Original paper

Lipoprotein(a) level assessment in internal medicine patients: cardiological vs. non-cardiological admissions

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Abstract

Introduction: Lipoprotein(a) [Lp(a)] is a lipid molecule similar to low-density lipoprotein cholesterol (LDL-C). Elevated Lp(a) has been associated with increased risk of atherosclerosis.
Aim of the research: The purpose of the study is to perform a screening analysis of Lp(a) levels in patients admitted to the Department of Internal Diseases, irrespective of whether their hospitalization is related to cardiological or non-cardiological reasons.
Material and methods: The study included 104 patients admitted to the Department of Internal Diseases and Clinical Pharmacology between 2 July 2021, and 3 December 2021, excluding those admitted due to SARS-CoV-2 infection. Patients were analysed based on the presence of atherosclerosis, Lp(a) levels, and other laboratory values. We divided them into two groups: those admitted due to cardiological complaints and those admitted due to other reasons.
Results: A total of 46 patients were affiliated with the group admitted for cardiological issues, while 58 were assigned to the group admitted for non-cardiological reasons. Upon further analysis of both groups regarding Lp(a) levels, it was observed that 14 out of the 46 patients (30.4%) admitted for cardiological reasons exhibited elevated lipoprotein levels ≥ 75 nmol/l. Thirteen (22.4%) out of the 58 patients in the group admitted for non-cardiological reasons demonstrated elevated Lp(a) levels. Except for HDL, no significant differences were observed between patients hospitalised due to cardiological and non-cardiological causes.
Conclusions: The findings emphasise the importance of universal Lp(a) screening, particularly in individuals without signs of atherosclerosis, to identify those at risk.

Introduction

Lipoprotein(a) is a proinflammatory, prothrombotic, and proatherogenic molecule. It is an undervalued but very important cardiovascular risk factor in patients with values above 75 nmol/l [1]. Lipoprotein(a) is a molecule consisting of an low-density lipoprotein (LDL)-like particle bound to apoB protein and apo(a) [2]. Due to these properties Lp(a) particles carry all atherogenic risk of low-density cholesterol although being more prone to oxidation and ability for endothelial penetration and promotion of foam cell production. Such properties are linked with homology between plasminogen and apolipoprotein(a), which both compete for binding sites on endothelial cells to inhibit fibrinolysis and promote intravascular thrombosis [2]. Lp(a) particle promotes formation of atherosclerotic plaque through various mechanisms. Lp(a) induces production of proinflammatory cytokines, including interleukins IL-1β, IL-6, and IL-8. Lp(a) enters the intima at similar rates to LDL cholesterol, although intensity of that process does not occur via lipoprotein receptor for LDL, but is dependent on Lp(a) plasma concentration, Lp(a) particle size, blood pressure, and arterial wall permeability [3]. Lp(a) tends to accumulate all over the intima, whereas LDL and other apo-B containing lipoproteins tend to accumulate only at atherosclerotic lesions. Atherosclerotic plaques in patients with elevated Lp(a) levels tend to have complex morphology [4]. These plaques tend to be prone to repeated ruptures and healing; these processes lead to fast progression of atherosclerosis. Cardiovascular incidents in patients with high Lp(a) tend to be severe and manifest as acute myocardial infarction rather than stable angina. What is more, according to studies patients with elevated Lp(a) levels have higher risk of recurring cardiovascular incidents due to the tendency of Lp(a) accumulation in injured sites in blood vessel walls, which results in restenosis of the coronary artery after revascularisation procedures [5]. Endothelial cell dysfunction followed by subendothelial lipoprotein accumulation and chronic inflammatory process often results in a wide spectrum of conditions, varying from mild valve thickening to severe calcification with stenotic occlusion.

Due to these properties, a high level of Lp(a) tends to be a major risk factor for ischaemic strokes, peripheral arterial disease, and calcific aortic valve disease [6]. Lp(a) concentration is considered as stable during the lifetime and determined by genetic factors, and there are no therapies dedicated to lowering its concentration [7]. Among available lipid-lowering drugs, inhibitors of PCSK9 or apheresis can lower Lp(a) concentration only by 30% [7]. In the newest guidelines it is recommended that the level of lp(a) be measured at least once in the lifetime of the patient. However, there is growing evidence that it may not be enough.

Furthermore, the evaluation of Lp(a) is more frequently conducted in patients with a cardiac history. The objective of this study is to perform a screening analysis of Lp(a) levels in patients admitted to the Department of Internal Diseases, irrespective of whether their hospitalisation is related to cardiological or non-cardiological reasons.

Aim of the research

We hypothesise that universal Lp(a) screening is crucial for identifying individuals at risk, particularly those without signs of atherosclerosis.

Material and methods

During this retrospective study, we analysed the data of 104 patients admitted to the Department of Internal Diseases and Clinical Pharmacology between 2 July 2021 and 3 December 2021, excluding those admitted due to SARS-CoV-2 infection. Patients were divided into two groups, patients admitted due to cardiological complaints (dyspnoea, decreased exercise tolerance, swelling of lower limbs, heart palpitations, angina pectoris, tightness of the chest, hypertension, weakness, syncope, dizziness, worsening of chronic heart failure, tachycardia, sweating, headaches, qualification and continuation of iPCSK9 therapy) and patients admitted due to other reasons (nausea, vomiting, dysphagia, cramping stomach ache, jaundice, increased body weight, diarrhoea, unwilling loss of body weight, increased abdominal circumference, chronic obstructive pulmonary disease, itchiness, loss of appetite, fever, anaemia, abnormal glomerular filtration rate (GFR), epileptic seizure, weakness of lower limbs, sepsis, renal failure, hyperkalaemia, presence of blood in stool, cough, constipation, abnormal labs, pneumonia). Further, cardiological patients were classified as follows: HF – heart failure, HA – arterial hypertension, arrhythmia, IHD – ischaemic heart disease, iPCSK9 – patients in B.101 drug program, follow-up on alirocumab and evolocumab. Non-cardiological patients were classified as follows: gastroenterological, neurological, anaemia, endocrinological, infections, pulmonological, others. Subsequently, we analysed Lp(a) levels among patients and the presence of atherosclerosis. We also analysed the following: total cholesterol, LDL, high-density lipoprotein (HDL), non-high-density lipoprotein (non-HDL), triglyceride, aspartate transaminase (AST), alanine transaminase (ALT), bilirubin, creatine kinase, thyroid-stimulating hormone (TSH), creatinine and eGFR level, history of atherosclerosis, cardiovascular incidents, and concurrent diseases. All laboratory measurements were performed routinely in patients during hospitalisation. Lp(a) level was assessed with the LPA 2 Tina-quant Lp(a) Gen.2 assay (Roche, Mannheim, Germany). An elevated Lp(a) ≥ 75 nmol/l cutoff point was selected based on Recommendations of the Experts of the Polish Cardiac Society (PCS) and the Polish Lipid Association (PoLA) on the diagnosis and management of elevated lipoprotein(a) levels [8].

Statistical analysis

The distribution of data was verified with the Shapiro-Wilk test. Most variables had a non-normal distribution. The relationships between pairs of groups were tested with Student's *t*-test (normal distribution) and the Mann-Whitney *U*-test (non-normal distribution). The relationships between categorical variables were assessed with the use of the χ^2 test or Fisher's exact test. A p-value < 0.05 was considered to be statistically significant. All the analyses were performed with PRISM 9.0.0 software.

Results

Throughout the study, we analysed results from a total of 104 patients. Subsequently, based on the criteria outlined in the Materials and Methods section, we categorised them into two groups based on

Table 1. Description of the study participants' classification

All patients 104							
Cardiological 46 (44.2%)				Non-cardiological 58 (55.7%)			
lp(a) elevated 14 (30.4%)		lp(a) not elevated 32 (69.6%)		lp(a) elevated 13 (22.4%)		lp(a) not elevated 45 (77.6%)	
Presence of atherosclerosis in patients							
Present 12 (85.8%)	Absent 2 (14.2%)	Present 19 (59.4%)	Absent 13 (40.6%)	Present 5 (38.5%)	Absent 8 (61.5%)	Present 19 (42.2%)	Absent 26 (57.8%)

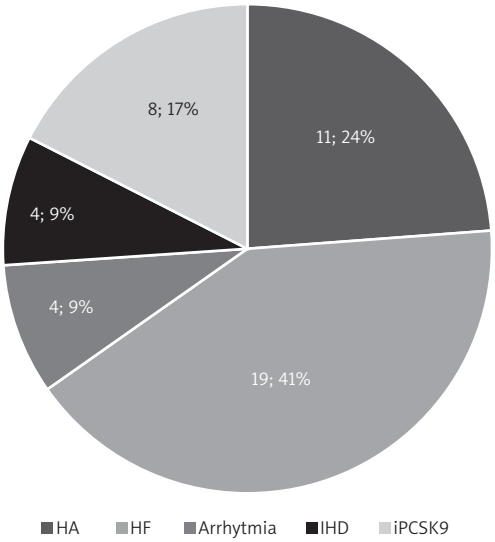

Figure 1. Cardiological reasons of admission, HA – arterial hypertension, *n* = 11; 24%, HF – decompensated heart failure *n* = 19; 41%, arrythmia *n* = 4; 9%, IHD – ischaemic heart disease *n* = 4; 9%, iPCSK9 – alirocumab, evolocumab treatment follow-up *n* = 8; 17%

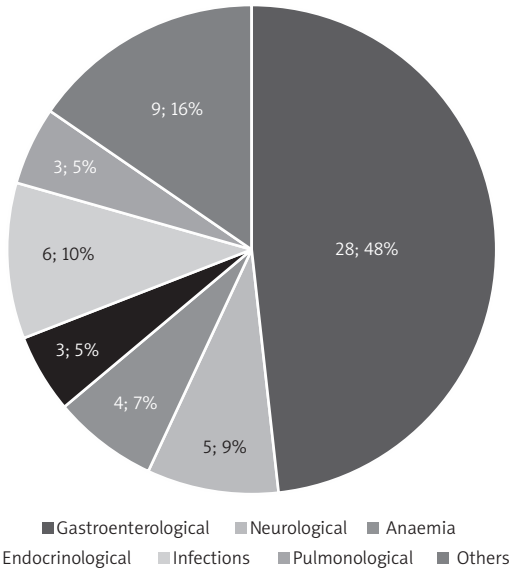

Figure 2. Non-cardiological reasons of admission, gastroenterological *n* = 28; 48%, neurological *n* = 5, 9%, endocrinological *n* = 3; 5%, infections *n* = 6; 10%, pulmonological *n* = 3; 5%, anaemia *n* = 4; 7%, and others *n* = 9; 16%

the cause of admission. Specifically, 46 patients were affiliated with the group admitted for cardiological issues, while 58 were assigned to the group admitted for non-cardiological reasons (Table 1).

Figure 1 presents the distribution of cardiology patients according to the cause of hospitalisation. Most cardiology patients were hospitalised due to heart failure exacerbation (HF group) – 19 (41%) patients. Eleven (24%) cardiology patients were hospitalised due to uncontrolled arterial hypertension (HA group), 8 (17%) were follow-up visits as part of the B.101 drug program for patients treated with alirocumab or evolocumab (iPCSK9 group), and 4 patients each (9%) were hospitalised due to ischaemic heart disease (IHD group) and arrhythmia (arrhythmia group) (Figure 1).

Among patients hospitalised for cardiac reasons, elevated Lp(a) levels were observed in 14 patients, accounting for 30.4% (Table 1). The proportion of patients with Lp(a) ≥ 75 nmol/l in the cardiology subgroups was as follows: HF – 3 out of 19 (33%), HT – 3 out of 9 (33%), and arrhythmia – 1 out of 4 (25%). None of the patients hospitalised due to ischaemic heart disease or pulmonary embolism had elevated Lp(a) levels. In the subgroup of patients hospitalised for follow-up within the B.101 drug program, the proportion of patients with Lp(a) > 75 nmol/l was as high as 7 out of 8 (88%).

Figure 2 presents the distribution of non-cardiology patients according to the cause of non-cardiac hospitalisation. The majority of non-cardiology patients were hospitalised for gastroenterological reasons – 28 (48%) patients. Six (10%) patients were hospitalised due to infections, 5 (9%) due to neurological causes, 4 (7%) due to anaemia, and 3 (5%) patients each due to endocrinological and pulmonological causes. Nine (16%) patients were hospitalised for other non-cardiac reasons (Figure 2).

Among patients hospitalised for non-cardiac reasons, elevated Lp(a) levels were observed in 13 patients, accounting for 22.4%. The proportion of patients with Lp(a) ≥ 75 nmol/l in the non-cardiac subgroups was as follows: gastroenterological – 5 out of 28 (17.9%), infections – 2 out of 6 (33.3%), neurological – 1 out of 5 (20%), endocrinological – 3 out of 3 (100%), and other non-cardiac causes – 2 out of 9 (22.2%). None of the patients hospitalised for pulmonological causes or anaemia had elevated Lp(a) levels.

Table 2 presents a comparative analysis of patients admitted for cardiological reasons versus those admitted for non-cardiological causes. Except for HDL, no significant differences were observed between patients hospitalised due to cardiological and non-cardiological causes.

Table 3 illustrates a comparison between patients with elevated Lp(a) levels and those without elevation within the groups hospitalised for cardiological and non-cardiological reasons.

Discussion

Given the association between elevated Lp(a) levels and increased cardiovascular risk, current guidelines recommend evaluating it at least once in a lifetime for all patients. Limited information is available regarding the patterns of Lp(a) testing in real-world clinical practice. According to a recent analysis,

Table 2. Comparative characteristics of patients hospitalised for cardiological and non-cardiological reasons

Data category	All patients *n* = 104	Non-cardiological patients *n* = 58	Cardiological patients *n* = 46	*P* for non-cardiological vs. cardiological
Age	68.0 ±14.8	68.8 ±16.8	66.9 ±12.0	0.1433
BMI	28.0 ±4.8	27.1 ±4.7	29.1 ±4.7	0.0620
Lp(a) [nmol/l]	74.7 ±120.1	56.3 ±93.7	97.8 ±144.7	0.0543
N of patients with Lp(a) ≥ 75 mmol/l	27 (25.96%)	13 (22.4%)	14 (30.4%)	0.3765
N of patients with Lp(a) ≥ 125 mmol/l	21 (20.2%)	9 (15.5%)	12 (26.1%)	0.2224
Total cholesterol [mg/dl]	169.6 ±82.9	175.0 ±102.9	164.0 ±55.5	0.6929
LDL [mg/dl]	91.0 ±65.8	94.2 ±77.8	86.7 ±51.4	0.8933
HDL [mg/dl]	44.9 ±17.2	38.2 ±15.0	51.8 ±16.8	0.0004
Non-HDL [mg/dl]	124.7 ±85.3	136.9 ±105.8	112.1 ±55.8	0.6240
TG [mg/dl]	144.0 ±126.5	167.4 ±169.9	120.1 ±45.3	0.3397
EF-ejection fraction	52.63 ±10.39	50.85 ±11.47	54.66 ±8.77	0.1240
ALT [U/l]	31.9 ±36.9	29.9 ±26.3	34.4 ±47.3	0.8941
AST [U/l]	49.9 ±78.2	48.3 ±47.1	52.5 ±112.9	0.1505
Bilirubin [mg/dl]	1.4 ±3.8	1.9 ±4.9	0.7 ±0.5	0.7735
CK [U/l]	151.8 ±170.6	121.6 ±74.8	191.7 ±243.1	0.8928
CK-MB mass [ng/dl]	3.06 ±2.43	3.40 ±2.47	2.61 ±2.34	0.0891
TSH [µ/l]	3.52 ±11.80	4.81 ±15.55	1.82 ±0.88	0.0811
fT3 [pmol/l]	3.31 ±1.13	3.20 ±1.25	3.72 ±0.36	0.4000
fT4 [pmol/l]	16.2 ±4.6	17.3 ±5.4	14.7 ±2.5	0.2453
Creatinine [mg/dl]	1.2 ±0.8	1.3 ±0.9	1.1 ±0.8	0.2362
GFR [ml/min/1.73 m^2]	69.7 ±33.2	70.1 ±40.3	69.2 ±21.7	0.8921

the testing of Lp(a) was most frequently conducted in patients with established atherosclerotic cardiovascular disease (ASCVD) and a history of multiple cardiovascular events [9]. There are no data on Lp(a) levels among patients without prior cardiovascular history. The aim of this study is a screening assessment of Lp(a) levels among patients hospitalised at the Department of Internal Diseases due to cardiological and non-cardiological indications. Interestingly, there was no significant difference in mean Lp(a) levels between patients hospitalised for cardiological reasons and those admitted for non-cardiological causes. Moreover, the number of patients with Lp(a) ≥ 75 nmol/l was also comparable between individuals hospitalised due to cardiological and non-cardiological reasons. What is more, if we exclude iPCSK9 patients from the analysis, the number of patients hospitalised due to cardiac reasons with Lp(a) ≥ 75 nmol/l is 7/38 (18.4%) in comparison to 13/58 (22.4%) in non-cardiological patients. This suggests that screening assessments for Lp(a) are equally relevant regardless of the reason for admission. In patients admitted for non-cardiological reasons with elevated Lp(a) levels, atherosclerosis was already present in 38.5% of individuals. This underscores the importance of universal Lp(a) screening and the necessity of further diagnosis of atherosclerosis including non-invasive methods, such as carotid ultrasound and angio-CT of coronary arteries, for individuals with elevated Lp(a) levels. The well-established genetic determination of Lp(a) levels underscores the significance of detecting elevated Lp(a) levels in patients without apparent signs of atherosclerosis [1]. Within patients exposed to elevated Lp(a) further examinations of lipoprotein levels in close family members are recommended, forming a crucial component of primary prevention strategies against atherosclerosis. According to recent studies, high levels of Lp(a) may be an intrinsic factor in development of atherosclerosis even if patients appear to be healthy [2, 10]. Screening for Lp(a) levels is crucial because elevated levels are often asymptomatic, and the initial manifestations may involve severe cardio-

Table 3. Comparative characteristics of patients with elevated vs. non-elevated Lp(a) levels within groups hospitalised due to cardiological and non-cardiological causes

Data category	Cardiological patients with normal Lp(a) levels *n* = 32	Cardiological patients with elevated Lp(a) levels *n* = 14	*P*-value	Non-cardiological patients with normal Lp(a) levels *n* = 45	Non-cardiological patients with elevated Lp(a) levels *n* = 13	*P*-value
Age	67.6 ±12.2	65.1 ±11.8	0.5355	71.4 ±13.8	59.7 ±22.6	0.2758
BMI	29.1 ±4.8	29.1 ±4.3	0.9545	27 ±5	27.4 ±3.6	0.8510
Lp(a) [nmol/l]	18.9 ±17.5	257.2 ±147.7	< 0.0001	15.5 ±16.7	200.3 ±108.2	< 0.0001
Tchol [mg/dl]	162.7 ±52.8	163.5 ±60.4	0.8415	169.8 ±98.5	191.3 ±115.1	0.6275
LDL [mg/dl]	89 ±48.4	82.7 ±55.9	0.8205	93.1 ±85	93.7 ±48.5	0.4838
HDL [mg/dl]	47.9 ±16.6	58.3 ±15.4	0.0095	38.7 ±16.9	40.7 ±13.9	0.6339
Non-HDL [mg/dl]	114.7 ±53.1	105.1 ±58.9	0.6831	132.1 ±104.9	147.4 ±109	0.4328
TG [mg/dl]	125.2 ±50.7	112.5 ±31.9	0.5315	148 ±92.9	229.7 ±301.1	0.4542
EF-ejection fraction	53.96 ±9.37	57.43 ±5.26	0.2523	49.93 ±12.21	55.00 ±6.45	0.6664
ALT [U/l]	35.6 ±54.7	31.3 ±25.3	0.9157	31 ±28.7	26.5 ±16.5	0.9124
AST [U/l]	57.2 ±130.4	39 ±37.6	0.5963	53.3 ±51.4	29.9 ±17.8	0.5158
Bilirubin [mg/dl]	0.8 ±0.5	0.6 ±0.3	0.1299	2.3 ±5.4	0.5 ±0.2	0.2060
CK [U/l]	123.2 ±128.8	264.5 ±294	0.4967	119.2 ±76.9	131.2 ±72.8	0.7936
CK-MB mass [ng/dl]	2.76 ±2.56	1.92 ±0.68	0.7946	3.60 ±2.49	2.53 ±2.35	0.2307
TSH [µ/l]	1.79 ±0.86	1.92 ±0.99	0.6963	2.99 ±7.30	10.42 ±28.91	0.7117
fT3 [pmol/l]	3.97 ±0	3.46 ±0	–	2.73 ±0.36	3.68 ±1.71	0.6626
fT4 [pmol/l]	13.8 ±3.1	15.0 ±2.5	0.8571	17.6 ±5.9	17 ±5.5	0.9113
Creatinine [mg/dl]	1.2 ±0.9	0.9 ±0.3	0.0049	1.2 ±0.6	1.6 ±1.5	0.5970
GFR [ml/min/1.73 m^2]	63.7 ±20.9	81.5 ±18.7	0.0089	68.2 ±39.3	77.3 ±45	0.5590

logical incidents or even sudden cardiac death. Among non-cardiological patients, screening of Lp(a) allowed us to identify 13 patients with Lp(a) ≥ 75 nmol/l. For some people, populational screening of Lp(a) is controversial due to a lack of targeted treatment. Awareness of elevated Lp(a) levels enables the implementation of measures to minimise the impact of other modifiable risk factors, particularly by further reducing LDL levels and addressing additional factors such as obesity, smoking, and sedentary lifestyle. Statistical estimation by Kelsey *et al.*, suggest that, to mitigate the cardiovascular risk associated with elevated Lp(a), initiating lipid-lowering therapy at an earlier stage is advisable [9]. This approach aims to further reduce LDL cholesterol and effectively compensate the cardiovascular risk associated with elevated Lp(a). Unfortunately, available lipid-lowering therapies do not lower Lp(a) directly, except for up to 30% reduction mediated by iPCSK9 [11]. Drugs aimed specifically at Lp(a) reduction are not available on the market yet; however, patients may receive them within clinical trials. Currently developed therapies include pelacarsen, a second-generation ASO drug conjugated with GalNAC3 and targeting apolipoprotein(a) mRNA, which demonstrated a reduction in Lp(a) levels by 35-80% in a phase 2 clinical trial [12]. Its potential to lower the risk of major adverse cardiovascular events (MACE) is currently being investigated in the HORIZON study. Similarly, Olpasiran, an siRNA, exhibited a 70.5% to 100.5% reduction in Lp(a) levels in the phase 2 OCEAN(a)-DOSE trial, with ongoing research exploring its impact on MACE [13]. Muvalaplin, an oral inhibitor of Lp(a) formation administered once daily, demonstrated a reduction in Lp(a) by 63–65% [14]. Population-wide screening for Lp(a) enables the identification of individuals who could potentially benefit from participation in clinical trials or those who may initiate therapy with novel drugs as soon as they become available in the market.

Conclusions

The findings emphasise the importance of universal Lp(a) screening, particularly in individuals without signs of atherosclerosis, to identify those at risk. Further research is recommended to explore the implications of elevated Lp(a) levels and to consider additional assessments for at-risk individuals.

Limitations

The limitations include the small sample size of patients. A longer observation period of patients would also bring important data. Analysis of subclinical atherosclerosis complications in a group of non-cardiological patients would also improve the study.

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Ethical approval

Approval number: RNN/180/25/KE.

Conflict of interest

The authors declare no conflict of interest.

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