Bristol Publishers

Case Reports and Case Series in Cardiology Journal

Case Report | Open Access | Volume: 2 Issue: 2 (April-June 2026) Pages: 36-41

ISSN: 3107-6416 (Online) | DOI: doi.org/10.64874/crcscj.v2i2.2026.024

Severe Elevation of Lipoprotein (A) in a Middle-aged Woman with Strong Familial Premature Coronary Disease: A Case Highlighting the Importance of Early Lp (A) Assessment

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Received: 05 April 2026; **Revised:** 19 May 2026; **Accepted:** 20 May 2026; **Published:** 28 May 2026

Academic Editor: Dr. Mohamed Ahmed Mostafa

Abstract

Lipoprotein(a) [Lp(a)] is genetically determined lipoprotein which independently contributes to atherosclerotic cardiovascular disease (ASCVD) and calcific aortic valve disease [1,2]. Despite its recognised causal role, Lp(a) measurement remains underutilised in routine practice. We present a woman (49 years old) with strong family history of premature coronary artery disease (PCAD) who was found to have a markedly elevated Lp(a) concentration of 753 nmol/L. Her LDL-cholesterol improved from 5.8 to 2.6mmol/L with high-intensity statin therapy, yet her persistently elevated Lp(a) placed her at very high lifetime cardiovascular risk. Comprehensive laboratory evaluation revealed pre-diabetes, normal renal and liver function, and no evidence of systemic inflammation. This case emphasises the importance of targeted Lp(a) testing, early risk stratification, and aggressive LDL-cholesterol reduction in individuals with inherited cardiovascular risk factors. Emerging Lp(a)-lowering therapies may further transform management in this high-risk population.

Introduction

Lipoprotein(a) is a cholesterol-rich particle structurally similar to LDL, distinguished by the presence of apolipoprotein(a), a highly polymorphic glycoprotein with homology to plasminogen [1]. This unique structure confers both atherogenic and pro-thrombotic properties, contributing to plaque formation, endothelial dysfunction, and impaired fibrinolysis [2]. More than 90% of circulating Lp(a) levels are genetically determined and remain stable in life [3].

Increased Lp(a) is now recognised as causal risk as well as independent feature for peripheral arterial disease, myocardial infarction, stroke, calcific aortic valve stenosis [4-6]. Roughly 20–25% of population worldwide has elevated Lp(a), with concentrations above 430 nmol/L associated with 2-to-4-fold rise in ASCVD risk [7]. Despite this, Lp(a) testing is not routinely performed, partly due to historical lack of targeted therapies [8].

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According to present guidelines, at least one lifetime measurement is advised, especially for people who have strong family history, premature ASCVD, or recurrent events even with optimum LDL-cholesterol control [9,10]. With the development of antisense oligonucleotide and small interfering RNA therapies capable of reducing Lp(a) by up to 80–90%, early identification of high-risk individuals is becoming increasingly important [11-13]. This case highlights the clinical relevance of severe Lp(a) elevation in a patient with strong familial cardiovascular risk and discusses contemporary management strategies.

Cases Presentation

A 49-year-old woman presented for cardiovascular risk assessment after relocating to Australia. Her medical history included hypertension managed with irbesartan and anxiety treated with paroxetine. She denied smoking, diabetes, or chronic inflammatory disease. Her family history was significant for PCAD: her father developed angina in his twenties, and her mother had established ischaemic heart disease.

A fasting lipid profile performed by her previous general practitioner revealed an LDL-cholesterol of 5.8 mmol/L. She was commenced on rosuvastatin 40 mg daily, which she tolerated well. On review in our clinic, repeat testing demonstrated improvement in total cholesterol of 4.9mmol/L, LDL-cholesterol to 2.6mmol/L, triglycerides of 2mmol/L, HDL-cholesterol of 1.51mmol/L.
Given her strong family history, an Lp(a) level was obtained and found to be markedly elevated at 753 nmol/L, placing her in the "very high-risk" category [14]. Comprehensive laboratory evaluation showed normal renal function (creatinine 66µmol/L, eGFR>90 mL/min/1.73m²), normal liver function tests, normal electrolytes, and a TSH of 1.9 mIU/L. HbA1c was 5.9%, consistent with pre-diabetes. Full blood count and iron studies were within acceptable limits, with mild iron saturation reduction. Urine albumin-creatinine ratio was 1.5mg/mmol, within normal limits.

She was counselled regarding the implications of elevated Lp(a) and the need for long term cardiovascular risk reduction. Ezetimibe 10 mg daily was added to further lower LDL cholesterol, with a target of <1.4 mmol/L because of her inherited risk profile [15]. A coronary artery calcium (CAC) score and transthoracic echocardiogram were arranged to assess for subclinical atherosclerosis and early aortic valve involvement. Her CAC score was 8, and echocardiography was unremarkable. Also, her Dutch Lipid Score was under 6.

Following discussion with the cardiology team, PCSK9 inhibitor therapy was considered appropriate to help achieve an LDL cholesterol level below 1.4 mmol/L, in line with Australian guidance for patients at high cardiovascular risk. At 8 week follow up, her LDL cholesterol had fallen further to 1.9 mmol/L on dual therapy. However, she did not meet Pharmaceutical Benefits Scheme (PBS) criteria for subsidised PCSK9 inhibitor therapy, and the anticipated out of pocket cost was a barrier to treatment. She therefore elected to continue current non pharmacological measures and intensive lipid lowering therapy, with ongoing monitoring.

Table 1: Clinical and Laboratory Findings.

Parameter	Result	Reference Range
Lipid Profile		
Total Cholesterol	4.9mmol/L	
LDL-C	2.6mmol/L	
HDL-C	1.51mmol/L	>1.09 mmol/L
Triglycerides	2mmol/L	
Non-HDL Cholesterol	3.39mmol/L	
Chol/HDL Ratio	3.2	

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Parameter	Result	Reference Range
Lipoprotein(a)	**753 nmol/L (very high)**	
Glycaemic Markers		
HbA1c (NGSP)	**5.9%**	
HbA1c (IFCC)	41 mmol/mol	
Fasting Glucose	6.6mmol/L (H)	3.6–6mmol/L
Renal Function		
Creatinine	66 µmol/L	45–85 µmol/L
eGFR	>90 mL/min/1.73 m²	>59
Urea	3.1mmol/L	2.5–7mmol/L
Urate	0.220mmol/L	0.150–0.400mmol/L
Urine ACR		
Urine Albumin	30 mg/L (H)	
Urine Creatinine	19.6 mmol/L	—
Albumin/Creatinine Ratio	**1.5 mg/mmol**	0.0–3.6 mg/mmol
Electrolytes		
Sodium	140mmol/L	135–145mmol/L
Potassium	4.3mmol/L	3.5–5.5mmol/L
Chloride	106mmol/L	95–110mmol/L
Bicarbonate	26mmol/L	20–32mmol/L
Anion Gap	8 mmol/L	
Corrected Calcium	2.35mmol/L	2.10–2.6mmol/L
Phosphate	1.16mmol/L	0.80–1.5mmol/L
Liver Function Tests		
ALT	27U/L	5–30U/L
AST	20U/L	10–35U/L
ALP	68U/L	20–105 U/L
GGT	16U/L	5–35U/L
Total Bilirubin	5µmol/L	
Albumin	41g/L	33–46g/L
Total Protein	68g/L	64–81g/L
Globulin	27 g/L	23–43 g/L
LD	98 U/L	
Thyroid Function		
TSH	1.9mIU/L	0.3–3.5mIU/L
Full Blood Count		
Haemoglobin	143 g/L	115–165 g/L
Haematocrit	0.42	0.35–0.47
RBC	4.5 ×10¹²/L	3.9–5.6
MCV	92 fL	80–100 fL
WCC	11.4 ×10⁹/L	3.5–12.0
Neutrophils	7.53 ×10⁹/L	1.5–8
Lymphocytes	2.68 ×10⁹/L	1.0–4

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Parameter	Result	Reference Range
Monocytes	0.61 ×10⁹/L	0–0.9
Eosinophils	0.52 ×10⁹/L	0–0.6
Basophils	0.05 ×10⁹/L	0–0.15
Platelets	308 ×10⁹/L	150–400
Iron Studies		
Iron	14 µmol/L	5–30 µmol/L
Transferrin	3.2 g/L (H)	1.9–3.1 g/L
TIBC	80 µmol/L (H)	47–77 µmol/L
Saturation	18% (L)	20–45%
Ferritin	66 µg/L	30–300 µg/L
Urine MCS	No pathogens	—
Leucocytes	110 ×10⁶/L (H)	
Erythrocytes		
Specific Gravity	1.021	1.005–1.030

Discussion

This case illustrates the importance of measuring Lp(a) in individuals having strong family history of PCAD. The patient's Lp(a) concentration of 753 nmol/L places her in the highest risk category, associated with significantly increased lifetime ASCVD risk [16]. Although her LDL-cholesterol responded well to high-intensity statin therapy, her genetically determined Lp(a) elevation represents a substantial source of residual risk.

Statins have minimal effect on Lp(a) and may slightly increase levels [17]. PCSK9 inhibitors reduce Lp(a) by approximately 20–30% and have demonstrated cardiovascular benefit in high-risk individuals [18,19]. However, access and cost may limit their use. Novel therapies targeting apolipoprotein(a) synthesis, including pelacarsen and olpasiran, have shown reductions of up to 80–90% in clinical trials and may soon offer targeted treatment options [11-13].

The patient's initial LDL-cholesterol raised the possibility of familial hypercholesterolaemia; however, the absence of clinical stigmata and the substantial response to statin therapy suggested a polygenic or mixed dyslipidaemia [20]. The coexistence of elevated LDL-cholesterol and markedly elevated Lp(a) significantly amplifies cardiovascular risk (21). CAC scoring is a valuable tool for refining risk assessment, as individuals with high Lp(a) but zero CAC may have lower short-term risk, whereas elevated CAC warrants more intensive intervention [22,23].

This case reinforces the need for clinicians to consider Lp(a) testing in patients having unexplained cardiovascular risk or strong family history. Early identification allows for personalised risk reduction strategies and timely monitoring for aortic valve disease, which is more common in individuals with elevated Lp(a) [24].

Conclusions

This report describes a middle aged woman with profound Lp(a) elevation and strong family history of PCAD. While high intensity statin therapy achieved a substantial reduction in LDL cholesterol, her Lp(a) level remains significant determinant of lifelong cardiovascular risk. Measuring Lp(a) in appropriately selected patients can prompt earlier, more intensive risk management, including ambitious LDL cholesterol targets and comprehensive modification of other risk factors. As dedicated Lp(a) lowering treatments progress toward clinical use, identifying patients with severe elevation will become increasingly relevant to enabling targeted intervention.

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Learning Outcomes:

Consider ordering Lp(a) in patients having premature "cardiovascular disease (CVD)" and/or a compelling family history suggestive of inherited risk.

Severe Lp(a) elevation is predominantly inherited and is linked to both atherosclerotic disease and calcific aortic valve stenosis as an independent risk contributor.

Current management is centred on lowering LDL cholesterol as far as feasible and addressing other modifiable risk factors (blood pressure, glycaemia, weight, and lifestyle).

Lp(a) specific drugs are emerging; earlier detection of very high levels may help clinicians and patients plan timely, targeted therapy once available.

REFERENCES

1. Tsimikas S. A Test in Context: Lipoprotein(a). J Am Coll Cardiol. 2017; 69: 692-711. doi:10.1016/j.jacc.2016.11.042.
2. Nordestgaard BG, Chapman MJ, Ray K, et al. Lipoprotein(a) as a Cardiovascular Risk Factor: Current Status. Eur Heart J. 2010; 31: 2844-2853. doi:10.1093/eurheartj/ehq386.
3. Clarke R, Peden JF, Hopewell JC, et al. Genetic Variants Associated with Lp(a) Lipoprotein Level and Coronary Disease. N Engl J Med. 2009; 361: 2518-2528. doi:10.1056/NEJMoa0902604.
4. Kamstrup PR, Tybjærg Hansen A, Nordestgaard BG. Elevated Lipoprotein(a) and Risk of Myocardial Infarction. JAMA. 2009; 301: 2331-2339. doi:10.1001/jama.2009.801.
5. Thanassoulis G, Campbell CY, Owens DS, et al. Genetic Associations with Valvular Calcification and Aortic Stenosis. N Engl J Med. 2013; 368: 503-512. doi:10.1056/NEJMoa1109034.
6. O'Donoghue ML, Fazio S, Giugliano RP, et al. Lipoprotein(a), PCSK9 Inhibition, and Cardiovascular Risk. Circulation. 2019; 139: 1483-1492. doi:10.1161/CIRCULATIONAHA.118.037184.
7. Erqou S, Kaptoge S, Perry PL, et al. Lipoprotein(a) Concentration and the Risk of Coronary Heart Disease. JAMA. 2009; 302: 412-423. doi:10.1001/jama.2009.1063.
8. Wilson DP, Jacobson TA, Jones PH, et al. Use of Lp(a) in Clinical Practice: A Biomarker whose Time has Come. Atherosclerosis. 2019; 291: 62-70. doi:10.1016/j.atherosclerosis.2019.10.007.
9. Mach F, Baigent C, Catapano AL, et al. 2019 ESC/EAS Guidelines for the Management of Dyslipidaemias. Eur Heart J. 2020; 41: 111-188. doi:10.1093/eurheartj/ehz455.
10. Grundy SM, Stone NJ, Bailey AL, et al. 2018 AHA/ACC Cholesterol Guideline. Circulation. 2019; 139: e1082-e1143. doi:10.1161/CIR.0000000000000625.
11. Viney NJ, van Capelleveen JC, Geary RS, et al. Antisense Oligonucleotides Targeting Apolipoprotein(a). Lancet. 2016; 388: 2239-2253. doi:10.1016/S0140 6736(16)310091.
12. Tsimikas S, Karwatowska Prokopczuk E, Gouni Berthold I, et al. Lipoprotein(a) Reduction in Persons with Cardiovascular Disease. N Engl J Med. 2020; 382: 244-255. doi:10.1056/NEJMoa1905239.
13. Nissen SE, Wolski K, Balog C, et al. Olpasiran for Lipoprotein(a) Reduction in ASCVD. N Engl J Med. 2022; 387: 1833-1844. doi:10.1056/NEJMoa2200460.
14. Australian Atherosclerosis Society. Lp(a) Risk Thresholds. Heart Lung Circ. 2023; 32: 287-296. doi:10.1016/j.hlc.2022.11.002.
15. Cannon CP, Blazing MA, Giugliano RP, et al. Ezetimibe added to Statin Therapy after ACS. N Engl J Med. 2015; 372: 2387-2397. doi:10.1056/NEJMoa1410489.

Citation: Gul A. Severe Elevation of Lipoprotein (A) in a Middle-aged Woman with Strong Familial Premature Coronary Disease: A Case Highlighting the Importance of Early Lp (A) Assessment. Case Rep Case Ser Cardiol J. April-June 2026; 02(02): 36-41. **DOI:** doi.org/10.64874/crcscj.v2i2.2026.024.

16. Boffa MB, Koschinsky ML. Lipoprotein(a): Truly a Direct Prothrombotic Factor? J Lipid Res. 2016; 57: 745-757. doi:10.1194/jlr.R067314.
17. Sahebkar A, Simental Mendía LE, Watts GF, et al. Effect of Statins on Lp(a): A Systematic Review. Br J Clin Pharmacol. 2016; 81: 646-655. doi:10.1111/bcp.12844.
18. Sabatine MS, Giugliano RP, Keech AC, et al. Evolocumab and Clinical Outcomes. N Engl J Med. 2017; 376: 1713-1722. doi:10.1056/NEJMoa1615664.
19. Schwartz GG, Steg PG, Szarek M, et al. Alirocumab and Cardiovascular Outcomes. N Engl J Med. 2018; 379: 2097-2107. doi:10.1056/NEJMoa1801174.
20. Sturm AC, Knowles JW, Gidding SS, et al. Familial Hypercholesterolemia Diagnosis and Management. J Am Coll Cardiol. 2018; 72: 662-680. doi:10.1016/j.jacc.2018.05.044.
21. Langsted A, Kamstrup PR, Nordestgaard BG. High Lp(a) and High LDL C: Double Risk. Atherosclerosis. 2016; 252: 193-198. doi:10.1016/j.atherosclerosis.2016.07.919.
22. Nasir K, Budoff MJ, Wong ND, et al. Coronary Artery Calcium and Cardiovascular Risk Prediction. J Am Coll Cardiol. 2012; 59: 434-447. doi:10.1016/j.jacc.2011.06.066.
23. Mehta A, Virani SS, Ayers CR, et al. Lipoprotein(a) and Coronary Artery Calcium. Circulation. 2020; 142: 1579-1589. doi:10.1161/CIRCULATIONAHA.120.046886.
24. Capoulade R, Chan KL, Yeang C, et al. Oxidized Phospholipids, Lp(a), and Aortic Stenosis Progression. J Am Coll Cardiol. 2015; 66: 1236-1246. doi:10.1016/j.jacc.2015.07.020.

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