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SERUM LIPID PROFILE AND NUTRIENT ANTIOXIDANTS IN HYPERTENSIVE PATIENTS OF SOUTHERN RAJASTHAN

Biochemistry

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ABSTRACT

Hypertension is a major health problem both in developed as well as developing countries with a common end result of elevated blood pressure and a silent killer. And it often goes unnoticed for prolonged periods resulting ultimately in many vascular complications. It is hypothesized that that high blood pressure is pathological state associated with loss of balance between peroxidation and antioxidant and multi factorial events participate in development of pathology.

KEYWORDS

Cholesterol, Cardiovascular Diseases, Lipids, Hypertension, Triglycerides

Introduction:-

Hypertension is the most common of the cardio-vascular diseases which is the leading cause of morbidity and mortality in the industrial world as well as becoming an increasing common disease in the developing countries (World Health organization, 1978). Hypertension is defined as SBP level higher than 140 mmHg and DBP higher than 90mmHg. Hypertension is characterized by abnormality of cardiac output systemic vascular resistance and arterial compliance. Approximately 25% of adult populations are affected (Giles TD *et al*, 2005). Hypertension is one of the ten leading reported causes of death and about4% deaths were due to hypertensive complication (Akuyam SA *et al*, 2009).Untreated hypertension is notorious for increasing the risk of immortality and is often described as a silent killer. Mild to moderate hypertension, if left untreated, is associated with a risk of atherosclerotic disease in 30% of people and organ damage in 50% of people after only 8-10 years of onset (Webster J *et al*, 1993).

Several risk factors are known to play a role in the progress of hypertension (Leone A, 2011). In an investigation on the different genetic and environmental risk factors of hypertension, Ruixing showed that age, hyperlipidemia, alcohol consumption, high Body Mass Index (BMI), and sodium intake were associated with hypertension (Ruixing *Y et al*, 2008). Several previous studies showed the relation between hyperlipidemia and hypertension (Hansen HS *et al,* 2009 and Feldstein CA. 2010). An excessive daily intake of saturated fats, cholesterol, and other sources of calories and subsequent disturbance of lipid profile leading to hypertriglyceridemia and hypercholesterolemia are associated with obesity and, consequently, hypertension (Kotsis V *et al*, 2010).

Impaired insulin function (Pastucha D *et al,* 2010), increased peripheral resistance, cardiac output, sympathetic tone, and salt congestion (Hall JE *et al,* 1999) are some of the responsible mechanisms which lead to hypertension. While the relation between hyperlipidemia and hypertension is clearly shown, there are only a few studies which have compared the lipid profiles of hypertensive and non-hypertensive cases.

Our study was designed to compare the serum triglyceride, cholesterol, HDL, and LDL and nutrient antioxidant levels in hypertensive and non-hypertensive individuals.

Material and Methods:-

The present case control study was carried out in the Department of Biochemistry, RNT Medical College, Udaipur (Rajasthan) Patients and controls were selected from Medicine outdoor of MB Govt. Hospital, RNT Medical College, Udaipur. Case history in detail was recorded on a proforma. An informed consent was taken from all the healthy control subjects and patients, under study apprising them the nature and objective of the study.

The subjects' blood pressures were measured according to the guidelines introduced by the World Health Organization (WHO). Patients with systolic blood pressure above 140 mmHg or diastolic blood pressure above 90 mmHg, or the ones receiving antihypertensive medications were considered as hypertensive.

The selected subjects were grouped as:-

Control group:- (n=133)

Age matched healthy controls, with no signs and symptoms of hypertension and other diseases were included. The selected control subjects were healthy family members, staff members and attendants of patients visiting M.B. Govt. Hospital and RNT Medical College, Udaipur.

Case group:- (n=87)

It includes the clinically established patients of hypertension.

Exclusion Criteria:-

1. Non willingness for participation.
2. Those suffering from complications of hypertension.
3. Those on lipid lowering drugs.
4. Known cases of renal failure, thyroid disorder and familial dyslipidemia.

Inclusion Criteria:-

1. Diagnosed cases of hypertension.
2. Patients on anti hypertensive medications.
3. Known cases of Diabetes mellitus.
4. Patients with concurrent history of IHD.
5. Smokers, alcoholics were also included.

Collection of blood samples of patients and control:-

10 ml of blood from the control, as well as the study group was drawn from antecubital vein and was collected in plain vials. Serum was separated by centrifugation of blood samples and Vitamin E, ascorbic acid, Total Cholesterol, Triglycerides, HDL- Cholesterol were estimated. Fasting blood sugar (FBS) was estimated from the fluoride vial.

Analysis of blood for various analytical parameters:-

1. Ascorbic Acid (Natelson, 1971).
2. α Tocopherol (Baker and Frank, 1968).
3. Blood Glucose (Trinder P, 1969). Estimated by enzymatic glucose kit (GOD/POD method).

Enzymatic kit methods are available for in vitro quantitative estimation of:-

A. Total Cholesterol (Allain C.C, *et al*., 1974).
B. Triglyceride (Foosati *et al*., 1982).
C. HDL- Cholesterol (Isezaki *et al*., 1996).

LDL- Cholesterol estimation (Friedwald, 1972).
LDL- Cholesterol = (Total Cholesterol)- T.G./5 – (HDL – Cholesterol).
VLDL – Cholesterol estimation (Friedwald, 1972) T.G./5

Classification

This was done on the basis of the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III).

Elevated Total Cholesterol	>200 mg/dl
Elevated Triglycerides	> 150 mg/dl
Elevated LDL - C	> 130 mg/dl
Low HDL - C	< 40 mg/dl

Total Cholesterol/HDL-Cholesterol Ratio And LDL-Cholesterol / HDL-Cholesterol Ratio (Castelli et al, 1977).

Coronary heart disease (CHD) risk factor can be calculated using total lipid profile as suggested by Castelli et al. The risk factor gives the most accurate and definite assessment of heart disease risk.+

Risk	Total / HDL-Cholesterol Ratio		LDL/HDL-Cholesterol Ratio	
	Men	Women	Men	Women
½ Avg.	3.43	3.27	1.00	1.47
Avg.	4.97	4.44	3.55	3.22
2xAvg.	9.55	7.05	6.25	5.03
3 x Avg.	23.99	11.05	7.99	6.14

P Value < 0.05 was considered as significant.

Result and Discussion:-

Table 1.0 Serum Lipid Profile and Nutrient Antioxidant Levels in Hypertensive and Controls

Parameters	Control (n=133)		Hypertensive (n=87)		t-value	P-value
	Mean	±SD	Mean	±SD		
Total Cholesterol (mg/dl)	161.90	39.16	190.13	54.28	4.19	< 0.001
Triglyceride (mg/dl)	102.55	36.63	217.60	84.35	12.00	< 0.001
HDL-Cholesterol (mg/dl)	45.88	8.07	40.87	6.90	4.92	< 0.001
LDL-Cholesterol (mg/dl)	92.90	29.7	104.96	32.38	2.79	< 0.001
VLDL-Cholesterol (mg/dl)	20.37	7.36	40.93	16.47	10.95	< 0.05
Total Cholesterol/HDL Cholesterol ratio	3.55	0.94	4.86	1.18	8.70	< 0.001
LDL Cholesterol /HDL Cholesterol ratio	2.07	0.70	2.86	0.83	7.33	< 0.001
Vitamin E	1.14	0.23	0.80	0.19	9.12	< 0.001
Vitamin C	1.03	0.27	0.81	0.33	5.19	< 0.001

The mean levels of total cholesterol in males and females were 188.27±34.60 and 195.58±46.94 mg/dL respectively, when compared to control, significantly higher values were observed in patients (P<0.001).

Similarly, the triglycerides were significantly raised in hypertensive patients (P<0.001). HDL-cholesterol was significantly lower, thereby intuitively suggesting that LDL-cholesterol were significantly higher (P<0.001). Consequently the total cholesterol, HDL-cholesterol ratio and LDL/HDL cholesterol ratio were also higher in patients, whereas defense system i.e. Vitamin E and C were significantly lower in all the group.

These observations further attest the finding of earlier worker (Hendre *et al*, 2006; Relan *et al*, 2004; Vasdev *et al*, 2003; Srinivas *et al,* 2000). The decreased levels of plasma ascorbic acid is related to its antioxidant property where it gets utilized. Ascorbic acid influences prostaglandin production, which in turn effects blood pressure. PGI_2 are vasodilators and are therefore hypotensive prostaglandins and are synthesized from polyunsaturated fatty acids and are susceptible to autooxidation. Antioxidant Vitamin C prevents the auto-oxidation of lipids that may have hypertensive effect.

Franco *et al,* 2003 found that decreased plasma ascorbic acid concentration of 6 Keto prostaglandin –F-1. This observation supports the theory that, dietary antioxidant enhances the production of prostacylin by scavenging the free radicals and peroxides, which inhibits the prostacyclin synthatase at concentration above a certain threshold, that could explain the lowered levels of vitamin C in hypertension.

Vitamin E is the major chain breaking antioxidant and is considered as first line of defense against lipid peroxidation. Increased levels of lipid peroxidation products and lipofuscin pigment results in inactivation of prostacylin and nitric oxide, hence an enhancement of peripheral vascular resistance and an increase in hypertension (Srinivas, 2000). No difference was observed between rural and urban, vegetarian and non-vegetarian and with and without family history. Smokers and alcohol consumers had lower levels of Vitamin E (P<0.01) (P<0.05).

Table 1.1 Serum Lipid Profile and Nutrient Antioxidant Levels in Diabetic and Non Diabetic Hypertensive

Parameters	Non Diabetic (n=60)		Diabetic (n=27)	
	Mean	±SD	Mean	±SD
Total Cholesterol (mg/dl)	184.26	38.43	197.14	43.11
Triglyceride (mg/dl)	191.39	80.11	247.28	78.27
HDL-Cholesterol (mg/dl)	41.01	8.03	37.71	8.92
LDL-Cholesterol (mg/dl)	102.94	26.45	111.24	33.24
VLDL-Cholesterol (mg/dl)	38.48	12.26	43.14	13.51
Total Cholesterol/HDL Cholesterol ratio	4.13	1.24	4.62	1.34
LDL Cholesterol /HDL Cholesterol ratio	2.21	0.91	2.73	0.98
Vitamin E	0.88	0.19	0.76	0.24
Vitamin C	0.81	0.32	0.74	0.31

In diabetes and hypertensive patients triglyceride requires special mention because the triglyceride were very high (247.28±78.27 mg/dL) than in patients with hypertension alone (191.39±80.11 mg/dL). This observation partly explains as why patients with HT plus DM are at greater risk of developing IHD. Besides this being under treatment these patients did not had adequate control of sugar might be responsible for exaggerated lipid profile. These observations are in tune with the finding of other research workers (Relan *et al*, 2004; Srinivas, 2000).

Conflict of Interest:- The author(s) declare(s) that there is no conflict of interest regarding the publication of this paper.

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