Serum Lipoprotein (A) in Patients with Fibromyalgia Syndrome

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Abstract

Background: Fibromyalgia syndrome (FMS) is the most common rheumatic cause of diffuse pain and multiple regional musculoskeletal pain and disability.
Objective: is to assess the contribution of serum lipoprotein (A) in the pathogenesis of FMS patients.
Methods: One hundred twenty two FMS patients were compared with 60 healthy control individuals who were age and sex matched. All FMS features and criteria are applied for patients and controls; patients with secondary FMS were excluded. Serum Lipoprotein (A): [Lp(A)], body mass index (BMI), & s.lipid profile were determined for both groups.
Results: There was a statistical significant difference between patients &controls in serum lipoprotein (A) (P=0.013). Also there was a statistical significant correlations between serum Lp(A) & FMS patients' age(r= 0.310, P=0.034); but not with: duration(r= -0.222, P=0.133) , BMI(r= 0.128,P=0.390) & s.lipid profile (p> 0.05) of FMS patients.
Conclusion: s.lipoprotein (A) may play an important role in pathogenesis of FMS patients.
***Key words*:** Fibromyalgia syndrome, S.Lipoprotein (A), S.lipoprotein (A) in fibromyalgia syndrome

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Introduction

Fibromyalgia (FM) is a debilitating and frustrating syndrome characterized by a chronic widespread pain and tenderness with prevalence rate of 4.9% in the general population [(1)]. Its pathogenesis has been linked to genetic &environmental factors; abnormal pain &sensory processing; hypothalamic-pituitary &autonomic dysfunction; and psychological &behavioral factors [(2)]. The American College of Rheumatology 1990 Criteria for Classification of FMS were applied for diagnosis [(3)].

Lipoprotein(A) [Lp(A)] is a heterogeneous macromolecule that consists of a glycoprotein, Apo lipoprotein(A) [Apo(A)], which is linked by a disulfide bridge to Apo lipoprotein B-100 on an LDL core [(4,5)]. It is clinically important because its concentrations are primarily genetically determined, associated with atherosclerotic disease, and less affected by lifestyle or medication [(6)].

There are previous reports about s.Lp(A) in rheumatological diseases like rheumatoid arthritis[(7)],& systemic lupus erythematosus [(8)]; but not in FMS.

As Lp(A) is an independent risk factor for cardiovascular disease.[(9)], & because FMS associated with cardiovascular manifestations, a possible association between the two might be present.

The aim of the current study was to assess the contribution of serum lipoprotein (A) in the pathogenesis of FMS.

METHODS

Across sectional study was carried out at the Baghdad Teaching Hospital / Rheumatology Unit from April 2008 to February 2009.

One hundred twenty two patients with primary FMS were included in the study & were diagnosed in the Rheumatology Unit on base of full history, & complete clinical examination. The American College of Rheumatology 1990 Classification Criteria [(3)] for fibromyalgia were applied to all fibromyalgia patients. Another 60 healthy individuals matched for age and sex were collected from healthy volunteers & were studied as a control group. Patients with secondary FMS have been excluded from the study.

S.Lipoprotein (A), s.lipid profile, & BMI were measured in all fasting subjects.

Patients' consent was taken & Ethical approval was obtained from the Ethics Committee of Baghdad University, College of Medicine, Medical Department.

Statistical analysis was done using statistical package for social science software (SPSS10). Association between different categorical variables was measured using Chi-square test. Difference between continuous variables was measured using t-test & Pearson correlation (r). P-values<0.05 were considered significant.

RESULTS

The 122 patients with FMS (21 males & 101 females) compared with 60 healthy individuals (12 males & 48 females) as a control group. The mean age was (41.56±10.82) years for the FMS patients group, and (42.93±12.72) years for the control group (P-value= 0.45) indicating no statistical significant difference between both groups **(Table 1).**

The mean of s.Lipoprotein (A) was (221.06 ± 22.35) mg/dl in 122 patients with FMS compared to (197.79 ± 32.63) mg/dl in 60 healthy individuals (P-value = 0.013) indicating a statistical significant difference between both groups as shown in Table 2.

There was a statistical significant correlation between s.Lp(A) & patients' age (r=0.310, P=0.034), but not with disease duration (r=-0.222, P=0.133); & patients BMI (r=0.128, P= 0.390) as shown in **Table 3.**

There was no statistical significant correlation between s.Lp(A) & s.lipid profile((TC, TG, HDL-C, LDL-C, VLDL-C**)** (r=0.071,P=0.636**;** r=-0.023, P=0.878**;** r=-0.188, P=0.206**;** r=0.132, P= 0.377**;** r= -0.028; P= 0.852) respectively as shown in **Table 4.**

Table 1: Distribution of the studied sample according to demographic characteristics:

Variables	Patients=122	Controls=60	p-value
Age(Mean ±SD) in years	41.56±10.82	42.93±12.72	0.45 n.s
Sex			
Male n.(%)	21(63.61)	12(36.4)	0.68 n.s
Female n.(%)	101(67.8)	48(32.2)	

ns, P-value is not significant

Table 2: Comparison of s. lipoprotein(A) between FMS patients and controls

Variables	FMS (n = 122) Mean ± SEM	Controls (n= 60) Mean ± SEM	P-value
Lp(a) (mg/dl)	221.06 ± 22.35	197.79 ± 32.63	0.013

**** P-value is significant***

Table 3: Correlations between Lp(A) and age, duration , & BMI of FMS

Paraneters	Lp(A)
Age	
r- value	0.310
P- value	0.034*
Duration	
r- value	-0.22
P- value	0.133
BMI	
r- value	0.128
P- value	0.390

**** P-value is significant***

Table 4: Correlations between Lp(A) and lipid profile of FMS patients

Paraneters	Lp(a)
TC	
r- value	**0.071**
P- value	**0.636** ^{NS}
TG	
r- value	**-0.023**
P- value	**0.878** ^{NS}
HDL-C	
r- value	**-0.188**
P- value	**0.206** ^{NS}
LDL-C	
r- value	**0.132**
P- value	**0.377** ^{NS}
VLDL-C	
r- value	**-0.028**
P- value	**0.852** ^{NS}

NS, P-value is not significant.

Discussion

In the present study we found a significant association between increased s.Lipoprotein (A) and FMS patients.

To our knowledge, this is the first study examining the relationship between FMS and s.Lp(A), so no previous results for comparison; but there are studies that have established the relationship between increased s.Lp(A) levels and rheumatoid arthritis (RA) [7] as well as systemic lupus erythematosus (SLE) [8].

The only link between s.Lp(A) and FMS is the work of Berg *et al* [10] on the hypercoagulation theory, as a proposed cause of FMS symptoms and other chronic illnesses. Since the hypercoagulability was decreased by heparin injections, the chronic illness symptoms have diminished. This was the first clue to the connection between coagulation and chronic illnesses.

In the present study, there was a statistically significant positive correlation between s.Lp(A) and patients' age. This agreed with other studies in which s.Lp(A) has been identified as a risk factor for atherosclerosis and increases with age [11,12].

Also in the present study, there was a negative non significant correlation between s.Lp(A) and duration of FMS, this possibly might be explained by occurrence of an inflammatory process in the early onset of the disease, which has elevated s.Lp(A) levels above normal values.

In the present study, there was a statistically non significant correlation between s.Lp(A) and: BMI, and s.lipid profile of FMS patients. This might be explained by the limited sample studied. This agreed with other studies [13,14].

Lp(A) has become a focus of attention due to the possibility that levels of circulating Lp(A) represent an independent risk factor for coronary vascular disease & of greater predictive potential than other lipoprotein traits [15].

Changes in plasma Lp(A) levels are associated with early atherosclerosis [16]. Lp(A) is an independent cardiovascular risk factor [17,18]. Some researchers have found a probable close relationship between FMS and cardiovascular alterations [19].

A number of limitations of the current study must be pointed out. We did not perform a detailed assessment of the effect of patients characteristics on s.Lp(A). More detailed analyses of these parameters would better characterize these aspects and would assist in the evaluation of the development of FM symptoms. The relatively small size of the study sample must be noted. Despite these limitations, our findings call attention to a previously unrecognized possible a relationship, and justify additional research for determining the risk factors for FMS.

We recommend to test s.Lp(A) to monitor hypercoagulability and early atherosclerosis in FMS patients.

Conclusion:

S.lipoprotein (A) may play an important role in pathogenesis of FMS patients.

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