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Lipoprotein(a) after acute myocardial infarction: what a written protocol changed in testing rates and prevalence estimates

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Introduction: Guidelines recommend measuring lipoprotein(a) [Lp(a)] at least once in every adult, yet in practice it is ordered selectively, and the prevalence figures that emerge from such testing reflect who was tested rather than who was admitted.[1] To quantify the change in Lp(a) testing after introducing a written protocol into the post-infarction care pathway and to describe the prevalence of elevated Lp(a) once testing became routine.

Patients and Methods: Single-center registry of consecutive acute myocardial infarction admissions (January 2017 - June 2026). From June 21, 2024, a written instruction required Lp(a) in the first-morning blood sample within 24 hours of admission. The pre-protocol and protocol eras were compared for testing rates and for the proportion of patients with Lp(a) ≥125 nmol/L. Selection into testing was addressed by comparing tested and untested patients and by inverse-probability weighting; exploratory analyses were adjusted for multiplicity.

Results: Testing rose from 3.9% (102/2604) to 78.0% (769/986) of admissions. In the protocol era through the end of 2025, Lp(a) ≥125 nmol/L was present in 22.0% of tested patients (131/595; 95% CI 18.9-25.5). Under earlier discretionary testing the apparent prevalence was about 1.7 times higher (ratio 1.74; 95% CI 1.30-2.32), indicating that clinicians had preferentially tested higher-risk patients. Weighting for non-testing did not materially change the protocol-era estimate (20.9% vs. 21.1%). No association with inflammatory or angiographic variables remained significant after correction.

Conclusion: A single written instruction, requiring no additional staff, equipment, or cost beyond the assay itself, turned Lp(a) measurement from an occasional decision into a default step of post-infarction care, raising uptake roughly twentyfold within months. The consequence was not only more testing but better information: once the choice of whom to test was removed from the clinician, the prevalence of elevated Lp(a) fell from an inflated, selection-driven figure to a stable estimate that weighting could not shift, roughly every fifth patient after myocardial infarction. That proportion is large enough to matter for secondary prevention and for identifying the population that future Lp(a)-lowering trials will need to recruit, even as the first outcome trial has left the therapeutic question open. Our findings suggest that the barrier to guideline-concordant Lp(a) testing is organizational rather than scientific, and that embedding the test in the care pathway is a simple and reproducible way to remove it.

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LITERATURE

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