (Supplementary Figure S2).

In multivariable analysis restricted to participants with CAP <248 dB/m, age, diabetes duration, and obesity remained independent predictors of fibrosis (Supplementary Table S3).

Further details about the cohort are available in the Supplementary Files.

Discussion

In this multi-centre study from India of more than 9000 adults with T2D, we found that one in four had clinically significant liver fibrosis on transient elastography, one in seven had advanced fibrosis, and one in twenty already met thresholds consistent with probable cirrhosis—despite being asymptomatic and recruited outside hepatology practices. The findings demonstrate a high burden of advanced liver disease among patients with T2D in India, carrying profound implications for clinical care, health policy, and global diabetes management. This also emphasizes that all physicians should actively screen for and appropriately investigate MASLD in every person with T2D and consider treatment allocation accordingly.⁸

	Univariable analysis			Multivariable analysis		
	Odds ratio	95% CI	p value	Odds ratio	95% CI	p value
Age	1.008	1.004–1.012	<0.0001	1.003	0.998–1.007	0.23
Male sex	1.078	0.980–1.186	0.12			
≥10 y of T2D	1.290	1.170–1.422	<0.0001	1.115	1.001–1.243	0.048
Obesity (BMI ≥25)	2.057	1.865–2.270	<0.0001	1.984	1.796–2.191	<0.0001
Hypertension	1.279	1.166–1.404	<0.0001	1.086	0.981–1.201	0.11
Dyslipidemia	1.381	1.259–1.516	<0.0001	1.212	1.098–1.338	0.0001
Hypothyroidism	0.976	0.866–1.099	0.68			
CAD	1.390	1.176–1.644	0.0001	1.188	0.996–1.416	0.055
eGFR <60	1.375	1.145–1.651	0.0006	1.233	1.022–1.489	0.029

Table 2: Univariable and multivariable logistic regression analyses of factors independently associated with clinically significant liver fibrosis (LSM ≥8 kPa).

The observed prevalence of clinically significant fibrosis (26% [2433/9202]) in this cohort is high. Prior Indian reports—often small or specialty-based—have shown wide variability, and even large datasets have been constrained by retrospective design or potential referral enrichment.^{16–18} Against this backdrop, the present study provides a pragmatic, cross-sectional, quality-assured benchmark for India. By systematically excluding hepatology referrals and outlier centres and mandating consecutive patient entry from routine diabetes clinics, our design minimizes selection bias and enhances external validity, making the results directly translatable into day-to-day diabetes care pathways.

Our multivariable analyses highlight that the key independent predictors of clinically significant fibrosis were obesity, dyslipidaemia, reduced renal function, and long duration of diabetes. However, the nuanced role of diabetes duration deserves emphasis. Although longer diabetes duration was independently associated with fibrosis in the full cohort, this effect disappeared in patients without metabolic or vascular comorbidities. In the comorbidity-free subgroup, age was the sole determinant of fibrosis risk. This finding suggests that diabetes per se is not sufficient to drive fibrosis in the absence of other metabolic insults, and that clustering of risk factors—rather than glycemia alone—propels progression to advanced disease.

Our findings suggest that liver fibrosis in patients with T2D arises through two overlapping but distinct pathways. In non-obese patients with T2D, especially those without metabolic comorbidities, age emerges as the key determinant of fibrosis, likely reflecting cumulative glycaemic injury, hepatocyte senescence, and age-related vulnerability, independent of adiposity. Even when non-obese patients have additional comorbidities, age remains the independent predictor, underscoring its primacy in “fat-light” diabetes. In contrast, when the overall cohort is considered—including obese patients—obesity, dyslipidaemia, and chronic kidney disease dominate as independent predictors, while age itself loses statistical weight, reflecting mediation through these metabolic stressors. Diabetes duration also becomes relevant in this broader context, capturing the chronicity of metabolic insults. The strong correlation between steatosis severity and fibrosis supports a lipotoxicity-driven pathway, yet the presence of fibrosis in patients with minimal steatosis (S0) highlights an additional “injury-first” pathway, where age, duration of diabetes, and obesity independently drive fibrogenesis in the absence of overt fat accumulation. Together, these data support a model in which diabetes and age confer a baseline fibrogenic risk, which is markedly amplified by obesity and metabolic comorbidities, while steatosis accelerates fibrosis progression but is not a prerequisite for its development.

A notable feature of our study is the regional variation in fibrosis prevalence, with data from centers in

southern and eastern parts of India showing the highest rates and central and western parts of India the lowest. Such heterogeneity needs further exploration and may reflect a confluence of dietary patterns, adiposity profiles, physical activity, and timing of diabetes detection and management across regions. Differences in healthcare access and practice patterns may also contribute.

From a public health standpoint, these findings may highlight the need for locally tailored screening thresholds and prioritisation to achieve cost-effectiveness. At the same time, the national burden remains high which may justify inclusion of fibrosis assessment in all diabetes programs, rather than reserving screening for hepatology clinics.

A key observation was the discordance between steatosis severity and fibrosis burden. Although fibrosis prevalence increased stepwise across CAP-defined steatosis grades, one third of participants with severe steatosis had no fibrosis, while 13% (370/2847) of those with CAP values below 248 dB/m already had clinically significant fibrosis, including 4% (107/2847) with LSM values consistent with probable cirrhosis. These findings indicate that hepatic fat content and fibrogenesis may diverge, particularly in individuals with type 2 diabetes, consistent with prior observations in metabolic liver disease.²³ Steatosis may diminish with progressive fibrosis (‘burnt-out’ steatohepatitis), while fibrogenesis continues, particularly in older individuals and those with longstanding metabolic disease. Accordingly, we deliberately did not estimate MASLD prevalence based solely on CAP thresholds, because absence of CAP-detected steatosis does not exclude MASLD and would misclassify individuals with ultrasound- or histology-detected steatosis, as well as those with advanced fibrosis in whom hepatic fat content may be reduced. Steatosis is dynamic and potentially reversible, whereas fibrosis reflects cumulative injury from lipotoxicity, oxidative stress, and vascular injury. Thus, reliance on ultrasound or CAP measurements as surrogates for fibrosis can be misleading and risks both under- and over-diagnosis. For clinical practice, this may indicate that fibrosis—rather than steatosis would be a beneficial target of screening in patients with T2D.⁸ Non-invasive fibrosis assessment should be integrated into diabetes care irrespective of steatosis grade.

Diabetes care has historically prioritized microvascular complications such as retinopathy, nephropathy, and neuropathy, along with macrovascular disease, while liver involvement has remained largely neglected owing to its insidious course and lack of systematic screening. Our data demonstrates that one in twenty patients with T2D already met cirrhosis-range LSM thresholds in community settings, most often undiagnosed until decompensation. This positions advanced liver disease as a “fourth major complication” of diabetes, demanding parity of attention in guidelines and

practice.^{6,24} For clinicians, systematic incorporation of non-invasive fibrosis assessment—using transient elastography or validated serum biomarkers—should be considered as routine as annual retinal examination or urine albumin testing.⁸ For policymakers, these results argue for integration of liver health into India's National Program for Prevention and Control of Cancer, Diabetes, Cardiovascular Diseases and Stroke (NPCDCS), which currently serves as the backbone of chronic disease care delivery across the country. Embedding liver fibrosis screening within NPCDCS algorithms would enable early identification of clinically significant fibrosis and advanced fibrosis, opening the window for timely lifestyle intervention and initiation of emerging therapies before progression to cirrhosis or hepatocellular carcinoma. At a global level, the study highlights that low- and middle-income countries, which shoulder the greatest diabetes burden, are simultaneously facing a silent epidemic of advanced liver disease—a dual challenge that threatens to overwhelm health systems unless addressed proactively.

The strengths of this study include its sample size, deliberate exclusion of hepatology referrals to minimize bias, and quality control. The physician-entered design enhances generalizability. Although the overall prevalence of clinically significant and advanced fibrosis in the DiaFib-Liver Study is broadly comparable to reports from Western cohorts of patients with T2D, several important distinctions emerge. First, a substantial burden of fibrosis was observed among non-obese individuals, consistent with lower adiposity thresholds and distinct body composition in South Asian populations. Second, fibrosis frequently occurred in the absence of CAP-detected steatosis, indicating a weaker coupling between hepatic fat content and fibrogenesis than is typically reported in western studies. Third, variation was noted among centres located in different parts of India. Finally, this cohort was derived from community diabetes practice, suggesting that clinically significant fibrosis is already prevalent before liver disease becomes clinically apparent. Collectively, these findings support the need for context-specific fibrosis screening strategies in people with T2D in India.

Several limitations merit acknowledgment. The cross-sectional design precludes assessment of outcomes such as progression to decompensation or mortality. Fibrosis assessment relied on transient elastography, which, while guideline-endorsed, has inherent variability. Histological confirmation was not feasible in a study of this scale. Lifestyle factors, medication use, and genetic data were not captured, limiting mechanistic interpretation. Individual-level data on social determinants of health, including education, income, and health-care access, were not systematically collected; therefore, the influence of socioeconomic factors on fibrosis risk and regional variation could not be directly assessed. Although

participants were asymptomatic for liver disease, VCTE was performed as part of routine clinical care based on physician discretion rather than population-based screening, introducing potential selection and spectrum bias. Consequently, the cohort may over-represent individuals perceived to be at higher metabolic or hepatic risk, and prevalence estimates should be interpreted as representative of real-world diabetes practice rather than the general population. Exclusion of gastroenterology-based practices was undertaken to avoid enrichment of the cohort with referral-driven liver disease; however, this approach may limit generalizability to settings where elastography is routinely performed within hepatology services rather than diabetes clinics. Although geographic representation was broad, participation was voluntary and may not fully reflect rural or underserved populations. Alcohol consumption was assessed using routine clinical history and pre-defined exclusion thresholds, and individuals with significant intake were excluded. However, under-reporting of alcohol use is a recognised limitation of real-world studies and may vary across regions within India. Residual misclassification of low-to-moderate alcohol exposure, including MetALD, cannot be fully excluded and may contribute modestly to geographic heterogeneity and to fibrosis observed in some non-obese or low-CAP individuals. Nevertheless, the consistent association of fibrosis with diabetes duration and metabolic comorbidities across regions, coupled with the exclusion of hepatology referrals, suggests that diabetes-related fibrogenesis represents the dominant signal in this cohort. Despite these caveats, the scale, rigor, and real-world nature of the dataset make the conclusions robust and clinically actionable.

An important consideration is the pragmatic sampling framework. Participants were asymptomatic individuals with T2D who had undergone elastography as part of routine clinical care, rather than a population-probability sample of all patients with diabetes. Although this introduces potential selection toward individuals perceived to be at higher metabolic risk, it reflects real-world diabetes practice where fibrosis assessment is most likely to be considered. Consequently, prevalence estimates should be interpreted as representative of contemporary diabetes care rather than the general diabetic population. Notably, even within this context, the high burden of clinically significant fibrosis—particularly among non-obese individuals and those without CAP-detected steatosis—suggests substantial under-recognition of advanced liver disease within existing care pathways.

In conclusion, in this multicentre study of over 9000 adults with T2D, one in four had clinically significant liver fibrosis and one in twenty already met cirrhosis-range LSM thresholds, despite being asymptomatic and recruited outside hepatology settings. These findings establish advanced liver disease as a major, under-

recognized complication of diabetes. Given the discordance between steatosis and fibrosis, reliance on fat detection alone is inadequate; fibrosis assessment—using transient elastography or validated serum biomarkers—should be systematically incorporated into diabetes care alongside existing complication screening. Actionable steps for practice include routine fibrosis testing in high-risk groups and recognition of cirrhosis as a preventable late-stage outcome of diabetes. Future studies should prospectively track outcomes to validate risk stratification models, evaluate cost-effectiveness of fibrosis screening in primary care, and examine the impact of lifestyle and emerging pharmacotherapies on fibrosis progression. Together, these efforts will be essential to shift liver disease from a neglected to a routinely managed complication of diabetes.

Contributors

AK conceptualized and supervised the study. AK, AM, PS, and AA designed the methodology. Contributing co-authors who provided 50 or more patient data—performed data curation and investigation. AK wrote the original draft. PS, AA, AM, VM and all co-authors reviewed and edited the manuscript. AK and PS accessed and verified the underlying data. AK was responsible for the decision to submit the manuscript. All authors had access to the manuscript, contributed to its revision, and approved the final version for submission.

Data sharing statement

The full study protocol is provided in the [Supplementary Appendix](#). De-identified participant-level data underlying the findings reported in this Article will be made available upon reasonable request to the corresponding author, following publication, subject to approval of a methodologically sound proposal and completion of a data access agreement. Requests should include a clear analysis plan. No additional study documents are available.

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Declaration of interests

AK reports receipt of speaking and lecture fees as well as travel reimbursement from Roche, Hetero, Viartis, La Renon, Torrent, and Sun Pharma. AM reports receipt of speaking and lecture fees as well as travel reimbursement from Eli Lilly, Lupin, Boehringer Ingelheim, Janssen, Cipla, AstraZeneca, Glenmark, Novo Nordisk, and Mankind. VM has acted as a consultant and speaker, and has received research or educational grants from Zydus, Abbott, Medtronic, Novo Nordisk, Sanofi, Servier, Boehringer Ingelheim, Eli Lilly, Johnson & Johnson, Lifescan, Roche, MSD, Novartis, Aventis, Bayer, USV, Dr. Reddy's, Sun Pharma, Intas, Lupin, Glenmark, Ipca, Torrent, Cipla, Biocon, Primus, Franco-Indian, Wockhardt, Emcure, Mankind, Fourrts, Apex, GSK, and Alembic. All other authors declare no competing interests.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.lansea.2026.100753>.

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Supplementary Material for: DiaFib-Liver Study

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Supplementary Tables

Supplementary Table 1

Univariable and multivariable logistic regression analyses of factors independently associated with clinically significant liver fibrosis (LSM ≥ 8 kPa) in non-obese patients with type 2 diabetes (BMI < 25 kg/m²; n=3,996).

	LSM <8 kPa (n=3,244)	LSM ≥ 8 kPa (n=752)	Univariable analysis			Multivariable analysis		
			Odds ratio	95% CI	P value	Odds ratio	95% CI	P value
Age	52.6 (SD 12.3)	54.6 (SD 11.9)	1.014	1.007 – 1.021	<0.0001	1.011	1.004 – 1.018	0.0032
Male sex	2,050 (63%)	505 (67%)	1.191	1.007 – 1.409	0.042	1.176	0.993 – 1.392	0.061
≥ 10 y of T2D	896 (28%)	245 (33%)	1.266	1.067 – 1.502	0.0067	1.060	0.878 – 1.281	0.54
Hypertension	1,429 (44%)	364 (48%)	1.192	1.016 – 1.397	0.031	1.030	0.866 – 1.225	0.74
Dyslipidemia	1,372 (42%)	359 (48%)	1.246	1.063 – 1.461	0.0067	1.138	0.960 – 1.350	0.14
Hypothyroidism	528 (16%)	130 (17%)	1.075	0.871 – 1.327	0.50			
CAD	204 (6%)	68 (9%)	1.481	1.112 – 1.973	0.0072	1.271	0.945 – 1.710	0.11
eGFR <60	177 (5%)	50 (7%)	1.234	0.892 – 1.707	0.20			

Supplementary Table 2

Univariable and multivariable logistic regression analyses of factors independently associated with clinically significant liver fibrosis (LSM ≥8 kPa) in non-obese patients with type 2 diabetes (BMI <25 kg/m²) without metabolic comorbidities (n=1,315).

	LSM <8 kPa (n=1,095)	LSM ≥8 kPa (n=220)	Univariable analysis			Multivariable analysis		
			Odds ratio	95% CI	P value	Odds ratio	95% CI	P value
Age	48.1 (SD 12.4)	51.5 (SD 12.3)	1.022	1.010 – 1.034	0.0002	1.025	1.012 – 1.037	<0.0001
Male sex	664 (61%)	144 (65%)	1.230	0.908 – 1.665	0.18	1.267	0.933 – 1.722	0.13
≥10 y of T2D	169 (15%)	33 (15%)	0.967	0.645 – 1.449	0.87	0.751	0.491 – 1.149	0.19

Supplementary Table 3

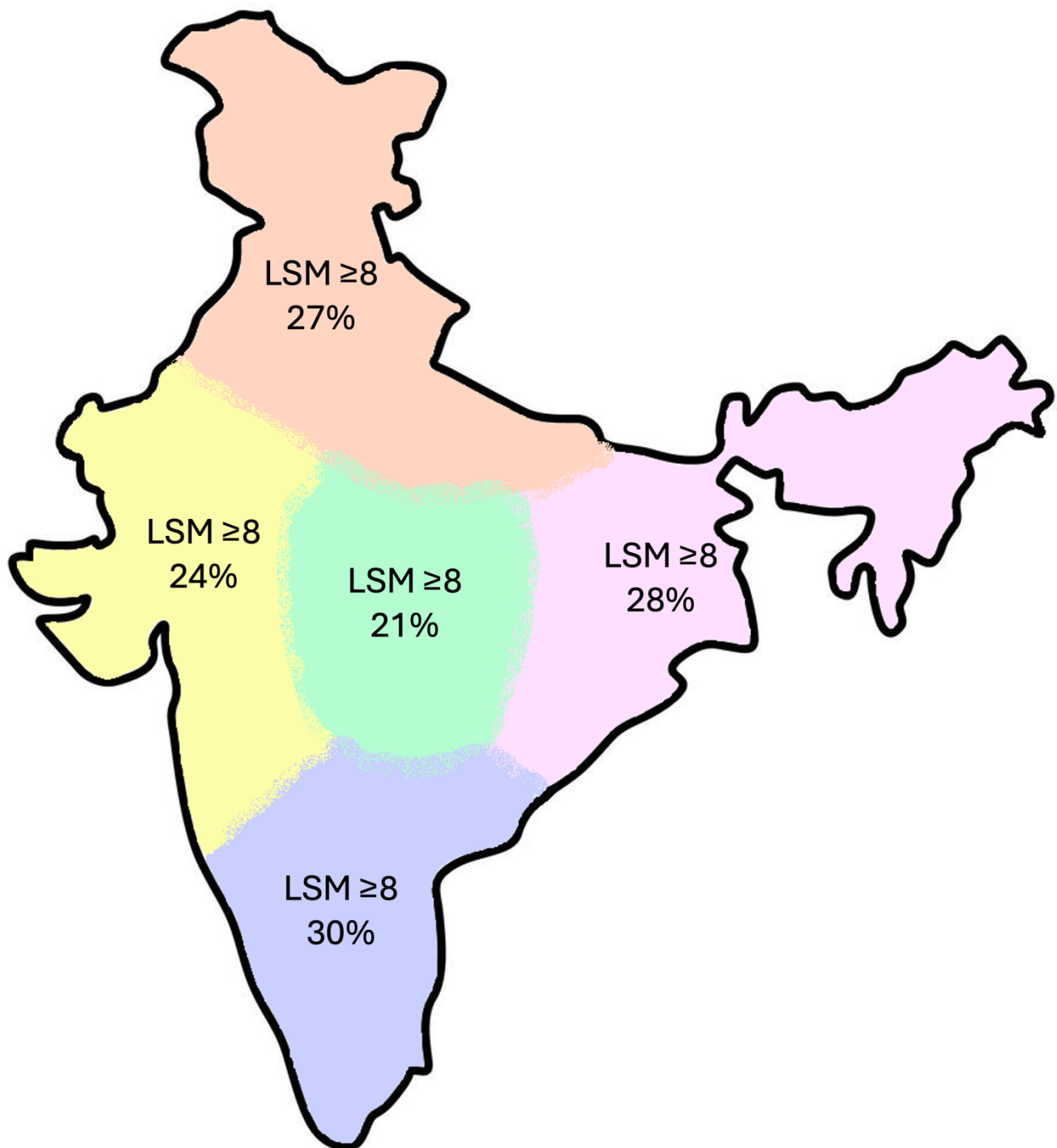
Univariable and multivariable logistic regression analyses of clinical factors associated with clinically significant liver fibrosis (LSM ≥ 8 kPa) among patients without CAP-detected steatosis (CAP < 248 dB/m; n=2,847).

	LSM < 8 kPa (n=2,477)	LSM ≥ 8 kPa (n=370)	Univariable analysis			Multivariable analysis		
			Odds ratio	95% CI	P value	Odds ratio	95% CI	P value
Age	52.6 (SD 12.2)	55.5 (SD 11.6)	1.020	1.011 – 1.029	< 0.0001	1.012	1.001 – 1.022	0.027
Male sex	1,505 (61%)	213 (58%)	0.873	0.700 – 1.090	0.23			
≥ 10 y of T2D	683 (28%)	145 (39%)	1.693	1.350 – 2.123	< 0.0001	1.367	1.064 – 1.758	0.015
Obesity (BMI ≥ 25)	959 (39%)	192 (52%)	1.707	1.371 – 2.126	< 0.0001	1.622	1.299 – 2.025	< 0.0001
Hypertension	1,117 (45%)	190 (51%)	1.285	1.033 – 1.599	0.025	0.980	0.770 – 1.248	0.87
Dyslipidemia	1,028 (42%)	181 (49%)	1.350	1.084 – 1.680	0.0072	1.171	0.928 – 1.479	0.18
Hypothyroidism	434 (18%)	57 (15%)	0.857	0.635 – 1.158	0.32			
CAD	158 (6%)	37 (10%)	1.631	1.120 – 2.375	0.011	1.232	0.829 – 1.831	0.30
eGFR < 60	155 (6%)	38 (10%)	1.715	1.181 – 2.490	0.0046	1.415	0.966 – 2.073	0.074

Supplementary Figures

Supplementary Figure 1

Geographic variation in the prevalence of clinically significant liver fibrosis ($\text{LSM} \geq 8 \text{ kPa}$) among individuals with type 2 diabetes across India. Each region is labeled with the corresponding fibrosis prevalence, illustrating the north-south gradient in disease burden.



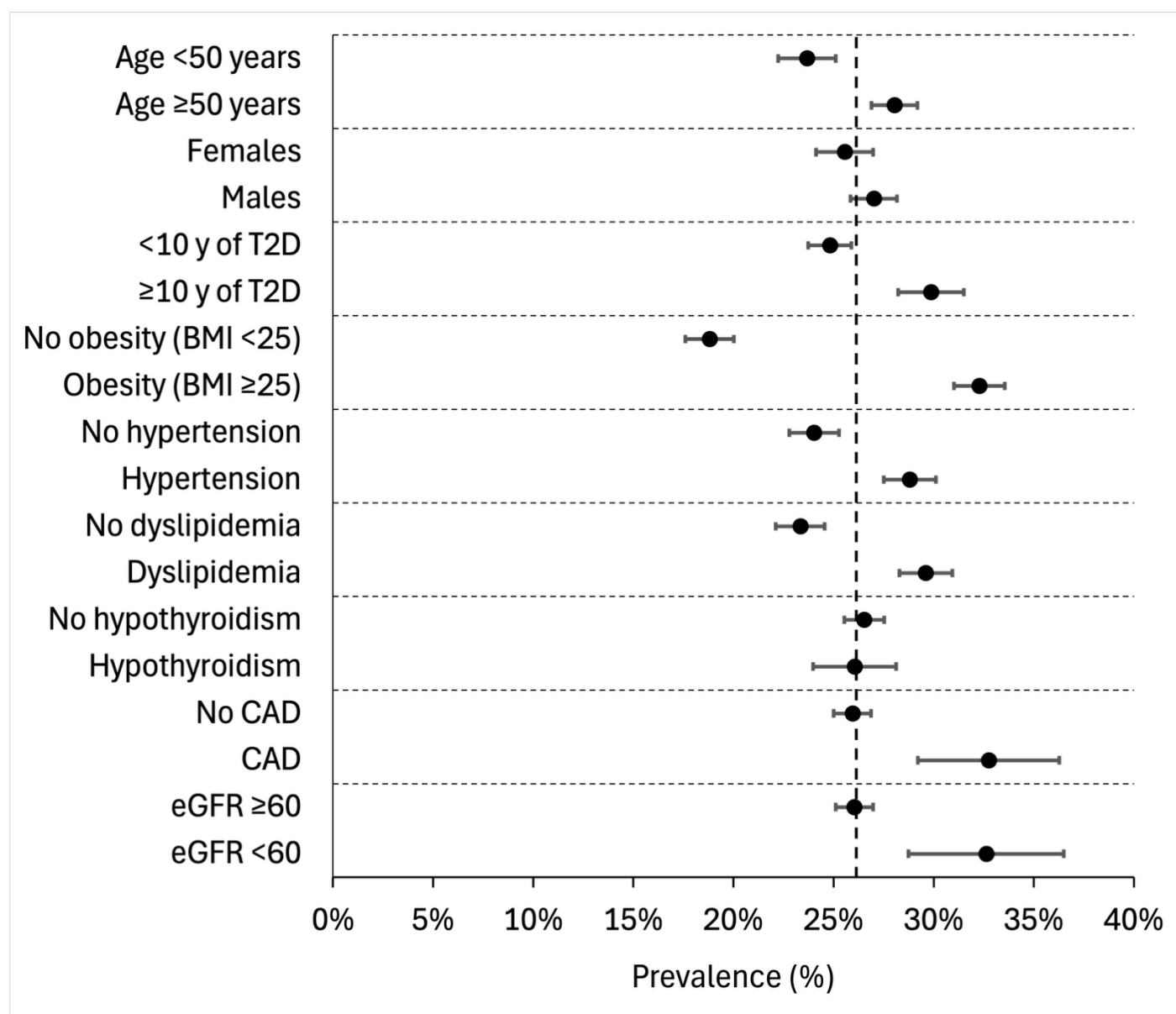
Prevalence of clinically significant fibrosis was 30% in centres from southern india (643 of 2,126), 28% in centres from the eastern part of India (74 of 261), and 27% in centres from the northern part of India (1,078 of 3,996), compared with 24% from centres in the western part of India (382 of 1,578) and 21% in central India (256 of 1,241) ($\chi^2=42.5$; $df=4$; $p<0.001$) (Supplementary figure 1).

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Supplementary Figure 2

Proportion of patients with clinically significant liver fibrosis (LSM ≥ 8 kPa) in each clinical subgroup. The figure shows the percentage of patients with clinically significant fibrosis (black dots) and their 95% confidence intervals (horizontal lines) for each subgroup. The vertical dashed line denotes the overall prevalence in the cohort (26%). Subgroups include demographic factors (age, sex), metabolic traits (obesity, hypertension, dyslipidemia), comorbidities (CAD, hypothyroidism, reduced eGFR), and duration of diabetes.



In those with hypertension (29% [1,338/4,645] vs 24% [1,095/4,557]; $p < 0.001$), dyslipidaemia (30% [1,349/4,557] vs 23% [1,084/4,645]; $p < 0.001$), coronary artery disease (33% [222/678] vs 26% [2,211/8,524]; $p < 0.001$), or reduced eGFR (33% [183/561] vs 26% [2,250/8,641]; $p < 0.001$). In contrast, fibrosis prevalence did not differ meaningfully between males and females (27% [1,517/5,617] vs 26%

[916/3,585]; $p=0.13$), or between those with and without hypothyroidism (26% [452/1,735] vs 26% [1,981/7,467]; $p=0.70$).

List of Contributing Physicians

Name of Doctor	Number cases	Clinic / Hospital	City
Dr. Jayant K. Panda	1106	Wellbeing Clinic	Cuttack
Dr. Prabhat Agrawal	1031	Agra Diabetes Research Center	Agra
Dr. Sunil Gupta	711	Sunil's Diabetes Care n' Research Centre	Nagpur
Dr. Dinesh Kumar	706	Harsha Clinic and Diabetes Centre	Lucknow
Dr. M. Shafi Kuchay	693	Medanta The Medicity	Gurugram
Dr. Jothydev Kesavadev	611	Jothydev's Diabetes Research Centre	Thiruvananthapuram
Dr. V. Mohan	463	Dr Mohan's Diabetes Specialities Centre	Chennai
Dr. Aravinda Jagadeesha	330	Dr Aravind's Diabetes Centre	Bengaluru
Dr. Bharat Saboo	329	Prayas Diabetes Center	Indore
Dr. Ajoy Tewari	303	Jai Clinic & Diabetes Care Centre	Lucknow
Dr. Kunal Das	270	Yashoda Super Speciality Hospital Kaushambi	Ghaziabad
Dr. K. P. Chandra	237	Dr Chandra's Diabetes Clinic	Lucknow
Dr. Shriram V. Kulkarni	231	Kulkarni Nursing home	Raigad
Dr. Shubhashree Patil	217	Diabetes & Wellness Clinic	Mumbai
Dr. Amit Gupta	214	Centre for Diabetes Care	Noida
Dr. Parikshit Goswami	174	Ayush Diabetes Clinic	Himmatnagar
Dr. Dhruvi Hasnani	144	RIMS Health care	Ahmedabad
Dr. Vipul Chavda	142	RIMS Super Speciality Hospital	Ahmedabad
Dr. Mayur Agrawal	141	Hormone India	Bhopal
Dr. Gaurav Chhaya	139	Shivam Medicare Clinic	Ahmedabad
Dr. C. Muralidharan	133	Vijaya Clinic Diabetes Care Centre	Dindigul
Dr. Aarathy Kannan	130	Sundaram Arulrhaj Hospital	Tuticorin
Dr. Parag Rana	123	Dev Medical Hospital	Vadodara
Dr. Ashu Rastogi	121	PGI Chandigarh	Chandigarh
Dr. Sangram Biradar	121	SB Medical Centre	Gulbarga
Dr. Khemeswar Agasti	113	SCB Medical College and Hospital	Cuttack
Dr. Prasanth Sankar	113	MGM Muthoot Hospitals	Pathanamthitta
Dr. L. Sreenivasamurthy	103	Life Care Hospital	Bengaluru
Dr. Deepak Suvarna	92	JSS Hospital	Mysuru
Dr. Rohit Kapoor	91	Carewell Heart and Super Speciality Hospital	Amritsar
Dr. Kiran Shah	70	The Consulting Room	Mumbai
Dr. Monica Gupta	68	Samvedna Hospital	Varanasi
Dr. Sandeep Suri	66	Holy Help Hospital	Hisar
Dr. Anubha Srivastava	65	MLN Medical College	Prayagraj
Dr. Debmalya Sanyal	62	Ananda Clinic	Kolkata
Dr. Seema Bagri	60	Bagri Diabetes Care Centre	Mumbai
Dr. Vitull K. Gupta	58	Kishori Ram Hospital and Diabetes Care Centre	Bhathinda
Dr. Alpana Sowani	56	Diabetes Speciality Centre	Mumbai

Dr. Purvi Chawla	55	Lina Diabetes Care Center	Mumbai
Dr. Manish HS	54	Manjunatha Hospital	Hassan
Dr. Nilesh M. Banthia	53	Neo Clinic and Echo centre	Panvel
Dr. Faraz Farishta	52	F S Endocrinology & Diabetic Center	Hyderabad
Dr. Shivam Kapoor	52	Carewell Heart and Super Speciality Hospital	Amritsar
Dr. Syed Haider Mehdi Husaini	50	JN Medical College, AMU	Aligarh
Dr. Rajesh Agrawal	45	Diabetes Overweight Thyroid Clinic	Indore
Dr. Rucha Mehta	44	EDMO Clinic	Ahmedabad
Dr. B. M. Makkar	40	Dr Makkar'S Diabetes & Obesity center	New Delhi
Dr. Prattay Ghosh	40	Apollo Clinic Beliaghata	Kolkata
Dr. Vandana Tomar	40	ECHS Polyclinic	Udhampur
Dr. R. K. Sahay	38	Osmnia General Hospital	Hyderabad
Dr. N. Kannan	31	Chandra Diabetes Care Centre	Chennai
Dr. Arijit Banerjee	29	Sanctoria Hospital	Asansol
Dr. Madhur Verma	29	AIIMS Bathinda	Bhathinda
Dr. Siddharth Agarwal	24	Saran Ashram Hospital, Dayalbagh	Agra
Dr. Akshay Kothari	23	S. S. Clinic	Mumbai
Dr. Shivani Sidana	23	AIIMS Bathinda	Bhathinda
Dr. Amrita Ghosh	20	Fortis CDOC Hospital	New Delhi
Dr. Ashish Dengra	19	Mahi Diabetes Thyroid Care and Research Center	Jabalpur
Dr. Deepak G. Pande	19	BLK Max Superspeciality Hospital/GDDC	New Delhi
Dr. Pooja Relia	17	Healers The Clinic	New Delhi
Dr. Anchin Kalia	15	Mahatma Gandhi Hospital	Jaipur
Dr. S. S. Dariya	13	Dr Dariya Diabetes Care clinic	Jaipur
Dr. Meena Chhabra	12	Dr Meena Chhabra Diabetes Clinic Delhi	New Delhi
Dr. Snehlata Verma	10	LLRM Medical College Meerut	Meerut
Dr. Ramkesh S. Parmar	9	Parmar Hospital	Jaipur
Dr. Abhishekh Raha	7	Lumding Divisional Railway Hospital	Lumding
Dr. Ketan K. Mehta	7	Health Harmony	Mumbai
Dr. Ranjit Khatua	5	Dr Ranjits Diabetes Specility clinic	Bhadrak
Dr. Pankaj N. Choudhry	4	Max Super Speciality Hospital Vaishali	Ghaziabad
Dr. Shailendra Kishore	4	IIT kanpur Health Centre	Kanpur
Dr. Md. Shahid Iqbal	3	Alhuda Clinic	Gaya
Dr. Swati Srivastava	3	Dr Swati's Diabetes and Metabolic Disorders Clinic	Jaipur
Dr. Arvind Kumar Ojha	2	ILS Hospitals, Dumdum	Kolkata
Dr. Ipsita Mishra	2	Personal Clinic	Cuttack
Dr. Mudit Sabharwal	2	Dharma Diabetes & Metabolic Clinic	New Delhi
Dr. Preeti Singh Dhoat	2	AIIMS Bathinda	Bhathinda
Dr. Rajiv Garg	2	ESI Hospital	Sahibabad
Dr. Atul Kakar	1	Sir Ganga Ram Hospital	New Delhi
Dr. Bhavya Koladia	1	Personal Clinic	Ahmedabad

Dr. Saurabh Srivastava	1	Government Institute of Medical Sciences	Greater Noida
Total	10,739		

STROBE checklist

Checklist of items that should be included in reports of **cross-sectional studies**

	Item No	Recommendation	Page No.
Title and abstract	1	(a) Indicate the study’s design with a commonly used term in the title or the abstract	Page 4 (Summary – Methods)
		(b) Provide in the abstract an informative and balanced summary of what was done and what was found	Page 4 (Summary)
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	Page 7
Objectives	3	State specific objectives, including any prespecified hypotheses	Page 7
Methods			
Study design	4	Present key elements of study design early in the paper	Page 8
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	Page 8
Participants	6	(a) Give the eligibility criteria, and the sources and methods of selection of participants	Page 8-9
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	Page 9-10
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	Page 8-10
Bias	9	Describe any efforts to address potential sources of bias	Page 9
Study size	10	Explain how the study size was arrived at	Page 11
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	Page 10-11
Statistical methods	12	(a) Describe all statistical methods, including those used to control for confounding	Page 10-11
		(b) Describe any methods used to examine subgroups and interactions	Page 11
		(c) Explain how missing data were addressed	Page 11
		(d) If applicable, describe analytical methods taking account of sampling strategy	Page 11
		(e) Describe any sensitivity analyses	Page 11
Results			

Participants	13*	(a) Report numbers of individuals at each stage of study—e.g. numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	Page 12, Figure 1
		(b) Give reasons for non-participation at each stage	Page 12, Figure 1
		(c) Consider use of a flow diagram	Figure 1
Descriptive data	14*	(a) Give characteristics of study participants (e.g. demographic, clinical, social) and information on exposures and potential confounders	Page 12, Table 1
		(b) Indicate number of participants with missing data for each variable of interest	Page 13
Outcome data	15*	Report numbers of outcome events or summary measures	Page 12-14
Main results	16	(a) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (e.g., 95% confidence interval). Make clear which confounders were adjusted for and why they were included	Page 12-14
		(b) Report category boundaries when continuous variables were categorized	Page 12-14
		(c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	Not applicable
Other analyses	17	Report other analyses done—e.g. analyses of subgroups and interactions, and sensitivity analyses	Page 13-14
Discussion			
Key results	18	Summarise key results with reference to study objectives	Page 14
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	Page 17
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	Page 15-18
Generalizability	21	Discuss the generalisability (external validity) of the study results	Page 16-17
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	Page 19