MDForLives

THE GLOBAL PULSE OF HEALTHCARE · CARDIOLOGY INSIGHT REPORT

LDL-C Targets in ASCVD: Why Secondary Prevention Still Falls Short

A cardiologist-focused look at how LDL-C goals translate into treatment intensification, where nonstatin access and statin intolerance slow progress, why long-term control weakens after acute coronary events, and how Lp(a) and EHR-supported follow-up are entering the next phase of secondary prevention.

LDL-C goal → Intensification → Access → Persistence → Residual risk

71
Survey records

6
Countries represented
USA · UK · Canada · Italy · France · Germany

69%
Completion rate

WHY THIS REPORT

Secondary prevention now has a lower LDL-C destination. The harder question is how consistently cardiology practice can get patients there and keep them there.

The 2026 ACC/AHA multisociety guideline on dyslipidemia reintroduced explicit LDL-C and non-HDL-C treatment goals and recommends LDL-C below 55 mg/dL for patients at very high risk of recurrent ASCVD events. It also recommends measuring lipoprotein(a), or Lp(a), at least once in adulthood. In Europe, the 2025 ESC/EAS focused update reinforced aggressive LDL-C lowering and early treatment intensification, including during an acute coronary syndrome admission when the existing regimen is unlikely to reach the patient's goal.

The therapeutic toolkit is broader than it was a decade ago. Statins remain foundational, while ezetimibe, PCSK9 monoclonal antibodies, bempedoic acid, and inclisiran can provide additional LDL-C reduction in appropriate patients. Yet a larger menu does not automatically produce better implementation. Every intensification decision still has to pass through patient tolerance, payer rules, prior authorization, affordability, dispensing, follow-up, repeat testing, and long-term persistence.

The MDForLives cardiology survey examines that delivery problem from the cardiologist's perspective. It asks how large the secondary-prevention population is, how often very-high-risk patients reach LDL-C below 55 mg/dL, what prompts nonstatin escalation, which administrative barriers dominate, which add-on therapy is chosen first, how common statin-associated symptoms appear in practice, how intolerance is managed, where post-ACS control is lost, how Lp(a) testing is being used, and what system change clinicians believe would close the LDL-C gap most effectively.

> **Methodology and data note.** Audience: cardiologists. Survey ID 8953232. The source export contains 71 survey records, a 69% completion rate, and responses from the USA, UK, Canada, Italy, France, and Germany. Question-level bases vary from 56 to 52 across the substantive items. The hidden country-punching item is used only for geography metadata. Administrative tracking, bad-ID, required-text, after-question, and honeypot fields are excluded from interpretation.

EXECUTIVE SUMMARY

Six signals that explain why secondary-prevention LDL-C control is still a pathway problem

INSIGHT 01

The LDL-C goal is reached inconsistently

46.4% estimate that only 40%–59% of their very-high-risk ASCVD patients achieve LDL-C below 55 mg/dL.

INSIGHT 02

Escalation is usually target-driven

55.6% say LDL-C remaining above target despite maximally tolerated statin therapy is the most common trigger for adding or initiating a nonstatin agent.

INSIGHT 03

Ezetimibe remains the leading first add-on

41.5% choose ezetimibe as their typical first nonstatin therapy after a high-intensity statin does not reach the LDL-C goal.

INSIGHT 04

Step therapy is the biggest access barrier

34.0% identify requirements to fail multiple statins and ezetimibe first as their single greatest administrative challenge.

INSIGHT 05

Rechallenge still leads statin-intolerance management

38.5% first try a different statin, lower dose, or alternate-day regimen when a patient reports statin intolerance.

INSIGHT 06

EHR alerts lead the proposed system fixes

36.5% say automatic EHR alerts for unachieved LDL targets would have the highest impact on closing the guideline gap.

01

SECONDARY-PREVENTION WORKLOAD

Secondary prevention is a major share of the cardiology panel, not a narrow subgroup handled only in specialized lipid clinics

39.3% estimate that 50%–74% of their adult patient panel requires secondary cardiovascular prevention.

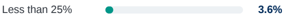
CONTEXT

Established ASCVD creates a recurring management problem rather than a one-time treatment decision. Patients with prior acute coronary syndrome, revascularization, peripheral artery disease, ischemic stroke, or other atherosclerotic disease return repeatedly for medication review, risk-factor control, and decisions about whether preventive therapy is sufficiently intensive.

WHAT THE DATA SHOWS

The largest group, 39.3%, say patients requiring secondary cardiovascular prevention make up 50%–74% of their adult panel. Another 19.6% place that share at 75% or higher, 37.5% at 25%–49%, and only 3.6% below 25%.

Share of the adult panel requiring secondary cardiovascular prevention

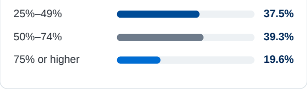


THE INSIGHT

For most respondents, secondary prevention occupies a substantial fraction of day-to-day cardiology practice. That matters because even modest workflow friction can affect many patients when the same intensification, authorization, adherence, and follow-up steps repeat across a large panel.

WHY IT MATTERS

The survey should therefore be read as a systems question as much as a pharmacotherapy question. A process that depends on one clinician remembering every LDL-C target, authorization deadline, repeat lipid panel, and tolerance discussion becomes difficult to scale when established ASCVD represents half or more of the clinic for many respondents.



02

LDL-C TARGET ATTAINMENT

The dominant estimate is that only 40%–59% of very-high-risk ASCVD patients reach LDL-C below 55 mg/dL

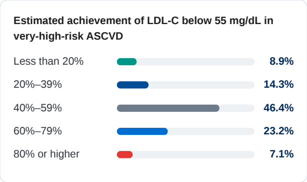
46.4% place LDL-C goal attainment in the 40%–59% range among their very-high-risk ASCVD patients.

CONTEXT

The 2026 ACC/AHA multisociety dyslipidemia guideline recommends an LDL-C goal below 55 mg/dL for very-high-risk secondary-prevention patients. That makes target attainment a useful lens on whether risk identification, medication selection, access, adherence, and monitoring are working together.

WHAT THE DATA SHOWS

46.4% estimate that 40%–59% of their very-high-risk patients achieve LDL-C below 55 mg/dL. 23.2% estimate 60%–79%, 14.3% estimate 20%–39%, 8.9% estimate less than 20%, and only 7.1% estimate that at least 80% reach the target.


Estimated achievement of LDL-C below 55 mg/dL in very-high-risk ASCVD

THE INSIGHT

The distribution is centered on partial control rather than near-universal control. The survey does not identify one cause for that gap, but the later questions show multiple plausible pressure points: delayed escalation, statin intolerance, payer rules, patient discontinuation, follow-up failure, and missed retesting.

WHY IT MATTERS

For cardiologists, the practical implication is to treat an LDL-C value above goal as the start of a workflow rather than the end of a clinic note. The next therapy, the access process, the follow-up interval, and the ownership of repeat testing all influence whether a numerical target becomes a sustained clinical result.

03

TREATMENT INTENSIFICATION

Cardiologists usually add nonstatin therapy because LDL-C is still above target, not because they have abandoned statin-based care

55.6% say LDL-C remaining above target despite maximally tolerated statin therapy is the most common trigger for nonstatin intensification.

CONTEXT

Modern dyslipidemia management increasingly assumes combination therapy when the amount of LDL-C reduction required exceeds what one agent can deliver. The 2026 ACC/AHA guideline incorporates several nonstatin options while continuing to position statins as foundational therapy for secondary prevention.

WHAT THE DATA SHOWS

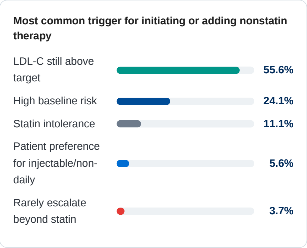
Remaining above LDL-C target despite a maximally tolerated statin leads at 55.6%. High baseline risk regardless of the starting LDL-C is next at 24.1%, followed by documented complete or partial statin intolerance at 11.1%, patient preference for a non-daily or injectable regimen at 5.6%, and rarely escalating beyond statin therapy at 3.7%.

THE INSIGHT

The leading trigger is explicitly goal-based: clinicians intensify because the patient is not where the risk profile says they should be. At the same time, nearly one quarter act primarily on the severity of baseline risk, suggesting that the treatment decision is not reducible to one laboratory value.

WHY IT MATTERS

This is a useful distinction in very-high-risk care. The cardiologist may need to think simultaneously about absolute LDL-C, percentage reduction, recurrent-event risk, prior treatment response, tolerance, and how quickly additional lowering is needed. A rigid one-size-fits-all escalation sequence may not fit every high-risk patient.



Most common trigger for initiating or adding nonstatin therapy

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04

ADMINISTRATIVE BARRIER

The clinical decision to intensify treatment often encounters a second decision layer controlled by step therapy, prior authorization, and cost

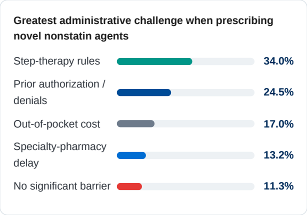
34.0% identify strict step-therapy rules requiring failure of multiple statins and ezetimibe first as the greatest administrative challenge.

CONTEXT

Nonstatin therapy is not delivered through a purely clinical pathway. For many patients, the next prescription also creates an access workflow that may involve documentation of prior therapies, payer-specific criteria, prior authorization, specialty-pharmacy routing, and patient cost discussions.

WHAT THE DATA SHOWS

Step therapy is the leading barrier at 34.0%. Prior-authorization paperwork and frequent initial denials follow at 24.5%, high out-of-pocket costs at 17.0%, specialty-pharmacy distribution or fulfillment delays at 13.2%, and no significant administrative barriers at 11.3%.


Greatest administrative challenge when prescribing novel nonstatin agents

THE INSIGHT

Nearly nine in ten respondents select a barrier rather than the no-barrier option. The specific friction varies, but the common pattern is that the therapy decision can be made before the treatment is actually obtainable. That gap creates opportunities for delay, abandonment, or a less intensive regimen than intended.

WHY IT MATTERS

In secondary prevention, access work is part of the treatment pathway even if it sits outside the traditional consultation. Clinics that can standardize documentation, track prior authorization, anticipate payer requirements, and surface patient cost issues early may reduce the time between recognizing an unmet LDL-C goal and achieving the intended escalation.

05

FIRST NONSTATIN CHOICE

Ezetimibe remains the most common first add-on, while PCSK9 therapy, bempedoic acid, and inclisiran create several alternative routes to the same LDL-C goal

41.5% choose ezetimibe as their typical first nonstatin add-on after high-intensity statin therapy does not reach LDL-C below 55 mg/dL.

CONTEXT

The choice of add-on therapy depends on how much additional LDL-C reduction is needed, the patient's risk, prior treatment response, route and frequency preferences, safety considerations, outcomes evidence, and whether the therapy can be obtained and continued. Contemporary guidelines increasingly recognize that more than one nonstatin route may be reasonable in high-risk care.

WHAT THE DATA SHOWS

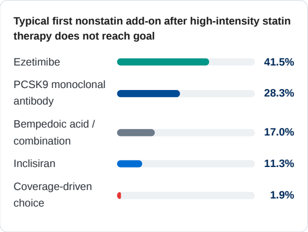
Ezetimibe leads at 41.5%. PCSK9 monoclonal antibodies are selected by 28.3%, bempedoic acid or a bempedoic-acid combination by 17.0%, inclisiran by 11.3%, and 1.9% say the choice varies entirely with insurance coverage rather than clinical preference.

THE INSIGHT

The treatment sequence is not uniform, but it is also not random. Ezetimibe remains the most common first add-on, while nearly six in ten respondents choose another option. That spread reflects the reality that the amount of LDL-C lowering required can differ substantially from patient to patient.

WHY IT MATTERS

A cardiologist who needs a small additional reduction, a large rapid reduction, a non-daily regimen, or a pathway around statin intolerance is solving different problems even when the LDL-C goal is the same. The useful question is not which agent wins overall, but whether the chosen agent can deliver the necessary reduction in a form the patient can access and sustain.


Typical first nonstatin add-on after high-intensity statin therapy does not reach goal

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06

STATIN INTOLERANCE

Statin-associated symptoms are common enough to shape practice, but the leading response is to preserve some statin exposure if possible

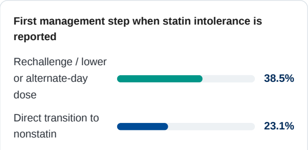
41.5% estimate that 5%–14% of patients report SAMS or statin intolerance that limits target statin dosing; 38.5% first try a different statin, lower dose, or alternate-day regimen.

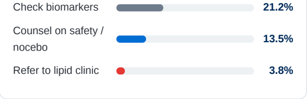
CONTEXT

Statin-associated muscle symptoms can complicate a high-intensity prevention plan because the clinician must balance two risks: leaving LDL-C above goal and continuing a regimen the patient cannot tolerate or no longer trusts. The National Lipid Association describes statin intolerance as complete or partial and recommends trying more than one statin, including a low-dose option, before concluding that no statin can be used.

WHAT THE DATA SHOWS

On prevalence, 41.5% estimate that 5%–14% of patients are limited by SAMS or intolerance, 24.5% estimate 15%–29%, 20.8% estimate 30% or higher, and 13.2% estimate less than 5%. On first management step, 38.5% rechallenge with a different statin, lower dose, or alternate-day dosing; 23.1% transition directly to nonstatin therapy; 21.2% check biomarkers; 13.5% counsel on statin safety and the nocebo effect; and 3.8% refer to a specialized lipid clinic.


First management step when statin intolerance is reported



THE INSIGHT

The survey portrays intolerance as a management problem rather than a single decision point. Most respondents do not immediately abandon statin therapy, but neither do they all follow the same sequence. That variation may reflect symptom severity, previous statin exposure, patient confidence, biomarker context, and the urgency of LDL-C reduction.

WHY IT MATTERS

The practical objective is to minimize time spent undertreated. A structured intolerance pathway can help clinicians investigate competing causes of symptoms, find a tolerable statin dose when possible, and add nonstatin therapy early enough that the patient's atherogenic-lipoprotein exposure does not remain unnecessarily high.

07

POST-ACS PERSISTENCE

Long-term LDL-C control appears most vulnerable after the acute phase, when ownership is shared across clinicians, patients, payers, and follow-up systems

28.8% identify PCP de-escalation or failure to intensify therapy as the greatest drop-off after an acute coronary event, closely followed by patient self-discontinuation at 26.9%.

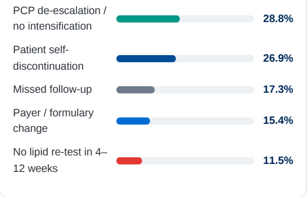
CONTEXT

An acute coronary syndrome creates a period of unusually high risk and unusually high clinical attention. The harder test comes after discharge, when medication regimens have to survive transitions between inpatient cardiology, outpatient cardiology, primary care, pharmacy, payer requirements, and the patient's own experience of treatment burden or side effects. The 2025 ESC/EAS focused update specifically emphasizes treatment intensification during the index ACS hospitalization when more LDL-C lowering is needed.

WHAT THE DATA SHOWS

PCP de-escalation or failure to intensify lipid therapy leads at 28.8%. Patient self-discontinuation because of side effects or pill burden is 26.9%, loss of follow-up or missed appointments 17.3%, payer coverage termination or formulary changes 15.4%, and lack of post-discharge lipid retesting within 4–12 weeks 11.5%.

Greatest drop-off in long-term LDL-C control after an acute coronary event



THE INSIGHT

The drop-off is distributed. No single actor owns all of it. The two largest responses involve clinical de-intensification and patient discontinuation, but missed visits, coverage changes, and delayed testing add additional failure points. The common feature is loss of continuity after the most intensively managed phase of care.

WHY IT MATTERS

A post-ACS lipid pathway may need explicit ownership of the next lipid panel, the next treatment decision, the prior-authorization process, and the conversation about side effects or pill burden. Without that ownership, even a strong discharge regimen can drift away from the long-term LDL-C goal.

08

LIPOPROTEIN(A)

Lp(a) screening is entering routine cardiovascular risk assessment, but the survey shows four different adoption patterns rather than one standard workflow

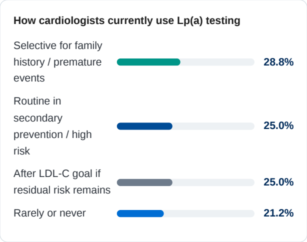
28.8% use Lp(a) selectively for strong family history or unexpected premature events, while 25.0% test routinely in secondary-prevention and high-risk patients.

CONTEXT

The 2026 ACC/AHA dyslipidemia guideline recommends measuring Lp(a) at least once in adulthood, and the 2025 ESC/EAS update also supports at least one adult lifetime measurement to refine risk. This changes the role of Lp(a) from a niche laboratory test toward a more routine risk-assessment tool, even though specific treatment decisions still depend on the overall clinical picture.

WHAT THE DATA SHOWS

Selective testing for family history or premature events leads at 28.8%. Routine testing for all secondary-prevention and high-risk patients is 25.0%, testing only after standard LDL-C targets are met but residual risk remains high is also 25.0%, and 21.2% rarely or never order Lp(a) because of the limited availability of specifically targeted therapies.


How cardiologists currently use Lp(a) testing

THE INSIGHT

The responses are unusually balanced. Rather than one dominant pattern, the survey shows a transition state: some cardiologists have normalized the test, others use it selectively, others reserve it for residual-risk questions, and a substantial minority still see limited actionable value.

WHY IT MATTERS

As Lp(a) becomes more routinely measured, the workflow question will move from whether to order the test to what happens after the result. The result may influence risk communication, intensity of LDL-C lowering and other prevention measures, family discussions, and future eligibility for targeted therapies as the evidence base evolves.

09

CLOSING THE GUIDELINE GAP

Cardiologists rank automatic EHR alerts above prior-

authorization reform, coverage expansion, and dedicated staff as the single highest-impact change

36.5% say automatic EHR alerts for unachieved LDL targets at follow-up would have the highest impact on closing the LDL-C guideline gap.

CONTEXT

The earlier findings show a pathway with multiple failure points: targets are not always reached, access can delay treatment, intolerance can change the regimen, and control can weaken after discharge. The final survey item asks which one system change would create the largest improvement across that pathway.

WHAT THE DATA SHOWS

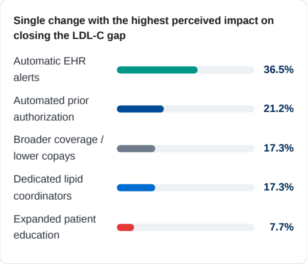
Automatic EHR alerts for unachieved LDL targets lead at 36.5%. Streamlined automated prior authorization is 21.2%, broader insurance coverage and lower copays 17.3%, dedicated lipid-optimization or prior-authorization staff 17.3%, and expanded patient education on “lower for longer” targets 7.7%.

THE INSIGHT

The leading answer is a workflow prompt, not a new drug. Cardiologists appear to see missed opportunities for intensification as a recurring operational problem that can be made visible at the point of follow-up. At the same time, more than half of responses are distributed across access and staffing solutions, so an alert alone would not eliminate every downstream barrier.

WHY IT MATTERS

The most useful digital intervention may be one that creates accountable next steps rather than another passive notification. An LDL-C alert becomes more meaningful when it can launch repeat testing, treatment review, prior authorization, or referral to a coordinator instead of simply adding another item to the inbox.


Single change with the highest perceived impact on closing the LDL-C gap

CROSS-QUESTION PERSPECTIVE

The survey points to a familiar cardiology paradox: the LDL-C target is becoming more explicit while the route to that target still depends on several loosely connected systems.

Taken together, the structured findings connect target attainment, treatment escalation, access, statin intolerance, post-ACS persistence, Lp(a), and workflow into one secondary-prevention pathway rather than a set of isolated decisions.

Target recognition is not the main gap

More than half say remaining above LDL-C target is the leading trigger for nonstatin escalation. The bigger challenge appears after recognition: choosing the next therapy, securing access, and confirming the patient actually reaches the goal.

Clinical choice and access are coupled

Ezetimibe leads first add-on therapy, but step therapy, prior authorization, out-of-pocket costs, and specialty-pharmacy logistics can still shape what is realistically delivered and how quickly.

Statin intolerance needs a pathway

Rechallenge leads the first response to intolerance, but many cardiologists use biomarkers, counseling, nonstatin transition, or specialty referral. A structured approach can reduce both unnecessary statin abandonment and prolonged undertreatment.

Persistence needs shared ownership

The post-ACS drop-off spans primary care, patient discontinuation, missed visits, payer changes, and repeat testing. No single reminder can solve every failure point unless responsibilities are clear.

> **What the cross-question pattern adds:** closing the LDL-C gap is not one treatment decision. It is a sequence of target recognition, intensification, access, tolerance management, retesting, and persistence that must remain connected across settings.

CARDIOLOGY PRACTICE LENS

What a more reliable secondary-prevention lipid pathway could look like in routine cardiology care

The survey does not define one universal treatment algorithm. It does identify where process design could reduce variation without replacing individualized cardiovascular risk assessment or clinical judgment.

Make the LDL-C goal visible at every transition

Carry the patient-specific LDL-C goal from discharge to cardiology and primary-care follow-up so treatment intensity is not lost when responsibility changes hands.

Link intensification to access work

When a nonstatin agent is chosen, start documentation, prior authorization, cost review, and specialty-pharmacy routing as part of the same workflow rather than as a later administrative task.

Use a structured statin-intolerance pathway

Evaluate competing causes of symptoms, test a tolerable statin strategy when appropriate, and add nonstatin therapy early enough to avoid a prolonged period above LDL-C goal.

Close the loop on retesting and residual risk

Use scheduled lipid panels, EHR prompts, Lp(a) results, and clear follow-up ownership to make missed targets and unresolved residual risk visible before the next event.

PEOPLE ALSO ASK

Frequently asked questions about LDL-C targets and secondary prevention

What LDL-C target is recommended for very-high-risk ASCVD in secondary prevention?

The 2026 ACC/AHA multisociety dyslipidemia guideline recommends an LDL-C goal below 55 mg/dL for patients at very high risk of recurrent ASCVD events. Individual treatment still depends on the patient’s overall risk, current therapy, tolerance, and clinical context.

When should nonstatin therapy be added after statin treatment?

Nonstatin therapy is considered when LDL-C remains above the patient-specific treatment goal despite maximally tolerated statin therapy, or when statin intolerance prevents adequate LDL-C lowering. The choice depends on the amount of additional lowering needed, risk level, evidence, access, adherence, and patient preference.

Which nonstatin therapies are used to lower LDL-C in ASCVD?

Common options include ezetimibe, PCSK9 monoclonal antibodies, bempedoic acid, and inclisiran. The 2026 ACC/AHA guideline incorporates these therapies into contemporary dyslipidemia management, with treatment selection guided by risk, required LDL-C reduction, tolerance, and practical factors.

How are statin-associated muscle symptoms usually managed?

Management commonly includes assessing other possible causes of muscle symptoms and trying a different statin, lower dose, or alternative dosing schedule when appropriate. Patients who cannot reach their treatment goal on a tolerable statin regimen may also need nonstatin therapy.

What is lipoprotein(a), and when should it be measured?

Lipoprotein(a), or Lp(a), is a largely inherited lipoprotein that can increase ASCVD risk. The 2026 ACC/AHA dyslipidemia guideline recommends measuring Lp(a) at least once in adulthood because lifestyle changes have little effect on the level and repeat testing is usually unnecessary.

Why can LDL-C control worsen after an acute coronary syndrome?

Long-term LDL-C control can weaken when follow-up, medication persistence, repeat lipid testing, treatment intensification, affordability, or coordination between hospital and outpatient clinicians breaks down. Effective secondary prevention therefore depends on both the prescription and the follow-through.

CLOSING PERSPECTIVE

Secondary prevention will close the LDL-C gap only when escalation, access, and long-term persistence work as one pathway

The MDForLives data describes a cardiology environment in which the treatment destination is increasingly clear but the journey remains fragmented. Very-high-risk ASCVD patients are expected to reach aggressive LDL-C goals, yet the most common estimate is that only 40%–59% achieve LDL-C below 55 mg/dL. That gap is clinically important precisely because the survey shows that cardiologists are not waiting passively for LDL-C to improve: remaining above target is the leading trigger for adding nonstatin therapy.

The problem is what happens next. Ezetimibe remains the most common first add-on, while PCSK9 monoclonal antibodies, bempedoic acid, and inclisiran create additional routes to greater LDL-C reduction. But therapy choice sits inside an access environment in which step therapy, prior authorization, out-of-pocket cost, and specialty-pharmacy delays are common. For the clinician, the treatment plan may therefore have two timelines: the clinical decision and the administrative path required to make that decision real.

Statin intolerance illustrates the same need for process. The leading first response is to preserve statin exposure through a different agent, lower dose, or alternate-day regimen, but a substantial share of cardiologists move directly to nonstatin therapy, check biomarkers, or focus on counseling. There is no single response that fits every patient. What matters is that symptom evaluation, tolerability, and LDL-C lowering remain connected so that the patient is not left undertreated while the team works out what can be sustained.

The post-ACS findings show where fragmentation becomes most visible. Respondents distribute the largest drop-offs across PCP de-intensification, patient self-discontinuation, missed follow-up, payer changes, and delayed lipid retesting. These are different causes with one shared consequence: the intensity achieved around an acute event can erode when the patient re-enters ordinary longitudinal care. The transition from hospital to clinic is therefore not just a scheduling issue. It is a treatment-persistence problem.

Lp(a) and the final system-change question point toward what may come next. The 2026 ACC/AHA guideline has moved Lp(a) toward at least one adult lifetime measurement, yet the survey still shows routine, selective, residual-risk, and rare-use patterns. At the same time, cardiologists rank EHR alerts for unachieved LDL targets as the single highest-impact system change. Together, these findings suggest that new risk information and new therapies will matter most when the workflow knows what to do with them.

The broader implication is that secondary prevention needs a closed loop. The LDL-C goal should be visible, the next treatment step should be obvious when the goal is missed, access work should begin immediately, intolerance should trigger a structured response, repeat testing should be scheduled, and responsibility should remain clear after discharge. Technology can support that loop, but it cannot substitute for ownership. The most effective system will be the one that turns every missed LDL-C target into an accountable next action.

1 · Make the target explicit

Keep the patient-specific LDL-C goal visible across cardiology, discharge, primary care, and follow-up encounters.

2 · Trigger intensification when the goal is missed

Connect above-target LDL-C to a treatment review rather than relying on memory or waiting for the next event.

3 · Treat access as part of therapy

Start prior authorization, step-therapy documentation, affordability review, and specialty-pharmacy routing with the prescription decision.

4 · Standardize statin-intolerance management

Use a repeatable pathway to assess symptoms, preserve tolerable statin exposure when possible, and add nonstatin therapy when needed.

5 · Protect the post-ACS handoff

Define who owns repeat lipids, treatment intensification, medication persistence, and access after discharge.

6 · Embed risk and follow-up in the EHR

Use alerts, Lp(a) results, and scheduled monitoring to surface missed targets and make the next step actionable.

Help future cardiology insights reflect what happens between an LDL-C target and long-term control.

Share your perspective with MDForLives and help build a clearer view of secondary prevention, treatment intensification, statin intolerance, access, Lp(a), post-ACS follow-up, and real-world cardiovascular risk reduction.

Join the Panel

Sources used for this report

1. **MDForLives Cardio Insight Survey_Aug, SGID 8953232.** Source survey report: 71 survey records, 69% completion rate, six countries, and question-level values used exactly as reported. Administrative and validation fields were excluded from interpretation.
2. **American Heart Association / American College of Cardiology.** 2026 Guideline on the Management of Dyslipidemia. The guideline recommends LDL-C below 55 mg/dL for very-high-risk secondary prevention and recommends measuring Lp(a) at least once in adulthood. https://professional.heart.org/en/science-news/2026-guideline-on-the-management-of-dyslipidemia/top-things-to-know
3. **American College of Cardiology / American Heart Association.** Updated dyslipidemia guideline announcement, March 13, 2026. Summarizes the return of LDL-C treatment goals, broader incorporation of nonstatin therapies, and once-in-adulthood Lp(a) testing. https://www.acc.org/About-ACC/Press-Releases/2026/03/13/18/01/ACCAHA-Issue-Updated-Guideline-for-Managing-Lipids-Cholesterol
4. **European Society of Cardiology / European Atherosclerosis Society.** 2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias. Includes updated guidance on intensifying lipid-lowering therapy during ACS hospitalization and on Lp(a) as a risk modifier. https://www.escardio.org/guidelines/clinical-practice-guidelines/all-esc-practice-guidelines/dyslipidaemias/
5. **National Lipid Association.** Scientific statement on statin intolerance: a new definition and key considerations for ASCVD risk reduction in the statin-intolerant patient. Recommends evaluating partial versus complete intolerance and trying strategies such as a different statin, dose, or dosing frequency when appropriate. https://www.lipid.org/resource/nla-scientific-statement-on-statin-intolerance-a-new-definition-and-key-considerations-for-ascvd-risk-reduction-in-the-statin-intolerant-patient/
6. **American College of Cardiology.** Statin Intolerance clinical tool, updated with content derived from the 2026 ACC/AHA multisociety dyslipidemia guideline. https://tools.acc.org/LDL/StatinIntolerance/

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