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CASE REPORT

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Elevated Lipoprotein(a) Linked to Recurrent Cardiovascular Events – A Case Report

Aurelian Roșca[1,2], Bianca Ion[2], Theofana Mihăilă[1,2]

[1] "George Emil Palade" University of Medicine, Pharmacy, Science and Technology, Târgu Mureș, Romania
[2] Clinic of Cardiology, Emergency Clinical County Hospital, Târgu Mureș, Romania

CORRESPONDENCE

Bianca Ion
Str. Gheorghe Marinescu nr. 38
540136 Târgu Mureș, Romania
Tel: +40 265 215 551
E-mail: biancapopovici12@gmail.com

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ABSTRACT

The role of lipoprotein(a) [Lp(a)] in the development of atherosclerosis has been recently recognized, and the current recommendation is to measure Lp(a) once in a lifetime in all individuals, in order to identify those at risk for developing an acute coronary syndrome or recurrent events, even in the absence of other cardiovascular risk factors. We present the case of a middle-aged patient with recurrent cardiovascular events, in whom we identified high levels of Lp(a) as a possible explanation of the recurrent events.

Keywords: atherosclerosis, lipoprotein(a), acute coronary syndromes

INTRODUCTION

Lipoprotein(a) [Lp(a)] is an established and genetically determined risk factor for atherosclerosis, coronary heart disease, stroke, thrombosis, and aortic stenosis. Structurally, it is a variant of LDL-cholesterol. Levels of Lp(a) above 50 mg/DL are correlated with an increased risk of cardiovascular disease. Screening patients to determine their Lp(a) levels could help identify those who need more aggressive lipid therapy and cardiovascular disease risk management.

It has been suggested that Lp(a) could provide a possible explanation for younger patients suffering from coronary artery disease with or without other risk factors. We present the case of a middle-aged patient with elevated Lp(a) levels resulting in multiple cardiovascular events.

CASE PRESENTATION

A 57-year-old hypertensive, diabetic patient with a history of post-inferior myocardial infarction, treated with primary revascularization and implantation of two pharmacologically active stents in the right coronary artery, presented with constrictive chest pain at rest, associated with fatigue and dyspnea, started two weeks prior to presentation and exacerbated on the day of admission. The electrocardiogram showed flattened T waves in V4–V6, DI,

Aurelian Roșca • Str. Gheorghe Marinescu nr. 50, 540136 Târgu Mureș, Romania. Tel: +40 265 372 653, E-mail: rosca_aurelian@yahoo.com

Theofana Mihăilă • Str. Gheorghe Marinescu nr. 50, 540139 Târgu Mureș, Romania. Tel: +40 265 372 653, E-mail: theofana_m@yahoo.com

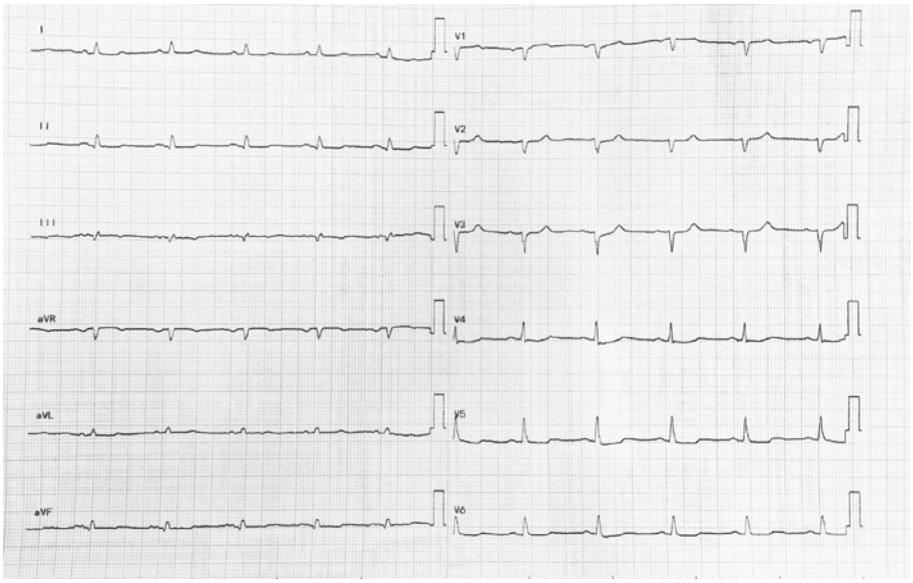


FIGURE 1. ECG at presentation, suggestive for diffuse ischemia

and DII, and amputated R waves in V1–V3. Troponin values were normal.

Transthoracic echocardiography showed slightly impaired systolic function, with basal interventricular septum hypokinesis, a left ventricular ejection fraction of 45%, diastolic dysfunction-type altered relaxation, minor mitral regurgitation, and minor tricuspid regurgitation. Urgent coronary angiography revealed a stenosis of the distal left main coronary artery, extending to the anterior descending artery and to the circumflex artery, and patent stents in the right coronary artery. Taking into account the repeated cardiovascular events of the patient, it was decided to measure Lp(a) levels, which were 0.77 g/L, considered very high.

The patient and the institution agreed with the publication of the case.

DISCUSSION

Lp(a) is considered an independent and unmodifiable risk factor for coronary heart disease, aortic valve stenosis, myocardial infarction, and ischemic stroke. The concentration of Lp(a) is strongly determined genetically, and its resemblance to plasminogen increases its atherogenicity.1 Moreover, unlike other lipoproteins characterized by constant masses, Lp(a) exhibits various isoforms of different sizes, inversely related to its concentration in plasma. Due to this particular characteristic, its measurement presents many challenges.1,2

The current recommendation is to perform a Lp(a) measurement once in a lifetime in all individuals. Although clinical data highlight the importance of reducing Lp(a) in order to reduce cardiovascular risk, there is no selective drug approved for Lp(a) hyperlipoproteinemia. Apheresis lowers both Lp(a) and LDL-cholesterol by removing proteins that contain apo-B. However, apheresis of lipoproteins does not reduce Lp(a) permanently and should be repeated every two weeks.3 Researchers are convinced that further international effort is needed in different ethnicities to assess the atherothrombotic risk due to Lp(a) on the one hand and apolipoprotein A on the other.4

In light of the introduction of new therapeutic approaches to lower Lp(a), the availability of well-standardized tests that provide comparability of results obtained by different laboratories is indispensable for the selection and classification of high-risk individuals.5 The introduction of new therapeutic approaches will require clinical trials to assess the clinical utility of Lp(a) decrease.6,7

CONCLUSIONS

In conclusion, elevated levels of Lp(a), a lipid molecule involved in atherosclerosis development, may be associated with the occurrence of acute coronary syndromes even at a young age, and even in patients with no major cardiovascular risk factors. Lp(a) determination should be performed in all patients presenting with chest pain, especially in those with no reasonable explanation for the acute cardiovascular event.

CONFLICT OF INTEREST

Nothing to declare.

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