Section: Biochemistry

Original Research Article

ASSOCIATION OF OXIDATIVE STRESS MARKERS WITH ANTIOXIDANT STATUS IN ESSENTIAL HYPERTENSION

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Received *: 26/11/2025*
Received in revised form *: 12/01/2026*
Accepted *: 30/01/2026*

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DOI: 10.70034/ijmedph.2026.1.268

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (1); 1538-1541

ABSTRACT

Background: Oxidative stress, resulting from an imbalance between reactive oxygen species and antioxidant defense mechanisms, plays a crucial role in the pathogenesis of chronic non-communicable diseases such as essential hypertension. **Objective:** To assess association of oxidative stress with antioxidant defense status in patients with essential hypertension and compare them with healthy controls.

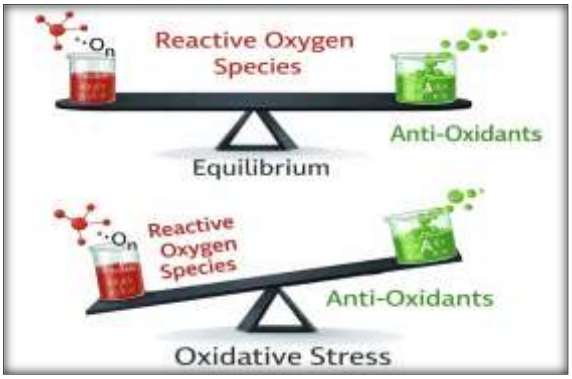
Materials and Methods: A cross-sectional study included 120 subjects aged 35–60 years, divided into controls (n=60) and more than 5 years' duration of essential hypertension patients (n=60). Serum malondialdehyde (MDA) was estimated as a marker of lipid peroxidation. Antioxidant defense was assessed by measuring superoxide dismutase (SOD), catalase, and reduced glutathione (GSH) using standard biochemical methods. **Results:** Hypertensive patients showed significantly higher MDA levels (4.2 ± 0.8 nmol/mL) compared to controls (2.1 ± 0.4 nmol/mL) ($p < 0.001$). Antioxidant enzymes SOD (3.6 ± 0.6 vs 5.6 ± 0.8 U/mL), catalase (45.2 ± 5.4 vs 62.4 ± 6.5 kU/L), and GSH (34.8 ± 3.5 vs 48.2 ± 4.1 mg/dL) were significantly reduced in hypertensive subjects ($p < 0.001$).

Conclusion: Essential hypertension is characterized by increased oxidative stress and compromised antioxidant defense. Assessment of oxidative stress markers and antioxidant enzymes may serve as useful adjuncts in risk stratification and management of hypertensive patients.

Keywords: Oxidative stress; Antioxidants; Free radicals; Essential Hypertension; Malondialdehyde (MDA); Superoxide dismutase (SOD).

INTRODUCTION

Aerobic metabolism is essential for human survival, yet it inevitably leads to the generation of reactive oxygen species (ROS) as by-products of oxidative phosphorylation. These ROS include free radicals, which are highly reactive molecules with unpaired electrons that can damage cellular components through chain reactions. Reactive intermediates such as superoxide anion, hydroxyl radical, and hydrogen peroxide are capable of damaging lipids, proteins, carbohydrates, and nucleic acids when produced in excess. Normally, cells maintain a balance between pro-oxidants, which promote free radical formation, and antioxidants, which neutralize them. When this balance is disrupted—either by increased pro-oxidants or reduced antioxidants— as "Oxidative Stress.[1-2] occurs, a central mechanism implicated in the pathogenesis of various diseases[3-5] like diabetes mellitus, hypertension, atherosclerosis, cancer, neurodegenerative disorders, etc.



Although the initial attack causes the free radical to become neutralized, another free radical is formed in the process, causing a chain reaction6 to occur. When the chain reaction occurs in a cell, it can cause damage or death to the cell. Antioxidants terminate these chain reactions by removing ROS intermediates, and inhibit other oxidation reactions. Antioxidants are employed to protect biomolecules from the damaging effects of such ROS.

There are various strategies [7] / lines of defenses applied by Antioxidants are:-

Table 1: Lines of defenses applied by Antioxidants and their outcome

Line of Defence	Main Purpose	Key Mechanisms / Examples	Outcome
Prevention	Prevent formation of ROS	Cytochrome oxidase prevents superoxide release; Ribonucleotide reductase (RNR) traps tyrosyl radical; Metal ion (Fe, Cu) binding; Protective pigments (melanin, carotenoids)	Reduces initiation of free radical formation
Interception	Neutralize ROS after formation	Superoxide dismutase ($O_2^- \rightarrow H_2O_2$); Catalase ($H_2O_2 \rightarrow H_2O + O_2$); Glutathione peroxidase ($H_2O_2 \rightarrow H_2O$); Radical transfer from membranes to aqueous phase	Stops propagation of oxidative damage
Repair	Reverse oxidative damage	DNA repair enzymes (SSB/DSB repair); Lipolytic enzymes for membrane damage; Proteolytic enzymes for damaged proteins	Restores normal cellular structure and function

MATERIALS AND METHODS

Study Design and Participant

This cross-sectional comparative research study was conducted at the Department of Biochemistry of our tertiary care hospital. A total of 120 subjects aged 35–60 years were enrolled and divided into two groups: - **Controls (n = 60):** Apparently healthy individuals with normal blood pressure - **Hypertension group (n = 60):** Patients diagnosed with essential hypertension (BP ≥140/90 mmHg or on antihypertensive therapy) for more than 5 years duration.

Inclusion Criteria

- Diagnosed essential hypertension for more than 5 years.
- Age between 35 and 60 years

Exclusion Criteria

- Diabetes mellitus
- Smokers and alcoholics
- Patients with renal failure, liver disease, malignancy, chronic infections, or inflammatory disorders.
- Individuals on antioxidant or vitamin supplementation

Sample Collection and Biochemical Analysis

After overnight fasting, 5 mL of venous blood was collected under aseptic precautions. Serum and plasma were separated for biochemical analysis.

- **Malondialdehyde (MDA):** Estimated by thiobarbituric acid reactive substances (TBARS) method
- **Superoxide Dismutase (SOD):** Measured by inhibition of auto-oxidation method
- **Catalase:** Estimated by hydrogen peroxide decomposition method
- **Reduced Glutathione (GSH):** Measured using Ellman's reagent

Statistical Analysis

Results were expressed as mean ± standard deviation. Statistical comparison between groups was performed using Student's *t*-test. A *p*-value <0.05 was considered statistically significant.

RESULTS

Baseline Characteristics of Study Participants

Table 2: Baseline demographic and clinical characteristics of study participants with essential hypertension and healthy controls.

Variable	Controls (n = 60)	Hypertension (n = 60)	p-value
Age (years)	51.2 ± 5.8	52.3 ± 6.1	>0.05
Gender (Male/Female)	34 / 26	36 / 24	>0.05
Systolic BP (mmHg)	118.6 ± 7.4	152.6 ± 11.8	<0.001
Diastolic BP (mmHg)	76.8 ± 6.2	96.4 ± 7.6	<0.001
Body mass index (kg/m²)	24.1 ± 2.6	26.8 ± 2.9	<0.001
Waist circumference (cm)	88.1 ± 7.3	94.2 ± 8.1	<0.001

The baseline characteristics revealed significant differences in several clinical and metabolic parameters between essential hypertension and healthy controls. As expected, systolic and diastolic blood pressures were markedly higher in the hypertensive group (152.6 ± 11.8 and 96.4 ± 7.6 mmHg, respectively) compared to controls (118.6 ± 7.4 and 76.8 ± 6.2 mmHg; $p < 0.001$). Hypertensive subjects also had significantly higher body mass

index and waist circumference, indicating greater adiposity ($p < 0.001$ for both)

Oxidative Stress Marker

Serum MDA levels were significantly elevated in hypertensive patients, indicating increased lipid peroxidation.

- Controls: 2.1 ± 0.4 nmol/mL
- Hypertension: 4.2 ± 0.8 nmol/mL ($p < 0.001$)

Antioxidant Enzyme Status

A significant reduction in antioxidant enzyme activity was observed in hypertensive subjects.

- **SOD:** 3.6 ± 0.6 U/mL vs 5.6 ± 0.8 U/mL in controls ($p < 0.001$)
- **Catalase:** 45.2 ± 5.4 kU/L vs 62.4 ± 6.5 kU/L in controls ($p < 0.001$)
- **GSH:** 34.8 ± 3.5 mg/dL vs 48.2 ± 4.1 mg/dL in controls ($p < 0.001$)

Graphical analysis using bar charts demonstrated a clear inverse relationship between oxidative stress markers and antioxidant defense in diseased states. [Figures 1-4]

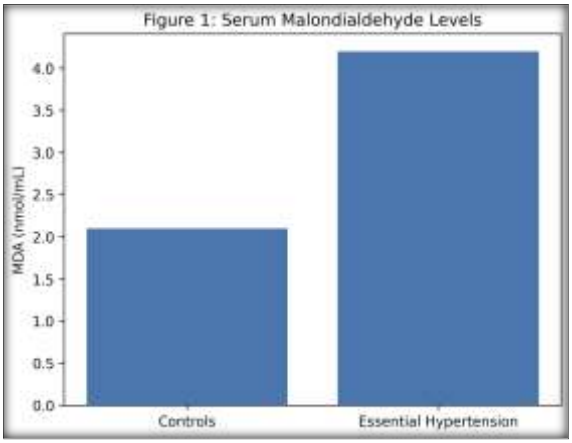


Figure 1: Serum Malondialdehyde levels

Figure 1: Bar diagram comparing serum malondialdehyde (MDA) levels between healthy controls and patients with essential hypertension. Elevated MDA levels in hypertensive patients indicate increased lipid peroxidation and oxidative stress.

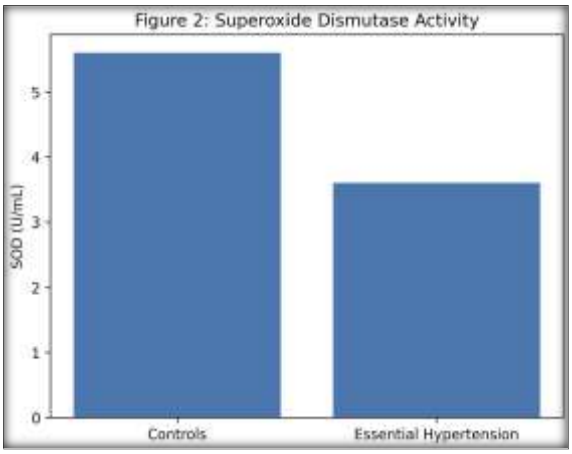


Figure 2: Superoxide Dismutase Activity

Figure 2: Bar diagram depicting superoxide dismutase (SOD) activity in controls and hypertensive patients. Significant reduction in SOD activity reflects impaired antioxidant defense in hypertension.

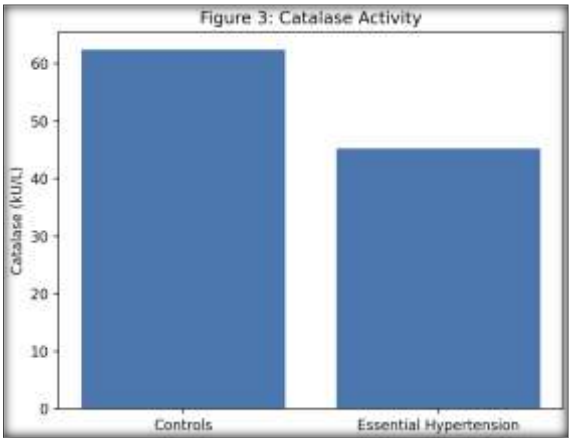


Figure 3: Catalase Activity

Figure 3: Bar diagram showing catalase activity among controls and hypertensive subjects. Decreased catalase activity suggests reduced detoxification of hydrogen peroxide.

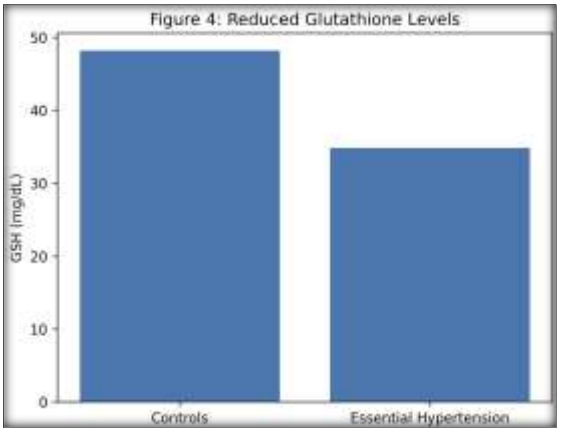


Figure 4: Reduced Glutathione Levels

Figure 4: Bar diagram representing reduced glutathione (GSH) levels in controls and patients with essential hypertension. Lower GSH levels indicate diminished intracellular redox buffering capacity.

DISCUSSION

The present study demonstrates a significant increase in oxidative stress and a concomitant decrease in antioxidant defense in patients with essential hypertension. Elevated MDA levels reflect enhanced lipid peroxidation due to excessive free radical,[8-9] generation. Reduced antioxidant enzyme activities suggest impaired neutralization of reactive oxygen species.

Oxidative stress contributes to hypertension by reducing nitric oxide bioavailability, increasing vascular tone, and promoting inflammation and

vascular remodeling. The findings of this study are consistent with earlier Indian and international studies reporting increased oxidative stress in hypertensive individuals.

Early detection of oxidative imbalance may help in preventing hypertension-related complications.[10-11] Lifestyle modifications, dietary antioxidant intake, and optimal blood pressure control may restore redox balance and improve vascular health.

CONCLUSION

Essential hypertension is associated with increased oxidative stress and compromised antioxidant defense mechanisms. Measurement of oxidative stress markers such as MDA and antioxidant enzymes including SOD, catalase, and glutathione may serve as useful adjuncts in the clinical evaluation and management of hypertensive patients.

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