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Managing Progressive Atherosclerotic Cardiovascular Disease in a Patient with Elevated Lipoprotein(a)

ABSTRACT

Lipoprotein(a) [Lp(a)] is a genetically determined, proatherogenic, and prothrombotic lipoprotein associated with an increased risk of atherosclerotic cardiovascular disease (ASCVD). Elevated Lp(a) levels are associated with progressive ASCVD even when guideline-recommended low-density lipoprotein cholesterol (LDL-C) targets are achieved under optimal lipid-lowering therapy. There is currently no approved pharmacological therapy specifically targeting Lp(a) reduction in routine clinical practice; therefore, current management strategies for patients with elevated Lp(a) primarily focus on aggressive control of modifiable cardiovascular risk factors and intensive LDL-C lowering. Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors provide a modest reduction in Lp(a) levels and have been associated with greater cardiovascular benefit in patients with high baseline Lp(a); however, this degree of reduction is often insufficient in individuals with markedly elevated Lp(a) levels and progressive ASCVD. At present, lipoprotein apheresis remains the only therapeutic option capable of achieving substantial and sustained reductions in Lp(a) concentrations and is recommended in selected high-risk patients with progressive ASCVD despite optimal medical therapy.

Meanwhile, Lp(a)-specific therapies, including antisense oligonucleotides and small interfering RNA agents, are in advanced clinical development and have shown marked reductions in Lp(a) levels in early phase trials. These emerging therapies are expected to significantly change future treatment strategies for patients with Lp(a)-driven residual cardiovascular risk.

Keywords: Atherosclerosis, cardiovascular events, cardiovascular risk factors, hyperlipidemia, lipoprotein, preventive cardiology

REVIEW

CASE

A 65-year-old man referred to a lipid clinic for progressive atherosclerotic cardiovascular disease (ASCVD) despite strict adherence to medical therapy. His cardiovascular history included left anterior descending artery stenting in 2010 and triple coronary artery bypass grafting (CABG) in 2024. Shortly after the CABG operation, he experienced a transient ischemic attack (TIA), although he reported no chest pain.

The patient did not smoke and had no history of hypertension or diabetes mellitus. His height was 177 cm and weight 85 kg, with a body mass index of 27 kg/m^2. His family history was remarkable: his father underwent CABG at age 51 years, his grandfather had a myocardial infarction at age 43 years, and his grandmother had a stroke at age 60 years.

Following the CABG, his low density lipoprotein cholesterol (LDL-C) level was 215 mg/dL (5.56 mmol/L), whereas Lipoprotein(a) [Lp(a)] had not been measured at that time. Based on his lipid profile, his treatment was re-adjusted, and he was started on rosuvastatin 40 mg, ezetimibe 10 mg, clopidogrel, and metoprolol. This treatment regimen was ongoing at the time of the TIA. At presentation with TIA, laboratory results under maximal lipid-lowering therapy were as follows: Total cholesterol: 111 mg/dL (2.8 mmol/L), triglycerides: 50 mg/dL (0.6 mmol/L), LDL-C: 54 mg/dL (1.4 mmol/L), HDL-C: 53 mg/dL (1.3 mmol/L), hs-CRP: <0.05 mg/dL,

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HbA1c: 5.5%, Lp(a): 214 mg/dL. Fibrinogen, D-dimer, Factor VIII, Protein C, and S levels were all within normal limits.

Despite achieving guideline-recommended lipid targets, markedly elevated Lp(a) levels and recurrent ASCVD events necessitated additional therapeutic interventions in this patient.

Treatment options, including proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibition and lipoprotein apheresis, were discussed with the patient. Evolocumab 140 mg every 2 weeks was initiated. Following treatment, LDL-C decreased further to 31 mg/dL, and Lp(a) was reduced to 145 mg/dL. The addition of lipoprotein apheresis to his management is currently under consideration to further lower Lp(a) since he has recurrent events while we wait for the Lp(a)-specific therapies to be available.

Case Review

Lipoprotein(a) [Lp(a)] is a uniquely structured atherogenic lipoprotein similar to LDL particles, which differ by having a covalently bound apolipoprotein(a) via a disulfide bond. Lipoprotein(a) [Lp(a)] carries the majority of circulating oxidized phospholipids (OxPL), which induce a potent inflammatory response and enhance monocyte recruitment.[1] Moreover, the apo(a) component structurally resembles plasminogen and competitively inhibits fibrinolysis, thereby creating a pro-thrombotic environment that increases the risk of acute cardiovascular events. The level of Lp(a) is mostly determined by genetic factors, and environmental factors don't play a major role in its level.[2] The Apo(a) component contains multiple kringle IV type 2 repeats, whose number is genetically determined and inversely correlated with plasma Lp(a) concentration. Although Lp(a) levels remain relatively stable throughout a lifetime, it has been shown that Lp(a) levels increase in women after menopause, and studies have reported higher median Lp(a) levels in women compared with men.[3,4] Now it is proven that Lp(a) levels are related to ASCVD risk independent of and additive to traditional risk factors.[5,6] Importantly, this risk relationship is continuous and linear; increases in Lp(a) across its distribution associate with proportionally increasing risk of myocardial infarction, stroke, and aortic valve stenosis.[7] Because levels are genetically determined and stable throughout life, Lp(a) has unique value as a lifetime risk indicator.

According to European guidelines and atherosclerosis experts, Lp(a) should be measured at least once during adult life. [8–11] In women, a second Lp(a) measurement may be considered after menopause.[8] The value of Lp(a) measurement in both primary and secondary prevention is increasingly recognized, particularly by clinicians specialized in this field, and approximately 75% of clinicians working in lipid clinics across Europe measure Lp(a) levels in their patients.[12] Screening to detect elevated Lp(a) levels in individuals without known cardiovascular disease is recommended, and high Lp(a) should be regarded as a risk-enhancing factor, particularly in patients at intermediate risk or those near treatment thresholds. The presence of markedly elevated Lp(a), typically defined as ≥50 mg/dL (≈125 nmol/L), supports reclassification of individuals with intermediate risk into higher risk categories.[7] Moreover, Lp(a) measurement is recommended in patients with premature ASCVD, those with a family history of premature ASCVD or elevated Lp(a), and younger patients with familial hypercholesterolemia.[8] In secondary prevention, clinical trials have demonstrated that among patients with established coronary artery disease, elevated Lp(a) levels are associated with higher cardiovascular event rates even when LDL-C is well controlled, highlighting the concept of residual cardiovascular risk.[13]

Despite the increasing recognition of the importance of Lp(a) measurement, there is currently no approved pharmacological therapy specifically targeting Lp(a) reduction; therefore, cardiovascular risk management in patients with elevated Lp(a) primarily relies on optimal control of other modifiable risk factors, particularly LDL-C levels. The EPIC-Norfolk study demonstrated that, in individuals with elevated Lp(a), effective control of other cardiovascular risk factors and adherence to a healthy lifestyle are associated with a reduction in cardiovascular risk.[14] In individuals without known ASCVD but with intermediate cardiovascular risk, elevated Lp(a) may justify earlier initiation of lipid-lowering therapy and more ambitious LDL-C targets. In the setting of secondary prevention, close clinical follow-up and stricter LDL-C goals should be considered. Although statins may modestly increase Lp(a) levels, intensive LDL-C reduction mitigates the overall atherogenic burden.[15,16] However, despite strict LDL-C control, elevated Lp(a) remains a source of residual cardiovascular risk and is associated with recurrent cardiovascular events.

To address this residual risk, specific therapies targeting Lp(a) reduction have been developed; however, they remain under investigation in clinical trials and have not yet been approved for routine clinical use. Currently, PCSK9 inhibitors are the only approved pharmacological agents that provide

HIGHLIGHTS

- Elevated Lp(a) is an important cause of residual cardiovascular risk despite optimal low-density lipoprotein cholesterol (LDL-C) lowering and is associated with progressive ASCVD even when guideline-recommended lipid targets are achieved.
- In the presence of elevated Lp(a), more aggressive LDL-C reduction is recommended to minimize cardiovascular risk.
- Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors provide only modest reductions in Lp(a) levels and may be insufficient in patients with very high Lp(a) concentrations.
- Lipoprotein apheresis remains the most effective currently available option for substantial Lp(a) lowering in selected high-risk patients.
- Lp(a)-specific pharmacological therapies are close to completing clinical development, and their approval is expected to represent a major advance in preventive cardiology.

a moderate reduction in Lp(a) levels. In the ODYSSEY trials, alirocumab reduced Lp(a) levels by approximately 23% at 4 months, with a greater absolute reduction observed in patients in the highest Lp(a) quartile. Moreover, individuals in the upper Lp(a) quartile had a greater reduction in major adverse cardiovascular events (MACE) with PCSK9 inhibition.[17] Similarly, in the FOURIER trial, evolocumab reduced Lp(a) levels by 26.9% at 48 weeks. Importantly, in patients with baseline Lp(a) levels above the median, more coronary events were prevented compared with those with Lp(a) levels at or below the median (23% vs 7%, respectively).[18] When either LDL-C or Lp(a) levels were reduced below the median, the relative risk reduction was 15%, whereas reductions in both LDL-C and Lp(a) were associated with a 28% reduction in cardiovascular events, indicating an Lp(a)-dependent benefit independent of LDL-C lowering. Data from the ORION trials demonstrated that inclisiran treatment results in a 15-28% reduction in Lp(a) levels.[19]

Nevertheless, the degree of Lp(a) reduction achieved with PCSK9 inhibition is generally insufficient in patients with very high Lp(a) levels and progressive ASCVD. Currently, lipoprotein apheresis remains the only therapeutic option capable of producing large and immediate reductions in circulating Lp(a) concentrations, with acute reductions of 60-75% per session.[20] When performed weekly or biweekly, mean interval Lp(a) concentrations decline by approximately 30-40%, resulting in sustained reductions in atherogenic and inflammatory burden.[21] Although its availability is limited and it is reserved for highly selected patients, lipoprotein apheresis plays an important role in European lipid clinics, particularly in Germany, Austria, and Switzerland. It currently represents the most potent intervention available for lowering Lp(a).

Lipoprotein apheresis is indicated in patients with progressive ASCVD who fail to achieve LDL-C targets despite maximally tolerated lipid-lowering therapy, as well as in patients with LDL-C at target but elevated Lp(a) levels (>60 mg/dL) in the presence of ASCVD, or in those with both elevated LDL-C and Lp(a). The Pro(a)LiFe study enrolled patients with a history of ASCVD, LDL-C levels close to guideline-recommended targets, and Lp(a) concentrations >60 mg/dL.[22] Lipoprotein apheresis resulted in a 67% reduction in Lp(a) levels during the first year and 68% during the second year compared with pre-apheresis values. Low-density lipoprotein cholesterol (LDL-C) levels were reduced by 66% in the first year and 68% in the second year, and these reductions were sustained for up to 12 years. The rate of major cardiovascular events decreased by 78% at 5 years and by 75% at 12 years compared with the pre-apheresis period.

Although these findings suggest that Lp(a) reduction is associated with a substantial decrease in cardiovascular events, it remains difficult to disentangle the relative contributions of Lp(a) and LDL-C lowering to the observed risk reduction. Further insight is provided by data from the German Lipoprotein Apheresis Registry, in which patients were categorized into 3 groups: isolated LDL-C elevation, isolated Lp(a) elevation, and combined elevation of LDL-C and Lp(a).[23] Overall, lipoprotein apheresis reduced LDL-C levels by 68.1% and Lp(a) levels by 75.6%. In patients with isolated Lp(a) elevation, MACE were reduced by 83% in the first year and by 86% in the second year. In contrast, patients with isolated LDL-C elevation experienced MACE reductions of 42% and 61% in the first and second years, respectively. In patients with combined elevation of LDL-C and Lp(a), MACE rates were reduced by 71% in the first year and by 78% in the second year compared with the pre-apheresis period. These findings underscore that the benefit of lipoprotein apheresis is greater in patients with elevated Lp(a), with or without concomitant LDL-C elevation.

Consistent with these data, the European Society of Cardiology and European Atherosclerosis Society (ESC/EAS) guidance recognizes lipoprotein apheresis as a therapeutic option for patients with progressive ASCVD and markedly elevated Lp(a) levels despite optimal medical therapy.[9] Until the approval of Lp(a)-lowering specific therapies, lipoprotein apheresis represents the most potent currently available intervention for lowering Lp(a). However, apheresis has several limitations. It is resource-intensive, requiring specialized equipment, dedicated vascular access, and regular treatment sessions lasting 2 to 3 hours. These logistical, economic, and patient-related considerations restrict its use.

Meanwhile therapies specifically targeting Lp(a) are advancing rapidly through clinical trials and are likely to become available for clinical use in the near future. These therapies directly target hepatic production of apolipoprotein(a) using antisense oligonucleotides or small interfering ribonucleoic asid (siRNA).[24] They achieve marked Lp(a) reductions of approximately 80-98%. Antisense oligonucleotides are single-stranded nucleic acids designed to be complementary to a specific target messenger RNA (mRNA). They contain a DNA-like gap region that can be recognized by RNase H, leading to degradation of the target mRNA and subsequent reduction in protein synthesis. Pelacarsen is an antisense oligonucleotide conjugated with GalNAc to selectively target hepatocytes and reduce the production of apolipoprotein(a) in the liver. When administered by subcutaneous injection, pelacarsen has demonstrated dose-dependent reductions in Lp(a) levels of approximately 80-90% in phase II trials, with good tolerability and sustained efficacy.[25] Importantly, the Lp(a) HORIZON study, a large cardiovascular outcomes trial enrolling more than 8000 patients with established ASCVD and elevated Lp(a), is currently ongoing.[26] The results, expected in 2026, are anticipated to address whether selective Lp(a) lowering translates into a reduction in major adverse cardiovascular events.

Developments in siRNA therapeutics have also been impressive. siRNAs are double-stranded RNA molecules that are incorporated into the RNA-induced silencing complex (RISC) within the cell. The guide strand directs the RISC complex to the complementary target mRNA, which is subsequently cleaved by Argonaute-2, a key component of RNA-induced silencing complex (RISC), leading to degradation of the target mRNA and inhibition of protein synthesis. Olpasiran, a GalNAc-conjugated apo(a) siRNA, administered every 12 to 24 weeks, achieves Lp(a) reductions of 90-98%, with many patients reaching nearly undetectable

levels.[27] Early-phase trials have demonstrated a remarkable long-term effect, with reductions persisting for months after the last injection. A clinical outcomes trial in patients with ASCVD evaluating the effect of olpasiran on major adverse cardiovascular events is currently ongoing, with results expected in 2026.[28] Other siRNA agents, such as lepodisiran and zerlasiran, are in various stages of clinical development and appear capable of achieving similar reductions with very infrequent dosing intervals, which may support long-term adherence.

If clinical trials are successful, clinicians may, for the first time, be able to effectively neutralize a major genetic risk factor that has until now remained clinically unmodifiable. If outcome trials confirm a reduction in major cardiovascular events, these therapies are expected to reshape prevention strategies, screening practices, and treatment algorithms across Europe. The integration of Lp(a)-specific therapies into clinical practice will require careful consideration of risk thresholds, prioritization strategies, and cost-effectiveness. Individuals with very high Lp(a) levels and those with elevated Lp(a) and progressive ASCVD despite low LDL-C levels and the absence of other major risk factors, similar to the patient described here, are likely to derive the greatest benefit.

In summary, the case presented in this review highlights a clinically important and increasingly recognized scenario in contemporary cardiovascular medicine: a patient with excellent control of traditional risk factors, optimal adherence to therapy, and LDL-C levels well below guideline-recommended targets, yet ongoing progression of atherosclerotic disease and recurrent cerebrovascular events. In this setting, elevated Lp(a) emerged as the dominant residual risk factor. Although PCSK9 inhibition provided only modest Lp(a) reduction, further lowering of Lp(a) was critical for secondary prevention.

At present, lipoprotein apheresis remains the only effective therapeutic option to substantially reduce Lp(a) levels in such patients. However, the rapid development of Lp(a) specific therapies is expected to fundamentally change the management of patients with residual cardiovascular risk driven by elevated Lp(a) and to offer new preventive strategies for individuals similar to the case described here.

AI Disclosure

The authors declare that no generative AI tools (such as large language models, chatbots, or image creators) were used in any stage of the preparation or writing of this manuscript.

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REFERENCES

1. Tsimikas S. Lipoprotein(a) in the year 2024: A look back and a look ahead. *Arterioscler Thromb Vasc Biol*. 2024;44(7):1485-1490. [CrossRef]
2. Reyes-Soffer G, Ginsberg HN, Berglund L, et al. Lipoprotein(a): A genetically determined, causal, and prevalent risk factor for atherosclerotic cardiovascular disease: a scientific statement from the American Heart Association. *Arterioscler Thromb Vasc Biol*. 2022;42(1):e48-e60. [CrossRef].
3. Derby CA, Crawford SL, Pasternak RC, Sowers M, Sternfeld B, Matthews KA. Lipid changes during the menopause transition in relation to age and weight: the Study of Women's Health across the Nation. *Am J Epidemiol*. 2009;169(11):1352-1361. [CrossRef].
4. Yurtseven E, Ural D, Gursoy E, et al. Is there a need for sex-tailored lipoprotein(a) cut-off values for coronary artery disease risk stratification?. *Clin Cardiol*. 2024;47(9):e70012. [CrossRef]
5. Patel AP, Wang M, Pirruccello JP, et al. [LP]. Lp(a) (lipoprotein[a]) concentrations and incident atherosclerotic cardiovascular disease: New insights from a large national Biobank. *Arterioscler Thromb Vasc Biol*. 2021;41(1):465-474. [CrossRef]
6. Gungor B, Simsek B, Cınar T, et al. Assessment of lipoprotein (a) levels in patients with atherosclerotic cardiovascular disease: Single center experience from Turkiye. *Atherosclerosis*. 2023;379:S49. [CrossRef]
7. Kronenberg F, Mora S, Stroes ESG, et al. Lipoprotein(a) in atherosclerotic cardiovascular disease and aortic stenosis: a European Atherosclerosis Society consensus statement. *Eur Heart J*. 2022;43(39):3925-3946. [CrossRef]
8. Mach F, Koskinas KC, Roeters van Lennep JE, et al. Focused Update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias: developed by the task force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS). *Eur Heart J*. 2025;46(42):4359-4378. [CrossRef]
9. Authors/Task Force Members, ESC Committee for Practice Guidelines (CPG), ESC National Cardiac Societies. 2019 ESC/EAS guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. *Atherosclerosis*. 2019;290(1):140-205. [CrossRef]
10. Tokgözoğlu L, Libby P. The dawn of a new era of targeted lipid-lowering therapies. *Eur Heart J*. 2022;43(34):3198-3208. [CrossRef]
11. Maher LL, Tokgözoğlu SL, Sanchez EJ, Underberg JA, Guyton JR. JCL roundtable: global think tank on lipoprotein(a). *J Clin Lipidol*. 2021;15(3):387-393. [CrossRef]
12. Catapano AL, Tokgözoğlu L, Banach M, et al. Evaluation of lipoprotein(a) in the prevention and management of atherosclerotic cardiovascular disease: a survey among the Lipid Clinics Network. *Atherosclerosis*. 2023;370:5-11. [CrossRef]
13. Small AM, Pournamdari A, Melloni GEM, et al. Lipoprotein(a), C-reactive protein, and cardiovascular risk in primary and secondary prevention populations. *JAMA Cardiol*. 2024;9(4):385-391. [CrossRef].

14. Perrot N, Verbeek R, Sandhu M, et al. Ideal cardiovascular health influences cardiovascular disease risk associated with high lipoprotein(a) levels and genotype: the EPIC-Norfolk prospective population study. *Atherosclerosis*. 2017;256:47-52. [CrossRef]
15. Tsimikas S, Gordts PLSM, Nora C, Yeang C, Witztum JL. Statin therapy increases lipoprotein(a) levels. *Eur Heart J*. 2020;41(24):2275-2284. [CrossRef]
16. Willeit P, Ridker PM, Nestel PJ, et al. Baseline and on-statin treatment lipoprotein(a) levels for prediction of cardiovascular events: individual patient-data meta-analysis of statin outcome trials. *Lancet*. 2018;392(10155):1311-1320. [CrossRef]
17. Bittner VA, Szarek M, Aylward PE, et al. Effect of alirocumab on lipoprotein(a) and cardiovascular risk after acute coronary syndrome. *J Am Coll Cardiol*. 2020;75(2):133-144. [CrossRef]
18. O'Donoghue ML, Fazio S, Giugliano RP, et al. Lipoprotein(a), PCSK9 inhibition, and cardiovascular risk. *Circulation*. 2019;139(12):1483-1492. [CrossRef]
19. Katsiki N, Vrablik M, Banach M, Gouni-Berthold I. Inclisiran, low-density lipoprotein cholesterol and lipoprotein (a). *Pharmaceuticals (Basel)*. 2023;16(4):577. [CrossRef]
20. Kayikcioglu M. LDL Apheresis and Lp (a) Apheresis: a Clinician's Perspective. *Curr Atheroscler Rep*. 2021;23(4):15. [CrossRef]
21. Waldmann E, Parhofer KG. Lipoprotein apheresis to treat elevated lipoprotein (a). *J Lipid Res*. 2016;57(10):1751-1757. [CrossRef]
22. Klingel R, Julius U, Bernhardt WM, et al. Lipoprotein apheresis for lipoprotein(a)-associated progressive atherosclerotic cardiovascular disease: 12-years follow-up. *Atherosclerosis*. 2025;410:120508. [CrossRef]
23. Schettler VJJ, Peter C, Zimmermann T, et al. The German lipoprotein Apheresis Registry—summary of the ninth annual report. *Ther Apher Dial*. 2022;26(suppl 1):81-88. [CrossRef]
24. Alhomoud IS. Emerging therapies targeting lipoprotein(a): the next frontier in cardiovascular risk reduction. *Front Med (Lausanne)*. 2025;12:1727918. [CrossRef]
25. Tsimikas S, Karwatowska-Prokopczuk E, Gouni-Berthold I, et al. Lipoprotein(a) reduction in persons with cardiovascular disease. *N Engl J Med*. 2020;382(3):244-255. [CrossRef]
26. Cho L, Nicholls SJ, Nordestgaard BG, et al. Design and rationale of Lp(a)HORIZON trial: assessing the effect of lipoprotein(a) lowering with Pelacarsen on major cardiovascular events in patients with CVD and elevated Lp(a). *Am Heart J*. 2025;287:1-9. [CrossRef]
27. O'Donoghue ML, Rosenson RS, Gencer B, et al. Small interfering RNA to reduce lipoprotein(a) in cardiovascular disease. *N Engl J Med*. 2022;387(20):1855-1864. [CrossRef]
28. Study details | NCT05581303 | Olpasiran trials of cardiovascular events and lipoprotein(a) reduction (OCEAN(a)) - outcomes trial. *Clinicaltrials.gov*. https://clinicaltrials.gov/study/NCT05581303. Accessed January 8, 2026.