

Diabetic Motor–Sensory Neuropathy, Retinopathy and Nephropathy in Indian Type 2 Diabetes: Why Complications Arrive Early, and What Can Be Done

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Abstract

Background: India carries the world’s second-largest diabetes burden, and type 2 diabetes mellitus (T2DM) in Indians is diagnosed a decade or more earlier, at lower body mass index, than in white populations. Microvascular complications therefore surface in the working-age years.

Objective: To synthesise current evidence on diabetic peripheral (motor–sensory) neuropathy, retinopathy and nephropathy in Indian T2DM, to explain why they appear early, and to outline feasible solutions.

Methods: Narrative review. PubMed and the Consensus academic search engine were searched for Indian population-based and clinic-based studies (2015–2026), landmark international trials and recent reviews.

Results: Roughly one in three Indian adults with T2DM has peripheral neuropathy, including a third of those who are newly diagnosed. National retinopathy prevalence is 12.5 percent (vision-threatening 4.0 percent); microalbuminuria affects about a quarter of urban patients. Only 36 percent of Indians with diabetes reach HbA1c below 7 percent and fewer than 8 percent meet combined glucose, blood-pressure and LDL targets. Each year of earlier diagnosis raises microvascular risk by about 5 percent. Intensive glucose lowering reduces kidney and eye events by 20 and 13 percent respectively, but not nerve events, which respond to multifactorial control.

Conclusion: The Indian “thin-fat”, insulin-deficient phenotype, aggressive glycaemic trajectory and late diagnosis explain early complications. Screening for

neuropathy, retinopathy and albuminuria from the day of diagnosis, task-shifted to primary care, together with combined metabolic control, is the practical way forward.

Keywords: Diabetic Neuropathy, Diabetic Retinopathy, Diabetic Nephropathy, Early-Onset Type 2 Diabetes, India, Glycaemic Control

Introduction

The ICMR-INDIAB national survey, which examined 113,043 adults across 31 states and union territories, estimated a weighted prevalence of diabetes of 11.4 percent and of prediabetes of 15.3 percent, corresponding to roughly 101 million and 136 million Indians respectively¹. Control is poor: among adults with self-reported diabetes only 36.3 percent achieved an HbA1c below 7 percent, 48.8 percent achieved blood-pressure control, and just 7.7 percent met all three “ABC” targets for glucose, blood pressure and LDL cholesterol². Against this backdrop, distal symmetric polyneuropathy is the most common complication of diabetes, developing in at least half of all patients over their lifetime³, and retinopathy and nephropathy remain leading causes of adult blindness and end-stage kidney disease.

This review addresses four linked questions raised in clinical practice: what is known about diabetic motor-sensory neuropathy in Indians; why complications arise early in this population; how retinopathy and nephropathy relate to glycaemic control; and what the rise of early-onset T2DM means for Indian diabetes care.

Methods

PubMed (via the National Library of Medicine interface) and Consensus, which indexes more than 220 million peer-reviewed papers, were searched in September 2026 using combinations of the terms diabetic peripheral neuropathy, retinopathy, nephropathy, albuminuria, young-onset or early-onset type 2 diabetes, Asian Indian

phenotype, glycaemic control and India. Preference was given to Indian population-based surveys (ICMR-INDIAB, SMART India, CURES), Indian clinic-based cross-sectional studies published from 2015 onwards, individual-participant meta-analyses of glucose-lowering trials, and recent Lancet, BMJ and Nature-family reviews. Evidence is cited by number in order of first appearance.

Why complications arrive early in Indians

• The thin-fat phenotype and early-life origins

For any given body mass index, Indians carry a higher percentage of body fat and more visceral fat than other populations, a pattern already present at birth and amplified by accelerated childhood growth and urban living⁴. Clamp studies of healthy, non-obese Asian Indian men showed that two-thirds were insulin resistant, with insulin action inversely related to visceral rather than subcutaneous fat^{5,6}. South Asians show greater abdominal obesity, more ectopic fat (particularly in the liver) and lower skeletal muscle mass than white people of similar age and BMI, and together with rapid beta-cell dysfunction this produces earlier onset and faster progression of hyperglycaemia⁸.

• Genetic predisposition to insulin deficiency and lipodystrophy

Partitioned polygenic-score analysis in more than 50,000 British South Asians found that genetic risk for beta-cell insufficiency and for unfavourable fat distribution was most strongly linked to T2DM and to a younger age at diagnosis; individuals at high risk on both scores were diagnosed 8.2 years earlier with a BMI 3 kg/m² lower, progressed faster to insulin dependence and microvascular complications, and responded less well to SGLT2 inhibitors⁷. The resulting greater aggregated hyperglycaemia is what drives more retinopathy, chronic

kidney disease and cardiovascular disease in this population⁸.

• **An adverse risk profile at young ages**

Pooled analysis of national surveys from India, the USA, Australia and Mauritius showed that the metabolic gap between people with diabetes and those with normal glucose tolerance is widest at younger ages for fasting glucose, HbA1c, triglycerides and diastolic pressure⁹. Diet contributes: Indian diets are dominated by low-quality carbohydrates, and the highest carbohydrate intakes carried a 30 percent higher odds of newly diagnosed T2DM in ICMR-INDIAB¹⁰. A meta-analysis of 26 studies (1.3 million participants) quantified the consequence: each one-year decrease in age at diagnosis was associated with 5 percent higher odds of microvascular disease and 4 percent higher mortality, adjusted for current age¹¹.

Diabetic motor–sensory neuropathy in Indian T2DM

Diabetic peripheral neuropathy (DPN) is length-dependent damage to sensory, motor and autonomic fibres that affects up to half of people with diabetes, causing pain, loss of protective sensation, imbalance, falls, foot ulceration and amputation¹². Sensory loss in a stocking distribution appears first; motor involvement follows with weakness and wasting of intrinsic foot muscles, loss of ankle reflexes and clawing of the toes, which together with sensory loss set the stage for plantar

ulcers. Indian estimates are consistently high (Table 1). A cluster-sampled community study of 526 adults with diabetes in Kerala found DPN in 34.2 percent (95 percent CI 30.2–38.4), with age above 60, low education, height above 160 cm, insulin use and coexisting nephropathy as independent predictors¹³. A multicentre facility-based study in rural Andhra Pradesh reported 39.3 percent, with 78.6 percent of participants having an HbA1c above 7 percent¹⁴. Most striking, among 350 newly diagnosed T2DM patients in Odisha (mean age 46 years) DPN was already present in 34 percent, and 35 percent had lost protective sensation on monofilament testing; each unit rise in HbA1c increased the odds of DPN by 23 percent and hypertension nearly tripled it¹⁵.

Two implications follow. First, because neuropathy is present at diagnosis in a third of Indian patients, foot examination cannot wait for the customary five-year mark used in type 1 diabetes. Second, glucose control alone is insufficient: whereas glycaemic control halts neuropathy progression in type 1 diabetes, its effect in T2DM is modest, and current guidance emphasises multi-targeted metabolic control including weight and lipids^{3,12}. For screening, the Michigan Neuropathy Screening Instrument has been translated into Tamil and validated against biothesiometry in Chennai, with 96.8 percent sensitivity and 85.7 percent specificity at a cut-off score of 2, making it suitable for routine primary-care use¹⁶.

Table 1: Prevalence of microvascular complications of type 2 diabetes in selected Indian studies

Study (year)	Setting	n	Complication	Prevalence (%)
Soman et al (2025) ¹³	Community, central Kerala	526	DPN (MNSI exam)	34.2
Darivemula et al (2019) ¹⁴	Facility, rural Andhra Pradesh	336	DPN (monofilament, VPT, reflexes)	39.3
Jadhao et al (2025) ¹⁵	Newly diagnosed T2DM, Odisha	350	DPN (DNS + NDS)	34.0

Study (year)	Setting	n	Complication	Prevalence (%)
Raman et al, SMART India (2022) ¹⁷	Population, 10 states + 1 UT, age ≥ 40 y	6133	Any DR / VTDR	12.5 / 4.0
Sabherwal et al (2025) ¹⁹	Population, rural Uttar Pradesh, age ≥ 50 y	224	Any DR / sight-threatening DR	22.8 / 5.8
Unnikrishnan et al, CURES (2007) ²⁰	Population, urban Chennai	1716	Microalbuminuria / overt nephropathy	26.9 / 2.2

Source: studies retrieved from PubMed and Consensus, September 2026. DPN, diabetic peripheral neuropathy; MNSI, Michigan Neuropathy Screening Instrument; VPT, vibration perception threshold; DNS, Diabetic Neuropathy Symptom score; NDS, Neuropathy Disability Score; DR, diabetic retinopathy; VTDR, vision-threatening DR; UT, union territory; CURES, Chennai Urban Rural Epidemiology Study.

Retinopathy and nephropathy in the context of glycaemic control

• Burden in India

The SMART India study screened 42,146 adults aged 40 years and older in ten states and one union territory and estimated a national prevalence of diabetic retinopathy of 12.5 percent and of vision-threatening retinopathy of 4.0 percent among people with diabetes, with no urban–rural difference; prevalence was almost twice as high in known as in undiagnosed diabetes, and hyperglycaemia was the strongest modifiable risk factor¹⁷. Extrapolated to 101 million people with diabetes, about 21 million Indians with diabetes have vision impairment and 2.4 million are blind¹⁸. A rural population survey in Uttar Pradesh found any retinopathy in 22.8 percent and sight-threatening disease in 5.8 percent, yet 84.8 percent of people with known diabetes had never had an eye examination and 76 percent had poorly controlled glucose¹⁹. For the kidney,

the Chennai Urban Rural Epidemiology Study found microalbuminuria in 26.9 percent and overt nephropathy in 2.2 percent of urban Asian Indians with T2DM; every unit of HbA1c raised the odds of microalbuminuria by 33 percent and of overt nephropathy by 48 percent²⁰.

• What glucose lowering achieves

An individual-participant meta-analysis of ACCORD, ADVANCE, UKPDS and VADT (27,049 participants) showed that a 0.9 percent lower HbA1c sustained for five years reduced kidney events by 20 percent (HR 0.80) and eye events by 13 percent (HR 0.87), but did not reduce nerve events (HR 0.98)²¹. In ADVANCE alone, targeting an HbA1c of 6.5 percent cut end-stage renal disease by 65 percent and new macroalbuminuria by 30 percent²². The message for Indian practice is that the eye and the kidney are highly glucose-responsive, whereas the nerve requires blood pressure, lipid and weight control alongside glucose. Given that only a third of Indians with diabetes achieve an HbA1c below 7 percent², the potential gain from closing this gap is large, and newer agents with kidney and cardiovascular benefit (SGLT2 inhibitors, GLP-1 receptor agonists) remain under-used mainly because of cost⁸.

Early-onset type 2 diabetes in India

Early-onset T2DM, diagnosed before 40 years, follows a more aggressive course than late-onset disease, with

faster beta-cell decline, worse insulin resistance, accelerated microvascular and macrovascular complications, and reduced life expectancy; management strategies extrapolated from older adults are inadequate and clinical inertia is common²⁴. Indian data illustrate this. At a tertiary centre in Lucknow, 400 patients with early-onset disease (median age at diagnosis 35 years) were matched for sex and duration with 400 late-onset patients: the younger group had higher HbA1c and worse lipid profiles, and despite being 13 years younger had the same prevalence of retinopathy and nephropathy; age at onset independently predicted neuropathy, nephropathy and atherosclerotic disease²³. Because complications track cumulative exposure, a person diagnosed at 30 who reaches 60 has lived with hyperglycaemia for three decades, exactly the duration at which retinopathy and nephropathy peak^{11,17}. Younger Indian patients also carry higher BMI and triglycerides than older patients, so the risk profile is not benign merely because blood pressure and albuminuria are lower at first⁹.

Solutions

• Prevent upstream

Because the phenotype begins in utero, prevention must span the life course: maternal nutrition, avoidance of rapid childhood weight gain, and adult diets that reduce refined carbohydrate and saturated fat while raising plant, dairy and egg protein^{4,10}. Population-wide preventive measures that account for the region's socioeconomic realities, together with affordable weight-loss and lifestyle programmes, are recommended for South Asia⁸.

• Screen for complications from the day of diagnosis

Given one-third prevalence of neuropathy at diagnosis¹⁵, every newly diagnosed Indian adult should have a foot examination (10 g monofilament, vibration by tuning fork or biothesiometer, ankle reflexes), a urine albumin–

creatinine ratio, and retinal imaging at baseline and then annually. The Tamil-validated MNSI provides a non-invasive tool for Health and Wellness Centres¹⁶, and community programmes rather than hospital referral are needed to reach the two-thirds who are asymptomatic¹³.

• Task-shift retinal screening to primary care

A pragmatic study in Punjab showed that non-ophthalmologist-led screening with non-mydriatic cameras at Health and Wellness Centres achieved 86 percent sensitivity for any retinopathy and 96 percent for referable retinopathy, while offline smartphone AI reached 93 percent sensitivity for referable disease, limited mainly by ungradable images from cataract²⁵. Combining both models can close the screening gap revealed by the 85 percent of rural patients never examined¹⁹.

• Treat to combined targets

Care should aim at the ABC triad rather than glucose alone, since kidney and eye outcomes respond to HbA1c while neuropathy responds to multifactorial control^{21,3}. Affordable generic metformin, sulfonylureas, statins and renin–angiotensin blockers remain the backbone; SGLT2 inhibitors and GLP-1 receptor agonists should be prioritised for those with albuminuria or cardiovascular disease as prices fall^{8,22}.

• Tailor care for young-onset disease

Young adults need proactive combination therapy, weight-centred management, cardiovascular risk assessment that does not underestimate lifetime risk, preconception care, and transition services to prevent disengagement²⁴. Structured exercise is effective: in a Manipal randomised trial of 84 adults aged 18–40 years, a 12-week combined aerobic and resistance programme significantly lowered HbA1c and systemic inflammation

compared with usual care²⁶. Screening for diabetes distress is also warranted in this group.

- **Strengthen the system**

Integrating complication screening into the National Programme for Prevention and Control of Non-Communicable Diseases, maintaining state diabetes registries, using ICMR-INDIAB state-level data to target resources, and ensuring uninterrupted supply of low-cost drugs at primary level are the enabling steps^{1,2,8}.

Conclusion

Indians develop T2DM early because of a genetically and developmentally programmed thin-fat, insulin-deficient phenotype, aggravated by carbohydrate-heavy diets and late diagnosis. As a result, one in three patients already has motor-sensory neuropathy when diabetes is first recognised, and retinopathy and nephropathy accrue at ages when people are still supporting families. The evidence is clear that glucose control protects the eye and kidney, that neuropathy needs multifactorial control, and that inexpensive screening tools can be delivered by non-specialists. The task now is implementation: screen at diagnosis, treat to combined targets, and design services around the young adult with diabetes.

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