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Investigating the Function of Lipoprotein (a) in Diabetes and Cardiovascular Disease in the Chinese Population

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Abstract

A number of cardiovascular diseases have been linked to high plasma levels of lipoprotein (a), and some other diseases are thought to be independently predicted. Recent research indicates that the Chinese population's Lp(a) concentration levels differ significantly from those of other populations. A high level of Lp(a) in the Chinese population indicates a higher rate of thrombogenicity, platelet aggregation, and revascularization following PCI. Higher levels of Lp(a) have been linked to an increased risk of atherosclerotic cardiovascular disease (ASCVD) in the Chinese population, according to studies. More specifically, higher levels of Lp(a) have been linked to an increased risk of coronary heart disease, severe aortic valve stenosis, deep vein thrombosis in spinal cord injury patients, central vein thrombosis in hemodialysis patients, and stroke in Chinese populations.

Lp (a) may also play a crucial role in a number of other conditions, including cancer, type 2 diabetes, and the metabolic syndrome, according to new and consistent data retrieved from a number of clinical trials. This audit investigates the clinical and epidemiological connections among Lp(a), cardiovascular sicknesses and diabetes in the Chinese populace as well as likely Lp(a) hidden systems in these illnesses. To better comprehend the role of Lp(a) in the Chinese population's cardiovascular diseases and diabetes, however, additional research is required.

Keywords: Lipoprotein (a) • Epidemiology • Cardiovascular diseases • Diabetes mellitus • Cancer

Introduction

The macromolecular complex known as lipoprotein (a) (Lp(a) is made up of the LDL particle, which contains apolipoprotein B-100 (apoB-100), and the large, highly polymorphic glycoprotein known as apolipoprotein (a) (apo(a)). The liver produces apo(a), which contains triple loop structures known as kringles. Two parts of Lp(a) are covalently linked by a disulfide bond between one of the kringle domains in apo(a) and apoB-100. A significant locus that regulates the concentration of Lp(a) is the LPA gene, which is located on the reverse side of chromosome 6q2. According to a few previous studies, Lp(a) has no useful role in physiological metabolism. However, recent research suggests that Lp(a) inhibits fibrinolysis, which reduces the risk of increased bleeding during childbirth and provides cholesterol for cell proliferation during tissue repair. In addition, Lp(a) is believed to be engaged with tissue fix and wound recuperating [1].

However, this association between Lp(a) and fibrinolysis was not supported by the European Atherosclerosis Society (EAS) consensus statement 2022. Due to its disordered plasma concentration, Lp(a) is thought to be an independent predictor of numerous cardiovascular and cerebrovascular diseases. Low Lp(a) concentrations were linked to an increased risk of type 2 diabetes (T2D), whereas high Lp(a) concentrations were linked to an increased risk of coronary heart disease (CHD). According to studies, Chinese people with higher levels of lipoprotein (a) have a lower risk of developing type 2 diabetes. The risk of developing type 2 diabetes may be reduced by lowering elevated Lp(a) levels below 30 ng/mL. Lp(a) may likewise act as an autonomous gamble biomarker for type 2 diabetes and foresee repetitive adverse results in type 2 diabetes patients with past cardiovascular occasions [2].

There are significant variations in plasma Lp(a) concentrations among individuals, within populations, and across populations. Lp(a) concentrations in humans range from less than 0.1 mg/dl to more than 200 mg/dl, and the mean Lp(a) values may differ by threefold between populations. For instance, people of African descent typically have higher levels of lipoprotein (a) than most Asian and European populations. It is essential to keep in mind that Lipoprotein (a) levels can be affected by genetic variants in the LPA gene as well as the size of apolipoprotein (a). It has been demonstrated that the plasma concentration of Lp(a) is inversely correlated with the size of apo(a), and genetic variants in the LPA gene may also contribute to variation in Lp(a) levels [3].

Literature Review

Plasma Lp(a) variance is estimated to be 30-40% explained by variations in LPA, while isoform size accounts for 40-70%. The varying Lp(a) levels observed in various populations can be explained by these factors. Variants in the LPA gene's frequency can vary from population to population. The frequency of elevated Lipoprotein (a) levels and the risk of associated health conditions in various populations may be affected by this variation. Some LPA variants, for instance, may be more prevalent in some populations, raising the likelihood of elevated Lipoprotein (a) levels and the health issues that go along with them. This demonstrates the significance of taking into account population-specific factors when determining the risk of health conditions linked to Lipoprotein (a) and developing targeted treatments. For instance, rs10455872 is prevalent in Caucasians and strongly linked to an increased risk of carotid artery disease and an increase in Lp(a) levels, but it is rare in Chinese people [2].

In addition, the European Society of Cardiology and the Canadian Cardiovascular Society recommend measuring Lp(a) in high-risk individuals, and the EAS 2022 consensus recommends measuring Lp(a) at least once in adults. Until now, no proposals for Lp(a) estimation from Chinese specialists have been given, nor have the exact removed upsides of serum Lp(a) levels for cardiovascular infection risk evaluation been formed. This review will

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summarize Lp(a)s with cardiovascular diseases and type 2 diabetes in the Chinese population in order to better understand the role of lipoprotein(a) in this population and develop more efficient prevention and intervention strategies to lessen the burden of these diseases [4].

A plasma lipoprotein known as the Lp(a) particle is made up of two distinct and significant components: 1) A particle that looks like LDL and contains apoB-100; and (2) a distinct glycoprotein apo(a) particle resembling plasminogen, whose size ranges from 200 to 900 kDa. Due to variations in KIV-2's copy number, the size of apo(a) is polymorphic. More than 40 isoforms are encoded by the variation in KIV-2 repetitions that is determined by the LPA gene. Plasma lipoprotein (Lp(a) concentrations are inversely correlated with the size of the apolipoprotein (apo(a)). The extended intracellular processing and increased degradation required for larger apo(a) isoforms may be the cause of this. Consequently, the size of apo(a) may have a significant impact on hepatic synthesis, secretion rate, and blood Lp(a) concentration [5].

Lipoprotein (Lp(a))'s precise physiological function in humans is still up for debate. Several previous studies have concluded that Lp(a) plays no significant role in physiological metabolis, despite the wide variation in plasma Lp(a) levels among individuals and the absence of metabolic abnormalities in individuals with low circulating Lp(a) levels. In any case, discoveries from a review propose that Lp(a) adds to the arrangement of cholesterol for cell expansion during tissue fix and furthermore assumes a part in lessening over the top draining by restraining fibrinolysis, especially during labor. Boffa et al., for example It has been reported that Lp(a) can compete with plasminogen for binding to carboxyl-terminal lysine-containing cell surface receptors like annexin and -enolase, thereby inhibiting fibrinolysis and reducing plasmin synthesis in vitro. Increased plasma levels of Lp(a) have been linked to an increased risk of venous thrombosis or venous thromboembolism in a number of genetic studies. Additionally, Lp(a) is involved in monocyte/macrophage chemotactic activation and angiogenesis regulation [6].

The fact that apo(a) isoform sizes alter based on the donor's genotype after liver transplantation demonstrates that LPA is primarily transcribed in the liver, which is the primary driver of apo(a) production. Despite these contradictory findings, the location of Lp(a) assembly remains a contentious issue. On the surface of hepatocytes, intracellularly, or even extracellularly, some studies suggest that assembly takes place. The difference in Lp(a) concentrations between arterial and venous blood in the renal circulation, the presence of apo(a) fragments in urine, and the significant impact that chronic kidney disease has on Lp(a) levels all point to a prominent role for the kidneys in Lp(a) catabolism. Lp(a)'s biological half-life is said to be comparable to that of LDL in some studies. On the other hand, another study came to the conclusion that Lp(a) has a fractional catabolic rate that is 30% lower than that of LDL [7].

Atherosclerosis is a pervasive cardiovascular sickness coming about because of the collection of plaques in the blood vessel walls. Lp(a) has been identified as a distinct risk factor for the development of atherosclerosis in a number of epidemiological and genetic studies. Numerous published studies in the Chinese population suggest that elevated Lp(a) levels may contribute to the development of atherosclerosis. There is evidence to suggest that elevated plasma Lp(a) levels may contribute to the accumulation of Lp(a) in the arteries, thereby exacerbating the progression of atherosclerosis, and that the progression of Lp(a) is regulated by low-density lipoprotein receptor (LDLr) and chemokine ligand 16 (CXCL16) in a retrospective clinical study of Chinese patients. This study compared a control group with Lp(a In addition, elevated levels of Lp(a) and an increased copy number of the KIV-2 gene were found to be significant risk factors for the onset of atherosclerosis in the Chinese Han population, and elevated levels of Lp(a) were found to be a sign of particular kinds of coronary plaques. Also, openness to oxidization, Lp(a) in endothelial cells could decrease the outflow of DSG1 and DSC2 by means of ROS age, which might work on the penetrability of endothelial cell monolayer to speed up the movement of atherosclerosis [8].

Discussion

Vascular occlusion and stenosis may be facilitated by atherosclerotic lesions, which may increase the risk of cardiovascular disease (CAD). Various cross-sectional examinations have shown that Lp(a) may be an autonomous indicator of the seriousness of computer aided design in the Chinese populace. Lp(a) levels were found to be significantly related to the severity of coronary artery disease, but not to cardiovascular events, in the findings of a Chinese cohort study. Additionally, the rs6415085 SNP in the Lp(a) gene was found to be associated with elevated plasma Lp(a) levels and an increased risk of developing CAD. Additionally, elevated Lp(a) levels were independently associated with a poor prognosis in individuals with non-obstructive CAD, suggesting that they might be useful in risk stratification for Chinese patients. Reduced myocardial perfusion is what causes acute myocardial infarction (AMI), which is typically brought on by the breaking or cracking of an atherosclerotic plaque. It has been accounted for that raised Lp (a) levels may be related with the AMI risk in Chinese patients. In patients with AMI, Lp(a) has also been identified as a potential biomarker for predicting coronary collateral circulation or cardiovascular mortality [9].

Besides, a huge communication was seen among Lp(a) and LDL-C levels according to episode Intense Myocardial Dead tissue (AMI) in the Chinese Han populace. This demonstrates that elevated Lp(a) and high LDL-C levels have a greater exacerbating effect on the risk of initial AMI than either factor alone. Another pivotal thought is that Lp(a) holds potential as a biomarker in the clinical determination and the board of coronary supply route illness (computer aided design). Clinical instability and the progression of CAD have been linked to elevated levels of Lp(a) in the Chinese patient population, particularly in patients with high fibrinogen levels. It is important to note that the level of LDL-C may affect the concentration of Lp(a) in Chinese patients. In real-world treated patients with CAD, circulating Lp(a) concentration may be a useful predictor of the risk of recurrent cardiovascular events, as demonstrated by a multi-center, prospective study of 7562 patients with angiography-diagnosed CAD [10].

Conclusion

The epidemiological and clinical connections between Lp(a) and other Chinese diseases, such as diabetes and cardiovascular disease, were demonstrated in this review. A number of specific Lp(a) mutations in the Chinese population were highlighted in this article. Higher Lp(a) values indicate an independent cardiovascular disease risk for Chinese patients. Moreover, new and reliable proof from various clinical examinations highlight the likelihood that Lp(a) might be critical in the improvement of extra circumstances, including type 2 diabetes and malignant growth. Lp(a) levels should be used in laboratory diagnostic testing for Chinese people who are at risk for cardiovascular diseases, according to the recommendations. Additionally, rather than the recommended cutoff value of 50 mg/dl (for the Caucasian population), serum Lp(a concentrations of >30 mg/dl should be considered a critical cutoff for assessing the risk of cardiovascular disease in Chinese patients. The mechanisms that link Lp(a) to various diseases in the Chinese population must still be investigated in the future. Additionally, in order to establish conclusive proof of causality and evaluate the clinical efficacy of the novel, potent Lp(a) lowering therapies, randomized outcome trials are required.

Acknowledgement

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Conflicts of Interest

None.

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