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Original article

Enalapril (ACE Inhibitor) attenuates Oxidative Redox - Imbalance, Inflammatory Response and improves Atherogenic Lipid Risk Indices more efficiently than *Atenolol* (Beta-Blocker) in Patients with Essential Hypertension. A *Comparative Case - Control Study*

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Abstract:

Background: Hypertension is an emerging serious public health burden, increasing the risk of heart disease, stroke, metabolic syndrome complications, suggesting effective preventive strategies essential. Oxidative stress, inflammatory response and atherogenic dyslipidemia intercalated the pathobiology of hypertension associated cardio-metabolic complications Though, all antihypertensive drugs primarily lower blood pressure, some drugs have favourable effects on metabolic profile, while others may have adverse effects. **Aim:** The present study aimed to investigate and compare the treatment efficacy of ACE inhibitor and beta-blocker on oxidative stress, systemic inflammatory response and atherogenic dyslipidemia in patient with essential hypertension. **Study Design:** 30 healthy volunteers, 60 newly diagnosed essential hypertensive patients were enrolled. Among 60 patients, 30 were treated with Enalapril (10mg/day) and 30 were treated with Atenolol (25mg/day) for two months. **Results & Conclusion:**Hypertensive patients displayed with oxidative stress, systemic inflammation and atherogenic lipid risk indices as evidenced by the elevated levels of TBARS, TOS, OSI and PABTNF-α, hsCRP and atherogenic lipid profile, AIP, CRI-I & II compared to control. Treatment with enalapril and atenolol significantly reduced all these metabolic derangements. Furthermore, enalapril treatment is more effectively attenuating these metabolic complications than atenolol, suggesting that enalapril contributing potent anti-oxidative, anti-inflammatory, and dyslipidemic benefits in addition to its standard antihypertensive activity than atenolol therapy.

Key Words: Oxidative Stress, Inflammatory Response, Atherogenic dyslipidemia, Essential Hypertension, ACE Inhibitor, Beta-Blocker.

Introduction:

Hypertension is a multi-factorial process, prevalent in developed as well as in developing countries, making urgent needs of effective preventive strategies. According to the guidelines of European Society of Cardiology 2024, hypertension is characterized by a sustained elevation of systolic blood pressure (SBP)of ≥ 140 mm Hg and a diastolic blood pressure (DBP)of≥90 mm Hg. In about 80% of the patients, the exact route cause for hypertension is unknown, called as essential hypertension. The emerging evident showed the global prevalence of hypertension

in 2025 was 33 % in the age group of 30 - 80 years [1]. If the hypertension remains untreated over the prolonged period, it can lead to the several cellular and tissues derangements, leading to metabolic syndrome and its pathological consequences; atherogenic coronary diseases and cardiovascular consequences [2].

Though several modifiable risk determinants have reported, hypertension, atherogenic dyslipidaemia and vascular inflammation form a triad of interrelated metabolic derangements which significantly and synergistically contribute to the pathogenesis of early and premature atherosclerosis and ultimately coronary heart diseases [3]. Maintaining the normal blood pressure is a critical approach for lowering the incidence of cardiovascular disability, and mortality. Hence, studying the biochemical basis of lipid derangements and atherogenic lipid risk in hypertension is essential in improving the better treatment strategy. Recently, studies have documented that, the atherogenic index of plasma (AIP), a newly formulated lipid metabolic index that is significantly associated with the incidence of insulin resistance, metabolic syndrome complications and cardiovascular diseases [4]. However, the association between AIP and the incidence of hypertension remains unexplored. Oxidative stress and inflammatory response are well known risk factors for vascular dysfunction, and drives hypertension and atherogenic dyslipidaemia [4, 5]. This suggesting the need for antihypertensive drugs that also target in improving the oxidative stress, vascular inflammation and mediated lipid derangements.

Currently, several classes of pharmacological strategies are available in the clinical medicine to treat hypertension, with varying effects on metabolic profile. Treating blood pressure alone can provide only limited benefit, although it is recommended for the management of overall cardiovascular risk. While all antihypertensive drugs primarily lower blood pressure, some classes of drugs have favourable effects on metabolic complications; inflammation, atherogenic dyslipidaemia, while others may have adverse effects [5]. Therefore, the study of the relative effectiveness of different classes of antihypertensive drugs with respect to metabolic outcomes will pave the way to improve the quality and effectiveness of hypertensive treatment strategies. Given the increasing burden of hypertension and the urgent need of effective preventive strategies, in the present study, we made an attempt to explore the comparative effectiveness of *Enalapril* (ACE inhibitor) and *Atenolol* (Beta-blocker) on oxidative stress, inflammatory response and atherogenic dyslipidaemia, a potent risk factors of cardiovascular consequences.

Materials and Methods

Study Subjects and Ethical Consideration

The Study was conducted in the Department of Biochemistry, Meenakshi Medical College Hospital and Research Institute, a tertiary care teaching hospital located at Enathur, Kanchipuram - 631552, Tamil Nadu, India., after obtaining the Institutional Human Ethical Committee approval. 60 freshly diagnosed Essential Hypertensive patients and 30 healthy volumeters were enrolled in the study. Hypertension was diagnosed according to the guidelines from the Indian Council of Medical Research (ICMR) and the Indian Society of Hypertension [6]. Subjects with a history of diabetic mellitus, kidney diseases, cardiovascular diseases, any infections, smokers, alcoholics and those who are on any kind of medications were excluded from the study. A written informed consent was obtained from the study subjects.

Blood Sample Collection

Five millilitres (5mL) of venous blood after over-night fasting of 12-14 hrs was collected in EDTA bottles. Plasma was separated by centrifuging the blood at 6000g for 5 mins. The plasma was used for the estimation of oxidative stress parameters; total anti-oxidant capacity (TAC), total oxidant status (TOS), Thiobarbituric acid reactive substances (TBARS), inflammatory markers such as tumor necrosis factor alpha (TNF-α), high sensitive C-reactive protein (hs CRP), IL-6, and for lipid profile parameters.

Anthropometric Measurement

Anthropometric parameters, including height and weight, were measured using standardized protocols. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters (kg/m^2).

Analysis of Plasma Inflammatory Markers

The plasma inflammatory markers; tumor necrosis factor alpha (TNF-α, Orgenium, Finland) and high sensitive C-reactive protein (hs-CRP, DBC, Canada) were measured by enzyme linked immunosorbent assay (ELISA) using commercially available standard kits according to manufacturer's instructions.

Evaluation of Oxidative Stress Indices

The plasma level of Thiobarbituric acid reactive substances (TBARS) was measured enzymatically using ELISA kit according to manufacturer's instructions (Cayman chemical company, USA). The plasma total anti-oxidant

capacity (TAC) and total oxidant status (TOS) were measured spectrophotometrically, developed by Erel [7]. The oxidative stress index (OSI) and pro-oxidant/anti-oxidant balance (PAB) were calculated as follows [8];
Oxidative Stress Index (OSI) = Total Oxidant Status (TOS)/Total Anti-oxidant Capacity (TAC). Pro-oxidant/Anti-oxidant Balance (PAB) = TBARS / TAC.

Estimation of Lipid Profile and Atherogenic Lipid Risk Indices

The fasting plasma lipid profile: total cholesterol (TC), triglyceride (TG) and high-density lipoprotein cholesterol (HDL-c) were estimated by standard diagnostic kit method (Agappe Diagnostics) using fully Automated Clinical Chemistry Analyzer (AU400, Olympus, USA). Low density lipoprotein cholesterol (LDL-c) and very low-density lipoprotein cholesterol (VLDL-c) were calculated mathematically using Friedwald's equation.

Calculation of Atherogenic Risk Indices

Coronary atherogenic lipid Indices such as Castelli's risk index I and II (CRI-I and II), the atherogenic coefficient (AC) and atherogenic index of plasma (AIP) were calculated mathematically using the formula as follows [9];
CRI - I = Total Cholesterol /HDL-C, CRI - II = LDL-C/HDL-C, AIP = log (TG)/HDL-C and AC = [(Total Cholesterol – HDL-C)/HDL-C]

Antihypertensive treatment:

In the group of patients with essential hypertension who were treated with ACE inhibitor, *Enalapril* was administered at a daily dose of 10 mg [10]. Meanwhile, in the essential hypertensive patients receiving a beta-blocker, *Atenolol* was given at a dose of 25 mg per day [11]. All study participants were followed for a period of sixty days, to evaluate all biochemical parameters whichever mentioned above.

Statistical Analysis

Results were expressed as Mean ± SD. One way analysis of variance (one-way ANOVA) with *Tukey post hoc test* was used to evaluate the statistically significant difference between the control and hypertensive patients before and after treatment with enalapril and atenolol. All statistical analysis were performed using licensed SPSS Statistical Package (Version 21.0). A P-value less than 0.05 considered as statistically significant.

Results:

Table 1: Effect of *Enalapril* (ACE inhibitor) and *Atenolol* (Beta-Blocker) on Anthropometric parameters and Blood Pressure in Patients with Essential Hypertension

S. No	Parameters	Control (n=30)	Essential Hypertension (Enalapril Treatment) (n=30)		Essential Hypertension (Atenolol Treatment) (n=28)	
			Before	After	Before	After
1	Age (years)	39.3 ± 5.81	38.9 ± 7.35	40.1 ± 7.29	40.53 ± 7.29	40.56 ± 7.31
2	BMI (Kg/m^2)	23.07 ± 2.08	26.2 ± 3.1	26.3 ± 2.9	24.8 ± 2.7	25.1 ± 2.5
3	SBP (mmHg)	101.7 ± 11.3	178.9± 18.5[a]	127.5 ± 11.3[b]	169.7 ± 18.2[a]	131.3 ± 5.2 [c]
4	DBP (mmHg)	74.3 ± 7.6	97.6 ± 4.5[a]	86.2 ± 8.9[bd]	90.4 ± 11.1[a]	91.5 ± 8.2[c]

Results were expressed as Mean ± S.D. A [a,b,c,d] P-value less than 0.05 considered statistically significant. a - in comparison with control, b - in comparison with before enalapril treatment, c - in comparison with before atenolol treatment, d - comparison between after enalapril treatment and after atenolol treatment. BMI - Body Mass Index, SBP - Systolic blood Pressure, DBP - Diastolic Blood Pressure.

In Table 1, we found that, bothsystolic and diastolic blood pressure were increased in essential HTN patients when compared to the control. Treatment with both enalapril and atenolol significantly reduces the systolic and diastolic pressure in all the groups. No significant difference was found in BMI of all the groups.

Table 2: Effect of *Enalapril* (ACE inhibitor) and *Atenolol* (Beta-Blocker) on Oxidative Stress Indices parameters in Patients with Essential Hypertension

S. No	Parameters	Control (n=30)	Essential Hypertension (Enalapril Treatment) (n=30)		Essential Hypertension (Atenolol Treatment) (n=28)	
			Before	After	Before	After
1	Plasma TBARS (µMol/L)	2.99 ± 0.93	3.85 ± 0.99 [a]	3.17 ± 0.85 [bd]	3.99 ± 0.42 [a]	3.54 ± 0.67 [c]
2	Plasma TAC (mmol Trolox Eq/L)	1.71 ± 0.45	1.57 ± 0.74	1.79 ± 0.56 [bd]	1.44 ± 0.41 [a]	1.53 ± 0.39
3	Plasma TOS (mmol Trolox Eq/L)	13.12 ± 4.43	22.43 ± 8.57 [a]	16.28 ± 6.01 [b]	20.07 ± 3.36 [a]	16.96 ± 0.39 [c]
4	OSI (TOS/TAC)	8.32 ± 3.9	19.07 ± 10.63 [a]	10.53 ± 5.49 [b]	14.93 ± 4.48 [a]	11.99 ± 5.31 [c]
5	TBARS/TAC	1.93 ± 1.03	3.68 ± 0.81 [a]	1.96 ± 0.82 [bd]	2.99 ± 0.91 [a]	2.51 ± 1.04

Results were expressed as Mean ± S.D. A [a,b,c,d] P-value less than 0.05 considered statistically significant. a - in comparison with control, b - in comparison with before enalapril treatment, c - in comparison with before atenolol treatment, d - comparison between after enalapril treatment and after atenolol treatment. TBARS – Thiobarbituric Acid Reactive Substances, TAC – Total Anti-Oxidant Capacity, TOS – Total Oxidant Status, OSI – Oxidative Stress Index.

Table 2 shows the oxidative stress indices of control and hypertensive patients before and after treatment with Enalapril and Atenolol. We found that, the essential HTN patient showed elevated levels of TBARS, TOS, calculated OSI and TBARS/TAC with decreased levels of TAC (not significant statistically) when compared to the control group. Treatment with enalapril significantly decreases these oxidative stress parameters and increased the TAC level. Whereas treatment with Atenolol significantly reduces the TBARS, TOS and OSI. No significance difference was found in TAC and TBARS/TAC by Atenolol treatment. In addition, enalapril treated patient showed more significant reduction in the TBARS, TBARS/TAC and increased TAC than Atenolol treated patient, indicating Enalapril more effectively improves oxidative stress than Atenolol.

Table 3: Effect of *Enalapril* (ACE inhibitor) and *Atenolol* (Beta-Blocker) on plasma Lipid Profile parameters in Patients with Essential Hypertension

S. No	Parameters	Control (n=30)	Essential Hypertension (Enalapril Treatment) (n=30)		Essential Hypertension (Atenolol Treatment) (n=28)	
			Before	After	Before	After
1	Total Cholesterol (TC, mg/dL)	162.63 ± 10.88	235.1 ± 30.86[a]	177.23 ± 19.15[bd]	244.77 ± 40.52 [a]	201.17 ± 30.08 [c]
2	Triglycerides (TG, mg/dL)	123.07 ± 13.01	165.7 ± 13.32 [a]	124.07 ± 15.21 [bd]	176.21 ± 12.88 [a]	160.83 ± 14.44 [c]

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3	HDL - cholesterol (HDL-C, mg/dL)	41.9 ± 6.33	33.87 ± 6.22 [a]	40.33 ± 5.82 [b]	35.91 ± 6.6 [a]	33.54 ± 6.34
4	LDL - cholesterol (LDL-C, mg/dL)	96.12 ± 13.51	168.69 ± 31.67 [a]	112.09 ± 19.65 [bd]	173.61 ± 36.75 [a]	135.43 ± 28.49 [c]
5	VLDL - cholesterol (VLDL-C, mg/dL)	24.63 ± 2.6	33.14 ± 2.66 [a]	25.81 ± 3.06 [b]	35.24 ± 2.58 [a]	32.17 ± 2.89

Results were expressed as Mean ± S.D. A [a,b,c,d] P-value less than 0.05 considered statistically significant. a - in comparison with control, b - in comparison with before enalapril treatment, c - in comparison with before atenolol treatment, d - comparison between after enalapril treatment and after atenolol treatment. HDL – High Density Lipoprotein, LDL – Low Density Lipoprotein, VLDL – Very Low-Density Lipoprotein

Table 3 shows that, the plasma total cholesterol, triglycerides, LDL-C were significantly higher, and HDL-C was significantly lower in essential HTN patients than control subjects. Both enalapril and atenolol treatment significantly total cholesterol, triglycerides and LDL-C in HTN patients, with more significant effect was observed in enalapril treatment than atenolol. No significant effect was found on HDL-C levels by atenolol treatment.

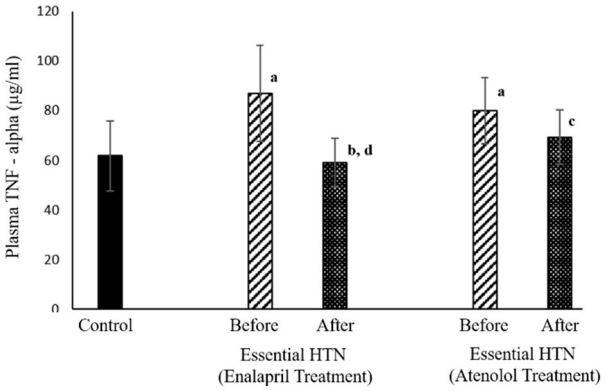
Table 4: Effect of *Enalapril* (ACE inhibitor) and *Atenolol* (Beta-Blocker) on Atherogenic Lipid Risk Indices in Patients with Essential Hypertension

S. No	Parameters	Control (n=30)	Essential Hypertension (Enalapril Treatment) (n=30)		Essential Hypertension (Atenolol Treatment) (n=28)	
			Before	After	Before	After
1	CRI - I	3.99 ± 0.78	7.15 ± 1.47 [a]	4.48 ± 0.81 [bd]	6.98 ± 1.40 [a]	6.15 ± 1.37
2	CRI - II	2.38 ± 0.68	5.14 ± 1.34 [a]	2.85 ± 0.73 [bd]	4.97 ± 1.28 [a]	4.18 ± 1.14
3	AC (TC-HDL/HDL)	2.99 ± 0.78	6.15 ± 1.47 [a]	3.48 ± 0.81 [bd]	5.98 ± 1.40 [a]	5.18 ± 1.31
4	AIP ((log(TG/HDL)	0.47 ± 0.08	0.70 ± 0.08 [a]	0.49 ± 0.09 [b]	0.70 ± 0.08 [a]	0.69 ± 0.09

Results were expressed as Mean ± S.D. A [a,b,c,d] P-value less than 0.05 considered statistically significant. a - in comparison with control, b - in comparison with before enalapril treatment, c - in comparison with before atenolol treatment, d - comparison between after enalapril treatment and after atenolol treatment.

Table 4 shows that, the atherogenic lipid risk indices; CRI-I, CRI-II, AC and AIP were significantly increased in essential HTN patients than control subjects. Treatment with enalapril significantly decreased all the atherogenic lipid risk indices in HTN patients. Whereas no significant effect was found on these parameters by atenolol treatment.

A)



B)

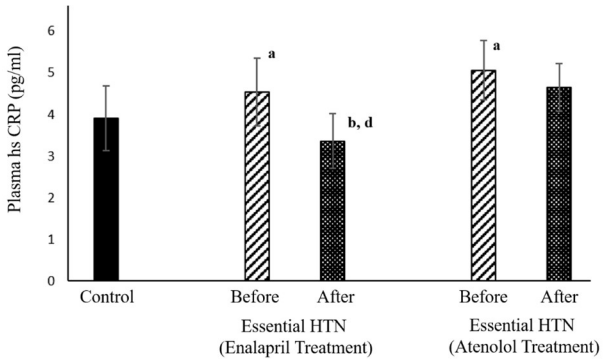


Figure 1:Diagrammatic representation of effects of Enalapril (ACE inhibitor) and Atenolol (Beta-Blocker) on Plasma Inflammatory Markers in Patients with Essential Hypertension.*A) Plasma Tumor Necrosis Factor – Alpha (TNF-α), B) Plasma high sensitive C-reactive protein (hs-CRP). A [a,b,c,d] P-value less than 0.05 considered statistically significant. a – in comparison with Control, b – in comparison with before enalapril treatment, c – in comparison with before atenolol treatment, d – comparison between after enalapril treatment and after atenolol treatment.*

Figure 1 shows that, the plasma inflammatory cytokines such as TNF-α and hsCRP were significantly elevated in essential HTN patients when compared to the control. Treatment with enalapril significantly reduces the TNF-α and hsCRP, whereas atenolol treatment significantly reduces TNF-α, but not hsCRP levels.

Discussion

Hypertension, an elevated blood pressure is emerging health burden in South Asian populations, caused by cluster of metabolic risk complications/factors such as oxidative stress, vascular dysfunction, inflammatory response. It contributes to premature mortality globally, affecting over a billion individuals, with the estimation of about 1 in 4 men and 1 in 5 women affected [12]. Studies reported that the prevalence of hypertension in India was 27.2% in 2025 and expected to rise 35.0% in 2030, India aims for a 25% reduction in hypertension prevalence by 2025 [12]. Maintaining the normal blood pressure is a critical approach for lowering the incidence of cardiovascular disability, and mortality. Currently, several types of pharmacological strategies are available in the clinical medicine to treat hypertension, with varying effects on metabolic profile. The report states that approximately 80% of individuals with hypertension lack sufficient treatment [13]. While all antihypertensive drugs primarily lower blood pressure, some classes of drugs have favourable effects on metabolic complications; inflammation, atherogenic dyslipidaemia, while others may have adverse effects [5,6]. present study explored the comparative effectiveness/efficacy of*Enalapril* (ACE inhibitor) and *Atenolol* (Beta-blocker) on oxidative stress, inflammatory response and atherogenic dyslipidaemia, a potent risk factors of cardiovascular consequences.

Present study, we found that the essential hypertensive patient had elevated levels of lipid peroxidation (TBARS), total oxidant status (TOS) and oxidative stress index (OSI) with decreased level of total antioxidant capacity (TAC), suggesting the occurrences of pro-oxidant/anti-oxidant imbalance (PAB), with the resultant oxidative stress. Furthermore, the plasma pro-inflammatory cytokines levels: TNFα and hsCRP levels were also elevated significantly in essential HTN patient, indicating the involvement of vascular endothelial dysfunction and

subsequent inflammation response. The possible mechanism(s) is that, the elevated blood pressure observed in the essential HTN might have triggers the Renin-Angiotensin System leading to the up-regulation of angiotensin converting enzyme (ACE) [14]. Which in turn caused increased conversion of Ang-I to Ang-II [14, 15, 16]. Ang-II activates several pro-oxidant enzymes like membrane-bound NADH/NADPH oxidases, which in turn enhance the production of free radicals by several mechanisms, including the increased activation of xanthine oxidase [16], and the inactivation of superoxide dismutase with the resultant stimulation of inflammatory signalling pathways and up-expression of inflammatory cytokines [17].

Treatment with ACE inhibitor (Enalapril) significantly improved oxidative stress and inflammatory response as evidenced by the decreased levels of plasma TBARS, TOS, OSI, PAB and decreased inflammatory cytokines; TNF-α and hsCRP, with the significant increment in the total anti-oxidant capacity. This effect may be due to the inhibition of the enzyme ACE, and subsequent inhibition of Ang-I/NADPH oxidase mediated production of free radicals (oxidative stress). Moreover, treatment with beta blocker (Atenolol) significantly reduces the lipid peroxidation, oxidative stress and TNF-α level, whereas no significance effects were seen in the levels of total anti-oxidant capacity, PAB and hs CRP levels. Interestingly we observed that, the reduction in the oxidative parameters and inflammatory cytokines in association with increment in the anti-oxidant defence capacity is significantly heightened in patients those were treated with enalapril when compared to the patients treated with atenolol. This suggests that, the treatment with *Enalapril* (ACE inhibitor) more effectively diminishes the oxidative stress and its triggered inflammatory response compared to the atenolol treatment (beta-blocker) along with its blood pressure lowering effects.

Recent studies in lipid profiling have developed novel non-traditional biomarkers such as atherogenic index of plasma (AIP), Castelli Risk Indices (CRI-I and CRI-II), and the atherogenic coefficient (AC) [18]; as key indicators of cardiovascular disease (CVD) risk. Among these, AIP demonstrates superior predictive efficacy for dyslipidemia and associated cardiometabolic complications. Meanwhile, CRI-I serves primarily as a surrogate marker for coronary plaque accumulation, whereas CRI-II offers targeted predictive value for broader cardiovascular events based on specific lipid ratios [18, 19].

In the current study, we observed that, the essential hypertensive patients had atherogenic dyslipidemia in comparison to the control group, as proved by higher plasma total cholesterol, triglycerides, and LDL cholesterol levels and lower HDL cholesterol levels. Additionally, it was found that essential HTN patients had significantly higher levels of the atherogenic lipid indices, Castelli's risk index I and II (CRI-I and II), the atherogenic coefficient (AC), and the atherogenic index of plasma (AIP). This suggests that the patient may experience cardiovascular problems in the future, such as coronary atherosclerosis. The results also align with earlier research that documented the interplay between inflammation, dyslipidemia, and hypertension in the development of cardiovascular diseases [20]. Elevated levels of inflammatory cytokines and increased oxidative stress may lead to the oxidation of low-density lipoprotein (LDL) cholesterol, which then modifies the lipid profile. Treatment with enalapril significantly reduced the plasma lipid profile and all the indices of atherogenic dyslipidemia. Whereas those group of essential hypertensive patients treated with atenolol, there is no significant restoration or improvement in the atherogenic lipid risk indices compared to the enalapril. The significant reduction in the oxidative stress and inflammatory cytokines observed in the enalapril treated patient might have reduced the lipid peroxidation, oxidised LDL and its induced lipids de-arrangements. Whereas, atenolol treatment improved oxidative stress and inflammatory response in some extend, but not sufficient to restore atherogenic lipid risk.

Fig.2, shows the schematic representation of possible biochemical and molecular mechanism of effects of Enalapril and Atenolol on Redox-Imbalance (Oxidative Stress), Inflammatory Response and Atherogenic Lipid Risk Indices in Essential Hypertension. Both Enalapril and Atenolol significantly improves oxidative stress, inflammation and atherogenic dyslipidemia, with more prone effect with Enalapril. Atenolol diminishes the blood pressure by reducing heart rate and decreasing cardiac workload, which reduces strain on blood vessels[20]. Whereas, Enalapril reduces the blood pressure by above said mechanisms, in addition to the direct inhibition of ACE and production of potent vasoconstrictor AngII, which in turn inhibits the activation of NADPH oxidase and subsequent production of reactive free radicals, with the resultant oxidative stress, inflammatory response and its induced adverse effects on atherogenic lipid risk indices. But this anti-oxidative, anti-inflammatory and anti-dyslipidemic effects were not observed by the Atenolol.

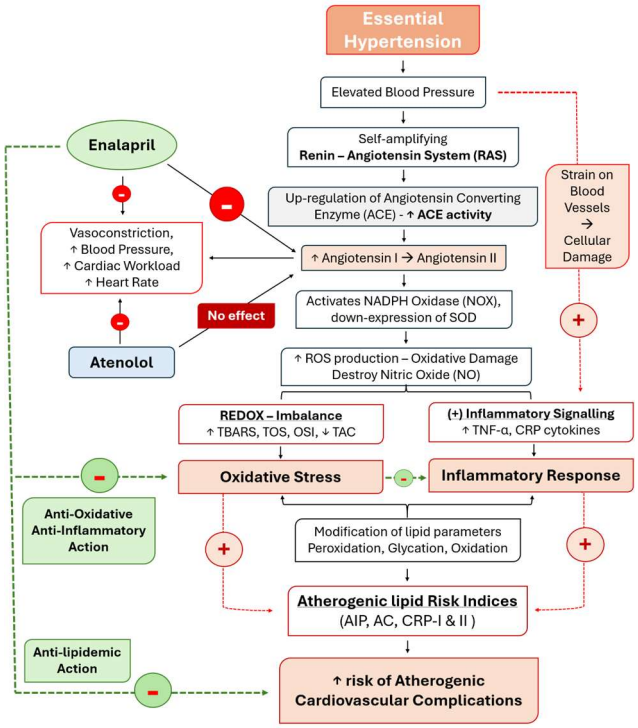


Fig 2: Graphical representation of Possible Biochemical and Molecular mechanisms underlying the of effects of *Enalapril (*ACE Inhibitor) and *Atenolol* (Beta-Blocker) on Redox-Imbalance (Oxidative Stress), Inflammatory Response and Atherogenic Lipid Risk Indices in Essential Hypertension. Red coloured arrows - indicate adverse effects, Red coloured dot arrows - indicate stimulatory adverse effects, Green colored arrow - indicates protective beneficial effects.

Conclusion:

Taken together, our findingsconclude that enalapril therapy is more effective than atenolol therapy in attenuating oxidative stress, suppressing systemic inflammation, and favourably modifying atherogenic lipid profiles in essential hypertension. These findings highlight enalapril's multifaceted pharmacological profile, demonstrating that its therapeutic value extends beyond blood pressure reduction to include marked anti-oxidative, anti-inflammatory, and anti-lipidemic effects.

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