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An Impact Of Diet On Oxidative Stress In Essential Hypertension

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Abstract: *Hypertension (HTN) is a major modifiable risk factor for Cardio Vascular Disease. Hypertension doubles the risk of cardiovascular diseases, including coronary heart disease (CHD), congestive cardiac failure (CCF), ischemic and hemorrhagic stroke, renal failure, and peripheral arterial disease. In India, the incidence of HTN occurs between 3rd and 6th decades of life. Also HTN leads to increased oxidative stress, in turn leads to atherosclerosis and dyslipidemia. Among the hypertensives, the oxidative stress is more marked in the non vegetarian groups. In this study, oxidative stress parameters serum uric acid and malon di aldehyde were estimated and was compared in both dietary groups. Routine investigations and the serum uric acid was estimated by standard kit method. Oxidative stress parameters (i.e) Plasma Malon dialdhyde (MDA) was estimated manually by Ester Bauer and Stein Berg method. The oxidative stress markers serum uric acid and MDA were markedly elevated in the non vegetarian groups and was positively correlated. In this study we found high serum uric acid, plasma MDA in both dietary groups but markedly elevated in non vegetarians than vegetarian subjects.*

Keywords: *HTN (Hypertension),MDA(Malan di aldehyde),CHD(coronary heart disease),CCF(congestive cardiac failure), LPO(lipid peroxidation).TC(Total cholesterol), TGL(Triglycerides)*

I. Introduction

Hypertension is one of the leading causes of the global burden of disease. Hypertension is sustained high blood pressure (≥140/90mmHg). Blood pressure itself is the pressure exerted by the blood on the walls of the blood vessels. Each time the heart beats (about 60-70 times a minute at rest), it pumps blood into the arteries. Blood pressure is at its highest when the heart beats, pumping the blood. This is called systolic blood pressure. When the heart is at rest, between beats, blood pressure falls. This is diastolic pressure[1].

Hypertension doubles the risk of cardiovascular diseases, including coronary heart disease (CHD), congestive cardiac failure (CCF), ischemic and hemorrhagic stroke, renal failure, and peripheral arterial disease .More than 25% of the adult population is affected by hypertension, and two thirds of those individuals reside in developing countries. The estimated total number of adults with hypertension in 2000 was 972 million. By 2025, the number of hypertension will increase by about 60% to a total of 1.56 billion as the proportion of elderly people will increase significantly[2].

The challenge to humanity is that out of all the hypertensive cases only 5-10% cases have definite cause and 90-95% cases are essential or primary or idiopathic hypertension i.e. for which nonspecific causes can be found.[3]In search for a causative factor for essential hypertension, uric acid and lipid peroxidation due to increased oxidative stress are considered. Increased vascular oxidative stress could be involved in the pathogenesis of hypertension , a major risk factor for cardiovascular disease mortality. In human, uric acid is the end product of purine metabolism and derives from the conversion of hypoxanthine to xanthine and of xanthine to uric acid, both reactions being catalysed by the enzyme xanthine oxidase.[4] Uric acid has been hypothesized to activate renin angiotensin system which can lead to injury to pre renal blood vessels. The increased oxidative stress and associated oxidative damage are mediators of renovascular injury in cardiovascular pathologies.[5],

Oxidative stress results from an imbalance between free radicals over-production and a lower degradation rate due to a decreased activity of endogenous defense systems.[6] These ROS damage all types of biomolecules especially lipids ie cholesterol, polyunsaturated fatty acids (PUFA) are a main target of oxidative attack and this leads to the formation and the accumulation of lipid per oxidation (LPO) products in particular oxysterols, hydroperoxides and endoperoxides.[7]

The link between hyper uricemia and essential hypertension may be due to lipid peroxidation.To assess the lipid peroxidation status in hypertensive patients, the breakdown products of lipid peroxides in serum or plasma, the malondialdehyde (MDA) is measured.[8]. In normal circumstances they are removed by the different scavenger systems present in blood and tissues. Significantly increased concentrations of malondialdehyde, is an index of lipid peroxidation, in hypertension and CHD patients.[9,10] On the other hand, changes in lifestyle includes a diet rich in sugar and high-fat processed foods and alcohol consumptison, obesity and sedentary behavior could also contribute to essential hypertension. Since the proportion of hypertensive people will increase dramatically worldwide, the prevention, detection, treatment and control of this condition should be a top priority.[2].

This study is designed to explain the role of oxidative stress in essential hypertension and also the impact of stress markers in essential hypertension.

II. Material And Methods

This study was carried out in the department of biochemistry, Vinayaka missions medical college & hospital, karaikal. The study encompasses 80 hypertensive cases and 51 age, sex matched normotensive controls. The following biochemical investigations were done on both cases and controls. The investigations are:

Estimated parameters.

1. Fasting Blood sugar-Glucose oxidase- Peroxidase method.
2. Serum Cholesterol-Cholesterol oxidase- Peroxidase method.
3. Serum Triglycerides-GPO-PAP method.
4. Serum HDL- cholesterol- Precipitating Reagent method
5. Serum urea- Berthelot method.
6. Serum creatinine-Jaffe's method.
7. Serum Uric acid- Uricase/ POD method.
8. Plasma MDA estimation by maual method-Esterbauer and Steinberg method 1989.[11]

III. Result And Analysis

The results obtained are presented as follows:

Table-1: Gender wise Distribution in the Subjects Studied

GROUPS	MALE	MEDIAN AGE	FEMALE
HYPERTENSIVE CASES(n=80)	49(61%)	48	31(39%)
CONTROLS (n=51)	30(59%)	46	21(41%)

The above TABLE shows in both the subjects, males were predominant. The mean age of hypertensives was around 48years. The mean age for controls was 46 years.

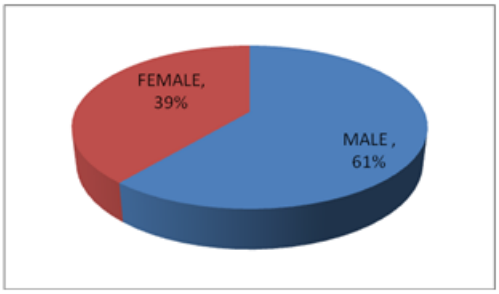

Figure 1: Gender wise Distribution of Cases

Figure 1 shows the gender wise distribution of study. Among the cases studied, 61% were males and 39% were females.

Table-2: Groups distribution based on Dietary Pattern;

DIETARY PATTERN	HYPETENSIVE (n=80)	NORMOTENSIVE (n=51)	X^2	p VALUE
NON VEG	53(66%)	17(33%)	3.925	0.04*
VEG	27(34%)	34(67%)		
TOTAL	80	51		

The TABLE 2 shows, out of 80 cases of hypertension, 53 (66%) cases were non vegetarian and 27(34%) were vegetarian. In controls, 34(67%) were non vegetarian and 17(33%) were vegetarian. By applying chi square, the dietary pattern in hypertension was statistically significant with p value <0.05.(* shows statistical significance)

Table-3: Routine Biochemical Parameters in Hypertensives and Controls:

PARAMETERS	HYPERTENSIVE CASES(n=80)	CONTROLS (n=51)	t value	p value
FASTING BLOOD SUGAR (mg/dl)	82.2 ± 8.7 (68-104)	79.7 ± 7.4 (68-93)	0.08	0.936(NS)
SERUM UREA (mg/dl)	27.1 ± 6.6 (16-42)	24.6 ± 5.3 (15-34)	0.015	0.98(NS)

SERUM CREATININE (mg/dl)	0.9 ± 0.1 (0.7-1.2)	0.8 ± 0.1 (0.7-1.1)	0.01	0.99(NS)

The TABLE 3 shows, the routine biochemical parameters in both groups studied. The fasting blood sugar, serum urea and creatinine in hypertensive cases and controls were observed within normal reference range and were not statistically significant ruling out diabetes, renal pathology.(NS- not significant)

Table- 4: Lipid Profile in Hypertensives and Controls

PARAMETERS (mg/dl)	HYPERTENSIVES(n=80) MEAN±SD	CONTROLS(n=51) MEAN±SD	t Value	p Value
TC(mg/dl)	203.1 ± 29.1 (150-285)	161.9 ± 24.6 (108-200)	2.421	0.01
TG(mg/dl)	203.2 ± 45.1 (128-340)	110.4± 27.6 (58-182)	2.400	**0.01***
HDL-c(mg/dl)	41.3 ± 5.5 (25-50)	51.3 ± 7.1 (32-63)	3.296	**0.001***
LDL-c(mg/dl)	120.8 ± 28.1 (54-198)	88.5 ± 24.8 (33-134)	4.529	**0.0001***
VLDL(mg/dl)	39.1 ± 9.0 (26-69)	22.1 ± 5.5 (12-36)	9.87	**0.0001***
N.HDL-c (mg/dl)	161.9 ± 28.8 (111-240)	110.6 ± 26.3 (53-161)	2.776	**0.006***

This table 4 shows the serum lipid profile in both the subjects. In the hypertensive cases, the serum TC, TG, LDL-c, VLDL-c and N.HDL-c were definitely on the higher side as compared to the controls who were in the normal reference range. The difference of these two subjects were statistically significant with p value 0.01, 0.01, 0.0001, 0.0001 and 0.006 respectively. The serum HDL-c was declined in cases as compared to controls and was statistically significant with p value of 0.001.(* shows statistical significance)

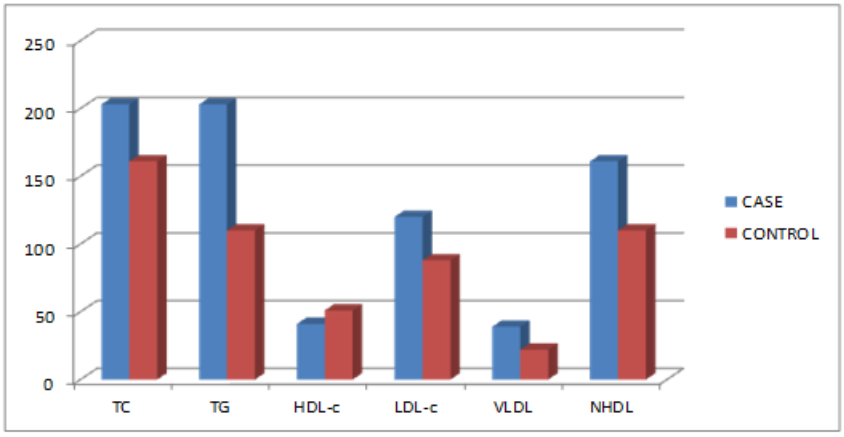

Figure 2: Lipid profile in hypertensives and controls

Fig.2 shows the lipid profile fractions TC, TGL, LDL-c, VLDL-c, non HDL-c were elevated in hypertensive cases compared to controls where lipid profile was within normal range

Table-5: Comparison of Oxidants in the Subjects Studied:

PARAMETERS	Cases(n=80)	Controls(n=51)	t Value	p Value
S.URIC ACID (mg/dl) MEAN ± SD	6.9±1.3	4.6±0.6	1.235	0.05
PLASMA MDA (µmol/l) MEAN±SD	5.7±1.4	3.0±0.6	5.694	**0.0001***

The TABLE 5, shows a comparison of oxidative stress parameters in both hypertensive and controls. The serum uric acid & the plasma MDA in the hypertensive were elevated as compared to controls and the difference was statistically significant with p value of 0.05, 0.0001 respectively.(* shows statistical significance)

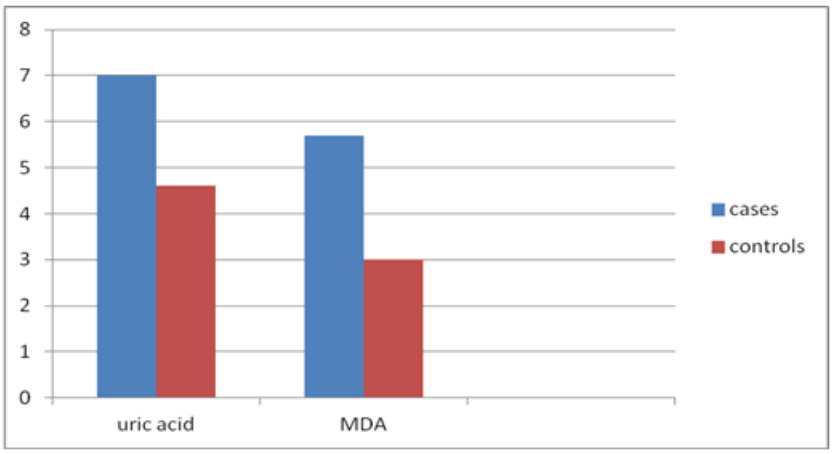

Figure 3: Comparison of Uric acid and MDA in cases and controls

The Fig.3 shows the oxidative stress parameters, the uric acid and MDA were elevated in cases than the controls.

Table-6: Comparison of Oxidative Stress parameters based on the Diet in both Subjects Studied.

PARAMETER	HYPERTENSIVE(n=80)			NORMOTENSIVE(n=51)		
	NON VEG (n=53)	VEG (n=27)	(n=80)	NON VEG (n=34)	VEG (n=17)	(n=51)
S.URIC ACID mg/dl	7.5 ±1.1	5.8 ± 0.9	6.9±1.3	4.4 ± 0.6	4.7 ± 0.5	4.60 ± 0.7
PLASMA MDA µmol/l	6.1 ± 1.3	4.9 ± 1.4	5.7±1.4	3.0 ± 0.6	3.0 ± 0.6	3.0±0.6

In the TABLE 6 shows the oxidative stress marker plasma MDA and serum uric acid are elevated in both non vegetarian and vegetarian group of hypertensive cases but markedly elevated in the non vegetarian group of hypertensive cases than the vegetarian groups. The control group shows within normal range in both dietary groups.

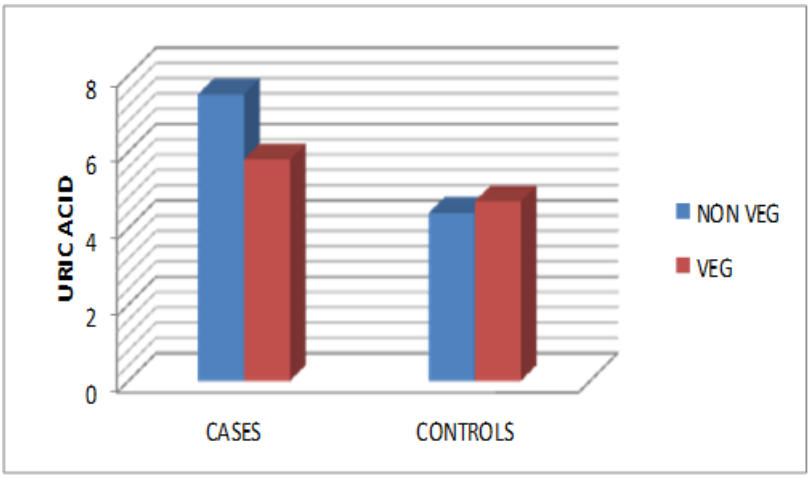

Figure 4: Comparison of uric acid based on diet in both cases and controls

The Fig.4 shows elevation of serum uric acid in the both non vegetarian and the vegetarian diet of cases. In controls both dietary groups have within normal range.

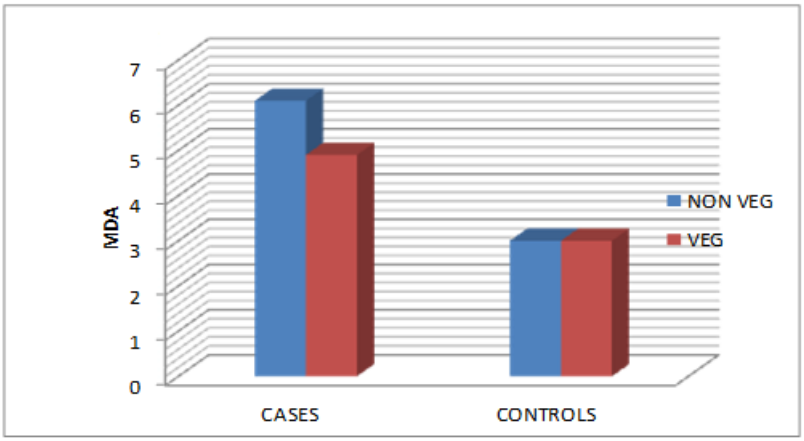

Figure 5: Comparison of MDA based on diet in both cases and controls

The Fig. 5 shows the marked elevation of plasma MDA in both non vegetarian and the vegetarian group of cases. In controls both dietary groups have within normal range.

Table-7: Comparison of Oxidative Stress Parameters Based on the Dietary Pattern in the Cases Studied.

PARAMETER	HYPERTENSIVE (n=80)	
	NON VEG (n=53)	VEG (n=27)
S.URIC ACID mg/dl (MEAN±SD)	7.5 ±1.1	5.8 ± 0.9
PLASMA MDA µmol/l (MEAN±SD)	6.1 ± 1.3	4.9 ± 1.4

In the TABLE 7, the oxidative stress marker plasma MDA(6.1 ±1.3) and serum uric acid(7.5 ±1.1) were markedly elevated in non vegetarian cases than the MDA(4.9 ± 1.4), serum uric acid (5.8 ± 0.9) of vegetarian of hypertensive cases. It strongly proves that the elevation of MDA, is due to increased oxidative stress in turn due to diet and more marked in the non vegetarian of hypertensive cases.

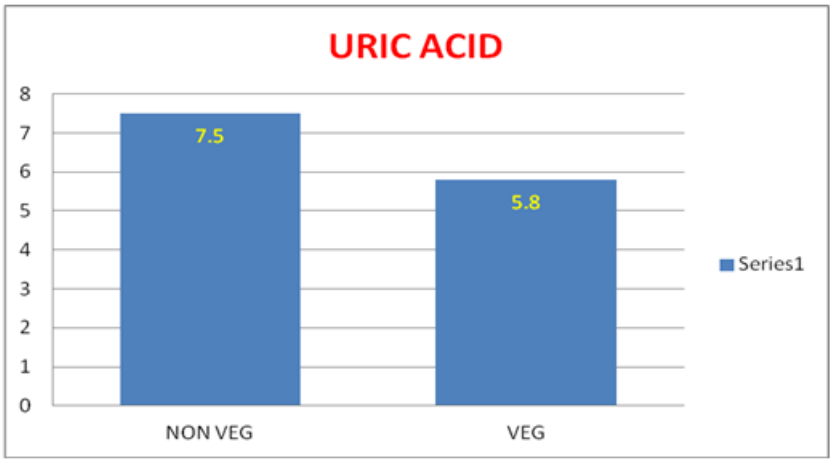

Figure 6: Comparison of serum uric acid in both Non Veg. and Veg. of hypertensive subjects

Fig. 6 shows increase of serum uric acid in both non vegetarian and vegetarian cases but markedly elevated in non vegetarian group(7.5 ±1.1)mg/dl than the vegetarian group.(5.8 ± 0.9)

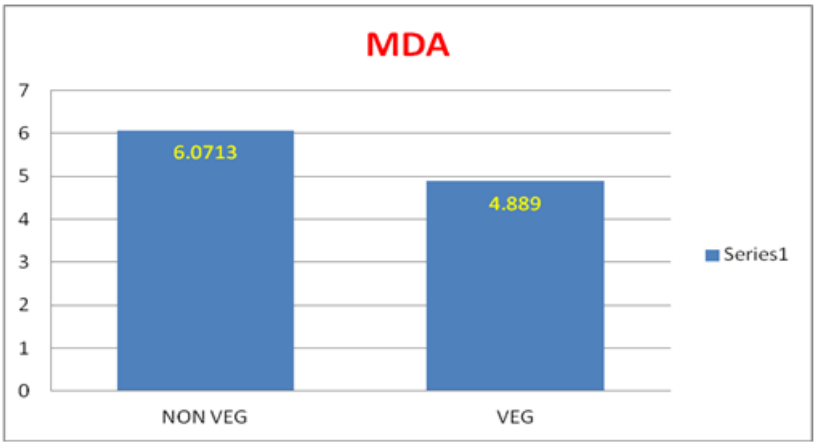

Figure 7: Comparison of plasma MDA in both Non Veg. and Veg. of hypertensive subjects

Fig.7 shows increase of serum uric acid in both non vegetarian and vegetarian cases but markedly elevated in non vegetarian group(6.1 ± 1.3) µmol/l than the vegetarian group.(4.9 ± 1.4) µmol/l

Table –8: Comparison of Lipid Profile Based on the Dietary Pattern in the Cases Studied

PARAMETER	HYPERTENSIVE(n=80)	
	NON VEG (n=53)	VEG (n=27)
TC (MEAN±SD)	210.0 ± 28.8	190.0 ± 25.0
TGL (MEAN±SD)	208.7 ± 49.8	192.4 ± 32.0
HDL-C (MEAN±SD)	42.2 ± 5.4	39.3 ± 5.4
LDL-C (MEAN±SD)	127.5 ± 26.2	113.4 ± 24.3
VLDL (MEAN±SD)	40.3 ± 9.9	36.9 ± 6.5

This TABLE 8, describes in the cases of non vegetarian subjects, the (Mean±SD) of TC, TG, LDL-c, VLDL-c, in the non vegetarian n subjects were markedly elevated as compared to vegetarian subjects.. In the hypertensive cases the lipid profile fractions were more marked among non vegetarian subjects than the vegetarian groups.

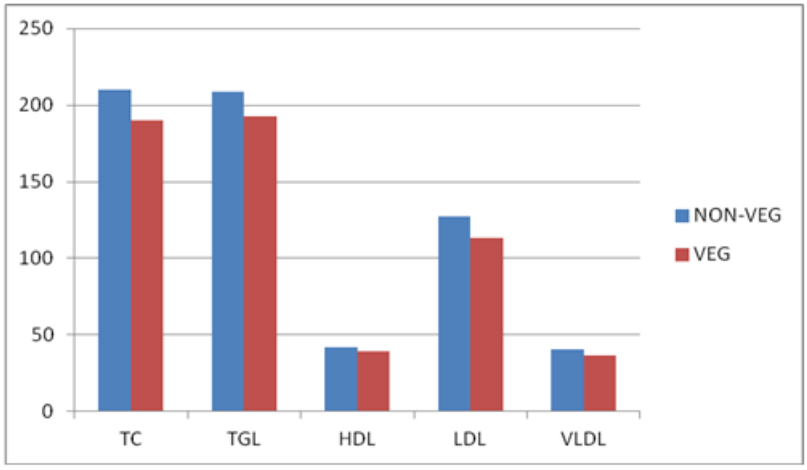

Figure 8: Comparison of lipid profile in both Non Veg. and Veg. subjects

Fig. 8 shows the lipid profile fractions were elevated in hypertensive cases of both non veg and veg subjects but more marked on the non vegetarian subjects compared to vegetarian groups.

IV. Conclusions And Suggestions

In this study, it is found that there was high serum uric acid, plasma MDA(lipid peroxidation marker) and dyslipidemia in non vegetarian group of hypertensives than the vegetarian group of cases.. The serum uric acid reflected the renal status as well as behaves as a pro-oxidant in excess. It was also correlated with lipid profile and lipid peroxidation. The increased serum uric acid and lipid peroxidation were observed in persons with significant modifying life style such as dietary pattern. The changes in dietary pattern such as non vegetarian has impact on increasing the oxidative stress which in turn increase the blood pressure So early Sintervention can be made by modifying the life style, dietary habits and also drugs aimed at treatment for hypertension as well as to prevent complication.

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