DYSLIPIDEMIC FEMALES HAVE EQUAL RISK TO CARDIAC DISEASES AS MALES - A NEUTROPHIL MEDIATED PATHWAY.

N.KARTHICK 1, K.N.POORNIMA 1, K.DILARA 2, G.THILIP KUMAR 1, A.SARAVANAN 3
1. Tutor, Department of Physiology, SRM Medical college Hospital & Research centre, Kattankulathur, 2. Professor, Department of Physiology, Sri Ramachandra Medical College & Research institute, Porur., 3. Professor and Head, Department of Physiology, SRM Medical college Hospital & Research centre, Kattankulathur, E mail id: karthick.poornima@gmail.com, karthick.nedu@gmail.com

Received: 3 May 2014, Revised and Accepted: 30 May 2014

INTRODUCTION

Coronary artery disease (CAD) is the leading cause of death and disability among adult women accounting for deaths of 1 in 3 women [1]. 1 in 9 women is diagnosed with some form of cardiovascular diseases between the age group of 45-64 [2]. According to WHO, by 2013 it is expected that 23 million people will die for cardiovascular disorders annually, women being the maximum victims. A premenopausal woman who has a heart attack has twice the death rate of a similarly aged man. The association of smoking, hypertension, dyslipidemia and diabetes with CAD is well established. Dyslipidemia, one of the prime risk factor for CAD, induces neutrophilia by promoting proliferation, mobilization and differentiation of Haemopoietic stem/progenitor cell (HSPC) and thereby increasing granulocytes and monocytes [3]. Increase in neutrophil and monocyte recruitment promotes early atherogenesis in the vessels and increases the risk of CAD in dyslipidemic individual. The excess CAD mortality among Asian Indians is greater in women than in men [4]. Traditionally epidemiological studies of CAD have been focused more on men therefore the present study aims to determine the effect of dyslipidemia on neutrophil count in both male and female dyslipidemic individuals.

MATERIALS AND METHODS

49 subjects with dyslipidemia defined by elevation of plasma cholesterol, triglycerides (TGs) or both, or a low high density lipoprotein level and 49 healthy age and sex matched subjects voluntarily attending our master health checkup clinic were enrolled in this study. Both control and dyslipidemia group contains 20 females and 29 males each.

Table 1 shows the comparisons of lipid parameter between the dyslipidemic subjects and the control subjects. The cholesterol, Low density lipoprotein (LDL) and cholesterol – High density lipoprotein ratio were significantly higher in group 1 when compared to group 2, hence group 1 called as dyslipidemic subjects and group 2 called as control subjects. The Triglyceride and High density lipoprotein (HDL) did not show any significant difference.

Table 2 shows the comparisons of Neutrophil between the dyslipidemic and the control group. Neutrophil were significantly higher in group 1 when compared to group 2.

RESULTS

Handling and storing of blood samples was done as per criteria furnished by national committee for clinical laboratory standard (NCCLS). Blood was collected after 12 hours fasting, in the morning between 8 – 9 am. Blood samples were collected in two tubes by observing universal precaution for venipuncture. To the first tube 2ml of blood was transferred and the blood was allowed to clot for serum separation. Serum was separated and stored at -70c until assay. SIEMENS kit was used for the analysis lipid profile. The second tube containing the anti-coagulant EDTA, 5ml of blood was added to that tube for the analysis of hematological parameters such as RBC, total and differential count of WBC, Hb, PCV and platelets and they are estimated by automatic cell counter. The study was approved by the institutional ethical committee.

Data are presented as the mean ± SD. Statistical analysis was performed by an IBM computer with the use of statistical package of social science (SPSS) ver. 21.0. Independent t test was used to showing differences between dyslipidemia group and controls. The level of significance was set at p < 0.05.
thanked with many factors such as obesity, family history, or hypertensive mice display reduced plaque sizes at early stage. In our study we be correlated with adverse cardiac events [13]. Furthermore these matrix degradation such as elastase, metalloproteinase are found to coronary diseases. HNPs form complexes with LDL and increases Human neutrophil peptides are markedly increased in acute neutrophils is associated with increased risk of cardiac events [11]. Myeloperoxidase, the most abundant protein in endothelium. Myeloperoxidase, the most abundant protein in chronic inflammation due to both loss of endothelial integrity and early atherogenesis and histopathologic features of rupture prone cardiovascular system as neutrophilia is found to be associated with neutrophilia [7]. Increase in LDL, cholesterol or triglycerides causes HSPC differentiation toward atherogenic associated with neutrophilia [7]. Increase in LDL, cholesterol or triglycerides causes HSPC differentiation toward atherogenic diabetes, dyslipidemia, hypertension, etc., our study suggests that the neutrophilia should be viewed as an independent risk factor for CAD in dyslipidemic individuals. Improved understanding of gender differences in clinical presentations, symptoms, treatment and outcome of CAD is needed. Future studies can be done to find out the possible therapeutic intervention to block the mechanism of dyslipidemia in proliferation of HSPCs.

**DISCUSSION**

Dyslipidemia is elevation of plasma cholesterol, triglycerides (TGs) or both or a low HDL, that contributes to the development of atherosclerosis. Dyslipidemia may be one of the first clues to the diagnosis of various diseases including CAD, subclinical hypothyroidism etc. [5, 6] and dyslipidemia is found to be associated with neutrophilia [7]. Increase in LDL cholesterol or triglycerides causes HSPC differentiation toward atherogenic monocyes and neutrophil [1, 8]. In our study we found that there is positive correlation between lipid levels and neutrophil count. As the lipid level increases there is increase in neutrophils. Dyslipidemia combined with neutrophilia impose a greater risk on cardiovascular system as neutrophilia is found to be associated with early atherogenesis and histopathologic features of rupture prone lesions [9]. The underlying pathogenesis of CAD is characterized by chronic inflammation due to both loss of endothelial integrity and sub endothelial retention of LDL. [10]. The mechanism of atherosclerosis in neutrophilia is mainly due to granules secreted and released from neutrophil which causes loss in integrity of endothelium. Myeloperoxidase, the most abundant protein in neutrophils is associated with increased risk of cardiac events [11]. Human neutrophil peptides are markedly increased in acute coronary diseases. HNPs form complexes with LDL and increases LDL binding to the endothelial surface which leads to endothelial dysfunction [12]. Other neutrophil specific proteases which link to matrix degradation such as elastase, metalloprotease are found to be correlated with adverse cardiac events [13]. Furthermore these granule proteins have shown to play a role in recruitment of inflammatory monocytes [14]. Studies have shown that neutrophenic mice display reduced plaque sizes at early stage. In our study we have found that there is no significant difference found in neutrophil count between male and female dyslipidemic subjects. Hence dyslipidemic females have equal risk to cardiac diseases as male. So, this proves that dyslipidemia mediated neutrophilia is an additional risk factor for CAD.

**REFERENCES**


