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EVALUATION OF OXIDATIVE STRESS AND DYSLIPIDEMIA IN DIAGNOSED HYPERTENSIVE PATIENTS

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ABSTRACT : Hypertension is the most prevalent chronic disorders in different ethnic populations. Hypertension and dyslipidemia are the independent risk factor for cardiovascular disease (CVD), which is the leading cause of morbidity and mortality worldwide. This study aimed to investigate the status of oxidative stress marker Malondialdehyde (MDA) and dyslipidemia in diagnosed hypertensive patients. In this case-control study, a total of 60 subjects (30 Hypertensive patients and 30 healthy controls) aged between 30-60 years were recruited. The plasma MDA levels were estimated in all subjects along with serum lipid profile; Total Cholesterol (TC), Triglyceride (TG), High-Density Lipoprotein-Cholesterol (HDL), Low-density Lipoprotein-Cholesterol (LDL), Very Low-density Lipoprotein-Cholesterol (VLDL) by using commercially available kits. All the data were compared between the two groups by using the unpaired t-test. Pearson correlation coefficient was calculated among hypertensive patients. A P value <0.05 was considered as statistically significant for all data analyzed. The mean age of case and control was found 53.77±10.07 and 34.40±3.28 years, respectively. The mean level of TC, TG, LDL, VLDL, and MDA was found significantly higher in hypertensive patients than healthy controls (P<0.001, 0.02, <0.001, 0.01, <0.001, respectively). However, the mean level of HDL was not found significantly different between hypertensive patients and healthy controls (P=0.09). A significant positive correlation was found between TC and LDL among hypertensive patients (r=0.92, P<0.01). In the same way, a significant positive association was found between TG and VLDL among hypertensive patients (r=0.99, P<0.01). However, there were no significant correlations found between MDA and lipid profile parameters TC, TG, HDL, LDL and VLDL among hypertensive patients. This study demonstrated that the patients with hyper-tension are more likely to exhibit dyslipidemia and oxidative stress including elevated MDA, TC, TG, LDL, VLDL and reduced HDL cholesterol levels than healthy controls. Results suggested that elevated blood pressure may predict certain disturbances in lipoprotein metabolism. Prevention strategies of hypertension and dyslipidemia may play a major role to reduce the prevalence of CVD. Further study is warranted.

Key words : MDA, oxidative stress, lipid profile, hypertension, dyslipidemia, CVD.

INTRODUCTION

Elevated blood pressure contributes to the leading risk factor for morbidity and mortality in India. Hypertension is contributed to 10.8% of all deaths in India (ICMR, 2018). It is contributed to more than 12% of global deaths (GBD, 2016). Several studies found the discrepancy in the prevalence of hypertension in Indian population. The Fourth National Family Health Survey reported that the overall prevalence of hypertension in India is 11.3% (NFHS-4). However, the Fourth District Level Household Survey reported that the overall prevalence of hypertension in India is 25.3% (Gupta *et al*, 2018). In addition, a study reported that about 33% of urban and 25% of rural Indian population having hypertension (Anchala *et al*, 2014).

Oxidative stress has been identified an important factor associated with hypertension (Montezano *et al*, 2012; Rodrigo *et al*, 2011; Baradaran *et al*, 2014). It is reported that atherosclerosis strongly linked with oxidized LDL-cholesterol (oxLDL) in elderly persons (Gradinaru *et al*, 2015). Various studies showed that oxLDL and higher arterial stiffness have a significant association and both are independent risk factors for CVD (Brinkley *et al*, 2009). A study reported that plasma oxLDL levels increases in women after the age of 50 years. In elderly individuals, the increase of oxLDL may amplify LDL atherogenicity because of the prooxidant and proinflammatory environments (Gradinaru *et al*, 2015; Liguori *et al*, 2018). In addition, Dyslipidemia and hypertension are two widely recognized independent key risk factors for the development of CVD (Brunelli *et al*, 2017) summarized in Fig. 1.