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FREQUENCY OF DYSLIPIDEMIA IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS FROM A TERTIARY CARE CENTER IN KARNATAKA, INDIA

Physiology

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ABSTRACT

Systemic lupus erythematosus (SLE) is the prototypic autoimmune disease characterized by multisystem involvement and the production of an array of autoantibodies. So, the primary presentation is skin and joint involvement and may go upto multi organ failure.

MATERIALS AND METHODS:This was a prospective hospital-based study conducted by the Department of Physiology at the Bangalore Medical College and Research Institute, Karnataka, India from October 2018 to March 2019. Sample size was calculated using the prevalence of dyslipidemia among SLE patients reported by a group of researchers from a tertiary care center in Orissa. Statistical analysis was done using SPSS version 16. Quantitative variables were described as mean +/-SD unless otherwise indicated.

RESULTS:Based on the inclusion and exclusion criteria, 74 patients with diagnosed SLE and same number of age and sex matched controls were selected for the current study. The mean age of the patients was 32.3 years with a standard deviation (SD) of 7.3 years. A total of 48 patients of SLE evaluated during the course of the study were diagnosed to have dyslipidemia on laboratory investigation. It comprised of 64.9% of the case population.

CONCLUSION: SLE confers a massive coronary artery disease (CAD) risk, which is far greater than that associated with other autoimmune diseases.

KEYWORDS

Dyslipidemia, Systemic lupus erythematosus

INTRODUCTION

Systemic lupus erythematosus (SLE) is the prototypic autoimmune disease characterized by multisystem involvement and the production of an array of autoantibodies. These autoantibodies damages self tissues from all over the body and leads to varied presentation. The organ involved constitute mainly of skin, joints, kidneys, heart, lungs and central nervous system. So, the primary presentation is skin and joint involvement and may go upto multi organ failure. [1]

Coronary artery disease due to pre mature atherosclerosis that is pertaining to underlying lipid abnormalities or the high dose steroid therapy is one of the most common cause of mortality and morbidity in patients with SLE. [2] Studies across the globe has shown that this pre mature atherosclerosis increases the risk of myocardial infarction to 5-8 folds. [3] Some of the associated factors that alter the prognosis of CAD among SLE patients have been reported as female sex, younger age, shorter SLE duration and black/African American population. They also reported that SLE itself alters the prognosis and increases mortality among CAD patients. [4] The exact mechanism behind this increased risk of dyslipidemia though remain unclear, there are two most common postulates. One is related to active disease process itself where there are elevated triglyceride (TG) and low levels of high density lipoprotein cholesterol (HDL-C) in association with up regulation of tumor necrosis factor-α (TNF-α)/tumor necrosis factor receptor system, while the second is related to high dose steroid therapy and not related to activity of SLE. [5, 6,7]

Many studies have reported varying prevalence of dyslipidemia in SLE patients and it ranges from 30% to 73%. [8, 9, 10] Many a times, dyslipidemia in lupus patients often go under-recognized till they develop CAD with underlying atherosclerosis. So, the need to diagnosis any abnormality in the lipid profile in these patients at the earliest. So, it is important to have data on the magnitude of the problem. There is a dearth of study evaluating serum lipid profile in SLE patients in this part of the country. Hence, the present study was undertaken to determine the prevalence of dyslipidemia in patients with SLE.

MATERIALS AND METHODS

This was a prospective hospital-based study conducted by the Department of Physiology at the Bangalore Medical College and Research Institute, Karnataka, India from October 2018 to March 2019. Sample size was calculated using the prevalence of dyslipidemia among SLE patients reported by a group of researchers from a tertiary care center in Orissa.[10] Thus, taking p as 57.4% and a relative error of 15%, minimum sample size was calculated to 74. So, 74 diagnosed SLE patients, who visited in patient department of the hospital were included in the study. Total duration of study was 6 months, of which 3 months was invested for data collection (October to March). Prior to the data collection, clearance from the Institutional ethical committee was duly obtained. Study participants comprised of both genders and were aged more than 18 years of age. Normal age and sex matched individuals were enrolled as control. Exclusion criteria considered for the study were diabetes mellitus or hypertension, family history of dyslipidemia, cases of hypothyroidism. Habitual drinker of alcohol or smokers were also excluded. Patients taking any hypolipidemic drugs or those on gonadal hormones were excluded.

All the study participants were explained about the purpose of the study and assured of the confidentiality of the information shared. Duly signed informed consent forms in local language were obtained from all the study participants. Data collection was done by detailed history taking and laboratory investigation. Age and gender were recorded. Data on duration of SLE, and treatment history of SLE were obtained fromeach of the patients. Thenafter, they were called the following morning after overnight fasting to collect the sample for lipid profile. Height and weight were recorded to nearest whole numbers and Body Mass Index was calculated later on.

Statistical analysis was done using SPSS version 16. Quantitative variables were described as mean +/-SD unless otherwise indicated. Qualitative variables were described by percentage. Comparison between two groups was done by unpaired chi-square test. For all statistical tests, p value has been reported. P value less than 0.05 were considered to be significant. Results have been shown in tabular forms.

RESULTS

Based on the inclusion and exclusion criteria, 74 patients with diagnosed SLE and same number of age and sex matched controls were selected for the current study. The mean age of the patients was 32.3 years with a standard deviation (SD) of 7.3 years. The age range was 19 to 49 years. Table 1 shows the baseline characteristics of the patients. Maximum number of patients belonged to age group of 19-39 years (89%). The age of control group ranged from 19 to 46 years with a mean of 29.6 with a standard deviation (SD) of 8.2 years. Majority of the cases as well as the controls were females. So, there is a female preponderance in the natural history of SLE. Females constituted 90.5% of the cases selected for the study while in the control population, 93.2% were females. Mean Body Mass Index in kg/m^2 of the cases and the control were 21.7 (±1.9) and 20.9 (± 2.3), respectively. Background characteristics of the study population has been depicted in tabular form (Table 1).

A total of 48 patients of SLE evaluated during the course of the study were diagnosed to have dyslipidemia on laboratory investigation. It comprised of 64.9% of the case population. While in the control

population only 15 (20.3%) were found to have hypercholesterolemia. In the group with diagnosed cases of SLE, hypercholesterolemia was among 59.2% cases, hypertriglyceridemia was found in 62.4%, raised LDL-C in 27.2% cases. Status of cases and controls with abnormality in various aspects of lipid profile has been shown in Table 2.

To establish the association between SLE and dyslipidemia, all the various components of lipid profile were evaluated among the cases and controls separately and their p value have been reported in Table 2.

Table 1: Background characteristics of the study population

Characteristics	Cases	Control
Total number	74	74
Age (in years)	32.3 (± 7.3)	29.6 (± 8.2)
Gender Male	7 (9.5)	5 (6.8)
Female	67 (90.5)	69 (93.2)
Average BMI (in kg/m^2)	21.7 (±1.9)	20.9 (± 2.3)

Table 2: Table showing distribution of the study population according to their status of various aspects of lipid profile and its association with SLE

Lipid profile (mg/dl)	Cases	Controls	P value
Cholesterol	183.4 ± 32.7	151.2 ± 18.6	< 0.001
Triglycerides	172.7 ± 44.6	139.6 ± 32.5	< 0.001
HDL	44.1 ± 5.3	48.1 ± 6.5	< 0.001
LDL	116.0 ± 42.5	80.2 ± 21.7	< 0.001
VLDL	32.6 ± 4.2	22.9 ± 4.2	< 0.001

DISCUSSION

SLE confers a massive coronary artery disease (CAD) risk, which is far greater than that associated with other autoimmune diseases.[11] CAD associated with SLE is premature inonsetand this significantly affects morbidity and mortality in SLE. Some of the factors that plays a key role in occurrence of CAD in SLE patients are age, gender, arterial hypertension, dyslipidemia, and smoking. Disease activity and duration, antiphospholipid antibodies, C-reactive protein, and renal disease are the some of the important disease-related factors. [12]

In the current study, the mean age of the patients was 32.3 years with a standard deviation of 3.2 years, which signifies that the patients were comparatively younger as compared with the natural history of CAD in absence of SLE. The female population predominates the cases selected for the study. The dyslipidemia seen in conjunction with SLE is similar to that described in the general population in relation to CAD, with elevations in TG, LDL-C and TC and a fall in HDL-C levels.

Dyslipidemia was reported among 57.4% cases in our study which is similar to the resukts observed by some of the other Indian researchers.[10,13] Mean fasting serum cholesterol, TG and LDL-C levels were significantly higher in SLE patients when compared to control group. Similar pattern has been observed by some of the previous researchers. [10,13,14] This associated dyslipidemia may be attributed to intake of steroids and statins in SLE patients, which a classical immunocomplex mediated inflammatory disease. This inflammation modulates Lipoprotein Lipase (LPL) enzyme that is attributed to significant down-regulation of LPL activity induced by TNF-α, IL-1 and IFN-γ.[15] This acute phase response also promotes an altered hepatic synthesis of an array of proteins that are involved in lipoprotein metabolism, as well as the coagulation pathway and in the complement system. Hence, it is acceptable that the inflammatory conditions in SLE induce these alterations in the lipid profile. An enhanced production of these cytokines (IL6) is characteristic of SLE, particularly during active disease, and it supports their role in lupus dyslipoproteinemia. This high level of TNF-α in the circulation of SLE patients is correlated with active disease process and triglyceride levels. Thus, all the autoimmune diseases including SLE produce a wide range of autoantibodies. These in turn form complexes with the enzymes like lipoprotein lipase (anti-LPL). It hence hinders their process of catabolism, leading to alterations in the lipid profile of the patients. varieties of dyslipidemia and hence a cause of a cause for autoimmune hyperlipidemia.

CONCLUSION:

SLE confers a massive coronary artery disease (CAD) risk, which is far greater than that associated with other autoimmune diseases. CAD associated with SLE is premature inonsetand this significantly affects morbidity and mortality in SLE. Some of the factors that plays a key role in occurrence of CAD in SLE patients are age, gender, arterial hypertension, dyslipidemia, and smoking. Disease activity and duration, antiphospholipid antibodies, C-reactive protein, and renal disease are the some of the important disease-related factors.

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