UDC 616.13-004.6-02:577.112.85]-038:616.12-008.331.1(048.8)
https://doi.org/10.31612/2616-4868.5.2026.19

LIPOPROTEIN(A) AS A MODIFIER OF VASCULAR REMODELING AND CARDIOVASCULAR RISK IN ARTERIAL HYPERTENSION: A LITERATURE REVIEW

Tetiana M. Solomenchuk[1], Rustam K. Zhurayev[1], Vasyl V. Protsko[1,2], Kateryna S. Hel[1,2], Viktoriia R. Nykoliak[1]

[1]Danylo Halytsky Lviv National Medical University, Lviv, Ukraine
[2]Municipal non-profit enterprise «Lviv Territorial Medical Union 'Multidisciplinary Clinical Hospital of Emergency and Intensive Care'», Lviv, Ukraine

Abstract

Introduction. Elevated lipoprotein(a) has emerged as an important determinant of cardiovascular risk because of its roles in atherosclerotic disease, vascular inflammation, endothelial dysfunction, arterial stiffening, and vascular calcification. Increasing evidence suggests that the clinical significance of Lp(a) extends beyond atherosclerosis, as higher concentrations have also been linked to vascular remodeling and adverse cardiovascular outcomes in patients with arterial hypertension.
Aim. To evaluate the current scientific evidence on the role of lipoprotein(a) in vascular remodeling and its effects on endothelial function, arterial stiffness, and cardiovascular risk among patients with arterial hypertension.
Materials and methods. The MEDLINE database via PubMed was searched for articles published between 2016 and 2026 using a predefined set of keywords. The search yielded 86 articles, which were initially screened and assessed based on their titles and abstracts. After evaluating relevance, we selected 56 articles for detailed assessment. Finally, 31 articles were included in the review.
Results. Elevated Lp(a) concentrations are linked to multiple mechanisms that drive vascular remodeling and cardiovascular injury. A substantial part of Lp(a)'s pathogenic activity is attributed to oxidized phospholipids, which contribute to endothelial dysfunction, vascular inflammation, extracellular matrix remodeling, fibrosis, and vascular calcification. Research indicates associations between elevated Lp(a) levels and increased pulse pressure, reduced nocturnal blood pressure decline, arterial stiffness, vascular calcification, and hypertensive target-organ damage. These findings suggest that elevated Lp(a) may influence the development of a less favorable vascular profile in arterial hypertension and contribute to cardiovascular risk beyond that explained by traditional lipid markers. The development of RNA-based therapeutic approaches, including antisense oligonucleotides and small interfering RNA agents, has enabled substantial reductions in circulating Lp(a) levels and may provide new opportunities for individualized cardiovascular risk management.
Conclusions. Elevated lipoprotein(a) levels should be regarded as an important genetically determined factor in vascular remodeling and cardiovascular risk in patients with arterial hypertension. Increased lipoprotein(a) concentrations contribute to endothelial dysfunction, vascular inflammation, arterial stiffness, vascular fibrosis, and calcification through multiple mechanisms, predominantly mediated by oxidized phospholipids.

Keywords: **lipoprotein(a), arterial hypertension, vascular remodeling, cardiovascular genetics, arterial stiffness, cardiovascular risk**

INTRODUCTION

Arterial hypertension remains a leading cause of cardiovascular morbidity and mortality worldwide and is strongly associated with ischemic heart disease, stroke, chronic kidney disease, heart failure, and premature cardiovascular death. Despite advances in antihypertensive therapy, a substantial proportion of hypertensive patients still have residual cardiovascular risk even after reaching recommended blood pressure

targets. This suggests that mechanisms beyond elevated blood pressure itself contribute to vascular injury and adverse cardiovascular outcomes [1, 24, 30].

Among genetically determined cardiovascular risk factors, lipoprotein(a) (Lp(a)) has attracted growing attention in recent years. Lp(a) is a lipoprotein particle composed of a low-density lipoprotein (LDL)-like core and apolipoprotein(a), which is structurally homologous to plasminogen. Plasma Lp(a) levels are largely determined by the LPA gene and remain relatively stable throughout life. Elevated Lp(a) concentrations are now recognized as an independent and causal risk factor for atherosclerotic cardiovascular disease and calcific aortic valve stenosis [2, 4, 10].

Recent evidence suggests that Lp(a) may contribute not only to atherosclerosis but also to vascular remodeling relevant to arterial hypertension. Experimental and clinical studies indicate that Lp(a) promotes endothelial dysfunction, oxidative stress, vascular inflammation, arterial stiffness, and vascular calcification by transporting oxidized phospholipids and activating pro-inflammatory signaling pathways. These mechanisms may exacerbate hypertensive vascular damage and contribute to a more severe cardiovascular phenotype in hypertensive patients [3, 15, 24].

Several studies have demonstrated associations between elevated Lp(a) levels and increased pulse pressure, abnormal nocturnal blood pressure profiles, arterial stiffness, and target-organ damage in hypertensive individuals. In particular, recent ambulatory blood pressure monitoring studies have shown that hypertensive patients with elevated Lp(a) have higher nocturnal systolic blood pressure and impaired nocturnal blood pressure dipping [5].

The clinical significance of Lp(a) has further increased with the development of novel targeted therapies, including antisense oligonucleotides and small interfering RNA (siRNA)-based agents that can substantially reduce circulating Lp(a) concentrations. These therapeutic advances have stimulated interest in identifying patient populations in whom elevated Lp(a) may contribute to residual cardiovascular risk, including individuals with arterial hypertension [6].

Currently, available data do not support Lp(a) as a direct cause of arterial hypertension. However, the prevailing scientific view increasingly regards Lp(a) as a genetically determined modifier of vascular remodeling and cardiovascular risk in hypertensive patients. Understanding these mechanisms is especially important given the rapid development of novel Lp(a)-lowering therapies, including antisense oligonucleotides and small interfering RNA agents, which may substantially reduce cardiovascular risk in selected high-risk populations [6, 18, 21].

Elevated Lp(a) should be viewed not merely as an abnormal lipid parameter but as an important contributor to vascular remodeling and persistent cardiovascular risk. Additional research is needed to clarify the relationships among Lp(a), endothelial impairment, arterial stiffening, and hypertension-related target-organ damage, especially in individuals with genetically elevated Lp(a) levels [7, 12, 25].

AIM

To evaluate the current scientific evidence on the role of lipoprotein(a) in vascular remodeling and its effects on endothelial function, arterial stiffness, and cardiovascular risk in patients with arterial hypertension.

MATERIALS AND METHODS

A literature search was conducted using open-access databases PubMed/MEDLINE and Google Scholar. The review included original research articles, systematic reviews, meta-analyses, and international clinical guidelines published up to May 1, 2026, using the following keywords: Lipoprotein(a); arterial hypertension; vascular remodeling; cardiovascular genetics; arterial stiffness; cardiovascular risk. The search was limited to publications in English and Ukrainian. Low-quality studies were excluded based on generally accepted criteria, including small sample sizes, isolated clinical cases, and a lack of statistically validated results compared with control or placebo groups. All authors independently reviewed all selected publications, and data were included after consensus was reached.

RESULTS

Lp(a) has become one of the most intensively studied genetically determined cardiovascular risk factors in modern cardiovascular medicine. Over the past decade, numerous experimental, epidemiological, and clinical studies have demonstrated that elevated Lp(a) concentrations are associated not only with accelerated atherosclerosis but also with endothelial dysfunction, vascular inflammation, arterial stiffness, vascular calcification, residual cardiovascular risk, and hypertensive target-organ damage. Increasing evidence indicates that Lp(a) may significantly contribute to vascular remodeling in patients with arterial hypertension, particularly in those with genetically elevated Lp(a) concentrations and persistent residual cardiovascular risk despite optimal therapy [1, 3]. Approximately 14-20% of the population has an Lp(a) level greater than 50 mg/dL. Lp(a) measurement is recommended at least once during an individual's lifetime as part of the initial cardiovascular risk assessment. Elevated Lp(a) levels ≥ 50 mg/dL (≥ 100 nmol/L) are considered an important cardiovascular risk modifier and may support initiation

or intensification of statin therapy in individuals with intermediate cardiovascular risk [6].

Lp(a) is a complex lipoprotein particle composed of a low-density lipoprotein (LDL)-like core covalently linked to apolipoprotein(a), a highly polymorphic glycoprotein structurally homologous to plasminogen. The LPA gene predominantly regulates plasma Lp(a) concentrations and remains relatively stable throughout life. Unlike traditional lipid fractions such as LDL cholesterol or triglycerides, Lp(a) levels are minimally influenced by diet, physical activity, or lifestyle changes, underscoring the major role of inherited cardiovascular risk [6]. Contemporary studies suggest that more than 90% of interindividual variability in Lp(a) concentrations is genetically determined, highlighting the importance of genetic background in the development of cardiovascular complications [2, 8, 29].

Several large epidemiological and Mendelian randomization studies have confirmed that elevated Lp(a) concentrations are independently associated with coronary artery disease, ischemic stroke, peripheral arterial disease, and calcific aortic valve stenosis [6, 7, 9, 14, 22]. Importantly, recent investigations increasingly suggest that elevated Lp(a) may also influence vascular remodeling and cardiovascular phenotype in hypertensive individuals [8]. This concept is particularly relevant because many hypertensive patients continue to demonstrate substantial cardiovascular risk despite adequate blood pressure control [8, 11, 16, 21].

One of the principal mechanisms underlying the pathogenic effects of Lp(a) is its ability to transport oxidized phospholipids (OxPL), which are recognized as potent mediators of inflammation and vascular injury. OxPL promote endothelial activation, oxidative stress, macrophage recruitment, inflammatory cytokine production, and vascular fibrosis [1, 5, 29]. OxPL-associated inflammatory signaling may reduce nitric oxide bioavailability, impair endothelium-dependent vasodilation, and contribute to endothelial dysfunction, which is considered one of the earliest stages of vascular remodeling in arterial hypertension [13, 17, 26].

Endothelial dysfunction is a key pathophysiological mechanism linking elevated Lp(a) to vascular remodeling and hypertensive vascular injury. Experimental studies have shown that OxPL-associated pathways stimulate adhesion molecule expression, activate inflammatory signaling cascades, and promote leukocyte recruitment to the vascular wall [3, 11, 20]. These processes drive chronic, low-grade vascular inflammation, progressive structural changes in the vasculature, and increased arterial stiffness [5, 23].

Recent investigations emphasized that elevated Lp(a) should be viewed not merely as an abnormal lipid parameter but as a multifunctional mediator of vascular injury. Lp(a) may amplify hypertensive vascular damage through pro-inflammatory, pro-fibrotic, and pro-calcific mechanisms, thereby contributing to residual cardiovascular risk despite optimal blood pressure control. The authors also emphasized the association of elevated Lp(a) with endothelial dysfunction, arterial stiffening, and progression of hypertensive target-organ damage [3, 5].

Plasma Lp(a) concentration is among the most strongly genetically determined cardiovascular risk factors identified to date. More than 90% of interindividual variability in circulating Lp(a) levels is controlled by the LPA gene on chromosome 6q26-27 [1, 3]. In contrast to traditional lipid fractions, Lp(a) concentrations are minimally influenced by dietary habits, physical activity, or environmental factors, underscoring the dominant role of inherited mechanisms in determining cardiovascular risk associated with elevated Lp(a) [2, 9, 19].

The LPA gene encodes apolipoprotein(a), a highly polymorphic glycoprotein structurally homologous to plasminogen [18]. The number of kringle IV type 2 (KIV-2) repeats in the LPA gene is one of the most important genetic determinants of Lp(a) concentration. Individuals with fewer KIV-2 repeats typically have significantly higher circulating Lp(a) levels, whereas larger apo(a) isoforms are generally associated with lower plasma concentrations [2]. This inverse relationship between apo(a) isoform size and Lp(a) concentration is a distinctive feature of Lp(a) metabolism [27].

Several Mendelian randomization studies have confirmed a causal association between genetically elevated Lp(a) and increased cardiovascular risk [3, 13, 15, 17]. Because genetic variants associated with elevated Lp(a) are randomly inherited at conception and remain stable throughout life, these studies provide strong evidence for a direct pathogenic role of Lp(a) in the development of cardiovascular disease [2, 3, 13, 17]. Elevated genetically determined Lp(a) concentrations were associated with increased incidence of coronary artery disease, myocardial infarction, ischemic stroke, peripheral arterial disease, and calcific aortic valve stenosis [1, 3, 7, 15, 22].

Recent investigations also suggest that genetic determinants of elevated Lp(a) may contribute to vascular remodeling and arterial stiffness in patients with arterial hypertension [1, 2, 4, 12, 16, 28]. Although elevated Lp(a) is not considered a direct cause of hypertension, inherited elevation of Lp(a) may amplify endothelial dysfunction, oxidative stress, inflammatory activation, and vascular calcification, thereby contributing to a more aggressive hypertensive vascular phenotype [1, 5, 6, 14, 17, 25].

The pathogenicity of genetically elevated Lp(a) is largely mediated by OxPL, which are preferentially transported by apo(a)-containing particles. OxPL trigger inflammatory signaling, endothelial

activation, macrophage recruitment, and vascular fibrosis [1, 5, 6, 17]. Consequently, individuals with inherited high Lp(a) concentrations may experience chronic vascular inflammation and accelerated vascular remodeling throughout life [1, 3, 14, 21].

Several genome-wide association studies (GWAS) have identified multiple LPA variants associated with elevated cardiovascular risk [2, 3, 13, 15, 19, 29]. Some of these variants are associated not only with higher Lp(a) levels but also with a greater burden of vascular calcification and aortic valve disease [1, 3, 8, 15]. These findings further support the role of genetically determined Lp(a) as an active mediator of vascular injury rather than a passive cardiovascular biomarker [1, 3, 6, 21].

An important aspect of Lp(a) genetics is ethnic variation in circulating concentrations. Population studies have shown substantial differences in Lp(a) levels among ethnic groups, with individuals of African ancestry generally exhibiting higher Lp(a) concentrations than those in Caucasian or Asian populations [3, 15, 21, 30]. These differences are primarily genetically determined and may partially contribute to variability in cardiovascular risk profiles across populations [2, 3, 13, 21].

In recent years, attention has increasingly focused on the association between genetically elevated Lp(a) and residual cardiovascular risk despite optimal lipid-lowering therapy. Several studies have shown that elevated Lp(a) may account for persistent cardiovascular risk even in patients who achieve target LDL cholesterol levels on intensive statin therapy. These findings underscore the importance of genetic assessment of cardiovascular risk beyond conventional lipid parameters [3, 6, 15, 21, 24].

Advances in RNA-targeted therapies that modulate LPA gene expression have substantially increased interest in the genetics of Lp(a). Antisense oligonucleotides and small interfering RNA (siRNA)-based therapies, including pelacarsen and olpasiran, directly suppress hepatic apo(a) synthesis and significantly reduce circulating Lp(a) levels. Clinical studies have shown 80-90% reductions in certain patient populations, offering new perspectives on personalized cardiovascular prevention based on genetic risk modification [6, 9, 21].

Contemporary evidence supports the concept that genetically elevated Lp(a) is not only an inherited lipid abnormality but also a complex mediator of vascular remodeling, endothelial dysfunction, arterial stiffness, vascular calcification, and residual cardiovascular risk [1, 3, 6, 12, 14, 15, 21]. Further studies are needed to clarify interactions among LPA genetic variants, vascular remodeling pathways, and hypertensive target-organ damage, particularly in patients with arterial hypertension and high inherited cardiovascular risk [2, 11, 17, 19, 29].

Increasing evidence supports an association between elevated Lp(a) concentrations and abnormal ambulatory blood pressure monitoring parameters. Vazquez-Agra et al. (2024) demonstrated that hypertensive individuals with elevated Lp(a) levels had significantly higher nocturnal systolic blood pressure, increased pulse pressure indices, and impaired nocturnal blood pressure reduction [5]. These findings suggest a potential interaction among elevated Lp(a), arterial stiffness, endothelial dysfunction, and disturbed circadian blood pressure regulation.

Pulse pressure and nocturnal blood pressure profile are increasingly recognized as important markers of vascular aging and arterial stiffness. Elevated pulse pressure reflects reduced elasticity of large arteries and is strongly associated with adverse cardiovascular outcomes. Several studies have proposed that chronic inflammatory activation mediated by OxPL pathways may accelerate vascular aging and structural remodeling of large arteries. Consequently, elevated Lp(a) may contribute to isolated systolic hypertension and increased left ventricular afterload in susceptible individuals [1, 5, 6, 16-18, 20, 28].

Arterial stiffness has become a major focus of investigation in the context of elevated Lp(a). It contributes not only to elevated systolic blood pressure and increased pulse pressure but also to impaired coronary perfusion, left ventricular hypertrophy, and progression of hypertensive target-organ damage. Contemporary studies indicate that chronic inflammatory and oxidative stress pathways associated with elevated Lp(a) may promote collagen deposition, extracellular matrix remodeling, and vascular fibrosis [1, 5, 6, 16-18, 20, 28].

Another important mechanism linking elevated Lp(a) to vascular remodeling is vascular calcification. OxPL carried by Lp(a) stimulates osteogenic signaling pathways in vascular smooth muscle cells, promoting vascular fibrosis and calcification of the vascular wall. These processes may partially explain the association between elevated Lp(a), arterial stiffness, calcific vascular disease, and increased cardiovascular risk in patients with arterial hypertension [1, 5, 6, 8, 14, 17, 25].

The role of Lp(a) in vascular calcification is particularly important because calcific changes in the vascular wall significantly contribute to increased arterial stiffness and reduced vascular compliance. Several studies suggest that Lp(a)-associated OxPL stimulates the expression of osteogenic transcription factors and promotes the differentiation of vascular smooth muscle cells toward an osteoblast-like phenotype. These mechanisms may accelerate vascular aging and the progression of calcific vascular disease in hypertensive individuals [1, 5, 6, 8, 14, 17, 25, 28].

Recent studies also suggest that elevated Lp(a) contributes to hypertensive target-organ damage independently of traditional lipid abnormalities. In high-risk hypertensive populations, increased Lp(a)

concentrations were associated with arterial wall thickening, endothelial dysfunction, microvascular impairment, and higher rates of cardiovascular events. These findings support the concept that Lp(a) acts as a modifier of the hypertensive vascular phenotype rather than a direct cause of arterial hypertension [1, 3, 5, 11, 14, 17, 21, 28].

Microvascular dysfunction may be another important mechanism linking elevated Lp(a) to hypertensive cardiovascular complications. Chronic endothelial activation and inflammatory signaling associated with elevated Lp(a) may impair microvascular perfusion, leading to tissue ischemia, fibrosis, and progression of organ damage. These changes may affect multiple organs, including the heart, kidneys, and brain [1, 5, 6, 11, 14, 17, 21].

The link between elevated Lp(a) and coronary microvascular dysfunction has drawn increasing scientific attention. Several studies suggest that OxPL-mediated endothelial injury and inflammation may impair coronary flow reserve and contribute to myocardial ischemia, independent of obstructive coronary artery disease. In hypertensive individuals, these mechanisms may exacerbate myocardial remodeling and increase the risk of heart failure [1, 5, 6, 14, 17, 18, 20, 21].

In addition to vascular inflammation and endothelial dysfunction, Lp(a) exhibits prothrombotic properties due to the structural homology between apolipoprotein(a) and plasminogen. This similarity may impair fibrinolysis and contribute to thrombogenic potential within the vascular wall. Consequently, elevated Lp(a) concentrations may simultaneously promote atherosclerosis, vascular inflammation, endothelial dysfunction, and thrombotic complications. The prothrombotic properties of Lp(a) may be particularly relevant in hypertensive patients because hypertension itself contributes to endothelial injury and activation of coagulation pathways. The coexistence of elevated Lp(a) and arterial hypertension may therefore create a highly proatherogenic and prothrombotic vascular environment, increasing cardiovascular risk [1, 3, 5, 6, 14, 18, 21, 22].

Recent investigations have underscored the importance of measuring Lp(a) for cardiovascular risk stratification. The European Atherosclerosis Society consensus statement recommended at least one lifetime assessment of Lp(a), particularly in individuals with premature cardiovascular disease, a family history of cardiovascular events, calcific aortic valve disease, or unexplained residual cardiovascular risk. Elevated Lp(a) may identify hypertensive patients at particularly high cardiovascular risk, despite otherwise acceptable lipid profiles. Several studies have shown that elevated Lp(a) levels may identify individuals with increased residual cardiovascular risk, even during intensive statin therapy. This concept is clinically important because many hypertensive patients continue to experience cardiovascular events despite achieving target LDL cholesterol levels. Elevated Lp(a) may therefore be an important explanation for persistent residual cardiovascular risk [1, 3, 6, 15, 21, 24, 26, 27].

The clinical significance of elevated Lp(a) is particularly relevant for patients with resistant hypertension, premature atherosclerosis, or calcific aortic valve disease. In these populations, elevated Lp(a) may indicate heightened vascular susceptibility to inflammation, fibrosis, calcification, and endothelial dysfunction. Consequently, assessing Lp(a) may improve cardiovascular risk stratification and support more personalized prevention strategies [1, 3, 5, 8, 14, 21, 25, 27].

Genetically elevated Lp(a) may also contribute to interindividual variability in hypertensive cardiovascular phenotypes. Several Mendelian randomization studies suggest that inherited elevation of Lp(a) is associated with increased cardiovascular risk, independent of conventional lipid parameters. These observations underscore the importance of genetic mechanisms in vascular remodeling and cardiovascular complications. The emergence of RNA-targeted therapies has substantially increased clinical interest in Lp(a). Antisense oligonucleotides and small interfering RNA (siRNA)-based therapies, including pelacarsen, olpasiran, zerlasiran, and other emerging agents, have demonstrated marked reductions in circulating Lp(a) concentrations in recent clinical trials [2, 3, 6, 9, 13, 15, 19, 21, 24, 30].

Pelacarsen, an antisense oligonucleotide that targets apolipoprotein(a) synthesis, demonstrated significant reductions in circulating Lp(a) levels in phase II clinical studies. Similarly, olpasiran and other siRNA-based therapies achieved 80-90% reductions in some clinical trials. These findings suggest that highly effective, targeted Lp(a)-lowering therapy may soon be available for cardiovascular prevention. The development of RNA-targeted therapies has transformed the view of Lp(a) from a passive cardiovascular biomarker to a potentially modifiable therapeutic target. Contemporary studies suggest that targeted reduction of Lp(a) may influence vascular inflammation, endothelial dysfunction, arterial stiffness, and the progression of vascular remodeling. These approaches may be particularly important for hypertensive patients with genetically elevated Lp(a) levels and persistent residual cardiovascular risk [6, 9, 21, 24, 30].

Recent investigations also emphasized the link between elevated Lp(a) and calcific aortic valve disease. Elevated Lp(a) concentrations were strongly associated with the progression of calcific aortic stenosis and with increased need for valve replacement. Because arterial hypertension itself contributes to vascular and valvular remodeling, the coexistence of elevated Lp(a) and

hypertension may accelerate the progression of calcific cardiovascular disease [1, 3, 8, 14, 21, 25].

The association between elevated Lp(a) and aortic valve calcification may further support the concept that Lp(a)-associated OxPL promote osteogenic and inflammatory pathways in cardiovascular tissues. These mechanisms may also contribute to arterial calcification and increased vascular stiffness observed in hypertensive populations. Several contemporary reviews emphasize that elevated Lp(a) contributes to residual inflammatory risk even among patients receiving intensive lipid-lowering therapy. Persistent inflammatory activation may be an important mechanism linking elevated Lp(a) levels to vascular remodeling and the progression of hypertensive target-organ damage [1, 3, 5, 6, 8, 14, 21, 25].

Another important aspect concerns the relationship between elevated Lp(a) and chronic kidney disease. Hypertensive patients with chronic kidney disease often have increased cardiovascular risk and accelerated vascular remodeling. Some studies suggest that elevated Lp(a) concentrations may further exacerbate endothelial dysfunction and vascular inflammation in these patients [1, 5, 11, 14, 17, 21, 26].

The interplay among elevated Lp(a), oxidative stress, and inflammatory signaling may also influence myocardial remodeling. Several studies have proposed that chronic vascular inflammation and endothelial dysfunction may contribute to myocardial fibrosis, impaired ventricular relaxation, and progression of heart failure. In hypertensive individuals, these mechanisms may amplify pressure overload-related myocardial remodeling [1, 5, 6, 14, 17, 18, 20, 21].

Recent evidence increasingly supports the concept that elevated Lp(a) contributes to a more aggressive cardiovascular phenotype in hypertensive patients. Elevated Lp(a) concentrations may be associated with increased arterial stiffness, vascular calcification, endothelial dysfunction, microvascular impairment, inflammation, fibrosis, and thrombogenicity. Despite substantial scientific progress, several aspects of the relationship between elevated Lp(a) and arterial hypertension remain incompletely understood. Available evidence does not support Lp(a) as a direct cause of hypertension. However, elevated Lp(a) should be considered an important modifier of vascular remodeling and residual cardiovascular risk in hypertensive individuals [1, 3, 5, 6, 11, 14, 17, 18, 21, 25, 28].

Further prospective investigations are needed to clarify the interactions among genetically elevated Lp(a), endothelial dysfunction, arterial stiffness, vascular calcification, inflammatory activation, and hypertensive target-organ damage. Additional studies are needed to determine whether targeted Lp(a)-lowering therapies may improve cardiovascular outcomes, particularly in hypertensive populations [1, 3, 5, 6, 9, 14, 21, 24, 30].

Contemporary evidence increasingly supports the concept that elevated Lp(a) contributes to vascular remodeling and an adverse cardiovascular phenotype in arterial hypertension through complex interactions involving oxidative stress, inflammation, endothelial dysfunction, fibrosis, calcification, and thrombogenicity. Improved understanding of these mechanisms may contribute to more accurate cardiovascular risk stratification and the development of personalized therapeutic approaches for hypertensive patients with genetically elevated Lp(a).

DISCUSSION

Elevated Lp(a) is increasingly recognized as one of the most important genetically determined cardiovascular risk factors, contributing to vascular remodeling and residual cardiovascular risk in patients with arterial hypertension [1, 3, 6, 12, 13, 15, 21, 24]. Although Lp(a) is traditionally associated with accelerated atherosclerosis and calcific cardiovascular disease, recent evidence suggests that its biological effects extend far beyond the classical accumulation of lipids in the vascular wall. Elevated Lp(a) concentrations are associated with endothelial dysfunction, oxidative stress, inflammatory activation, arterial stiffness, fibrosis, vascular calcification, myocardial remodeling, and hypertensive target-organ damage [1, 5, 6, 14, 17].

One of the most important aspects of Lp(a)-associated cardiovascular risk is its strong genetic basis. The LPA gene largely controls plasma Lp(a) concentrations and remains relatively stable throughout life [2, 3, 15]. Contemporary genetic studies have demonstrated that variability within the LPA gene extends beyond the kringle IV repeat polymorphism and significantly influences circulating Lp(a) levels, cardiovascular phenotype, and susceptibility to vascular remodeling [2, 19, 29]. Mendelian randomization studies have confirmed that inherited elevation of Lp(a) is causally associated with increased cardiovascular risk, independent of traditional lipid parameters [3, 15, 21].

Several epidemiological and observational studies have demonstrated associations between elevated Lp(a) concentrations and arterial hypertension [4, 10, 11, 12]. Data from the Multi-Ethnic Study of Atherosclerosis (MESA) revealed a significant association between elevated Lp(a) and hypertension in primary cardiovascular prevention populations [4]. Similarly, increased Lp(a) levels were observed in patients with newly diagnosed hypertension, and higher concentrations were associated with greater severity of hypertension [10]. Recent evidence has also demonstrated associations between elevated Lp(a) and hypertension-mediated organ damage,

suggesting that Lp(a) may contribute to an adverse hypertensive phenotype [11, 31].

The pathogenicity of Lp(a) is closely linked to oxidized phospholipids (OxPL), which are carried by apo(a)-containing particles and trigger inflammatory activation, oxidative stress, endothelial dysfunction, macrophage recruitment, fibrosis, and vascular calcification [1, 6, 17]. Endothelial dysfunction is a key mechanism linking elevated Lp(a) to vascular remodeling and the progression of arterial hypertension [17]. Chronic inflammatory activation driven by OxPL reduces nitric oxide bioavailability, increases vascular stiffness, and promotes progressive vascular injury [1, 5, 17].

Several studies have emphasized the relationship between elevated Lp(a) and arterial stiffness, a major determinant of adverse cardiovascular prognosis in hypertensive individuals [5, 16, 28]. Elevated plasma Lp(a) concentrations were independently associated with arterial stiffening in patients with hypertension [28]. Increased arterial stiffness contributes to elevated systolic blood pressure, increased pulse pressure, vascular aging, impaired coronary perfusion, and left ventricular overload [16, 28]. Recent investigations have also demonstrated associations between elevated Lp(a), abnormal nocturnal blood pressure patterns, and impaired nocturnal blood pressure reduction [5].

Another important mechanism linking elevated Lp(a) to cardiovascular injury involves vascular and valvular calcification [1, 6, 14, 31]. OxPL stimulates osteogenic signaling pathways in vascular smooth muscle cells and promotes calcification of the vascular wall [1, 6]. These mechanisms may partially explain the association between elevated Lp(a), vascular stiffness, calcific vascular disease, and increased cardiovascular risk in hypertensive patients. Furthermore, elevated Lp(a) concentrations were associated with dilatation of multiple aortic segments in individuals with arterial hypertension, suggesting an additional role in vascular wall remodeling and aortopathy [25].

Recent investigations also demonstrated important associations between elevated Lp(a) and cardiac remodeling [8, 18, 20, 23]. Elevated Lp(a) concentrations were associated with left ventricular systolic dysfunction in hypertensive patients without coronary artery disease [18]. Large cross-sectional studies also reported significant associations between elevated Lp(a) and left ventricular hypertrophy, as assessed by electrocardiography [20, 23]. Moreover, recent data from the Multi-Ethnic Study of Atherosclerosis demonstrated associations between elevated Lp(a) and cardiac remodeling across racial and ethnic groups [8]. These findings suggest that Lp(a) may contribute not only to vascular remodeling but also to structural alterations in the myocardium and an adverse cardiac phenotype.

The relationship between elevated Lp(a) and panvascular disease has also attracted increasing scientific attention [7, 14]. Elevated Lp(a) concentrations have been associated with peripheral arterial disease, cerebrovascular disease, coronary artery disease, and generalized vascular remodeling [7, 14]. Large population studies have also confirmed associations between elevated Lp(a) and increased incidence of myocardial infarction and stroke [22]. These observations support the concept that Lp(a) should be considered a systemic cardiovascular risk factor affecting multiple vascular territories.

Residual cardiovascular risk remains a major challenge in modern cardiovascular medicine. Several studies have demonstrated that elevated Lp(a) may explain persistent cardiovascular risk despite intensive lipid-lowering therapy and attainment of target LDL cholesterol levels [3, 6, 21]. This observation is particularly relevant in hypertensive individuals who continue to show increased cardiovascular morbidity despite adequate blood pressure control. Elevated Lp(a) may therefore be an important marker of residual inflammatory and vascular risk [1, 6, 21].

An important additional aspect is the frequent underdiagnosis of elevated Lp(a) in routine clinical practice [26, 27]. Several studies have shown that elevated Lp(a) levels often go undetected even in high-risk cardiovascular populations [26]. Clinical and epidemiological analyses have also emphasized the importance of broader implementation of Lp(a) screening for cardiovascular risk stratification [27]. Contemporary recommendations support at least one lifetime measurement of Lp(a), particularly in patients with premature cardiovascular disease, a family history of cardiovascular events, calcific vascular pathology, resistant hypertension, or unexplained residual cardiovascular risk [3].

Recent advances in molecular and RNA-targeted therapies have substantially increased interest in Lp(a) as a therapeutic target [6, 9]. Small interfering RNA (siRNA) therapies and antisense oligonucleotides have demonstrated marked reductions in circulating Lp(a) concentrations and have opened new perspectives for personalized cardiovascular prevention [6, 9]. These therapies may be particularly important for patients with genetically elevated Lp(a), arterial stiffness, vascular calcification, and progressive hypertensive organ damage.

Despite substantial progress in understanding the role of Lp(a) in vascular remodeling and cardiovascular disease, several aspects remain incompletely understood [12, 13, 24]. Current evidence does not support elevated Lp(a) as a direct cause of arterial hypertension. However, elevated Lp(a) should be considered an important modifier of vascular remodeling, endothelial dysfunction, arterial stiffness, calcification,

thrombogenicity, and residual cardiovascular risk in hypertensive individuals [1, 3, 6, 12, 21].

Contemporary evidence increasingly supports the concept that elevated Lp(a) contributes to adverse vascular and cardiac remodeling through complex interactions involving oxidative stress, inflammation, endothelial dysfunction, fibrosis, calcification, thrombogenicity, and genetic susceptibility [1, 11, 30]. Improved understanding of these mechanisms may enable more accurate cardiovascular risk stratification and the development of personalized therapeutic approaches for patients with arterial hypertension and genetically elevated Lp(a).

CONCLUSIONS

This review demonstrates that elevated Lp(a) plays an important role in vascular remodeling and residual cardiovascular risk among patients with arterial hypertension. Increased Lp(a) concentrations are associated with endothelial dysfunction, arterial stiffness, vascular inflammation, vascular calcification, and hypertension-mediated organ damage. Plasma Lp(a) levels are predominantly regulated by the LPA gene, underscoring the importance of genetic predisposition in cardiovascular risk and vascular remodeling. Contemporary evidence suggests that assessing Lp(a) may improve cardiovascular risk stratification in hypertensive individuals, while emerging RNA-targeted therapies offer promising approaches to personalized cardiovascular prevention for patients with genetically elevated Lp(a).

Perspectives for further research. Future research should clarify the role of elevated Lp(a) in vascular remodeling, endothelial dysfunction, arterial stiffness, and hypertension-mediated organ damage. Additional studies should examine the relationships among LPA genetic variants, circulating Lp(a) concentrations, and individual cardiovascular phenotypes in patients with arterial hypertension. Future research is also needed to evaluate the long-term clinical effects of RNA-targeted Lp(a)-lowering therapies on vascular remodeling, arterial stiffness, and cardiovascular outcomes in hypertensive populations.

COMPLIANCE WITH ETHICAL REQUIREMENTS

The article drew on publicly available publications from open databases such as PubMed/MEDLINE, including original data, systematic reviews, and meta-analyses, as well as published guidelines and recommendations from international medical societies. All sources are cited in the text. No proprietary clinical research data requiring ethical committee approval were used in preparing this article. The work was conducted in accordance with the uniform requirements developed by the International Committee of Medical Journal Editors (ICMJE). All sources are properly cited, and plagiarism or data fabrication is excluded. This work did not employ artificial intelligence for text generation, data analysis, or image creation. All content was prepared by the authors, who bear full responsibility for its accuracy, originality, and adherence to ethical standards.

Declaration of the use of generative AI in manuscript preparation. The authors of the manuscript certify that, in the process of conducting the research and preparing this manuscript, no artificial intelligence was used for text generation, data analysis, or final editing. All stages of the work were performed exclusively by the authors.

FUNDING AND CONFLICT OF INTEREST

The authors' own resources funded the study. The authors declare no conflict of interest or financial interests that could be interpreted as influencing the results or interpretation of the manuscript. All authors have given their consent for the publication of the manuscript.

AUTHOR CONTRIBUTIONS

Solomenchuk T. M.[A, D, E, F]

Zhurayev R. K.[A, B, D, E, F]

Protsko V. V.[A, B, F]

Hel K. S.[B, C, D]

Nykoliak V. R.[B, C, D]

All authors read and approved the submitted version of the article.

REFERENCES

1. Brosolo, G., Da Porto, A., Marcante, S., Picci, A., Capilupi, F., Capilupi, P., Bulfone, L., Vacca, A., Bertin, N., Vivarelli, C., Comand, J., Catena, C., & Sechi, L. A. (2023). Lipoprotein(a): Just an innocent bystander in arterial hypertension? International Journal of Molecular Sciences, 24(17), 13363. https://doi.org/10.3390/ijms241713363
2. Coassin, S., & Kronenberg, F. (2022). Lipoprotein(a) beyond the kringle IV repeat polymorphism: The complexity of genetic variation in the LPA gene.

Atherosclerosis, 349, 17-35. https://doi.org/10.1016/j.atherosclerosis.2022.04.003
3. Kronenberg, F., Mora, S., Stroes, E. S. G., Ference, B. A., Arsenault, B. J., Berglund, L., Dweck, M. R., Koschinsky, M., Lambert, G., Mach, F., McNeal, C. J., Moriarty, P. M., Natarajan, P., Nordestgaard, B. G., Parhofer, K. G., Virani, S. S., von Eckardstein, A., Watts, G. F., Stock, J. K., Ray, K. K., Tokgözoğlu, L. S., & Catapano, A. L. (2022). Lipoprotein(a) in atherosclerotic cardiovascular disease and aortic stenosis: A European Atherosclerosis Society consensus statement. European Heart Journal, 43(39), 3925-3946. https://doi.org/10.1093/eurheartj/ehac361
4. Rikhi, R., Bhatia, H. S., Schaich, C. L., Ashburn, N., Tsai, M. Y., Michos, E. D., Chevli, P. A., Herrington, D. M., Tsimikas, S., & Shapiro, M. D. (2023). Association of Lp(a) (Lipoprotein[a]) and hypertension in primary prevention of cardiovascular disease: The MESA study. Hypertension, 80(2), 352-360. https://doi.org/10.1161/HYPERTENSIONAHA.122.20189
5. Vazquez-Agra, N., Cruces-Sande, A., Barbosa-Gouveia, S., Lopez-Paz, J. E., Camafort, M., Casariego-Vales, E., Pose-Reino, A., & Hermida-Ameijeiras, A. (2024). Assessing the relationship between lipoprotein(a) levels and blood pressure among hypertensive patients beyond conventional measures: An observational study. Scientific Reports, 14(1), 14433. https://doi.org/10.1038/s41598-024-65231-w
6. Kaur, G., Abdelrahman, K., Berman, A. N., Biery, D. W., Shiyovich, A., Huck, D., Garshick, M., Blankstein, R., & Weber, B. (2024). Lipoprotein(a): Emerging insights and therapeutics. American Journal of Preventive Cardiology, 18, 100641. https://doi.org/10.1016/j.ajpc.2024.100641
7. Mwipatayi, B. P., Dodd, J. E., Ahmad Bazlee, A. H., Stratford, G., Lee, B., Mori, T., Watts, G. F., Armari, E., Wong, J., & Schlaich, M. P. (2025). Lipoprotein(a) and its role in peripheral arterial disease: A narrative review. Vascular Health and Risk Management, 21, 965-981. https://doi.org/10.2147/VHRM.S555127
8. Abdollahi, A., Ostovaneh, A., Chehab, O., Hoballah, M., Matuck, B. R. S., Wu, C. O., et al. (2026). Association between lipoprotein(a) and cardiac remodeling across race and ethnicity in the Multi-Ethnic Study of Atherosclerosis. Circulation: Cardiovascular Quality and Outcomes, 19(3), e013261. https://doi.org/10.1161/CIRCOUTCOMES.125.013261
9. Sarzani, R., Spannella, F., Di Pentima, C., Giulietti, F., Landolfo, M., & Allevi, M. (2023). Molecular therapies in cardiovascular diseases: Small interfering RNA in atherosclerosis, heart failure, and hypertension. International Journal of Molecular Sciences, 25(1), 328. https://doi.org/10.3390/ijms25010328
10. Mahto, S. K., Sheoran, A., Gadpayle, A. K., Gupta, K., Gupta, P. K., Chitkara, A., & Agarwal, N. (2022). Evaluation of lipoprotein(a) [Lp(a)] and lipid abnormalities in patients with newly detected hypertension and its association with severity of hypertension. Journal of Family Medicine and Primary Care, 11(4), 1508-1513. https://doi.org/10.4103/jfmpc.jfmpc_989_21
11. Nardoianni, G., Tocci, G., Pala, B., Russo, M., Dutti, G. M., Fogacci, F., Cicero, A. F. G., Volpe, M., & Barbato, E. (2026). Lipoprotein(a) in essential hypertension: Associations with blood pressure and hypertension-mediated organ damage. High Blood Pressure & Cardiovascular Prevention. Advance online publication. https://doi.org/10.1007/s40292-026-00783-8
12. Ward, N. C., Nolde, J. M., Chan, J., Carnagarin, R., Watts, G. F., & Schlaich, M. P. (2021). Lipoprotein(a) and hypertension. Current Hypertension Reports, 23(12), 44. https://doi.org/10.1007/s11906-021-01161-6
13. Katsiki, N., Al-Rasadi, K., & Mikhailidis, D. P. (2017). Lipoprotein(a) and cardiovascular risk: The show must go on. Current Medicinal Chemistry, 24(10), 989-1006. https://doi.org/10.2174/0929867324666170112111948
14. Xu, R., Wang, Z., Dong, J., Yu, M., & Zhou, Y. (2025). Lipoprotein(a) and panvascular disease. Lipids in Health and Disease, 24(1), 186. https://doi.org/10.1186/s12944-025-02600-y
15. Kamstrup, P. R. (2021). Lipoprotein(a) and cardiovascular disease. Clinical Chemistry, 67(1), 154-166. https://doi.org/10.1093/clinchem/hvaa247
16. De la Maza-Bustindui, N. S., León-Álvarez, M., Ponce-Acosta, C., Zarco-Morales, K. P., Fermín-Martínez, C. A., Antonio-Villa, N. E., & Bello-Chavolla, O. Y. (2025). Impact of cardiometabolic risk factors and their management on the reversion and progression of arterial stiffness. NPJ Cardiovascular Health, 2(1), 36. https://doi.org/10.1038/s44325-025-00074-6
17. Ramírez-Melo, L. M., Estrada-Luna, D., Rubio-Ruiz, M. E., Castañeda-Ovando, A., Fernández-Martínez, E., Jiménez-Osorio, A. S., Pérez-Méndez, Ó., & Carreón-Torres, E. (2025). Relevance of lipoprotein composition in endothelial dysfunction and the development of hypertension. International Journal of Molecular Sciences, 26(3), 1125. https://doi.org/10.3390/ijms26031125
18. Wang, Y., Ma, H., Yang, J., Chen, Q., Lu, L., & Zhang, R. (2017). Lipoprotein(a) is associated with left ventricular systolic dysfunction in a Chinese population of patients with hypertension and without coronary artery disease. Archives of Medical

Science, 13(5), 1078-1085. https://doi.org/10.5114/aoms.2016.59875
19. Fogacci, F., Di Micoli, V., Avagimyan, A., Giovannini, M., Imbalzano, E., & Cicero, A. F. G. (2023). Assessment of apolipoprotein(a) isoform size using phenotypic and genotypic methods. International Journal of Molecular Sciences, 24(18), 13886. https://doi.org/10.3390/ijms241813886
20. Yan, X., Gong, J., Wang, Z., Wang, F., & Qi, C. (2023). Association of lipoprotein(a) with left ventricular hypertrophy assessed by electrocardiogram in adults: A large cross-sectional study. Frontiers in Endocrinology, 14, 1260050. https://doi.org/10.3389/fendo.2023.1260050
21. Duarte Lau, F., & Giugliano, R. P. (2022). Lipoprotein(a) and its significance in cardiovascular disease: A review. JAMA Cardiology, 7(7), 760-769. https://doi.org/10.1001/jamacardio.2022.0987
22. Brandt, E. J., Mani, A., Spatz, E. S., Desai, N. R., & Nasir, K. (2020). Lipoprotein(a) levels and association with myocardial infarction and stroke in a nationally representative cross-sectional US cohort. Journal of Clinical Lipidology, 14(5), 695-706.e4. https://doi.org/10.1016/j.jacl.2020.06.010
23. Wang, Z. W., Xiao, S. J., & Liu, N. F. (2023). Association of lipoprotein(a) with left ventricular hypertrophy in patients with new-onset acute myocardial infarction: A large cross-sectional study. Clinica Chimica Acta, 540, 117226. https://doi.org/10.1016/j.cca.2023.117226
24. Kotani, K., Serban, M. C., Penson, P., Lippi, G., & Banach, M. (2016). Evidence-based assessment of lipoprotein(a) as a risk biomarker for cardiovascular diseases – Some answers and still many questions. Critical Reviews in Clinical Laboratory Sciences, 53(6), 370-378. https://doi.org/10.1080/10408363.2016.1188055
25. Wang, L., Ma, C., Liu, X., & Han, W. (2025). Association between lipoprotein(a) and dilatation of different aortic segments in hypertensive patients. Echocardiography, 42(1), e70061. https://doi.org/10.1111/echo.70061
26. Chua, F., Lam, A., Mak, Y. H., Lee, Z. H., Dacay, L. M., Yew, J. L., Puar, T., Khoo, J., Chow, W., Tan, V. H., Tong, K. L., Liew, B. W., Yeo, C., & Loh, W. J. (2023). Undiagnosed cardiovascular risk factors including elevated lipoprotein(a) in patients with ischaemic heart disease. Frontiers in Epidemiology, 3, 1207752. https://doi.org/10.3389/fepid.2023.1207752
27. García de Prada, M., Costa Segovia, R., de Castro Martínez, M., Valdeolivas Hidalgo, N., Sánchez López, M. B., & Blanco Echevarría, A. (2024). Clinical-epidemiological analysis of patients with elevated lipoprotein A in a third level hospital. Clínica e Investigación en Arteriosclerosis, 36(3), 118-125. https://doi.org/10.1016/j.arteri.2023.12.001
28. Brosolo, G., Da Porto, A., Bulfone, L., Vacca, A., Bertin, N., Colussi, G., Cavarape, A., Sechi, L. A., & Catena, C. (2021). Plasma lipoprotein(a) levels as determinants of arterial stiffening in hypertension. Biomedicines, 9(11), 1510. https://doi.org/10.3390/biomedicines9111510
29. Di Maio, S., Grüneis, R., Streiter, G., Lamina, C., Maglione, M., Schoenherr, S., Öfner, D., Thorand, B., Peters, A., Eckardt, K. U., Köttgen, A., Kronenberg, F., & Coassin, S. (2020). Investigation of a nonsense mutation located in the complex KIV-2 copy number variation region of apolipoprotein(a) in 10,910 individuals. Genome Medicine, 12(1), 74. https://doi.org/10.1186/s13073-020-00771-0
30. Schrader, B., Shakoor, A., Schmidt, A., Schrader, J., Vaske, B., Haller, H., Koziolek, M., Elsässer, A., & Lüders, S. (2021). Relationship between lipoprotein(a) and cardiovascular risk factors: Data from 4602 participants of the ELITE study. Reviews in Cardiovascular Medicine, 22(4), 1569-1575. https://doi.org/10.31083/j.rcm2204162
31. Zhurayev, R., Proost, D., Zerbino, D., Fedorenko, V., Meester, J. A., Van Laer, L., & Loeys, B. L. (2016). Identification of FBN1 gene mutations in Ukrainian Marfan syndrome patients. Genetics Research, 98, e13. https://doi.org/10.1017/S0016672316000112

Резюме

ЛІПОПРОТЕЇН(А) ЯК МОДИФІКАТОР СУДИННОГО РЕМОДЕЛЮВАННЯ ТА СЕРЦЕВО-СУДИННОГО РИЗИКУ ПРИ АРТЕРІАЛЬНІЙ ГІПЕРТЕНЗІЇ: ОГЛЯД ЛІТЕРАТУРИ

Тетяна М. Соломенчук[1], Рустам К. Жураєв[1], Василь В. Процько[1,2], Катерина С. Гель[1,2], Вікторія Р. Николяк[1]

[1]Державне некомерційне товариство «Львівський національний медичний університет імені Данила Галицького», м. Львів, Україна
[2]Комунальне некомерційне підприємство «1 Територіальне медичне об'єднання м. Львова», м. Львів, Україна

Вступ. Ліпопротеїн(а) сьогодні розглядають як найважливіший генетично детермінований фактор серцево-судинного ризику, пов'язаний із прискореним розвитком атеросклерозу, ендотеліальною дисфункцією, судинним запаленням, підвищенням артеріальної жорсткості та кальцифікуючим ураженням судин. Наукові дані останніх років свідчать про те, що підвищений рівень Lp(a) асоціюється не лише з атеросклеротичними серцево-судинними захворюваннями, а й із процесами судинного ремоделювання та збереженням підвищеного серцево-судинного ризику при артеріальній гіпертензії.

Мета. Проаналізувати сучасні наукові публікації та визначити роль ліпопротеїну(а) у судинному ремоделюванні, ендотеліальній дисфункції, ригідності артерій, артеріальній жорсткості та оцінці серцево-судинного ризику в пацієнтів із підвищеним артеріальним тиском та артеріальною гіпертензією.

Матеріали та методи. Ми здійснили пошук статей у MEDLINE, опублікованих у період 2016-2026 років, за допомогою комбінації заздалегідь визначених ключових слів. У результаті пошуку було виявлено 86 наукових публікацій, які первинно оцінювалися за назвами та анотаціями. Після аналізу релевантності для детального опрацювання було відібрано 56 статей. До фінального огляду включено 31 публікацію.

Результати. Підвищені концентрації Lp(a) асоціюються не лише зі зростанням серцево-судинного ризику, а й з низкою механізмів, залучених до процесів судинного ремоделювання. Одним із ключових патогенетичних механізмів є дія окиснених фосфоліпідів, що транспортуються Lp(a) та спричиняють оксидативний стрес, активацію запалення, ендотеліальну дисфункцію, судинний фіброз і кальцифікацію. Сучасні дослідження також продемонстрували зв'язок підвищеного рівня Lp(a) зі збільшенням пульсового тиску, порушенням нічного зниження артеріального тиску, артеріальною жорсткістю, судинною кальцифікацією та ураженням органів-мішеней при артеріальній гіпертензії. Таким чином, підвищений рівень Lp(a) може сприяти формуванню несприятливого судинного фенотипу при артеріальній гіпертензії та посилювати залишковий серцево-судинний ризик незалежно від традиційних ліпідних показників. Останні досягнення у сфері РНК-таргетної терапії, включаючи антисенсові олігонуклеотиди та препарати на основі малих інтерферуючих РНК, продемонстрували значне зниження концентрації Lp(a) у крові та відкрили нові перспективи персоналізованої серцево-судинної профілактики.

Висновки. Підвищений рівень Lp(a) слід розглядати не лише як порушення ліпідного обміну, але і як важливий генетично детермінований модифікатор судинного ремоделювання та серцево-судинного ризику у пацієнтів з артеріальною гіпертензією. Сучасні дані свідчать про те, що підвищений Lp(a) бере участь у розвитку ендотеліальної дисфункції, судинного запалення, артеріальної жорсткості, судинного фіброзу та кальцифікації.

Ключові слова: **ліпопротеїн(а), артеріальна гіпертензія, судинне ремоделювання, кардіологічна генетика, ригідність артерій, серцево-судинний ризик**

Received: 15.04.2026
Accepted: 11.06.2026
Published: 13.08.2026