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EVALUATION OF STATIN UTILISATION PATTERN AND ADVERSE EFFECTS IN PATIENTS WITH DYSLIPIDEMIA

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ABSTRACT

Dyslipidemia contributes to atherosclerotic cardiovascular disease (ASCVD), and statins remain central to lowering low-density lipoprotein cholesterol (LDL-C) and preventing cardiovascular events in patients whose risk warrants treatment. This narrative review evaluates statin prescribing patterns, adherence and discontinuation, adverse effects, and approaches to safer long-term use. Priority was given to current guidelines, randomized trials and meta-analyses, authoritative safety statements, and observational utilisation studies, including Indian hospital-based evidence. Indian studies frequently reported atorvastatin and rosuvastatin, but observed prescribing patterns varied by setting, population, period, and design and should not be pooled into one prevalence estimate. Rational use requires individualized intensity, interaction review, appropriate baseline assessment, symptom-directed investigations, lipid-response follow-up, and shared decision-making when intolerance is suspected. Contemporary Indian evidence remains geographically and clinically limited, highlighting the need for multicentre studies that standardize indication, intensity, adherence, outcomes, and adverse-event definitions.

KEYWORDS: Statins; Dyslipidemia; Drug utilisation; Adverse drug reactions; Statin-associated muscle symptoms; Pharmacovigilance

INTRODUCTION

Dyslipidemia describes abnormalities in the concentration or composition of circulating lipids and lipoproteins, including elevated low-density lipoprotein cholesterol (LDL-C), raised triglycerides, reduced high-density lipoprotein cholesterol (HDL-C), and increased atherogenic remnant or apolipoprotein B-containing particles. Its clinical importance lies not in a laboratory value alone but in its contribution to the development and progression of atherosclerotic cardiovascular disease (ASCVD), including coronary heart disease, ischemic stroke, and peripheral arterial disease. Contemporary guidance increasingly emphasizes a person's overall cardiovascular risk, the magnitude and duration of exposure to atherogenic lipoproteins, and the presence of established vascular disease when selecting treatment.[1,2]

Statins inhibit hepatic 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase, the rate-limiting enzyme in cholesterol synthesis. The resulting decrease in hepatic cholesterol stimulates increased expression of LDL receptors, enhancing the removal of circulating LDL particles. Statins also modestly influence triglycerides and, to varying degrees, HDL-C, but their principal proven clinical benefit is reduction in LDL-C and ASCVD events. Statin intensity refers to the expected LDL-C reduction of a regimen rather than to a universal dose equivalence between every molecule. Guideline recommendations also vary by country, risk calculator, health-system context, and the evidence considered at the time of publication; accordingly, guidance must be interpreted within its intended population.[1,2,17]

Statin-associated adverse effects influence clinical discussions because muscle pain, weakness, laboratory changes, and concerns about diabetes may be attributed to treatment. Muscle symptoms deserve a careful assessment, but symptom reports alone do not establish a causal relationship. Blinded trial evidence suggests that statins produce a small excess of muscle symptoms compared with placebo, concentrated particularly soon after treatment initiation, while many symptoms that occur during therapy also occur without pharmacological exposure. Randomized evidence also supports a small, dose-dependent glycemic effect, which should be considered against the cardiovascular benefit expected for the individual patient. Severe myopathy, rhabdomyolysis, and clinically apparent liver injury are uncommon, but require timely recognition when suspected.[6-9,15]

1. MATERIALS AND METHODS

This manuscript was developed as a narrative literature review, not as a systematic review or meta-analysis. Searches were undertaken using PubMed/MEDLINE and Google Scholar, supplemented by targeted searches of journal platforms, professional-society guideline pages, government regulatory information, and the National Library of Medicine's LiverTox resource. The literature search was updated on 16 October 2026. Scopus and Web of Science were considered relevant discovery sources but were not independently searched through a structured, exportable database workflow for this manuscript; no claim is made that the retrieval was exhaustive. The approach follows the organization expected for a narrative scientific review and does not claim a PRISMA screening process.[1,2,6,17,18]

Sources were prioritized if they were peer-reviewed original research, randomized trials, systematic reviews or meta-analyses, clinical practice guidelines, authoritative scientific statements, or regulatory resources directly relevant to statin use, safety, adherence, and monitoring. Indian studies were considered particularly relevant to the utilisation section, provided that their design, setting, and study population could be identified. Sources were excluded from the synthesis when they were off-topic, duplicated another report, lacked a verifiable bibliographic record, or made clinical claims unsupported by accessible evidence. No article counts are reported because a formal deduplication, title/abstract screening, full-text screening, and data-extraction log was not completed.[10-18]

2. RESULTS AND DISCUSSION

2.1. Overview of Statins and Their Pharmacological Basis

Therapeutic targets and decision thresholds should be interpreted according to the guideline used. The 2026 ACC/AHA multisociety guideline retires the 2018 US cholesterol guideline and updates risk assessment and LDL-C goals; the European Society of Cardiology/European Atherosclerosis Society guidance places emphasis on risk categories and LDL-C targets; and NICE guidance uses a UK-specific framework for primary prevention and secondary prevention. These documents share the core principle that treatment intensity should be proportional to risk, but they differ in risk estimation tools, thresholds, targets, and health-system context. A review of prescribing patterns must therefore state which recommendation is used to judge appropriateness, rather than applying one national threshold to all settings.[1,2,17]

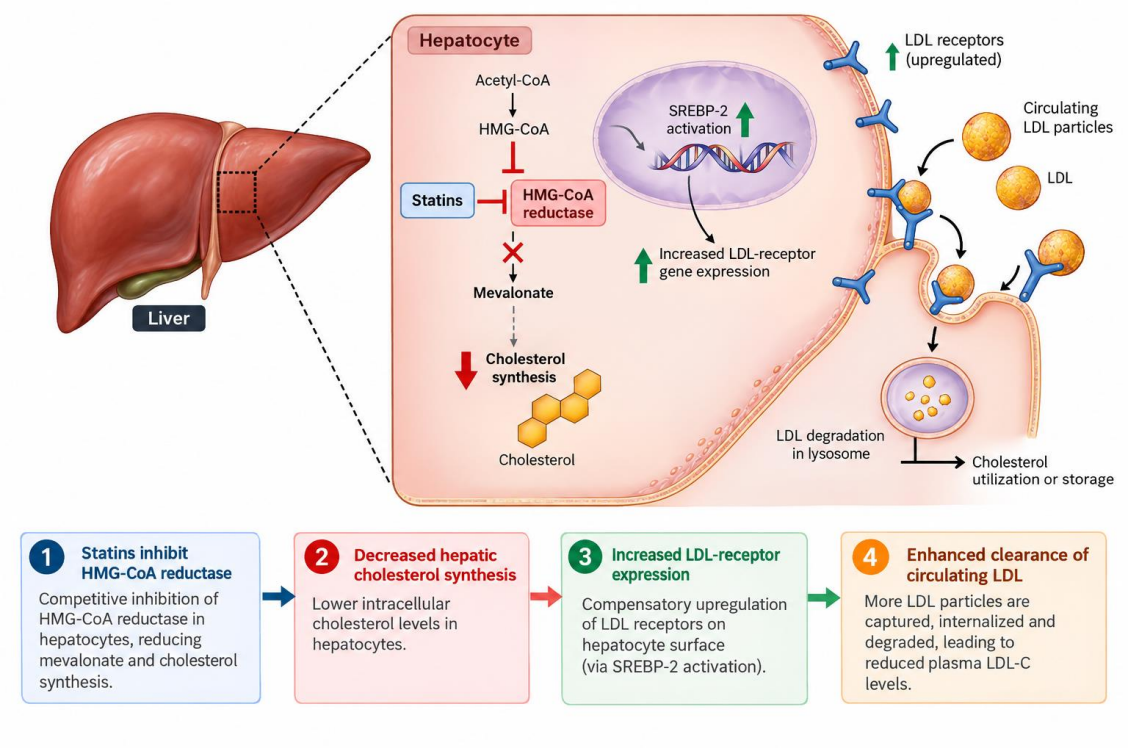

Figure 1. Mechanism of Action of Statins and Their LDL-Cholesterol-Lowering Effect.

2.2. Classification of Statins and Treatment Intensity

Seven statins are used in clinical practice in many countries: atorvastatin, rosuvastatin, simvastatin, pravastatin, fluvastatin, lovastatin, and pitavastatin. Atorvastatin and rosuvastatin have high-potency regimens capable of reducing LDL-C by approximately 50% or more at appropriately selected high doses. Other agents are commonly used at low- or moderate-intensity doses, and no routinely used dose of pravastatin, fluvastatin, lovastatin, or pitavastatin is generally categorized as high-intensity under the conventional US intensity framework. Expected percentage reductions are population estimates, not guarantees for an individual patient.[1,2]

TABLE 1. CLASSIFICATION OF COMMONLY USED STATINS AND THEIR CLINICAL CHARACTERISTICS.

Statin	Representative adult dose range	Conventional intensity classification	Pharmacokinetic / interaction considerations	Clinical prescribing notes
Atorvastatin	10–80 mg once daily	Moderate: 10–20 mg; High: 40–80 mg	CYP3A4 substrate; exposure may rise with strong inhibitors and some interacting drugs.	Widely used high-potency option; select intensity according to risk and response.

Statin	Representative adult dose range	Conventional intensity classification	Pharmacokinetic / interaction considerations	Clinical prescribing notes
Rosuvastatin	5–40 mg once daily	Moderate: 5–10 mg; High: 20–40 mg	Limited CYP metabolism; transporter interactions and renal impairment remain relevant.	Useful when high-intensity lowering is required; dose limitations may apply in severe renal impairment or specific populations.
Simvastatin	10–40 mg once daily	Low: 10 mg; Moderate: 20–40 mg	CYP3A4 substrate; multiple drug interactions; exposure-related myopathy risk.	Avoid new initiation at 80 mg; check product-specific restrictions with interacting medicines.
Pravastatin	10–80 mg once daily	Low: 10–20 mg; Moderate: 40–80 mg	Minimal CYP metabolism; renal function may affect dosing.	May be considered when CYP3A4 interactions are a concern; potency is lower than atorvastatin or rosuvastatin.

2.3. Clinical Indications and Guideline-Based Prescribing

In secondary prevention, statins are indicated for most patients with established ASCVD unless contraindicated or not tolerated, because recurrent-event risk is high and randomized evidence supports clinically meaningful benefit. The intensity chosen depends on guideline recommendations, age, comorbidities, drug tolerance, LDL-C response, and the need for additional therapy. For patients with very high residual LDL-C despite maximally tolerated statin treatment, contemporary guidance supports considering evidence-based non-statin therapies rather than leaving substantial risk untreated. The choice to add a non-statin agent should account for achieved LDL-C, absolute risk, expected benefit, cost, access, and patient preference.[1,2,4]

2.4. Statin Utilisation Patterns in Patients with Dyslipidemia

Drug-utilisation studies describe actual prescribing within defined settings and are essential for assessing whether statins are selected at appropriate intensity, used for suitable indications, and followed up adequately. Their results are difficult to compare when one study samples all patients receiving lipid-lowering drugs, another samples patients with

diabetes, and another focuses on individuals with established coronary disease. Prescriptions may be counted per patient, per encounter, or per dispensed item; these units produce different estimates. A percentage reported below should therefore be read with its study population and denominator in view, rather than interpreted as a national or global prescribing prevalence.[10-14]

TABLE 2. PUBLISHED EVIDENCE ON STATIN UTILISATION PATTERNS IN PATIENTS WITH DYSLIPIDEMIA.

Country / setting	Design and population	Reported utilisation finding	Interpretive limitation
Tertiary-care teaching hospital, North India	Prospective, noninterventional study; 200 dyslipidemic patients; June–August 2000.	Fibrates accounted for 53.5% and statins for 46.5% of lipid-lowering prescriptions; 40% of treated patients did not meet the study's NCEP-II initiation criteria.	Historical prescribing period; criteria and practice differ from current guidelines.
Tertiary-care teaching hospital, southern India	Hospital prescription-pattern assessment of hypolipidaemic medicines.	Atorvastatin was the leading monotherapy (53.4%); atorvastatin plus aspirin was the most frequent co-prescription pattern (20%). Dosing discrepancies were reported by the authors.	A single hospital; aspirin is not a second lipid-lowering agent; "under/overdosing" depends on criteria used.
Tertiary-care teaching hospital, Bengaluru, India	Cross-sectional observational study; 237 adults with type 2 diabetes, July 2021–June 2022.	Of 164 participants reported to have dyslipidemia, 129 were on lipid-lowering treatment; among statins described, rosuvastatin was reported in 54% and atorvastatin in 40%.	Diabetes-focused, single-centre sample; not a national estimate.

2.5. Factors Influencing Statin Prescribing and Utilisation

Clinical risk is the principal determinant of the indication and desired intensity, but prescribing occurs within a broader context. Baseline LDL-C, prior ASCVD, diabetes, hypertension, smoking, chronic kidney disease, family history, and other risk-enhancing factors affect expected benefit. The same LDL-C concentration may lead to different decisions in a young adult with few other risk factors and in a patient with prior myocardial infarction. Guideline tools can standardize the initial assessment, but patient-specific factors

and shared decision-making remain important, particularly when the benefit is modest or the potential burden of treatment is high.[1,2,17

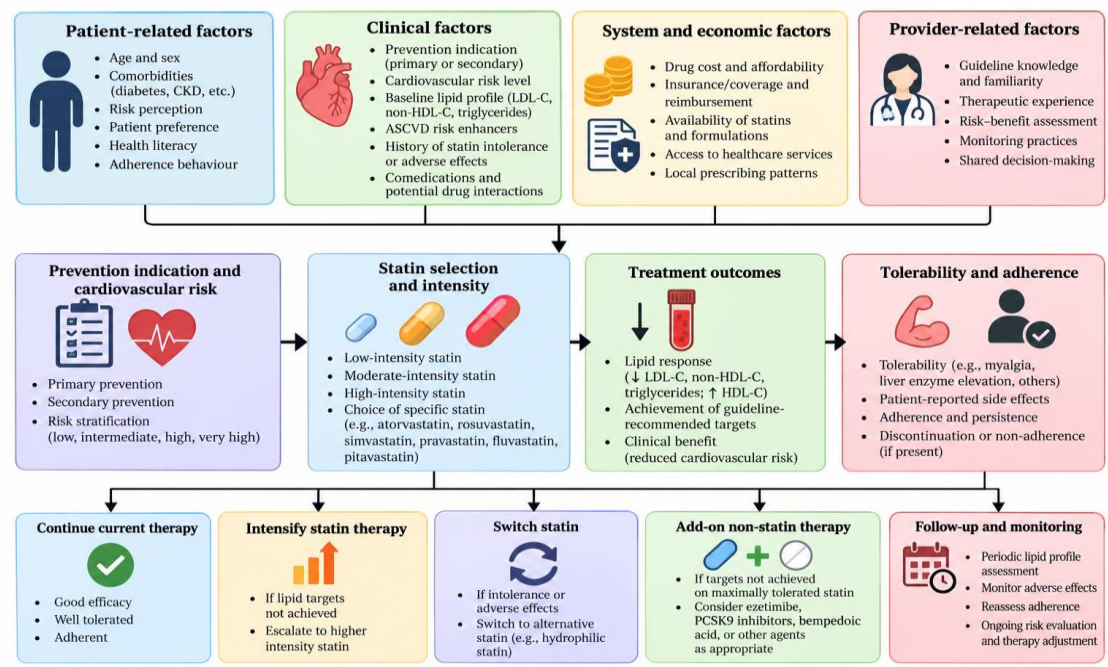

Figure 2. Overview of Statin Utilisation Patterns in Clinical Practice.

2.6. Statin Adherence, Persistence, and Discontinuation

Adherence describes how closely a patient's medicine-taking behavior follows the agreed treatment regimen; persistence describes the duration from initiation until treatment discontinuation. These concepts are related but not interchangeable. A patient may remain on a statin for years but miss frequent doses, indicating poor adherence with persistence, whereas another patient may take every dose correctly for a short period and then stop. Claims-based studies often approximate adherence through proportion of days covered or medication possession ratio, while persistence may be defined by a permissible gap between refills. Different thresholds and data sources limit comparisons between studies.[14]

2.7. Adverse Effects Associated with Statins

A. Muscle-related adverse effects

Statin-associated muscle symptoms (SAMS) are typically described as muscle pain, tenderness, cramps, or weakness, often without a marked creatine kinase (CK) increase. Myalgia is a symptom description, whereas myopathy generally implies clinically meaningful muscle dysfunction or biochemical injury; definitions vary among trials and clinical settings. Rhabdomyolysis is a rare, severe syndrome involving extensive muscle breakdown with risk of myoglobinuria, acute kidney injury, electrolyte disturbance, and systemic complications.

This terminology matters because counting any pain as myopathy can overstate severe toxicity and lead to misleading comparisons across studies.[6,7,9]

B. Hepatic effects

Statins may cause mild, asymptomatic elevations in alanine aminotransferase or other liver enzymes. Such changes are not equivalent to clinically significant drug-induced liver injury and often resolve without progression. Clinically apparent hepatic injury attributable to a statin is rare relative to the large number of exposed patients. In reported cases, the presentation can be hepatocellular, cholestatic, or mixed, and other causes—including viral hepatitis, alcohol, metabolic dysfunction-associated steatotic liver disease, biliary disease, and concomitant medicines—should be considered before causality is assigned.[6,18]

C. Glycemic effects

Randomized evidence supports a small increase in new-onset diabetes diagnoses and a modest worsening of glycemia with statin treatment, with a greater effect generally observed with more intensive regimens. The absolute effect depends on baseline metabolic risk: patients already near the diagnostic threshold are more likely to cross it than metabolically healthy patients. The 2024 CTT individual-participant meta-analysis strengthened the evidence for a dose-related association. This risk should be discussed accurately without suggesting that statins routinely cause diabetes in every user.[8]

D. Other reported adverse effects

Gastrointestinal complaints, headache, sleep concerns, rash, and other nonspecific symptoms are reported during statin treatment, but causal evidence and frequency vary by symptom and agent. Randomized data do not support treating every subjective complaint that occurs after statin initiation as a drug effect. Neurocognitive complaints have received public attention, yet controlled evidence has not established a consistent clinically meaningful pattern of persistent cognitive harm from statins. New, severe, or progressive symptoms still deserve assessment on their own merits and should not be dismissed based on the average trial result.[6,15,23]

2.8. Risk Factors and Clinically Important Drug Interactions

Statin-associated toxicity is influenced by systemic exposure, patient susceptibility, and the pharmacological properties of the individual agent. Important risk factors include higher prescribed dose, recent initiation or escalation, older age in the presence of frailty or polypharmacy, renal or hepatic impairment, untreated hypothyroidism, acute infection or systemic illness, previous statin-associated muscle symptoms, and concurrent medicines that increase statin concentrations. These factors do not necessarily prohibit treatment; they

prompt a more deliberate selection of molecule and dose, explicit counselling, and closer assessment when symptoms occur.[6,9,16]

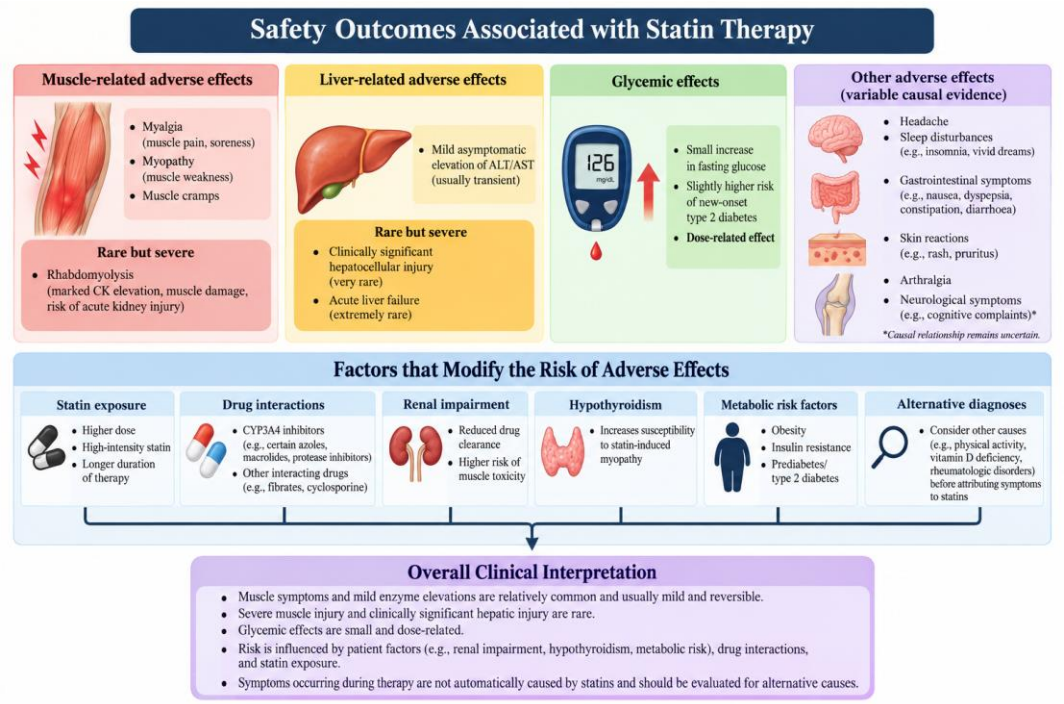

Figure 3. Major Statin-Associated Adverse Effects and Contributing Risk Factors.

2.9. Monitoring, Prevention, and Management of Adverse Effects

Before initiation, the prescriber should document the indication, cardiovascular risk context, baseline lipid profile, relevant medical history, previous intolerance, pregnancy-related considerations when applicable, and a review of interacting medicines. Baseline liver transaminases are commonly recommended; renal function and thyroid testing are guided by the patient's condition and by suspected risk factors rather than automatically required for every patient. CK is not generally a routine screening test in asymptomatic people, but baseline measurement may be reasonable where there is a previous history of muscle disease, prior significant statin-associated symptoms, or another specific clinical concern.[6,9,16,17]

TABLE 3. PREVENTION, MONITORING, AND MANAGEMENT OF STATIN-ASSOCIATED ADVERSE EFFECTS.

Clinical issue	Assessment or approach	When relevant
Before starting treatment	Document indication and cardiovascular risk; obtain baseline lipid profile and liver transaminases; review history, pregnancy considerations, and all medicines.	Routine pre-treatment assessment; renal/thyroid testing when patient-specific factors indicate.

Clinical issue	Assessment or approach	When relevant
Response to treatment	Repeat lipid profile and liver transaminases according to local guideline; review dose, adherence, secondary causes, and target response.	After initiation or treatment change; then intervals specified in guideline or clinical circumstances.
Muscle pain, tenderness, or weakness	Characterize symptoms, examine alternative causes and interactions; measure CK when unexplained symptoms occur.	Symptom-directed, not routine repeated CK testing in all asymptomatic patients.
Severe muscle injury suspected	Urgent assessment; CK, renal function, urine findings, electrolytes, and clinical monitoring as indicated.	Marked weakness, severe pain, dark urine, systemic illness, or concern for rhabdomyolysis.

2.10. Clinical Benefits and the Risk–Benefit Assessment

The evidence base for statins includes large randomized trials and meta-analyses showing reductions in major coronary and vascular events. The 4S trial demonstrated benefit of simvastatin in patients with coronary heart disease, while the Heart Protection Study showed benefit in a broad high-risk population, and JUPITER demonstrated benefit in a selected primary-prevention population with elevated high-sensitivity C-reactive protein and LDL-C below traditional treatment thresholds. The populations and trial endpoints differed, so their event rates should not be compared directly as if the trials were interchangeable. Their collective value is that they show benefit across a range of risk settings when patients match the studied indication.[22-24]

CONCLUSION

Statins remain a cornerstone of dyslipidemia management because lowering LDL cholesterol reduces atherosclerotic cardiovascular events, with the greatest absolute benefit generally expected in patients at highest baseline risk. The available utilisation literature, including Indian hospital-based studies, shows frequent use of atorvastatin and rosuvastatin in the populations examined but does not support a single estimate of prescribing patterns across all patients with dyslipidemia. Appropriate use must be judged in relation to the patient's indication, cardiovascular risk, required LDL-C reduction, selected dose intensity, treatment response, and the guideline relevant to the setting.

Muscle symptoms are an important reason for concern and discontinuation, but they should not automatically be classified as statin-caused myopathy. Severe muscle injury and clinically apparent hepatic injury are uncommon; a small, dose-related glycemic effect is better

established and should be balanced against the vascular benefit of treatment. A careful medication history, interaction review, appropriate baseline assessment, symptom-directed investigations, shared decision-making, and follow-up of lipid response can help reduce preventable harm. When intolerance is suspected, clinician-guided dose adjustment, switching, or use of additional lipid-lowering therapy may preserve protection more effectively than unplanned permanent discontinuation.

The major evidence gap is the limited availability of contemporary, representative, longitudinal utilisation data that link prescriptions with adherence, achieved lipid levels, adverse events, and cardiovascular outcomes, particularly across diverse Indian care settings. Future work should use standardized definitions and transparent denominators to improve comparisons and guide interventions. Ultimately, the goal is evidence-based, individualized, appropriately monitored statin therapy that maintains cardiovascular protection while responding responsibly to each patient's clinical circumstances and preferences.

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