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Prevalence of elevated lipoprotein(a) among women consulting the Women's Hospital in Karbala, Iraq: A hospital-based study

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Abstract

Background: Lipoprotein(a) [Lp(a)] is an emerging, genetically determined lipid parameter recognized as an independent risk factor for atherosclerotic cardiovascular disease. The study aimed to determine the prevalence of elevated Lp(a) levels among Iraqi women attending the Women's Hospital in Karbala Iraq. Although elevated Lp(a) is associated with higher rates of cardiovascular disease (CVD), limited data exist regarding Lp(a) values among Iraqi women of reproductive and middle age.

Objectives: The aim of this study was to document the prevalence of elevated Lp(a) levels among women attending Al-Saida Khadija Al-Kubra Women's Hospital in Karbala City Iraq in the period April 2024 to March 2025.

Methods: We conducted a cross-sectional observational study. Women aged 18-50 years who attended the hospital's outpatient clinics for consultation on various non-critical conditions underwent Lp(a) testing (only funded test) We determined the proportion of women with Lp(a) values ≥50 mg/dL.

Results: Lp(a) was measured in 1013 women (age 37.48±8.93 years) who attended outpatient clinics (endocrinology, gastroenterology, nutrition and cardiovascular) during the period from April 2024 to March 2025 was 1013. Median IQR levels of Lp(a) were 75.5(61.9, 90) mg/dl and the prevalence of elevated Lp(a) was 11.15% (n=113).

Conclusions: Approximately 1 in 9 women aged 18-50 years attending a secondary care hospital in Karbala City had elevated Lp(a). This finding highlights the importance of incorporating Lp(a) testing into cardiovascular risk assessment, especially for women with a family history of premature ASCVD or unexplained dyslipidemia.

Introduction

Cardiovascular Disease (CVD) remains a leading cause of morbidity and mortality worldwide, despite all the progress achieved regarding both prevention and treatment [1,2]. Atherosclerosis is a complex, multifactorial disorder and multiple risk factors including increasing age, cigarette smoking, hypertension, inflammation, elevated Low-Density Lipoprotein Cholesterol (LDL-C) and decreased High-Density Lipoprotein Cholesterol (HDL-C) increase the likelihood of a person developing atherosclerosis. Besides these traditional risk factors elevated lipoprotein(a) (Lp(a)) can also significantly increase the risk of developing atherosclerosis [3].

Lp(a) is made up of an LDL-like particle to which apolipoprotein(a) [apo(a)] is covalently bound by a disulfide bond [4]. Both observational and genetic studies have shown an independent association between elevated levels of Lp(a) and the development of atherosclerotic cardiovascular disease (CVD) and calcific aortic valve stenosis [5].

There is a continuous association between Lp(a) blood levels and ASCVD risk. However, values greater than 50 mg/dL are generally regarded as 'high' and clinically relevant, but even levels above 30 mg/dL may increase risk.

Lp(a) levels are mainly genetically determined and can vary several hundredfold between individuals. Additionally, there are ethnic variations, with levels in the black population being 2-3 fold higher than those in white populations. In addition, Lp(a) levels remain relatively constant throughout life and are not influenced by age, gender, diet or lifestyle [6].

There have been no prior study determining the epidemiology of Lp(a) in Iraqi women (This study is the first). This study aims to estimate the prevalence of high Lp(a) in Iraqi Women consulting the Women's Hospital in Karbala Governorate.

Patients and methods

This prospective cross-sectional study was conducted over a 12-month period from April 2024 to March 2025 at Al-Saida Khadija Al-Kubra Women's Hospital, Karbala City, Iraq.

The inclusion criteria were being female, aged 18 to 50 years, and consulting a subspecialty medical clinic at the hospital.

The exclusion criteria were:

- Clinically overt atherosclerotic cardiovascular disease
- Diabetes mellitus or hypothyroid
- Receiving medications known to affect the lipid profile, including oral contraceptive pills, isotretinoin, oral corticosteroids, atypical antipsychotics, or immunosuppressive therapies, pregnancy, renal impairment, severe liver disease, sepsis, and coagulopathy.

A complete medical history was obtained from all patients with particular emphasis on the presence of renal insufficiency, known coronary artery disease, and previously diagnosed hyperlipidemia. A positive family history of early ASCVD was defined as the occurrence of AMI or sudden death in a first-degree male relative before the age of 55 years or in a first-degree female relative before the age of 65 years, or a history of stroke occurring before the age of 50 years in a first-degree relative.

Lp(a) concentrations were determined from non-fasting serum samples. Following collection, samples were immediately aliquoted and stored at −80°C to ensure protein stability and prevent degradation. To maintain structural integrity, samples underwent only a single freeze-thaw cycle prior to analysis.

Analytical platform and reagents

Quantitative measurements were performed on the Abbott Architect Plus ci4100 integrated clinical chemistry analyzer (Abbott Diagnostics, Abbott Park, IL, USA). The assay utilized a latex-enhanced immunoturbidmetric method (Abbott List No. 06P33). The reagent system consisted of a suspension of uniform-sized latex particles covalently coated with polyclonal rabbit anti-human Lp(a) antibodies (IgG).

Principles of operation

The assay logic is based on the principle of antigen-antibody agglutination. Upon mixing the serum sample with the latex reagent, the Lp(a) antigens in the specimen react with the anti-Lp(a) antibodies, resulting in the formation of insoluble immune complexes. The resulting increase in turbidity was measured spectrophotometrically at a primary wavelength of 700 nm. The change in absorbance is directly proportional to the concentration of Lp(a) in the sample, which was quantified against a multi-point calibration curve stored on the system.

Standardization and units

The assay was calibrated using values traceable to the WHO/IFCC SRM 2B international reference material, ensuring global standardization. While the assay is designed to be minimally sensitive to the apolipoprotein(a) isoform size heterogeneity, results were expressed in mass units of milligrams per deciliter (mg/dL).

Analytical performance and quality control

The laboratory maintained strict quality assurance protocols. Accuracy and precision were monitored using two levels of liquid Quality Control (QC) materials (low and high) processed at the beginning of each analytical run. All results remained within two standard deviations (2 SD) of the target values. Potential interferences from bilirubin, hemoglobin, and intralipid were monitored according to the manufacturer's technical specifications to ensure analytical specificity.

Statistical analysis

The data of the current study were entered into and analyzed using the Statistical Package for the Social Sciences (SPSS 23.0 for Windows). Descriptive statistics were used in term of frequency and percentage for categorical variables and mean±SD for continuous variables in appropriate tables. Lp(a) levels are presented as median (1st and 3rd quartiles). The Kruskal-Wallis test was used to assess the difference in Lp (a) level. The Significance level was set at $p<0.05$.

Results

Overall Lp(a) was measured in 1,013 women. The mean (SD) age of participants was 37.48±8.93 years (range 18-50 years) and 31.9% were in the 25-34 years age group. The mean (SD) ages were 37.48±8.93 years for elevated age group and 36.32±5.19 years for non-elevated group. The median (quartiles) of elevated LP(a) level was 75.5(61.9, 90) mg/dl and for non-elevated Lp (a) 31.6 (20.5, 46) mg/dl as shown in (Table 1).

There were 113 (11.15%) participants with elevated Lp(a) (≥50 mg/dl) as shown in (Figure 2). In addition, there was no association between age and elevated level of Lp(a) and a non-significant difference between age groups regarding Lp(a) level as shown in (Table 2).

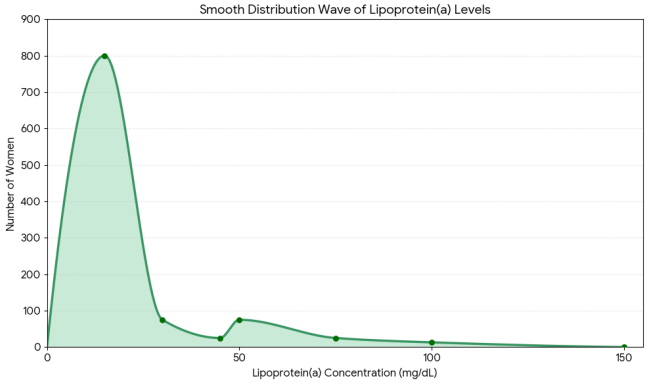

Figure 1: Lp(a) frequency distribution in mg/dl in all participants.

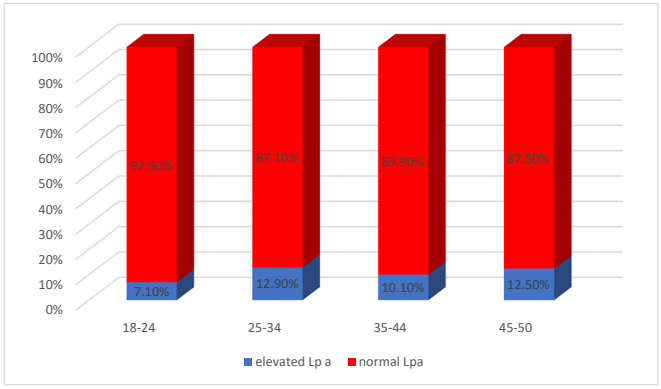

Figure 2: Distribution of study women by age.

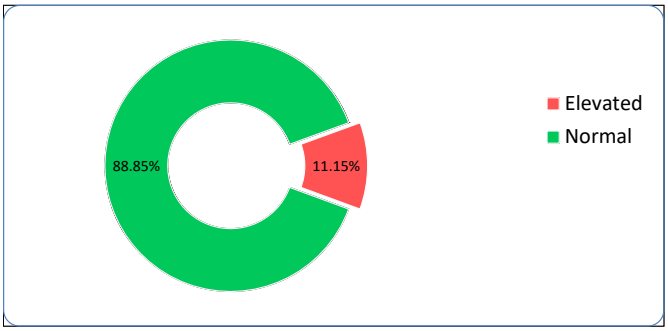

Figure 3: Proportion of elevated Lp(a) among study women.

Table 1: Lp(a) levels according to age strata.

		Elevated Lp(a)		Non elevated Lp(a)		
	Categories	**Frequency**	**Percent**	**Frequency**	**Percent**	**Total**
Age group	18-24	10	7.1%	131	92.9%	141
	25-34	36	12.9%	242	87.1%	278
	35-44	30	10.1%	268	89.9%	298
	45-50	37	12.5%	259	87.5%	296
	Total	113		900		1013
Age	Mean ± SD		37.48±8.93	36.32±5.19		
Lp(a) mg/dl	Median (1st and 3rd quartiles)		75.5 (61.9, 90)	31.6 (20.5, 46)		

Table 2: Show the difference in Lp (a) median within age group.

Categories Age group	No.	Mean rank	p-value
18-24	10	46.7	0.6
25-34	36	60.22	
35-44	30	58.28	
45-50	37	55.61	
Total	113		

Discussion

The primary objective of this study was to establish the prevalence of elevated Lp(a) among women of reproductive and perimenopausal age (18-50 year) in Karbala, Iraq. Our findings revealed a prevalence of Lp(a) >50 mg/dL of 11.15%. While this is significant, it sits at the lower end of the global estimated average, where it is generally accepted that approximately 20% of the world's population has Lp(a) levels >50 mg/dL [7].

The lower prevalence found at Al-Saida Khadija Women's Hospital suggests that while elevated Lp(a) is a concern in the Iraqi population, levels above 50 mg/dL may be less prevalent than in other populations.

Studies in Western cohorts often report higher prevalence rates, but ethnic variations are well-documented. For instance, individuals of African descent often show significantly higher levels compared to Caucasians or Asians [8]. Our study is the first step in defining what "normal" looks like for the Iraqi female population specifically.

Data on Lp(a) in the Middle East are scarce. Our results provide a vital baseline for the Iraqi health system, suggesting that roughly 1 in 9 women in this age bracket are at an increased, often "hidden," risk for (ASCVD).

Conventional lipid panels (LDL-c, HDL-c and Triglycerides) generally miss the risk posed by Lp(a), which is largely genetically determined and resistant to traditional lifestyle changes or statin therapy [9].

In addition to being rich in cholesterol, Lp(a) carries oxidized phospholipids that may enhance vascular inflammation and plaque instability [10].

These mechanisms may be particularly relevant in women, who often exhibit different plaque characteristics and a greater propensity for thrombotic and microvascular dysfunction compared with men [11].

Since the sample focused on women aged 18-50, identifying these 113 women allows for early aggressive management of other modifiable risk factors (blood pressure, glucose, and LDL-c) to reduce the risk of high Lp(a). One limitation of this study is that women in higher age groups, and therefore after the menopause, were not included. Lp(a) may rise after the menopause; therefore, it would be important in future studies to evaluate its levels in older women [12].

The implementation of Lp(a) screening at Al-Saida Khadeeja Women's Hospital has provided the first localized data on the prevalence of Lp (a) among Iraqi women in their reproductive and perimenopausal years. The identified prevalence of 11.15% confirms that elevated Lp(a) is a significant, yet previously unrecognized, cardiovascular risk factor within this population. While this rate is lower than the global estimated average of 20%, it still involves a sizable group of over 100 women who might have been misclassified as "low risk" under traditional lipid screening protocols. This study effectively demonstrates the feasibility of integrating advanced lipid testing into the Iraqi primary health system and highlights the need to go beyond standard LDL-C assessments to better identify genetic cardiovascular risk.

Conclusion

In conclusion, Lp(a) levels showed a skewed distribution in our population, with 11% presenting elevated levels. More data are needed in older women and men to better characterize Lp(a) concentrations in Iraqi people. Also, one need to test if higher Lp(a) concentrations are associated with a higher ASCVD risk in Iraqi populations.

Declarations

Funding: This study received no funding.

Ethical consideration: Ethical approval for this study was granted by [Al-Saida Khadija Al-Kubra Women's Hospital, Karbala, Iraq] Institutional Review Board (Protocol No. N/A). This study was conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent prior to their inclusion in the study.

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