

±Association of Oxidative Stress, Serum Nitric Oxide, Plasma Fibrinogen in Hypertensive Males of Southern odisha

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Abstract

Background And Objectives: Various studies have implicated the association of oxidative stress and nitric oxide depletion in essential hypertension. This condition aggravates endothelial dysfunction and leads to atherosclerosis. Hypertension is a major risk factor and plasma fibrinogen is a consistent predictor of cardiovascular diseases. Hence this study was designed to evaluate the correlation of oxidative stress, nitric oxide and plasma fibrinogen in hypertensive patients of Southern Odisha.

Material And Methods: This case-control study included 92 hypertensive males and an equal number of matched controls. Oxidative stress was measured by FOX2, anti oxidant status by FRAP, serum nitric oxide by Griess method and plasma fibrinogen by commercial kits from Tulip diagnostics.

Observation: We observed statistically significant rise in oxidative stress, decreased nitrosative stress and increased plasma fibrinogen levels in hypertensive patients. There is a positive correlation between oxidative stress and plasma fibrinogen and negative correlation between NO, oxidative stress and plasma fibrinogen.

Conclusion: These findings suggest a strong association of oxidative stress, increased plasma fibrinogen and decreased NO levels in hypertensive patients.

I. Introduction

Hypertension (HTN) is defined as systolic blood pressure more than 140 mm Hg and diastolic blood pressure more than 90 mm Hg (1) HTN has been associated with oxidative stress (2, 3). Various studies have suggested initiation of generation of reactive oxygen species (ROS) by increased blood pressure. Exposure to ROS leads to lipid peroxidation of cell membranes. ROS is known to inactivate nitric oxide (NO), which is a potential vasodilator. Studies have also supported a variety of antioxidant treatments in HTN. (4,5,)

Oxidative stress creates a prohypertensive environment in the body by involving both hemodynamic (vasoconstrictive) and structural (vascular remodeling) mechanism. (6,7) ROS stimulates vascular smooth muscle remodeling in resistance arteries, leading to increased vascular wall rigidity and narrowing of arterial lumen. (8,9) . NO is metabolized rapidly to form nitrite. ROS also stimulates non specific inflammation and plasma fibrinogen is a positive acute phase protein. (10,11) Hence this study was designed to evaluate Oxidative stress (OS), Nitric oxide (NO) and Fibrinogen (Fib) in hypertensive patients of Southern Odisha and to find a correlation between OS, NO and Fibrinogen

II. Material And Method

This case-control study was carried out in the department of biochemistry MKCG Medical College, Berhampur during April 2014 to May 2015. The study included 92 HTN male patients and an equal number of age and sex matched controls. All the hypertensive patients were selected as per the WHO/ISH guidelines (12). HTN patients having associated diseases such as DM, CVD, and Renal disease were excluded. Patients who were on antioxidant therapy or vitamin supplements were also excluded. The study was approved by the institutional ethical committee. The oxidant load was measured by FOX2 method and antioxidant capacity by FRAP method (14,15). NO was estimated by Griess method (16). Plasma fibrinogen was estimated by commercial kits from Tulip diagnostics. (17) All data are represented as mean ± SD. Statistical analysis was done by unpaired student-t test. Correlation analysis was done by Pearson's correlation analysis. All statistical analysis was done by SPSS version 19.

III. Observation

Table 1 Demographic characteristics of hypertension in cases and controls

Characteristics	HTN Patients	Controls	p value
AGE	48.5±10.2	49.2±9.6	NS
SBP	160±5.6	120±2.2	0.01
DBP	106±2.6	80±2.1	0.01
BMI	23±0.02	22±0.8	NS

We observed a significantly higher SBP and DBP in cases as compared to controls. There was no significant difference in age and BMI between cases and controls.±

Table 2 Depicts biochemical parameters of cases and controls

Biochemical parameters in mg%	HTN	CONTROLS	p value
FBS	98.6±4.2	100±2.0	NS
Total cholesterol (TC)	260.1±43.8	152±16.4	<0.001
Triglyceride(TG)	298.7 ±69.2	150±30.2	<0.001
HDL-C	36.5±2.5	45.8±3.9	<0.001
wLDL-C	168.2±5.8	100.8±4.72	<0.001
VLDL-C	59.74±13.8	30±6.0	<0.001
Plasma Fibrinogen	314.3 ±62.1	115±26.4	<0.001
Nitric Oxide	37.59±4.19	73.8±5.65	<0.001

We observed a significantly increased Lipid profile, plasma Fibrinogen and lower NO levels in HTN patients.

Table 3 shows the oxidative stress and serum nitric oxide levels in cases and controls.

Biochemical parameters	HTN	CONTROL	p value
FOX 2(Equivalence of H ₂ O ₂)	14.2± 1.4	3.17 ±1.49	<0.001
FRAP (Equivalence of Ferrous Sulphate)	98.89±42.6	440.2±14	<0.001
NO	37.59±4.19	73.8±5.65	<0.001

We observed a significantly higher oxidant load (FOX 2) and a low anti oxidant status (FRAP) in HTN cases.. The nitric oxide level was significantly higher in HTN cases.

IV. Discussion

In this case- control study, age, sex matched HTN patients and healthy volunteers were taken. The HTN patients had significantly increased levels of Serum lipid profile. This is in concurrence with previous studies (14). The increased LDL-cholesterol leads to enhanced free radical production and endothelial dysfunction. (14) The increased ROS causes reduction in endothelial dependent vasodilatation and increase the peripheral resistance in HTN patients (21). Oxidation of lipid biomembranes by the action of generated free radicals is termed as lipid peroxidation. Elevated FOX2 is an indicator of increased lipid peroxidation. The enhanced lipid peroxidation product might have caused the peroxidative damage to vascular endothelium. Therefore it can be stated that, lipid peroxidation may contribute as one of the cause of essential hypertension. Endothelial dysfunction is closely associated with essential hypertension. Endothelial cells produce the biologically active molecule, NO, which plays a major role in controlling vascular diameter and its tone. However it was observed that, superoxide ion can inactivate NO (18). Hence, it can be suggested that superoxide anion may affect vascular resistance by inactivating NO. This explains low NO in essential hypertension. Another significant observation was, raised level of plasma fibrinogen which correlated positively with OS and NO levels. This is similar to the finding of (19). The elevated Fibrinogen level also indicates the increase in shear stress, and endothelial dysfunction, and a progress towards peripheral vascular disease in severe uncontrolled HTN. (22) It has been suggested by various studies that oxidative stress and NO levels increase the release of inflammatory cytokines. Fibrinogen, an acute phase protein along with the inflammatory condition of HTN causes a rise in fibrinogen level. (20)

V. Conclusion

Antioxidant therapy should be included along with lipid lowering drugs. Plasma Fibrinogen should be estimated at regular intervals and steps to maintain it within the normal range should be initiated to prevent cardiac and peripheral vascular diseases in HTN patients.

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