Demographic Analysis and Lipid profile in Fibromyalgia Syndrome

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Summary:

Background: Fibromyalgia syndrome (FMS) is a very common cause of multiple regional musculoskeletal (MSK) pain and disability; it is characterized by chronic widespread for at least three months and tender points identified by the American Collage of Rheumatology (ACR).The cause of FMS is currently unknown. However, several hypotheses have been developed including genetic predisposition. This study aims to evaluate the contribution of serum lipid profile to the pathophysiology of FMS.

Patients & Methods: The study has included 160 patients with FMS with age range (18-72) years and 60 control individuals who were age and sex matching with FMS patients: 29 patients with chronic musculoskeletal complaints but without FMS and 31 healthy controls. Colorimetric method was used to determine serum lipid profile. Results were evaluated using descriptive and inferential statistics; data were expressed as (mean ± SEM). P value of <0.05 was accepted as significant.

Results: There were no significant differences among the three subject groups in serum lipid profile.

Conclusion: Lipid profile has no role in FMS patients as a cause or result of this syndrome.

Key words: Fibromyalgia, lipid profile, Demographic analysis.

Introduction:

Biochemical approaches are often fundamental in illuminating the cause of disease, and designing appropriate therapies (1). Among these diseases a very common cause of multiple regional musculoskeletal (MSK) pain and disability; that is "Fibromyalgia syndrome" (FMS) (2). FMS is characterized by strong female predominance with peak incidence at ages (20-60) years old, it has been observed in up to 15% of rheumatology patients and 5% of patients from a general medical practice (2,3). FMS is characterized by chronic widespread pain for at least three months and tender points identified by the American Collage of Rheumatology with associated symptoms including stiffness, fatigue, sleep disturbance, emotional distress and functional impairment with evidence of pain amplification (4-6). Although several hypotheses have been developed; the cause of FMS is currently unknown (7).

Subjects & Methods:

This is a cross-sectional study, which was carried on 122 FMS patients who have attended the out patient clinic in Medical City – Baghdad Teaching Hospital – Rheumatology & Rehabilitation Consultation Unit during the period from April 2008 to February 2009. These 122 patients (101females+21males); with age range (18-72) years (*FMS (+) patients group);* fulfilled ACR 1990 criteria for the diagnosis of FMS and 60 control individuals (48females+12males), who were age and sex matching with FMS (+) patients: 29 patients (25females+4males) with chronic musculoskeletal complaints but without FMS (RA + OA + SLE) (*FMS (-) patients control group)* and 31 healthy volunteers (23females+8males) without musculoskeletal complaints *(healthy control (HC) group).* From each subject the medical and social history was taken along with special set of rules that deem certain epidemic and clinical related variables.

Criteria of exclusion have included Diabetes mellitus (DM), Sleep apnea, Hypercortisolism, Thyroid problems, and other rheumatic disorders.

The anthropometric tests (to evaluate body mass index 'BMI') were performed in the out patient clinic in Medical City – Baghdad Teaching Hospital – Rheumatology & Rehabilitation Consultation Unit, and, the biochemical tests were done in Medical City – Teaching Laboratories and the College of Medicine – Department of Physiological Chemistry.

Disposable plastic syringes of (23 G) needles were used to aspirate five milliliters of venous blood from each patient and control after (12-16) hours fasting from 08.00 a.m. to 12.00 a.m.

Serum total cholesterol (TC) and serum triglycerides (TG) were determined by enzymatic colorimetric test with lipid clearing factor (a lipoprotein-metabolizing). Kit used was from HUMAN-(CHOLESTEROL liquicolor, CHOD-PAP-Method)-Germany and HUMAN-(TRIGLYERIDES liquicolor ^{mono}, CPO-PAP-Method)-Germany respectively. Serum HDL-C was determined by enzymatic colorimetric test after

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precipitation. Kit used was from HUMAN-(HDL cholesterol)-Germany.

Serum LDL-C level was calculated from TC, HDL-C and TG levels according to Friedewald equation.

$$\text{LDL-C} = \text{TC} - (\text{HDL-C} + \frac{\text{TG}}{5}) \quad [\text{mg/dl}]$$

Results were evaluated using descriptive and inferential statistics; data were expressed as (mean ± SEM). Chi-Square (χ^2), Student test (t-test), ANOVA & LSD test (F-test), and Person correlation (r) were used to accept or reject the statistical hypotheses. All the statistical analyses were done by using Pentium-4 computer through the SPSS program (version-10) and Excel application. P value of <0.05 was accepted as significant.

Results:

Many parameters have been measured in this study; we have performed a demographic analysis for the subjects. Lipid profile test was done for all subjects. There was a highly significant difference in age within FMS (+) group (P = 0.00). There was no significant difference in age among the three groups: FMS (+), FMS (–), and HC (P = 0.169) (Table-1).

The (mean ± SEM) values of age for the three groups FMS (+), FMS (–), and HC were: (39.95 ± 1.10), (40.93 ± 2.40), (42.81 ± 2.16) respectively. There were no significant differences in age among the three groups: FMS (+), FMS (–), and HC (P = 0.42).

The number of females in FMS (+) group was 101 (82.8%) vs. 21 (17.2%) males while in FMS (–) group was 25 (86.2%) vs. 4 (13.8%) males, and in HC group was 23 (74.2%) vs. 8 (25.8%) males. There was a highly significant difference in gender within FMS (+) group (P = 0.00). There was no significant difference in gender among the three groups: FMS (+), FMS (–), and HC (P = 0.434).

Obesity was expressed by body mass index (BMI) a simple anthropometric measure that provides a marker of nutritional status, which has been calculated by the equation:

$\text{BMI} = \text{Kilograms} / \text{meters}^2$

BMI was divided into four classes, and, the body weight was classified according to them:

BMI	Classification
< 18.5	Lean
8.5 – 24.9	Normal weight
25.0 – 29.9	Overweight
> 30	Obese

As observed from (Table-1); the majority of FMS (+) patients in our study were obese. There was a highly significant difference in BMI within FMS (+) group (P = 0.00). There was no significant difference in BMI among the three groups: FMS (+), FMS (–), and HC (P > 0.05).

Table-1: Age and BMI Distribution in the Study.

Parameters	FMS (+) (n = 122) n (%)	FMS (–) (n = 29) n (%)	HC (n = 31) n (%)	P-value	Sig.
Age (y) <20 20-40 41-60 61-80	 5 (4.1%) 52 (42.6%) 61 (50%) 4 (3.3%)	 – 9 (31%) 16(55.2%) 4 (13.8%)	 – 12(38.7%) 18(58.1%) 1 (3.2%)	0.169	NS
BMI (kg/m^2) Lean Normal weight Overweight Obese	 2 (1.6%) 35 (28.7%) 41 (33.6%) 44 (36.1%)	 – 8(27.6%) 11(37.9%) 10(34.5%)	 1 (3.2%) 8 (25.8%) 10(32.2%) 12(38.7%)	0.102	NS

The (mean ± SEM) values of BMI of the three groups FMS (+), FMS (–), and HC were: (28.53 ± 0.56) Kg/m^2, (29.11 ± 1.05) Kg/m^2, (27.64 ± 0.89) Kg/m^2 respectively. There was no significant difference in (mean ± SEM) values of BMI among the three groups: FMS (+), FMS (–), and HC (P = 0.21).

The number of smokers in FMS (+) group was 34 (27.9%) while in FMS (–) group was 5 (17.2%), and in HC group was 3 (9.7%). There was a highly significant difference in smoking within FMS (+) group (P = 0.00). There was no significant difference in the smoking among the three groups: FMS (+), FMS (–), and HC (P = 0.072).

Table-2 has revealed the (mean ± SEM) values of lipid profile of the three groups in the study. All of them were within normal values. There was no significant difference in lipid profile among the three groups: FMS (+), FMS (–), and HC (P > 0.05) in all parameters except for HDL-C.

Table-2: Statistical Data for Lipid Profile

Parameters (mg/dl)	FMS (+) (n = 122) Mean ± SEM	FMS (–) (n = 29) Mean ± SEM	HC (n= 31) Mean ± SEM	P-value	Sig.
TC	176.28 ± 2.99	180.48 ± 5.24	176.71 ± 5.97	0.819	NS
TG	139.53 ±5 .56	126.34 ± 8.06	130.61 ± 12.33	0.062	NS
HDL-C	55.45 ± 1.06	62.79 ± 2.40	65.39 ± 1.98	0.000*	HS
LDL-C	104.25 ± 2.65	99.52 ± 6.07	93.90 ± 5.37	0.210	NS
VLDL-C	27.71 ± 1.11	25.24 ± 1.61	26.10 ± 2.46	0.077	NS

*** Significant Differences between FMS (+) and each of FMS (–) (P < 0.004) and HC (P < 0.004).**

Discussion:

To our knowledge, this is the first study examining the relationship between FMS and Lipid profile in Iraq. Most people are diagnosed during middle age, but the incidence may increases earlier than this age because in their 20s and 30s they might be more

susceptible to pain than those in their 40s to 60s due to several factors. They are dealing with the stress brought on by a chronic physical problem at the same time they are trying to accomplish the time-consuming tasks of young adulthood and establishing themselves in a career (8). The mechanisms of gender differences in FMS are not fully understood, but are likely to involve an interaction between biology, psychology, and sociocultural factors (9). FMS is much more common in women than in men. As estimated about 80% of suffers are women. Pain severity, global severity and physical functioning were not significantly different between the sexes, nor were psychological factors, e.g., anxiety, stress, and depression (10). Obesity is one of FMS risk factors (11,12). On the other hand FMS symptoms discourage exercise, thus contributing to a sedentary lifestyle that results in an elevated BMI; which has serious health problems as well as endocrinological ramifications due to the impact of obesity on the somatotrophic axis (13). Previous studies have found that more than 24% of FMS patients are obese; furthermore, it has been shown that a 5% loss of body weight results in a mild improvement of FMS symptoms (14). Smoking is detrimental for FMS patients for several reasons. Smoking reduces blood-oxygen content and impairs the already compromised muscle oxygenation further. Nicotine is a potent muscle contractor and aggravates muscle tension and spasm, leading to increased pain. It is also stimulant and increases the mental tension, which in turn intensifies pre-existing muscular tension (15). Nicotine was thought to increase pain intensity by increasing substance P in the cerebral spinal fluid, which helps transmit pain signals. At the same time, smokers have lower endorphin levels (natural pain killers). In addition, the connection between smoking and personality disorders may render FMS pain worse (16). A number of studies have suggested that there is an association between hyperlipidemia and MSK manifestations (17-19). In these studies, most of the patients had myalgia and arthralgia, tendo Achilles tendinitis, oligoarthritis or migratory polyarthritis, which are all associated with hyperlipidemia. However, the pathogenesis of the MSK system manifestations in hyperlipidemia is not fully understood (17,18). In the literature, musculoskeletal changes, which were mentioned; are not chronic pain syndromes such as FMS. On the other hand; the lipid levels determined in these studies were well above the upper ranges found in our study. Some drugs could potentially affect lipid and lipoprotein levels (20-22), and because of this many patients in our study who had been previously diagnosed and were using these drug types were excluded. In our study 15 (12.3 %) FMS patients interestingly their TC levels were well above 200 mg/dl. Reduced physical activity caused by many painful conditions may be a confounding factor in terms of the measured serum lipids. Accordingly, most of FMS patients in the present study were sedentarily housewives who lead sedentary lives and had similar daily physical activities. The etiology of the chronic pain syndromes has not been completely clarified. The typical features of these syndromes are painful tender points in muscles and tendon insertions (23).

Conclusion:

Lipid profile has no role in FMS patients as a cause or result of this syndrome.

References:

1. Doherty M, Lanyon P, Ralston S. Musculoskeletal disorder. In: Boon N, College N, Walker B, Hunter J. (Eds.) Davidson principle and practice of medicine, 20th ed. Churchill Livingstone, 2006; 1065-1144.

2. Sellami M, Guermazi M, Ghrobi S, et al. Validated translation in Arab of a questionnaire to screen fibromyalgia patient. Ann Rheum Dis: 21st EULAR Congress 2006:65:560.

3. Malucci Cerinic M, Zoppi M, Taib C, et al. Prevalence of fibromyalgia in Italy: uptated results. Ann Rheum Dis: 21st EULAR Congress 2006:65:557.

4. Baker K, Barkhuizen A. Pharmacologic Treatment of Fibromyalgia. Current Pain and Headache Reports 2005, 9:301–306.

5. Yunus M, Masi A, Calabro J et al. Primary fibromyalgia (fibrositis): clinical study of 50 patients with matched normal controls. Semin Arthritis Rheum 1981; 11:151-171.

6. Theadom A, Cropley M, Humphrey K. Exploring the role of sleep and coping in quality of life in fibromyalgia. J Psychosom Res 2007; 62:145–151.

7. Geenen R, Jacobs J. Fibromyalgia: diagnosis, pathogeneses, and treatment. Curr Opin Anesthesiol 2001; 14:533-9.

8. Burckhardt C, Clark S, Bennett R. Pain coping strategies and quality of life in women with fibromyalgia: Does age make a difference? J Musculoskel Pain 2001; 9: 2: 5-18.

9. Yunus M. Gender differences in fibromyalgia and other syndromes. J Gend Specific Med 2002; 5:42-47.

10. Yunus M. The role of gender fibromyalgia syndrome. Curr Rheumatol Rep 2001:3:128-34.

11. Holick M. Vitamin D: importance in the prevention of cancers, type 1 diabetes, heart disease, and osteoporosis Am J Clin Nutr 2004; 79: 362–371.

12. Kop W, Lyden A, Berlin A, et al. Ambulatory Monitoring of Physical Activity and Symptoms in Fibromyalgia and Chronic Fatigue Syndrome. Arthritis Rheum 2005; 52: 1: 296–303.

13. Thompson D, Lettich L, Takeshita J. Fibromyalgia: An overview. Curr Psychiatry Rep 2003; 5: 3: 211-217.

14. Saber A, Boros M, Mancl T, et al. The Effect of Laparoscopic Roux-en-Y Gastric Bypass on Fibromyalgia. Obes Surg 2008; 18: 6: 652-655.

15. Yunus M. Fibromyalgia and Overlapping Disorders: The Unifying Concept of Central

Sensitivity Syndromes. Semin Arthritis Rheum 2007; 36:6:339-356.
16. Weingarten T. Impact of Tobacco Use in Patients Presenting to a Multidisciplinary Outpatient Treatment Program for Fibromyalgia. In Clin J Pain 2009; 25: 1: 39-43.
17. Klemp P, Halland A, Majoos F, et.al. Musculoskeletal manifestations in hyperlipidemia: A controlled study. Ann Rheum Dis 1993; 52: 44-48.
18. Careless D, Cohen M. Rheumatic manifestations in hyperlipidemia and antihyperlipidemia drug therapy. Semin Arthritis Rheum 1993; 23: 90-98.
19. Seruthers G, Scott D, Bacon P, et.al. Musculoskeletal disorders in patients with hyperlipidemia. Ann Rheum Dis 1983; 42: 519-523.
20. Young D, Peterson C, Basch C, Halladay S. Effects of naproxen and nabumetone on serum cholesterol levels in patients with osteoarthritis. Clin Ther 1995; 17: 231-240.
21. Andrews J, Nemenoff C. Contemporary management of depression. Am J Med 1994; 97: 24S-32S.
22. Vinokur V, Gubacheulu M. The effect of antidepressants on lipid metabolism and the clinical course in IHD. Ter Arkh 1994; 66: 76-80.
23. Fricton J. Myofascial pain syndrome: Characteristics and epidemiology. In: Fricton J, Awad E. (Eds.) Advances in pain research and therapy. New York, Raven Press Ltd., 1990; 107-129.