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Original Research Article

Assessment of plasma ascorbic acid and marker of lipid peroxidation in Essential Hypertension patients

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Abstract: Oxidant mediated biomolecular damage including lipid peroxidation has been implicated in the pathogenesis of series of health related complications including essential hypertension (HT). Antioxidants provide protection from these free radicals in an effective manner but under pathophysiological condition these antioxidant level changes leading to disease development. The present study was carried out to estimate the levels of plasma ascorbic acid, an aqueous medium containing non-enzymic antioxidant and malondialdehyde (marker of lipid peroxidation) in Hypertensive subjects of different grades as per JNC 7th norms. In the present study, plasma ascorbic acid levels and malondialdehyde levels were measured in 120 hypertensive subjects (30-60 years), categorized into three groups as prehypertension, Stage I HT and Stage II HT, depending upon their blood pressure and statistically compared it with that of 40 healthy individual, served as control. Plasma ascorbate level was found to be significantly low in each patient group as compared to control ($P < 0.001$) and whereas malondialdehyde levels were increased significantly each patient group as compared to control ($P < 0.001$) and with subsequent increase in blood pressure. Plasma ascorbate levels were also low among three groups of essential hypertension. These findings suggest that plasma ascorbate is consumed in scavenging free radical to reduce oxidative stress mediated destructions with subsequent rise in blood pressure as characterized by increased production of malondialdehyde. Therefore, regular monitoring of oxidative stress markers should be encouraged along with consumption of diet rich in vitamin C and development of light physical exercise habit with increase in blood pressure.
Keywords: Vitamin C, malondialdehyde, oxidative stress, free radical.

INTRODUCTION:

Hypertension (HT), a multifactorial disease often called as silent killer, remains the most common risk factor for cardiovascular morbidity and mortality with essentially unknown aetiology [1-3]. Among various risk factors and biochemical events associated with the etiopathogenesis of essential hypertension, increased production of oxygen free radicals have been identified as an important inducer of vascular disorder leading to HT [4, 5]. Lipid peroxidation is a free radical mediated process in which the polyunsaturated fatty acids contained in the LDL or present in the cell membrane are degraded to variety of aldehydes (mainly malondialdehyde). These aldehydes may alter endothelial function and inhibit NO synthase activity leading to the development of HT [6].

Antioxidants reduce or eliminate these free radicals. Among non enzymic antioxidant, vitamin C is an exogenous water soluble antioxidant functions as primary defense against free radicals in plasma and disappeared more quickly. In the immune system, the major role of vitamin C seems to be as a physiological antioxidant, protecting host cells against oxidative stress caused by infections. In various experimental settings, vitamin C increased the functioning of phagocytes, the proliferation of T-lymphocytes and the production of interferon, and decreased the replication of viruses [7, 8]. Kumar and Das also observed that free radicals are

produced in increased amount in HT and altered levels of vitamin C are significantly related with both systolic and diastolic blood pressure [9].

Although limited information is available on MDA level and plasma ascorbate level in the patients with essential hypertension, alteration in its level with severity of disease is still obscure. Therefore, the objectives of present study were to estimate erythrocyte MDA and plasma ascorbate levels in patients with essential hypertension and to determine the variation in these levels with increase in blood pressure.

MATERIALS & METHODS:

In the present study, 120 subjects of essential hypertension of either sex were recruited in the patient group and 40 age matched healthy individuals were recruited in control group (30-60 years) after taking their informed consent form and approval of the study from Ethical committee of the college. The patients were classified into 3 groups (40 patients in each group), according to "Seventh Report of Joint National Committee on High Blood Pressure". These include Group I or pre-hypertension [Systolic Blood Pressure (SBP) 120-139 mm Hg and Diastolic Blood Pressure (DBP) 80-89 mm Hg], Group II or Stage I HT (SBP 140-159 & DBP 90-99 mm Hg) and Group III or Stage II HT (S.B.P. ≥ 160 mm Hg & D.B.P. ≥ 100 mm Hg). Venous blood was collected in EDTA vial from each subject after measurement of blood pressure. In addition, hypertensive patients with other systemic diseases were excluded. Samples were processed immediately.

Erythrocyte malondialdehyde (MDA) levels were measured as thiobarbituric acid reactive substances, after preparation of hemolysate. The heat induced reaction of malondialdehyde (MDA) with thio barbituric acid (TBA) in the acid solution forms a trimethine coloured substance, which is measured spectrophotometrically at 532 nm [10]. Plasma ascorbic acid levels were estimated by Mc Cormick and Greene method. Ascorbic acid in plasma is oxidized by Cu (II) to form dehydro ascorbic acid which reacts with acidic 2,4–dinitrophenyl hydrazine to form a red bis hydrazone, which is measured at 520 nm [11].

STATISTICAL ANALYSIS:

The data from both the study group subjects and controls were expressed as Mean ± SD and compared by using Student's t-test and distribution of probability (P).

RESULTS:

The mean plasma ascorbate level and lipid peroxide level (i.e. malondialdehyde) of the patient and control group are depicted in Table.1.0 and Figure 1.0 respectively. The mean plasma ascorbate levels in hypertensive subjects were significantly low (P<0.001) as compared to controls i.e. 23.8%, 33.06% and 40.32% in pre-hypertension, stage I HT and stage II HT patients respectively. On the other hand, erythrocyte malondialdehyde levels were found to be significantly high (P<0.001) 15.80%, 27.20% and 32.35% in prehypertension, stage I HT and stage II HT patients as compared to healthy controls. These variations were increased continuously with increase in blood pressure.

Table 1: Plasma ascorbic acid and erythrocyte malondialdehyde levels in patients and control group (Mean±SD)

S.No.	Particulars	Control Group	Patient Group		
			Group I	Group II	Group III
1.	No. of Samples	40	40	40	40
2.	Age (years)	30 – 60	30 – 60	30 – 60	30 – 60
3.	Blood Pressure (SBP/DBP mm Hg)	100-119/ 70-79	120-139/ 80-89	140-159/ 90-99	≥ 160/ ≥80
4.	Ascorbic acid (mg/dl)	1.24 ± 0.21	0.95* ±0.18	0.83** ±0.17	0.74** ±0.15
	i) % Decrease	-	23.38%	33.06%	40.32%
	ii) P – value	-	P < 0.05	P < 0.05	P < 0.001
5.	Malondialdehyde (µ mol MDA/ml)	2.72 ± 0.09	3.15* ±0.12	3.46* ±0.13	3.60** ±0.15
	i) % Increase	-	15.80%	27.20%	32.35%
	ii) P – value	-	P < 0.05	P < 0.05	P < 0.001

Where,
* P < 0.05: Significant
** P < 0.001: Significant

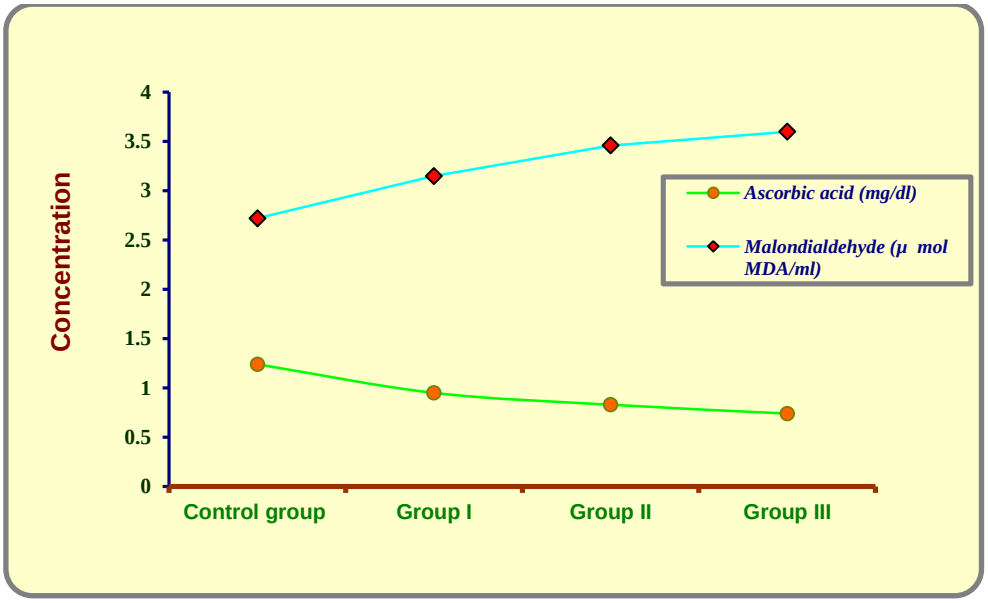

Fig-1: Plasma ascorbic acid and erythrocyte MDA levels in study group subjects

DISCUSSION:

Hypertension is a major risk factor in the development of ischemic heart disease and cerebrovascular disease probably by mechanical injury to arterial wall. It has now been proved that oxidative stress is increased in patients with essential hypertension owing to increase in free radical production [2]. Involvement of free radicals in membrane damage via lipid peroxidation and its resultant products such as lipid radicals (L◦), lipid peroxides (LOO◦), lipid hydro peroxides (LOOH) and highly reactive aldehydes plays a crucial role in the development and progression of inflammatory and age related disorders [12]. Free radicals via lipid peroxidation play an etio pathogenic role in the development of HT patients as obvious in the findings of present study. Marked elevated levels of malondialdehyde (i.e. marker of lipid peroxidation) were observed in each patient group ($P<0.001$) as compared to healthy control which rises continuously with increase in blood pressure. Our findings were in concordance with the findings of Tandon *et al.;* According to them, lipid peroxides are toxic to the cellular components and lipid peroxidation may be responsible for vascular disorder in HT [13]. It has also documented that excess endogenous aldehyde plays a major role in HT by binding sulphydryl groups of membrane proteins, altering Ca^{2+} channels and increasing cytosolic free Ca^{2+} that cause further extensive membrane damage due to the action of phospholipases and proteases, the activation of contractile proteins and the accumulation of mitochondrial calcium resulting in a vicious cycle of damage extension, peripheral vascular resistance and hypertension [14, 15].

The production of free radicals and its culprit effect on biomolecules is effectively regulated by antioxidant reserves of body. Among non-enzymic antioxidants, vitamin C is an effective water soluble antioxidant and a stabilizer of biological membranes, prevent accumulation of free radicals and alone can afford protection against the oxidant mediated damage to LDL [16]. In the present study, plasma ascorbate levels were significantly low ($P<0.05$, $P<0.001$, $P<0.001$) in each patient group as compared to controls. However, they did not differ significantly with each other. Reduction in vitamin C levels could not be only due to its free radical scavenging action but also in maintaining the body antioxidant reserve and in normalization of vascular superoxide formation which prevent endothelial dysfunction. Salonen *et al.;* in their study also observed that low ascorbate level is significantly related with both diastolic and systolic blood pressure [17]. Furthermore, reduction in vitamin C & E level with increased levels of lipid peroxides were also reported in previous study on hypertensive subjects and concluded it as a contributory event in the development of cardiovascular disease in HT patients [18]. In addition, Singh et al. in their study on different age group subjects observed that vitamin C levels decreases continuously with advancing of age due to

augmented oxidative stress, making individual more susceptible to develop age related complications including hypertension [19].

CONCLUSION:

On the basis of present study and consistent findings of previous studies we can conclude that plasma ascorbate level is inversely related to increase in blood pressure and lipid peroxidation plays an etiopathological role in the development of HT. As the blood pressure rises, plasma vitamin C level decreases continuously not only due to its free radical scavenging action but also in maintaining body's antioxidant reserve and in limiting the lipid peroxidation. Thus, both vitamin C and malondialdehyde levels may be an excellent marker of oxidative stress in essential hypertension. In addition, our study suggests that the diet with high vitamin C contents along with consumption of fruit, vegetables and grains is essential for patients suffering with essential hypertension in order to combat its deleterious effect and future complications.

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