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Real-world Consensus on ADD Management: Insights from an Indian Physician Survey

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ABSTRACT

Background: Atherogenic diabetic dyslipidemia (ADD), defined by elevated triglycerides, reduced high-density lipoprotein cholesterol, and predominance of small dense low-density lipoprotein particles, is a highly prevalent phenotype among individuals with type 2 diabetes mellitus (T2DM), particularly within South Asian populations. Despite the availability of international guidelines, significant gaps persist in the realworld diagnosis and management of atherogenic diabetic dyslipidemia in India.

Objective: To establish expert consensus recommendations for the diagnosis and management of atherogenic diabetic dyslipidemia in Indian clinical practice.

Method and Result: A structured survey was conducted between April and August 2025 among 146 Indian physicians to generate consensus statements addressing epidemiology, pathophysiology, screening, diagnostic criteria, lipid targets, lifestyle interventions, and pharmacological management of atherogenic diabetic dyslipidemia in T2DM. Predefined agreement thresholds defined consensus. Strong agreement of 66.44% was achieved on the high prevalence of atherogenic diabetic dyslipidemia in Indian patients with T2DM, the necessity of lipid assessment beyond low-density lipoprotein cholesterol (LDLC) (with emphasis on triglycerides, nonHDLC, and apolipoprotein B), and the universal role of lifestyle modification as foundational therapy. Statins were endorsed as essential for highrisk patients irrespective of baseline LDLC, with frequent recognition of the need for combination therapy to mitigate residual atherogenic risk.

Conclusion: This consensus underscores the importance of a phenotypebased, comprehensive approach to dyslipidemia management in Indian patients with diabetes. Early identification of atherogenic diabetic dyslipidemia and individualized treatment strategies are critical to reducing cardiovascular risk in this population.

Keywords: Atherogenic diabetic dyslipidemia, Type 2 diabetes mellitus, Hypertriglyceridemia, Residual cardiovascular risk, Saroglitazar

Introduction

Atherogenic diabetic dyslipidemia (ADD) is a distinct lipid phenotype that contributes substantially to residual cardiovascular risk in patients with type 2 diabetes mellitus (T2DM). Characterized by elevated triglycerides (TG) (≥150 mg/dL), low high-density lipoprotein cholesterol (HDLC) (<40 mg/dL in men, <50 mg/dL in women), raised apolipoprotein B (apo B), and a predominance of small dense low-density lipoproteins (sd-LDL), ADD is particularly prevalent among South Asians. This pattern reflects the interplay of hyperglycemia, adipocytokine imbalance, and early, severe insulin resistance, further compounded by truncal obesity and high carbohydrate intake, making ADD both highly prevalent and clinically challenging in Indian practice.[1]

Epidemiological data underscore ADD's high burden in Indian diabetics, with prevalence reaching 34% in treatment-naïve patients, 73% of whom had poor glycemic control (HbA1c >8%) and 89% of newly diagnosed cases showing dyslipidemia at presentation.[2] The diabetes epidemic has surged from 90 million cases in 2011 to 140 million by 2025, amplifying ADD as a primary complication and mortality in T2DM patients compared to non-diabetics.[3] The Research Society for the Study of Diabetes in India (RSSDI) 2022 consensus guidelines emphasize annual screening and comprehensive profiling (including non-high-density lipoprotein [HDL]-C and apo B) for all diabetics, recognizing ADD's unique atherogenicity in South Asians versus Caucasians.

Persistent uncertainties surround ADD management in India, including optimal TG cutoffs (>150 mg/dL for diagnosis, >200 mg/dL for cardiovascular disease (CVD) risk intervention, >500 mg/dL for pancreatitis prevention), fasting versus non-fasting TG reliability, and residual risk strategies beyond statins despite LDL-C control. While global guidelines predominantly prioritize LDL-C lowering as the primary strategy for CVD risk reduction, emerging evidence indicates that residual CVD risk persists despite achieving optimal LDL-C levels. Evidence also supports hypertriglyceridemia, particularly in the context of insulin resistance and sd-LDL predominance contributing to CVD risk. Lipoprotein(a) further elevates risk dose-dependently, yet population-specific thresholds remain underexplored (4).

The current study addressed these gaps through a consensus survey capturing opinions from 146 Indian physicians on ADD prevalence, diagnosis, and management in diabetic patients. Experts reviewed RSSDI guidelines alongside emerging evidence on insulin resistance-driven Very low-density lipoprotein (VLDL) overproduction, Triglyceride-Glucose (TyG) index utility, and therapies targeting residual TG risk (e.g., saroglitazar dual lipid-glycemic benefits).

This survey establishes practical, evidence-based recommendations tailored to Indian diabetics, validating RSSDI frameworks while resolving real-world ambiguities in ADD care. Near-universal consensus (>90% in key areas) underscores the need for ADD-specific screening, multi-marker assessment, and stepwise intensification beyond LDL-C targets to mitigate India's escalating CVD burden in diabetes.

Method

1.1 Study design and questionnaire development

A structured survey was employed to facilitate optimal consensus among a broad panel of 146 expert physicians from diverse backgrounds. This systematic approach provided comprehensive, expert-driven opinions to ensure robust, evidence-based agreement.[5] The expert panel comprised physicians from across India with expertise in diabetes and dyslipidemia management, including clinicians from key specialities involved in cardiometabolic care [internal medicine (n=84), endocrinology (n=39), cardiology (n=20), nephrology (n=3)], representing diverse clinical practice settings and supporting a multidisciplinary, practice-oriented consensus process. The consensus survey was conducted manually, with responses collected and compiled by the research team, ensuring broad geographic participation across India [North zone (n=24), South zone (n=37), West zone (n=25), East zone (n=60)], complete response capture, and standardized recording of all consensus items. The scientific committee, constituted based on members' extensive expertise and professional experience, collaborated with practising physicians to design the survey questionnaire. Survey development was guided by a structured literature search that prioritized systematic reviews and other critical evidence syntheses from databases such as MEDLINE, EMBASE, and PubMed, supplemented by manual review of relevant articles identified through keywords including *atherogenic dyslipidemia* and *cardiovascular risk*. Each item was drafted as a clear statement, affirmative or negative, intended to capture professional opinion or clinical recommendations and to address areas of uncertainty, debate, or practical relevance in the management

of atherogenic dyslipidemia. The final questionnaire comprised consensus statements grouped into three domains: disease prevalence (4 items), diagnosis (4 items), and disease management (8 items). The expert panel reviewed these statements (Table 1) and voted using predefined criteria ("strongly agree," "agree," "disagree," "undecided," or "not my area (NA) of expertise"), providing comments for refinement. Responses were analyzed descriptively, with consensus defined as ≥80% respondents selecting 'Agree' or 'Strongly agree' in line with established survey methodologies. The response option "Not my area of expertise" was included to allow panellists to abstain from statements outside their clinical scope and to preserve the validity of responses. For consensus calculations, NA responses were excluded from the denominator; thus, agreement was expressed as the proportion of substantive responses selecting "Strongly agree" or "Agree" (i.e., among non-NA responses). Iterative rounds incorporated feedback, with revised statements recirculated until convergence was achieved, followed by final approval. The approved statements informed the manuscript and were endorsed by all authors.

Table 1. Expert consensus levels of agreement (N = 146 experts).[a]

Statements	Strongly agree (%)	Agree (%)	Disagree (%)	Strongly disagree (%)	Undecided (%)	Consensus (% SA+Agree)
Disease prevalence						
1. Indian diabetic patients with TG ≥150 mg/dL, low HDLC (<40 mg/dL in men, <50 mg/dL in women), and abnormal sdLDL should be classified as ADD patients	66.44	28.77	0.68	–	3.42	95.21
2. In South Asians, insulin resistance is more common and occurs earlier, contributing to the increasing ADD incidence	72.60	23.97	–	–	2.74	96.57
3. Elevated TG is more common than LDL-C in Asians; hypertriglyceridemia should be considered a significant CVD risk factor	48.63	41.10	0.68	–	8.90	89.73
4. In patients with controlled LDL-C on statins, elevated TG increases residual CVD risk	40.41	44.52	2.74	–	10.27	84.93
Diagnosis						
1. Fasting TG is more reliable than non-fasting TG, though non-fasting can be used if fasting is unavailable.	38.36	45.89	6.85	0.68	7.53	84.25
2. TG ≥150 mg/dL should be used as the cut-off for hypertriglyceridemia in Indian patients	36.99	41.10	5.48	–	13.70	78.09
3. Non-HDLC and apoB should be considered secondary markers for CVD risk along with TG and LDLC	48.63	43.84	1.37	–	5.48	92.47
4. The TyG index is a validated marker for insulin resistance and CVD and should be regularly calculated	34.25	43.84	3.42	–	17.81	78.09

Statements	Strongly agree (%)	Agree (%)	Disagree (%)	Strongly disagree (%)	Undecided (%)	Consensus (% SA+Agree)
Management						
1. Lifestyle modifications (weight reduction, diet, stress reduction) are first-line before medical therapy	78.08	15.75	1.37	–	1.37	93.83
2. Statin therapy is the first-line medication for managing dyslipidemia in diabetes	76.71	19.18	–	0.68	1.37	95.89
3. TG >200 mg/dL should be the threshold to initiate medication for CVD risk reduction	38.36	40.41	4.11	–	14.38	78.77
4. TG >500 mg/dL should be treated to prevent pancreatitis	86.30	10.27	–	–	0.68	96.57
5. In patients with controlled LDL-C post statin, if TG >200 mg/dL, non-statins agents (ezetimibe, bempedoic acid, PCSK9 inhibitors) should be added	39.04	34.93	11.64	1.37	9.59	73.97
6. In patients with controlled LDL-C on statins, if TG >200 mg/dL, TG-lowering drugs (saroglitazar, fenofibrate, icosapent ethyl, niacin) should be added	48.63	41.78	3.42	–	3.42	90.41
7. In patients with TG >500 mg/dL, TG-lowering drugs should be started (with or without statins) to prevent pancreatitis	79.45	15.07	0.68	–	0.68	94.52
8. Saroglitazar is the preferred add-on to statins for hypertriglyceridemia, given insulinsensitizing and glycemic benefits	67.81	26.03	0.68	–	2.74	93.84

Note: [a] Responses marked as Not applicable (NA) were excluded from the analysis as they were considered analytically non-informative and did not contribute meaningfully to the assessment of consensus. The results presented include only valid responses, and consensus statements were derived based on the proportion of respondents selecting "Agree" and "Strongly agree," which represented the predominant statements observed.
The proportion of NA responses across individual statements ranged from 0.68% to 4.11%, indicating minimal item-level abstention. ADD = atherogenic diabetic dyslipidemia; apoB = apolipoprotein B; CVD = cardiovascular disease; HDLC = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; sdLDL = small dense low-density lipoproteins; TG = triglycerides; TyG = triglyceride-glucose index.

1.2 Disease prevalence

Hyperglycemia, insulin resistance, adipocytokine imbalance, and insulin deficiency disrupt lipid metabolism in diabetes, creating a distinct profile common among South Asians.[6] Compared to Caucasians, Indians typically exhibit only modestly elevated LDL-C levels but a higher proportion of sd-LDL particles, alongside raised fasting triglycerides (>150 mg/dL), increased apo B, and lower HDL-C, collectively defining atherogenic diabetic dyslipidemia.[4] These features are further aggravated by abdominal adiposity (waist circumference exceeding 90 cm in men or 80 cm in women) and features of metabolic syndrome. Atherogenic diabetic dyslipidemia affects 34% of treatment-naïve Indian diabetics, with 73% exhibiting poor glycemic control (HbA1c >8%) and 89% demonstrating dyslipidemia across the studied groups.[7]

Beyond LDL-C alone, comprehensive screening with non-HDL-C, apolipoprotein B (apo B), fasting/non-fasting triglyceride profiles, and ethnicity-adjusted QRISK3 proves essential for diabetics stratified as high/very high CVD risk per RSSDI guidelines (considering comorbidities and target organ damage).[8] Single-time lipoprotein(a) assessment merits recommendation, particularly with a family history of premature CVD.[9] Risk enhancers that shift low or moderaterisk diabetics into higher categories, requiring stricter LDLC targets, include triglyceride elevations above 150 mg/dL (or >175 mg/dL in the nonfasting state), intermediate lipoprotein(a) levels (20–49 mg/dL), impaired fasting glucose in the 100–125 mg/dL range, and highsensitivity Creactive protein values exceeding 2 mg/L.[10] Additional modifiers comprise air pollution exposure, inflammatory joint disease, premature menopause, preeclampsia, gestational diabetes, polycystic ovary syndrome, elevated polygenic risk scores, and chronic infections.[11]

High-density lipoprotein particles confer cardiovascular protection by facilitating reverse cholesterol efflux from peripheral tissues to the liver and promoting endothelium-mediated vasorelaxation, plus anti-inflammatory, antithrombotic, and antioxidant mechanisms. Diabetes markedly impairs HDL's anti-inflammatory function, making isolated HDL-C levels unreliable for CVD risk assessment.[12] The HDL concentration, composition, metabolism, and functionality diverge substantially from nondiabetic norms; type-1 diabetes mellitus (T1DM) with nephropathy or T2DM frequently displays low HDL-C, even as some T1DM cases maintain normal/elevated levels despite heightened CVD incidence from distorted cholesteryl ester/triglyceride ratios, diminished phospholipids, impaired macrophage cholesterol efflux, reduced anti-inflammatory/antioxidant capacity, and other proatherogenic traits. T2DM produces triglyceride-laden HDL prone to rapid catabolism.[13]

Lipoprotein(a), structurally akin to plasminogen, binds its receptors to heighten thrombosis and aids CVD risk reclassification among moderate/high-risk individuals.[14] In patients with T2DM who develop CVD or related complications, ADD is frequently characterized by altered apolipoprotein profiles together with genetic polymorphisms that modulate lipid metabolism and amplify diabetesassociated CVD risk.[15] Apolipoprotein B reveals high-risk dyslipidemia patterns overlooked by routine lipid panels, informing lipid-lowering therapy initiation to curb cardiovascular morbidity/mortality, particularly among hypertriglyceridemia or coronary heart disease (CHD) patients achieving LDL-C/non-HDL-C goals.[16] The European Society of Cardiology (ESC) guidelines position apo B as superior to LDL-C for assessing atherogenic lipoprotein burden, especially in diabetes, obesity, hypertriglyceridemia, or very low LDL-C contexts.[17]

1.3 Diagnosis

Diagnosing ADD demands comprehensive lipid profiling that surpasses traditional LDL-C evaluation alone. This phenotype manifests through persistently low HDL-C, raised triglycerides, and a predominance of sd-LDL particles collectively forming atherogenic lipid triad typical of diabetic patients. The pattern proves especially prominent among South Asians, where unremarkable LDL-C concentrations often mask substantial loads of proatherogenic lipoproteins.[18]

Both fasting and non-fasting lipid assessments serve for initial screening; however, fasting profiles gain preference amid hypertriglyceridemia since triglyceride levels fluctuate markedly with meals and insulin resistance, while postprandial surges may distort LDL-C computations. Fasting triglycerides thus offer superior risk estimation, whereas apo B and non-HDL-C function as robust indicators of particle number irrespective of prandial state. Triglyceride-enriched remnants including chylomicrons and VLDL characterize hypertriglyceridemia in diabetes and metabolic syndrome, with their prolonged circulation driving atherogenesis and constituting residual risk despite optimized LDL-C.[19]

ApoB measurement holds particular utility in South Asian populations, precisely quantifying total atherogenic particles and associating more robustly with cardiovascular outcomes than LDL-C amid elevated triglycerides or adiposity. Advanced techniques quantifying sd-LDL subfractions, when accessible, enhance diagnostic precision, given sd-LDL enrichment as a signature of South Asian diabetic dyslipidemia.[20] Screening merits initiation at diabetes diagnosis, with annual repetition and accelerated intervals for suboptimal glycemic regulation, manifest CVD, or lipid-modifying treatments. Prompt phenotype recognition proves vital, frequently antedating clinical events and sustaining residual risk post-glycemic optimization, thus underscoring targeted diagnostics for effective management.[21]

Fasting ≥8 hours isolate predominantly hepatic lipoproteins, contrasting non-fasting profiles that incorporate intestinal remnants for a comprehensive 24-hour atherogenic exposure snapshot. Contemporary assays enable reliable non-fasting LDL-C derivation, with evidence affirming equivalent cardiovascular risk prediction across prandial states. While fasting triglycerides represent the benchmark in mild, moderate, or severe hypertriglyceridemia, permitting non-fasting evaluations

streamlines routine practice, surmounting adherence obstacles and broadening diabetic risk appraisal.[22]

Accurate ADD evaluation hinges on strategic fasting/non-fasting threshold application.[23] Fasting lipid panels become imperative when non-fasting triglycerides surpass 440 mg/dL, signaling potential secondary etiologies or severe familial hypertriglyceridemia. Likewise, non-fasting non-HDL-C ≥220 mg/dL raises familial lipid disorder suspicion, prompting fasting confirmation, especially alongside premature cardiovascular disease family history. Fasting testing endures as the cornerstone for familial hyperlipidemia screening and monitoring.[24]

Moderate non-fasting triglyceride elevations (~175 mg/dL) require no mandatory fasting verification but necessitate lifestyle guidance. Non-fasting triglycerides ≥200 mg/dL justify repeat fasting profiling within 2–4 weeks to validate persistence and inform intervention. Established lipid thresholds provide a practical framework for identifying heightened atherogenic burden in patients with diabetes.[25] Fasting triglyceride concentrations at or above 150 mg/dL, or nonfasting values ≥175 mg/dL, are considered abnormal and signal residual risk. NonHDLC levels ≥145 mg/dL in the fasting state or ≥150 mg/dL when nonfasting denote excess atherogenic lipoproteins, while LDLC values ≥115 mg/dL are clinically relevant for risk stratification.[26] Apo B concentrations ≥100 mg/dL further identify highrisk dyslipidemia patterns not captured by routine lipid panels. In addition, remnant cholesterol levels ≥30 mg/dL (fasting) or ≥35 mg/dL (nonfasting) reflect increased residual risk.[16, 27] Collectively, these thresholds underscore the importance of comprehensive lipid assessment in ADD and provide a framework for intensifying therapy to reduce cardiovascular morbidity and mortality. Incorporating prandialspecific cutoffs further strengthens diagnostic workflows by balancing feasibility with accuracy, thereby ensuring that highrisk South Asian patients with diabetes are appropriately prioritized for early identification and targeted intervention.[28,29]

1.4 Management

Effective dyslipidemia management in diabetes requires an integrated strategy targeting lipid derangements, metabolic abnormalities, and modifiable cardiovascular risks.[30] Lifestyle optimization forms the foundation, particularly for hypertriglyceridemia, encompassing smoking cessation, routine exercise, structured nutrition, adequate sleep, and mental health support.[31] Recent American Diabetes Association (ADA) guidance advises intensified lifestyle measures and glycemic control in diabetics with elevated triglycerides or low HDLC. Alcohol intake should be restricted to ≤2 drinks/day in men and ≤1 drink/day in women when triglycerides are 150–499 mg/dL, with complete abstinence mandated at ≥500 mg/dL to prevent pancreatitis. Achieving 5–10% weight loss reduces triglycerides (~20%), enhanced by high-protein, lower-carbohydrate diets while avoiding refined sugars, high-glycemic foods, and fructose beverages (>10% energy intake). Maintain total fat at 30–35% calories (favoring monounsaturated fatty acids [MUFAs]/polyunsaturated fatty acids [PUFAs]) for mild-moderate hypertriglyceridemia, reducing to 20–25% (500–999 mg/dL) or <5% (≥1000 mg/dL), potentially incorporating medium-chain triglycerides. The ADA guidance prescribes at least 150 minutes per week of moderateintensity or 75 minutes per week of vigorousintensity exercise, with recognition of variability in individual responses.

Pharmacotherapy becomes necessary when lifestyle and metabolic optimization fail to meet lipid goals.[32] Attaining very low LDL-C levels through statins represents the primary means to counter ADD's effects. Tailor therapy to the dominant lipid phenotype using agents with established cardiovascular outcomes benefits. Initiate high-intensity statins in high/very high CVD-risk diabetics regardless of baseline lipids.[33] Given dyslipidemia's multifactorial etiology, combination regimens often prove essential for target achievement. Statins serve as the primary pharmacologic therapy for dyslipidemia control in diabetic individuals. Their use extends beyond genetic dyslipidemias (e.g., familial hypercholesterolemia) to primary and secondary CVD prevention, with established benefits in high-risk diabetics, including those with >40 years of disease duration.[34] In the absence of contraindications, moderate-to-high-intensity statins are recommended as first-line therapy according to current American Association of Clinical Endocrinology (AACE) guidelines.[35] Despite intensive statin regimens, significant residual cardiovascular risk persists in primary prevention populations harbouring multiple risk factors. Landmark statin clinical trials enrolling substantial diabetic cohorts demonstrate clear reductions in CVD events.[36]

Hypertriglyceridemia management primarily aims to mitigate atherosclerotic cardiovascular risk and prevent pancreatitis. Cardiovascular outcome data supporting triglyceride-lowering lags substantially behind LDL-C reduction evidence, prioritizing LDL-C target attainment for CVD prevention. Conversely, aggressive TG reduction remains essential for pancreatitis prophylaxis, as risk escalates sharply above 500 mg/dL. In statin-treated diabetics attaining LDL-C goals yet exhibiting persistent hypertriglyceridemia (≥200 mg/dL), therapy must distinguish LDL particle-mediated versus triglyceride-rich lipoprotein-driven residual risk. Ezetimibe, bempedoic acid, and PCSK9 inhibitors primarily target LDL-C and suit

scenarios of suboptimal LDL-C control or incremental LDL-related risk reduction; their triglyceride-lowering effects remain modest.[37]

Ezetimibe inhibits intestinal cholesterol absorption and, combined with statins, produces additive LDL-C lowering with improved target attainment. This enables lower statin doses while maintaining efficacy and minimizing adverse effects.[38] Ezetimibe shows neutral or favorable glycemic effects in the long term. Guidelines recommend its addition when LDL-C goals persist despite maximal statins; the American Heart Association (AHA) endorses statin-ezetimibe for (≥50% LDL-C) reduction in high-risk diabetics (10-year CVD risk ≥20%).[1] Bempedoic acid, a liver-selective ATP citrate lyase inhibitor prodrug, reduces hepatic cholesterol synthesis upstream of 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase, ideal for statin-intolerant patients.[39,40] PCSK9 inhibitors (alirocumab, evolocumab) achieve 55–72% LDL-C reductions in high-risk groups, including diabetic dyslipidemia, without glycemic detriment. Guidelines support their use when LDL-C (≥70 mg/dL) remains despite maximal statins ± ezetimibe, demonstrating safety and superior efficacy beyond statins alone.[41,42]

In ADD patients with several co-existing risk factors and triglyceride concentrations above 150 mg/dL on statins, icosapent ethyl provides incremental cardiovascular benefit.[43] For triglycerides (≥500 mg/dL), prioritize statins and ezetimibe as an add-on treatment, initially to avert pancreatitis before fibrates or alternatives.[44] Icosapent ethyl addresses residual risk in statin-treated patients with persistent triglycerides, alongside glycemic optimization.[46] Fenofibrate and saroglitazar (dual peroxisome proliferator-activated receptor alpha/ Gamma [PPAR-α/γ] agonist) effectively reduce triglycerides and improve atherogenic profiles in diabetic dyslipidemia, with South Asian trials confirming substantial TG lowering and good tolerability as statin add-ons.[45] Saroglitazar has demonstrated robust efficacy in both real-world and trial settings for diabetic dyslipidemia.[48,49] Niacin lowers TG and raises HDL-C but is limited by flushing, glycemic worsening, and lack of cardiovascular benefit atop statins.[47] Agent selection considers renal function, glycemic/edema risks, interactions, and lipid phenotype, with non-HDL-C and apoB monitoring.

Findings/discussion from consensus survey

This national consensus survey provides robust, practice-oriented guidance for identifying and managing atherogenic dyslipidemia in Indian patients with diabetes, emphasizing phenotype-based strategies that move beyond traditional LDL-C-centric approaches. In this Consensus Statement, the authors report the final statements and recommendations, together with a summary of the broader literature on the prevalence, diagnosis, and pharmacological management of ADD.

2.1 Disease Prevalence

The survey demonstrated a strong and consistent expert consensus on the definition and clinical significance of ADD in Indian diabetic patients, as presented in Figure 1. Overall, 95.21% of respondents expressed consensus that the coexistence of raised triglycerides (≥150 mg/dL), reduced HDLC (thresholds of <40 mg/dL in men and <50 mg/dL in women), and abnormal sdLDLC levels should define patients as having ADD. This marked level of agreement establishes a clear and clinically relevant diagnostic framework for identifying this high-risk dyslipidemia phenotype in the Indian population.

Experts prodigiously recognized insulin resistance as a central pathophysiological abnormality in South Asians, with 96.57% agreeing that it is more prevalent and occurs earlier in this population. This consensus underscores the importance of considering ethnic-specific metabolic characteristics when assessing cardiovascular risk in Indian patients, as traditional lipid-centric models may inadequately capture the underlying risk driven by insulin resistance.

Hypertriglyceridaemia was widely endorsed as a major cardiovascular risk factor, with 89.73% of respondents agreeing that elevated triglycerides should be considered as clinically important as LDL-C in cardiovascular risk assessment. This reflects an evolving understanding that triglyceride-rich lipoproteins represent an independent and meaningful contributor to atherogenic risk, particularly in Asian populations characterized by higher carbohydrate consumption and greater insulin resistance.[51]

Importantly, 84.93% of experts agreed that elevated triglyceride levels contribute significantly to residual cardiovascular risk, even among patients who reach recommended LDLC thresholds with statin therapy. This finding highlights the limitations of an LDL-C-driven treatment paradigm and reinforces the need for comprehensive lipid management approaches that address triglyceride abnormalities to achieve optimal cardiovascular risk reduction in Indian diabetic patients.

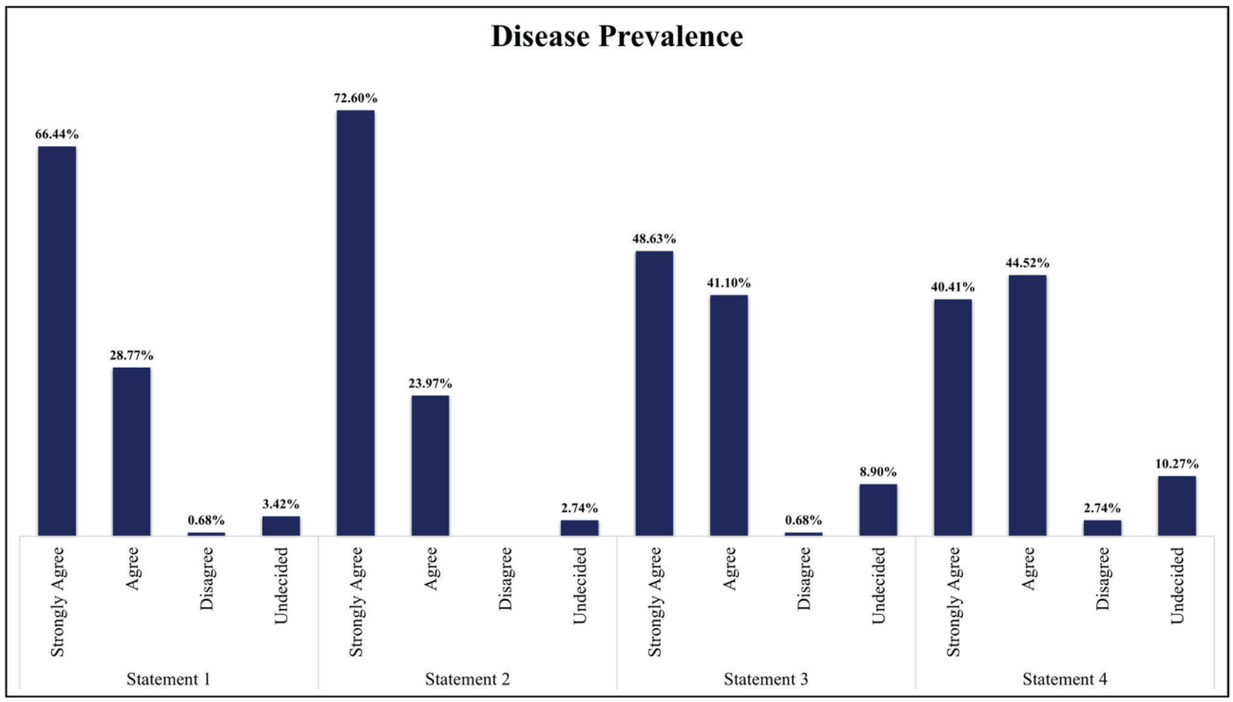

Fig 1. Disease prevalence and agreement levels on dyslipidemia-related cardiovascular risk in Indian and South Asian populations.

Consensus Statements

1. Indian diabetic patients with elevated triglycerides 150 mg/dL, low HDL-C (40 and 50 mg/dL for male and female, respectively) and abnormal sd-LDL levels should be considered as ADD patients.
2. In the South Asian population, insulin resistance is more common and occurs at an earlier age. It is one of the root causes contributing to the increasing incidence of ADD in India.
3. Considering the Asian population has higher insulin resistance and greater carbohydrate consumption, elevated TG is more common than elevated LDL-C. So, like LDL-C, hypertriglyceridemia should be considered a significant CVD risk factor.
4. In patients with controlled LDL-C on statin therapy, elevated triglycerides play a crucial role in increasing residual CVD risk.

2.2 Diagnostic Considerations

The diagnostic component of the survey demonstrated a strong yet pragmatic consensus among experts as presented in Figure 2. Fasting triglyceride measurements were considered more reliable by 84.25% of respondents; however, there was clear acknowledgment of the clinical utility of non-fasting triglyceride values when fasting samples are not feasible, reflecting a balance between diagnostic accuracy and real-world applicability. A triglyceride threshold of ≥150 mg/dL for defining hypertriglyceridemia in Indian patients achieved 78.09% agreement, indicating broad acceptance while also highlighting ongoing discussion regarding population-specific cut-off values. There was robust support (92.47%) for incorporating non-HDL-C and apo B as ancillary determinants of cardiovascular risk alongside LDL-C and triglycerides, consistent with a comprehensive, multi-marker approach to atherogenic risk assessment in diabetic dyslipidaemia.[52] Additionally, the TyG was endorsed by 78.09% of respondents acting as a surrogate measure for insulin resistance and cardiovascular risk, although the relatively higher proportion of undecided responses suggests that its routine clinical adoption remains an evolving practice.

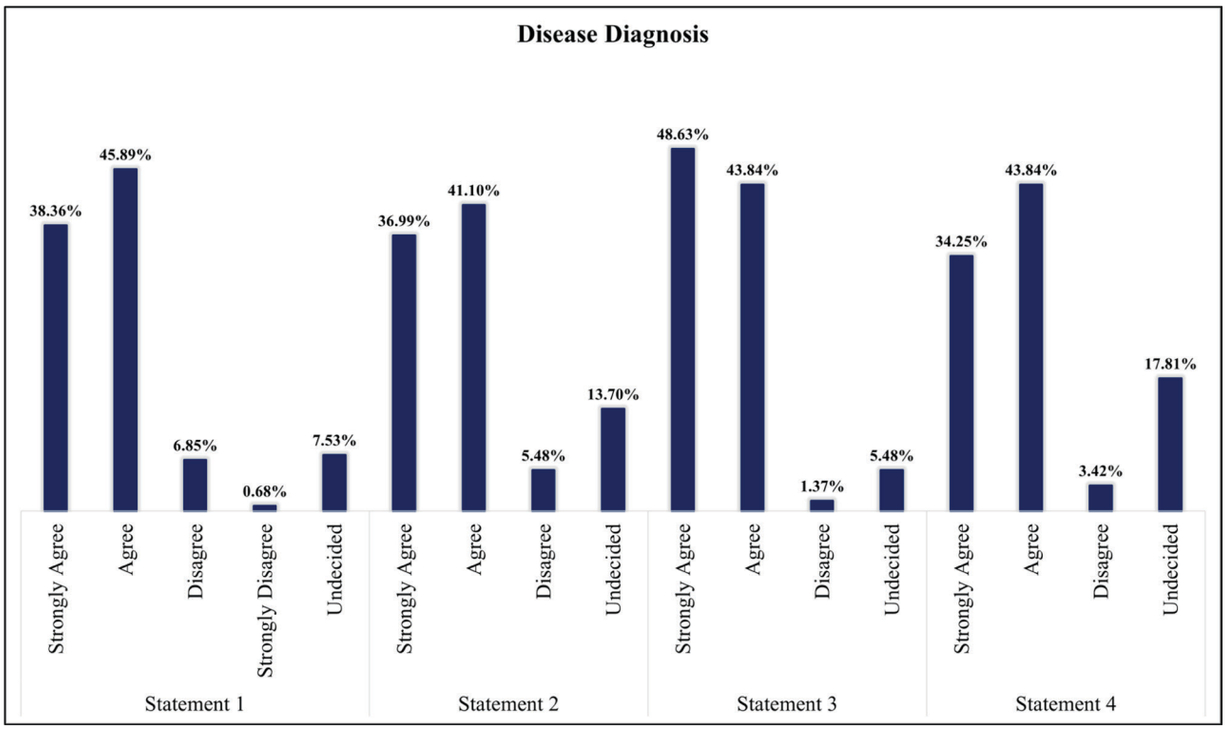

Fig 2. Agreement levels on diagnostic approaches and biomarkers for dyslipidemia and cardiovascular risk assessment in Indian patients.

Consensus Statements

1. Fasting TG should be considered more reliable than non-fasting TG for detecting dyslipidaemia. However, if fasting TG is not available, non-fasting TG can be used as an alternative.
2. Given that elevated TG plays a key role in CVD risk, TG levels above 150 mg/dL should be used as the cut-off for defining hypertriglyceridemia in Indian patients.
3. In Indian diabetic patients, non-HDL-C and Apo B should be considered as secondary markers for CVD risk assessment, along with TG and LDL-C.
4. The TyG is a validated marker for predicting insulin resistance and CVD. It should be regularly calculated in Indian diabetic patients suffering from dyslipidaemia.

2.3 Management Strategies

The therapeutic management section demonstrated the strongest consensus as presented in Figure 3. An overwhelming 93.83% of respondents endorsed lifestyle modifications as first-line therapy, with 78.08% expressing strong agreement. This reflects evidence-based prioritization of non-pharmacological interventions before initiating medical management. Statin therapy as first-line pharmacological management achieved near-universal acceptance at 95.89%, consistent with global guidelines and extensive clinical trial evidence supporting statins in diabetic populations. Treatment thresholds for hypertriglyceridemia showed interesting patterns. For initiating medication to reduce CVD risk, 78.77% agreed with a threshold of >200 mg/dL, while extreme hypertriglyceridemia (>500 mg/dL) requiring treatment to prevent pancreatitis achieved the highest consensus in the entire survey at 96.57%. This clear differentiation between cardiovascular risk reduction and acute pancreatitis prevention reflects evidence-based threshold distinctions (53). The add-on therapy section revealed more nuanced perspectives. When LDL-C is controlled, but triglycerides remain elevated (>200 mg/dL), 73.97% supported adding TG-lowering drugs (saroglitazar, fenofibrate, icosapent ethyl, niacin) along with statins.

In contrast, a similar proportion (73.97%) supported non-statin agents (ezetimibe, bempedoic acid, PCSK9 inhibitors), but with notably higher disagreement (13.01%) compared with TG-lowering drugs. This suggests greater clinical confidence in agents with specific triglyceride-lowering mechanisms rather than primarily LDL-lowering agents for managing hypertriglyceridemia. For severe hypertriglyceridemia (>500 mg/dL), 94.52% agreed that TG-lowering drugs should be initiated regardless of statin use, prioritizing acute pancreatitis prevention. Saroglitazar achieved exceptional endorsement (93.84%) as the preferred add-on option for managing hypertriglyceridemia in diabetic patients. This strong preference reflects the drug's dual benefits of insulin sensitization and glycaemic control alongside triglyceride reduction, making it particularly suitable for the diabetic dyslipidaemia phenotype prevalent in Indian patients.

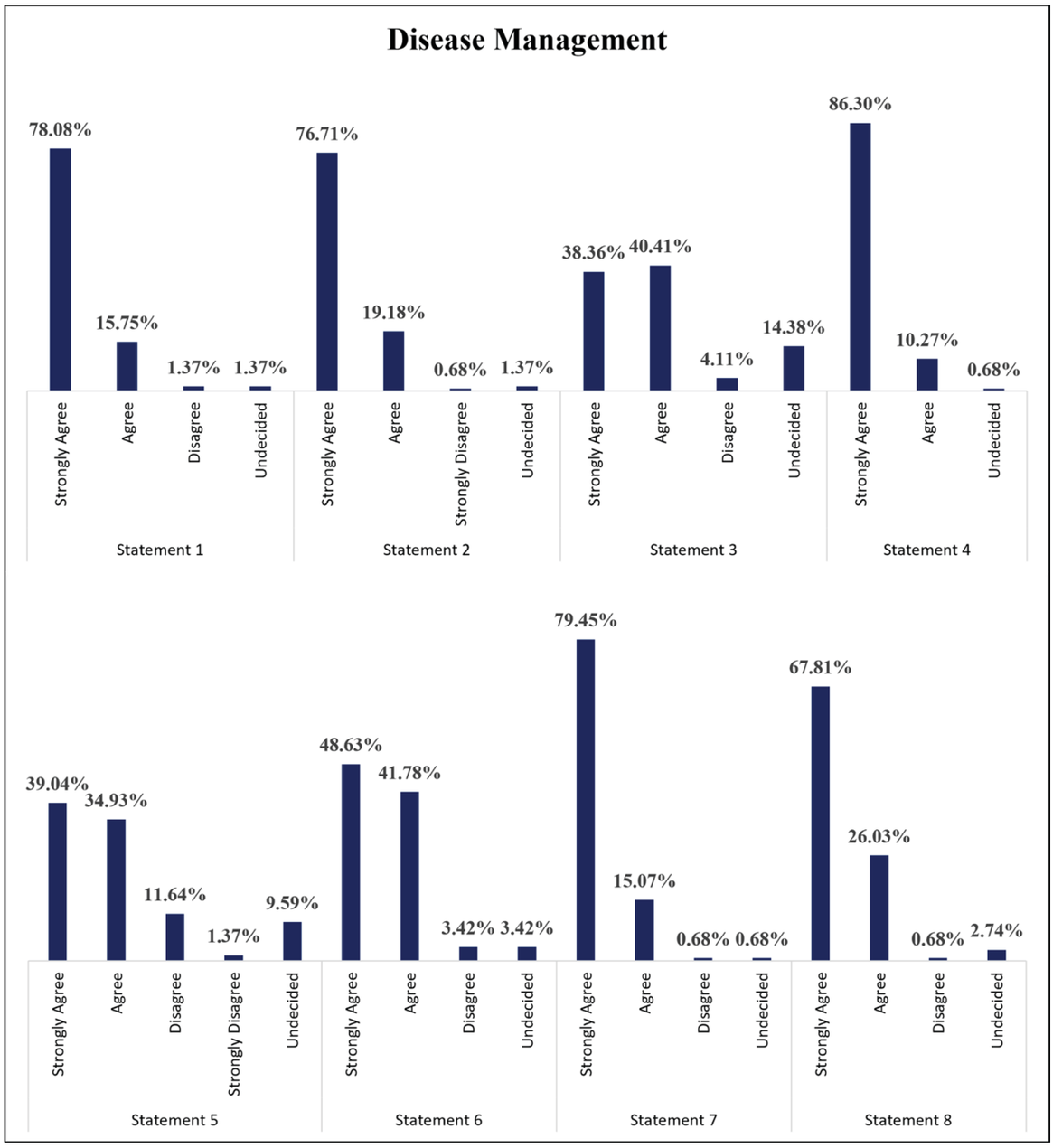

Fig 3. Agreement levels on therapeutic strategies for the management of diabetic dyslipidemia and hypertriglyceridemia in Indian patients.

Consensus Statements

1. In the management of diabetic dyslipidemia, lifestyle modifications like weight reduction, low-calorie intake, meditation, and stress reduction are always considered as a first-line therapy before considering medical management.
2. Statin therapy is the first-line medication for managing dyslipidaemia in diabetes patients.
3. In India, the ideal threshold level for TG should be 200 mg/dL to initiate medication to reduce the risk of CVD.
4. If TG levels exceed 500 mg/dL, they should be treated with appropriate medication to prevent the risk of pancreatitis.
5. In patients with controlled LDL-C post statin therapy, if TG is >200 mg/dL, non-statin agents (ezetimibe, bempedoic acid, PCSK9 inhibitors) should be added to reduce TG level along with statin.
6. In patients with controlled LDL-C on statin therapy, if TG is >200 mg/dL, a TG-lowering drug (Saroglitazar, Fenofibrate, Icosapent ethyl, Niacin) should be added along with statin.
7. In patients with TG levels >500 mg/dL, TG-lowering drugs should be started (with or without statin) to prevent acute pancreatitis.
8. Saroglitazar is the preferred add-on option to statin therapy for managing hypertriglyceridemia, as it has insulin-sensitizing properties and improves glycemic control along with a reduction in TG in diabetic dyslipidaemia patients.

This consensus survey represents expert opinion requiring prospective validation; it does not provide empirical evidence from interventional or outcomes-based studies. The panel may not fully represent all Indian clinicians, particularly those practising in resource-limited settings. Findings are specific to the Indian/South Asian context and may not be directly generalizable to other populations. Recommendations are based on the current evidence base and should be periodically updated as new data emerge.

Conclusion

Consensus expert physicians recognized ADD as a major determinant of residual cardiovascular risk in Indian and broader South Asian patients with T2DM. The panel endorsed a phenotype-driven approach for diagnosis and management, emphasizing early identification and targeted treatment of hypertriglyceridemia in conjunction with LDL-C reduction, and acknowledged non-HDL-C and apo B as complementary indicators. Practical guidance on therapeutic sequencing and intervention thresholds supports integrated metabolic risk management tailored to ethnicspecific metabolic characteristics and real-world clinical practice, with lifestyle measures prioritized, statins as first-line pharmacotherapy, and TG-lowering agents added when triglycerides remain elevated. Consensus further highlighted saroglitazar as a preferred adjunct in patients with persistent hypertriglyceridemia despite statin therapy, reflecting evidence of improvements in triglycerides, glycemic parameters, and insulin sensitivity, and reinforcing its role in cardiovascular risk reduction in ADD.

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