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Can Elevated Lipoprotein(A) Drive Severe Atherosclerosis in the Complete Absence of Cardiovascular Risk Factors?

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Abstract

Background: Lipoprotein(A) (Lp(a)) is an inherited and independent risk factor for atherosclerotic cardiovascular disease. Because levels are genetically determined and unaffected by lifestyle, its contribution is often overlooked. Markedly elevated Lp(a) can cause aggressive coronary atherosclerosis even without traditional cardiovascular risk factors. This case describes sudden cardiac arrest attributed to isolated Lp(a)-mediated coronary disease.

Methods: This case report was developed through retrospective review of the patient's electronic medical record. Clinical history, examination findings, laboratory data, cardiac imaging, and coronary angiography were assessed. Diagnostic evaluation included electrocardiography, transthoracic echocardiography, computed tomography pulmonary angiography, computed tomography of the head and neck, and invasive coronary angiography. Laboratory tests included lipid profile, inflammatory markers, high-sensitivity troponin, glycated haemoglobin, renal function tests, and quantitative Lp(a) measurement reported in nmol/L.

Results: A 67-year-old man with no past cardiovascular disease, diabetes, smoking history, hyperlipidaemia, hypertension, or family history of premature coronary disease suffered an out-of-hospital ventricular fibrillation arrest. Post-resuscitation evaluation showed preserved ventricular function and no reversible acute pathology. Coronary angiography demonstrated severe diffuse stenosis of the left anterior descending artery, additional disease in the left circumflex artery, and collateral supply from a non-dominant right coronary artery. Routine lipid parameters were within normal limits, while Lp(a) was markedly elevated at 216 nmol/L. The patient underwent urgent triple coronary artery bypass grafting and recovered well.

Conclusions: This case illustrates that markedly elevated Lp(a) can act as the primary driver of severe coronary artery disease and may present as sudden cardiac arrest in individuals without conventional risk factors. Measuring Lp(a) in unexplained or premature coronary presentations may support earlier recognition of this underdiagnosed condition.

Keywords: Lipoprotein(A); Atherosclerosis; Coronary artery disease; Sudden cardiac arrest; Diffuse coronary disease; Genetic dyslipidaemia; Premature ASCVD; Coronary artery bypass surgery.

Introduction

Lipoprotein(A) [Lp(a)] is a genetically determined risk factor for atherosclerotic cardiovascular disease (ASCVD), including coronary artery disease, ischaemic stroke, and aortic valve stenosis. Structurally, Lp(a) is a variant of low-density lipoprotein (LDL), consisting of apolipoprotein B-100

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covalently bound to apolipoprotein(a). The presence of apolipoprotein(a) contributes to its proatherogenic and prothrombotic properties [1-5]. Lp(a) levels are largely genetically determined and are not significantly influenced by diet or environmental factors. A series of three prospective studies in Copenhagen demonstrated a causal association between elevated Lp(a) levels and myocardial infarction [6]. It is estimated that approximately 1 in 5 individuals globally are at high risk of developing ASCVD due to elevated Lp(a) levels [7]. The distribution of Lp(a) is generally similar between men and women [8], although levels tend to rise slightly in women after menopause [9]. Ethnic differences in Lp(a) concentrations have also been observed, likely due to underlying genetic variation [10]. Recommendations for screening vary across guidelines. However, the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS) recommend measuring Lp(a) levels at least once in a person's lifetime, especially in those with family history of premature ASCVD, and those with moderate to high risk of ASCVD [11,12]. Currently, there are no approved therapies specifically targeting elevated Lp(a), although ongoing research is exploring potential treatment options[1]. We present an unusual case of cardiac arrest as a first presentation of advanced coronary atherosclerosis with elevated lipoprotein A as the only cardiovascular risk factor.

History of Presentation

A 67-year-old man, non-smoker, with no prior history of cardiovascular disease, diabetes, or hyperlipidaemia collapsed suddenly at home. Emergency medical services found him in ventricular fibrillation. He received an initial defibrillation shock, which converted the rhythm to asystole. He was haemodynamically stable upon arrival to the hospital and admitted to the ICU.

Past Medical History

The patient was previously fit and well, on no regular medications, and had no history of chest pain, exertional symptoms, or prior cardiovascular evaluations. No family history of premature cardiac disease was reported. The patient was physically active and jogged once a week. He was a slim build with a body mass index of 22. There was no history of recreational drug use or comorbidities that could be pro-inflammatory.

Investigations

Post-arrest ECG showed no ischaemic changes or conduction abnormalities. Laboratory results are summarised in table 1, demonstrating elevated troponin, normal HbA1c, and largely normal lipid profile. CRP was mildly elevated, likely reflective of the acute event.

Echocardiography demonstrated preserved left ventricular systolic function (EF 55-60%), normal diastolic function, and normal right ventricular function, with no significant valvular disease apart from aortic sclerosis.

Table 1: laboratory results for patient.

Test	Result	Reference Range
HbA1c	36 mmol/mol	<41
Troponin	20 → 490 ng/L	<14
Total cholesterol	3.4 mmol/L	<5.0
LDL	2.1 mmol/L	<3.0
HDL	0.7 mmol/L	0.9–2.3
Triglycerides	1.5 mmol/L	<1.7
Non-HDL cholesterol	2.7 mmol/L	<4.0
Lipoprotein(a)	216 nmol/L	<75
CRP	7 mg/L	<5
Sodium	143 mmol/L	133–146
Potassium	3.8 mmol/L	3.5–5.3
Creatinine	93 µmol/L	59–104
eGFR	73 mL/min/1.73m²	>60
Albumin	33 g/L	35–50

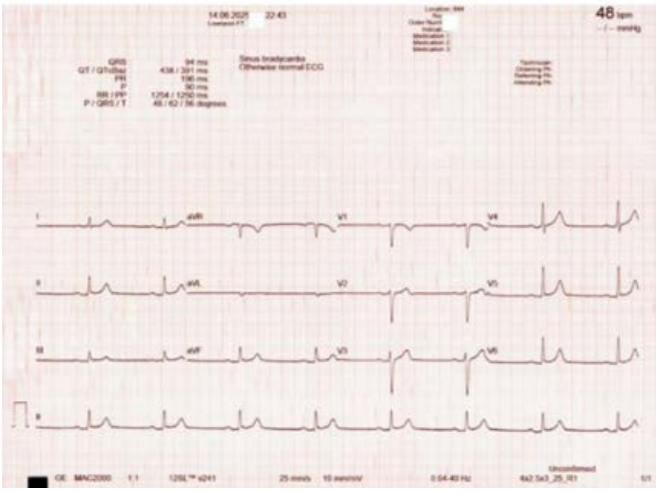

Figure 1: Twelve-lead electrocardiogram post return of spontaneous circulation.

CT pulmonary angiogram (figure 2) excluded pulmonary embolism or right heart strain; minimal basal consolidation and CPR-related costomanubrial fractures were observed. There was, however, evidence of atherosclerotic calcifications in proximal left anterior descending artery (LAD) (arrows).

Computed tomographic Axial (figure 2a left) cut at basal level of ascending aorta (left main artery origin) and long axis cut of left ventricle (right, figure 2b).

Computed tomography of head/neck excluded acute intracranial or cervical spine pathology. Invasive coronary angiography (figure 3) revealed severe diffuse disease of the proximal and mid left anterior artery branch of left coronary artery (LAD), severe stenosis of the first diagonal ostium, further mid-LAD disease , and severe distal disease in the

Abuzahra T, et al., Arch Clin Biomed Res 2026
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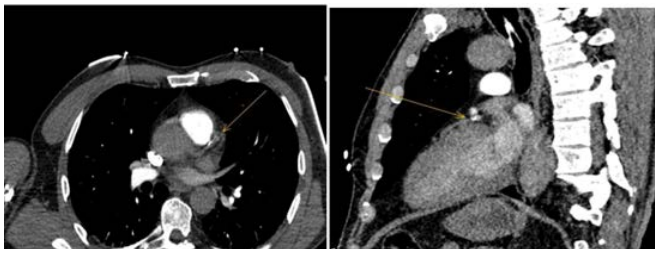

left circumflex artery (LCx) with impaired phasic flow to the posterior descending artery (PDA). The Right coronary artery (RCA) was nondominant but provided retrograde collateral flow to the PDA. Given the extent of coronary atherosclerosis identified on both CT imaging and invasive angiography in an individual without conventional cardiovascular risk factors, lipoprotein(a) was measured and found to be markedly elevated. This was considered the most likely driver of the patient's diffuse coronary disease.

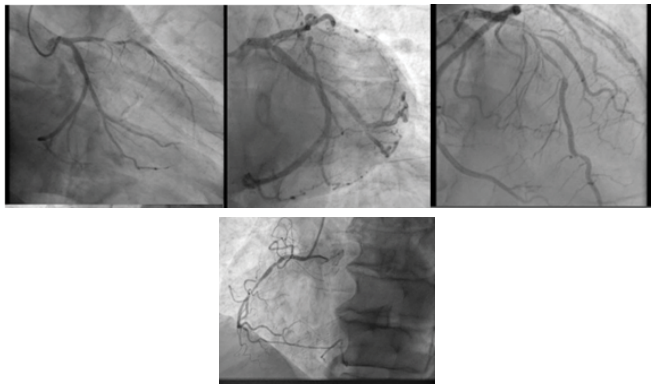

Figure 3a-c (from left to right, RAO caudal projection, PA caudal and PA cranial projection of left coronary artery demonstrating severe proximal and mid left anterior descending artery disease with severe ostial diagonal narrowing. Figure 3d (below)- demonstrates severe diffuse atherosclerotic disease in a nondominant right coronary artery.

Management

The patient was initially managed in the intensive care unit without inotropic support and was subsequently transferred to the Acute Cardiac Care Unit. In view of the extensive coronary disease, he underwent urgent triple coronary artery bypass surgery. The patient was discharged home with cardiology follow-up and secondary prevention therapy, including statins, beta-blockers, and antiplatelet therapy. Genetic counselling for Lp(a) elevation and family screening was recommended.

Discussion

Lp(a) is known to contribute to atherosclerosis through a combination of proatherogenic, prothrombotic, and pro-inflammatory mechanisms. Despite a normal lipid profile and HbA1c, the patient's elevated Lp(a) likely drove the diffuse and severe coronary disease identified on angiography. This underscores the importance of considering Lp(a) in patients with stroke or acute myocardial infarction who have an absence of cardiovascular risk factors. The absence of ECG changes or preceding symptoms further complicates the diagnosis, demonstrating that occult coronary disease should be considered despite an absence of risk factors or preceding symptoms. Early invasive coronary imaging was critical in identifying the culprit lesion and expediting surgical revascularisation. Current guidelines vary regarding Lp(a) testing, but the 2016 ESC and 2019 ESC/EAS guidelines advise measuring Lp(a) at least once in a person's lifetime, especially in individuals with a family history of premature ASCVD or moderate to high risk [11,12]. However, routine testing remains underutilised in many clinical settings. This case highlights the need for heightened clinical awareness and a lower threshold for Lp(a) measurement in patients with myocardial infarction in whom traditional cardiovascular risk factors are absent or minimal. Currently, no therapies are approved to specifically lower Lp(a), though promising developments are underway. Clinical trials involving antisense oligonucleotides [13] and small interfering RNAs such as lepodisiran [14] have shown significant reductions in Lp(a) concentrations and may offer future therapeutic options. Until then, risk mitigation relies on aggressive management of modifiable factors and appropriate secondary prevention strategies [3-15].

Conclusion

This case highlights the role of elevated Lipoprotein(A) as a potential driver of severe coronary artery disease and sudden cardiac arrest in patients without traditional risk factors.

Learning Objectives / Take-Home Messages

- Elevated Lipoprotein(A) can be an isolated but significant risk factor for severe, premature coronary artery disease.
- Lp(a) testing should be considered in unexplained or premature ASCVD, even with a normal lipid profile.

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