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# Comparative Study of Atenolol And Nebivolol on Hypertension And Heart Rate in Hypertensive Patients

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***Abstract:*** *Hypertension is the leading cause of death and a major risk factor for cardiovascular diseases. Beta blockers play an important role in the reduction of both systolic and diastolic blood pressures. Newer beta blockers like nebivolol bear much of future as mono therapeutic anti hypertensive agents due to their better patient compliance, better efficacy with negligible side effects. A comparative, prospective, randomised open labelled study was conducted in patients with mild to moderate hypertension patients attending medical OPD in OGH, Hyderabad for 6 months. A total of 104 patients were enrolled in the study. Group A contained 58 patients, was advised to take atenolol 50 mg once daily. Group B contained 46 patients, was advised to take nebivolol 5 mg once daily. Patients were followed after 3 and 6 months. On each visit blood pressure and heart rate were recorded. Paired t test to compare within the group and unpaired t test for intergroup analysis was used. In the present study, level of significance is considered to be 0.001. Nebivolol caused lesser reduction in heart rate and had a better antihypertensive effect when compared to atenolol.*

***Keywords:*** *Hypertension, Beta blockers, Atenolol, Nebivolol, Heart rate.*

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## I. Introduction

Essential hypertension accounts for 90% of all cases of hypertension. Hypertension is a major public health problem, being one of the leading causes of death and disability worldwide and a major risk factor for cardiovascular diseases [1]. There is a close relationship between blood pressure levels and the risk of cardiovascular events, strokes, and kidney disease. The risk of these outcomes is lowest at a blood pressure of around 115/75 mm Hg and above 115/75 mm Hg. For each increase of 20 mm Hg in systolic blood pressure or 10 mm Hg in diastolic blood pressure, the risk of major cardiovascular and stroke events doubles [2].

Beta blockers play an important role in the reduction of both systolic and diastolic blood pressures, and can be efficiently used as mono therapeutic agents [3]. Meta analysis done by **B O Carlberg et al.,** have showed that atenolol when compared with other anti hypertensives showed a higher cardiovascular mortality. Though there was no difference in anti hypertensive effect when compared with other drugs, atenolol has shown to increase the risk of stroke [4]. **Laura M Kuyper et al.,** have conducted a Meta analysis in which atenolol have been proved to increase the risk of stroke compared with other anti hypertensives [5]. **Samuelsson et al.,** in their Meta analysis showed that atenolol is ineffective in reducing the cardiovascular mortality and also caused increased mortality in hypertension [6].

Hypertension is sometimes associated with endothelial dysfunction which is caused by production of oxygen free radicals that destroy nitric oxide (NO) and impair its beneficial and protective effects on vessel wall [7]. The endothelial dysfunction is thus a mechanism promoting atherosclerosis and thrombosis, contributing to cardiovascular events. It is now considered as an important target for cardiovascular treatment. The role of beta blockers like atenolol and some older short acting beta blockers seem to offer less protection against such factors. Nebivolol is a selective $\beta_1$-adrenoreceptor blocker that possesses vasodilation property [8]. Nebivolol possesses a distinct hemodynamic profile, including reduced peripheral vascular resistance and negligible impact on cardiac output. Nebivolol also provides significant dose-dependent BP reduction and is considered to be safe and well tolerated.

**Table 1:** Classification of hypertension [9]

| Category | Systolic blood pressure (mm of Hg) | Diastolic blood pressure(mm of Hg) |
|---|---|---|
| Optimal | <120 | <80 |
| Normal | <130 | <85 |

| | | |
|---|---|---|
| High Normal | 130-139 | 85-89 |
| Stage 1(mild hypertension) | 140-159 | 90-99 |
| Stage 2(moderate hypertension) | 160-179 | 100-109 |
| Stage 3 (severe hypertension) | ≥180 | ≥110 |

1.1)Atenolol: Atenolol was discovered by Imperial Chemical Industries (ICI) in 1976, while searching for a specific Beta-1 cardioselective adrenoreceptor blocking agent. Atenolol received approval in the United States August 19, 1981.

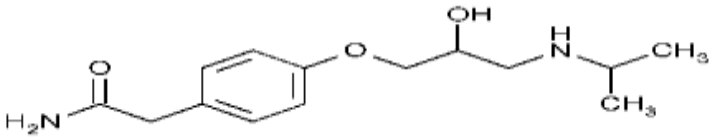


**Fig 1:** Structure of atenolol [10]

Atenolol may defined by IUPAC nomenclature [10]:

- 4-[2'-hydroxy-3'-[(1-methylethyl)amino]propoxy-benzeneacetamide
- Benzeneacetamide, 4-[2'-hydroxy-3'-[(1-methylethyl) amino] propoxy]

It is a hydrophilic drug, with chemical formula $C_{14}H_{22}N_2O_3$. It is freely soluble in strongly acidic solution [11].Atenolol competes with sympathomimetic neurotransmitters such as catecholamines for binding at beta (1)-adrenergic receptors in the heart and vascular smooth muscle, inhibiting sympathetic stimulation. This results in a reduction in resting heart rate, cardiac output, systolic and diastolic blood pressure, and reflex orthostatic hypotension. It has an oral bioavailability of 50% and its protein binding is less than 5%. It is widely distributed, apparent volume of distribution is 50 to 75 L after 5 mg through iv route. Over 90% of the absorbed drug is excreted in unchanged form into urine. The elimination half-life of atenolol is about 6 hr. The drug accumulates in patients with renal failure and dosage should be adjusted for patients whose creatinine clearance is less than 35 mL/min/1.73m$^2$.

1.2)Nebivolol: On December 18th 2007 NEBIVOLOL is approved by FDA for the treatment of Hypertension [12].

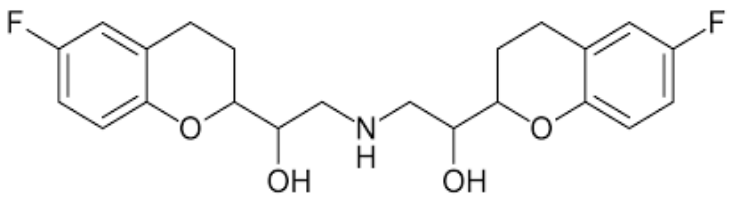


**Fig 2:** Structure of nebivolol [13]

**Iupacname:** 1-(6-fluorochroman-2-yl)-{[2-(6-fluorochroman-2-yl)-2-hydroxy-ethyl]amino} ethanol OR 2,2'-azanediylbis(1-(6-fluorochroman-2-yl)ethanol) [14]. Nebivolol is a highly selective beta 1 adrenoceptor antagonist, lipophilic drug which is devoid of membrane stabilizing and intrinsic sympathomimetic activity [14].Nebivolol is administered as racemic mixture of equal proportions of "d" and "l" isomers. Nebivolol has 4 asymmetric centres, d- isomer refers to (S,R,R,R)-nebivolol and lisomer to (R,S,S,S)-nebivolol. Nebivolol is rapidly absorbed following oral administration, reaching peak plasma concentrations within 0.5–4 hours after a dose. Nebivolol is extensively metabolized via hydroxylation in the hepatic system to active and inactive metabolites. The half-life of nebivolol is approximately 10 hours. Average volume of distribution of nebivolol is 10L/kg. Less than 1% of the drug is excreted unchanged in the urine. Nebivolol is highly protein bound intravascularly, predominately to albumin.

The present study is done to evaluate the effectiveness of nebivolol as a potent anti hypertensive agent by comparing the reduction of blood pressure and heart rate with atenolol in patients with essential hypertension. The present study also emphasizes on evaluating the long term effects of nebivolol in comparison to atenolol.

**2**) **Patients and methods:** Patients with mild to moderate hypertension patients attending medical outpatient department in Osmania General Hospital, Hyderabad were enrolled. A prospective, randomised open labelled and comparative study carried out at osmania general hospital, Hyderabad. Subjects included in the present study are patients with mild to moderate hypertension patients attending medical outpatient department in Osmania General Hospital.

A total of 104 patients are enrolled in the study after meeting the following inclusion and exclusion criteria. Both male and female patients are included in the study, in the age group greater than 40 years. Inclusion criteria: Age group greater than 40 years, Either sex,Patients diagnosed with mild and moderate hypertension.Exclusion criteria: Pregnant and lactating females,Patients with Secondary hypertension, Patients with severe hypertension, Patients with bronchial asthma,Patients with Peripheral arterial disease, Patients with heart block, Patients with Diabetes mellitus, Patients taking any other medication for hypertension, Patients who have not given informed consent.

Approval from Institutional Ethics Committee of Osmania Medical College, Hyderabad was obtained. A proforma containing the detailed information about patient's medical history and general examination was prepared. Demographic profile of the patient, detailed general examination of the patient, medical history of the patient, family history of the patient, and addictions if any were documented. After meeting the above mentioned inclusion and exclusion criteria, a total of 136 patients were enrolled in to the study. The patients were randomised based on the computer based technique. After randomisation they were divided in to two groups, group A and group B. The patients were explained in detail about the purpose of the study, procedure of the study and also explained about the follow up. Written informed consent was taken from the patients who volunteered to participate in the study. Patients of either gender in the age group greater than 40 years were included in the study.

Systolic and diastolic blood pressure was measured in right arm, sitting posture by auscultatory method using standard mercury sphygmomanometer. Two recordings of blood pressure were taken at an interval of 15 min. The heart rate was measured by electrocardiogram. Group A contained 76 patients. Group B contained 60 patients. Group A was advised to take atenolol in the dose of 50 mg once daily. Group B received nebivolol in the dose of 5 mg once daily. The patients were asked to come for follow up after 3 months and after 6 months. 20 patients from group A and 12 patients from group B failed to follow up. Excluding the drop outs, the study was continued with a total number of 104 patients. Group A contained 56 patients and group B contained 48 patients.On each visit blood pressure was recorded. Electrocardiogram was taken to record the heart rate. Adverse effects if any are noted. Data will be presented as mean ± standard deviation and will be analyzed using Student t-test (unpaired t-test) and student t-test (paired t-test) using Graph pad prism version 6.5.
In the present study, p value is taken as < 0.001 – significant, > 0.001 - not significant.

## II. Results

Table 2: Comparison of atenolol and nebivolol on heart rate:

| | Group A(Atenolol 50 mg) | Group B(Nebivolol 5 mg) | P value |
|---|---|---|---|
| Base line | 85.6±2.4 bpm | 84.65±3.6 bpm | * |
| After 3 months | 76.23±2.3 | 77.4±3.3 | * |
| After 6 months | 67.2±2.14 | 70.1±3.1 | ** |

p < 0.001 -- ** significant; p > 0.001 --- * not significant, Test - unpaired student's t test .

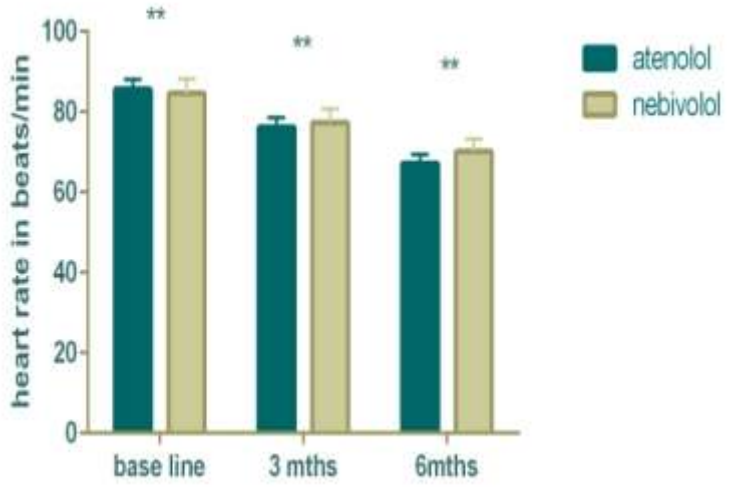


**Fig 3:** Effect of atenolol and nebivolol on heart rate after three and six months

**Table – 3:** Comparison of effect of atenolol and nebivolol on mean reduction of heart rate

| | Group A<br>Atenolol 50 mg | Group B<br>Nebivolol 5 mg | P value |
|---|---|---|---|
| 0 – 3 months | 9.35±2.32 | 7.25±18 | ** |
| 3 - 6 months | 9.69±1.8 | 7.25±2.6 | * |
| 0 – 6 months | 19.05±3.04 | 14.5±3.19 | ** |

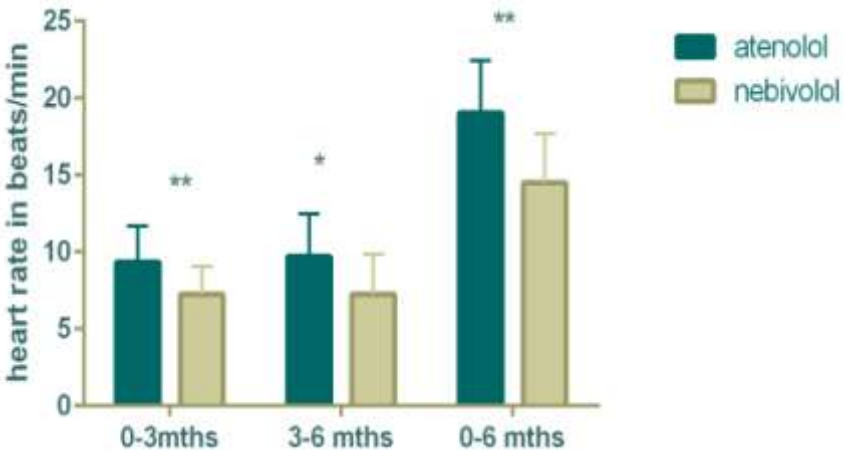


**Fig 4:** Comparison of effect on heart rate by atenolol and nebivolol on mean reduction of heart rate

**Table – 4** Comparison of effect of atenolol and nebivolol on blood pressure

| S no | Parameters | Effect on SBP | | | Effect on DBP | | |
|---|---|---|---|---|---|---|---|
| | | Atenolol | Nebivolol | P value | atenolol | nebivolol | P value |
| 1 | Baseline | 142.48±4.9 | 145.16±8.7 | * | 89.07±5.6 | 88.2±5.73 | * |
| 2 | After 3 months | 137.39±4.7 | 136.83±7.1 | * | 83.57±4.36 | 81.16±4.18 | * |
| 3 | After 6 months | 132.32±4.12 | 131.12±6.5 | ** | 80.25±2.12 | 77.08±4.5 | ** |

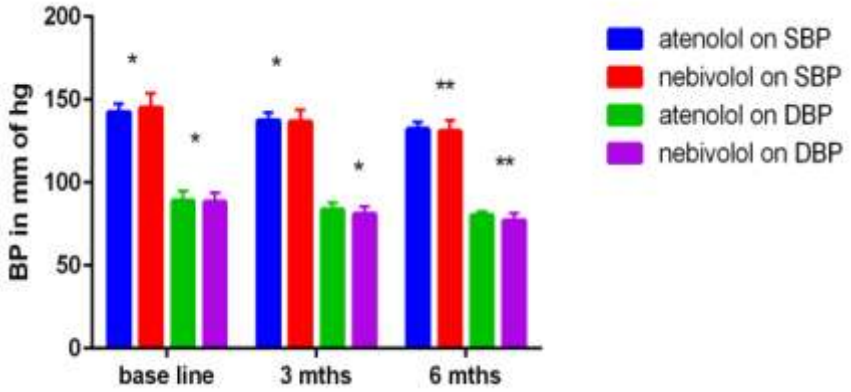


**Fig –5** Comparison of effect of atenolol and nebivolol on blood pressure

**Table 5:** Comparison of effect of atenolol and nebivolol on mean reduction of blood pressure

| S no | Parameters | Mean Reduction of SBP | | | Mean Reduction of DBP | | |
|---|---|---|---|---|---|---|---|
| | | atenolol | Nebivolol | P value | atenolol | nebivolol | P value |
| 1 | 0 – 3 months | 5.39±2.95 | 8.33±4.74 | * | 5.51±3.21 | 7.04±5.21 | * |
| 2 | 3 – 6 months | 5.07±2.05 | 5.70±4.6 | * | 3.32±1.15 | 4.08±5.23 | * |
| 3 | 0 – 6 months | 10.04±3.07 | 14.04±5.75 | ** | 8.82±4.66 | 11.12±6.65 | ** |

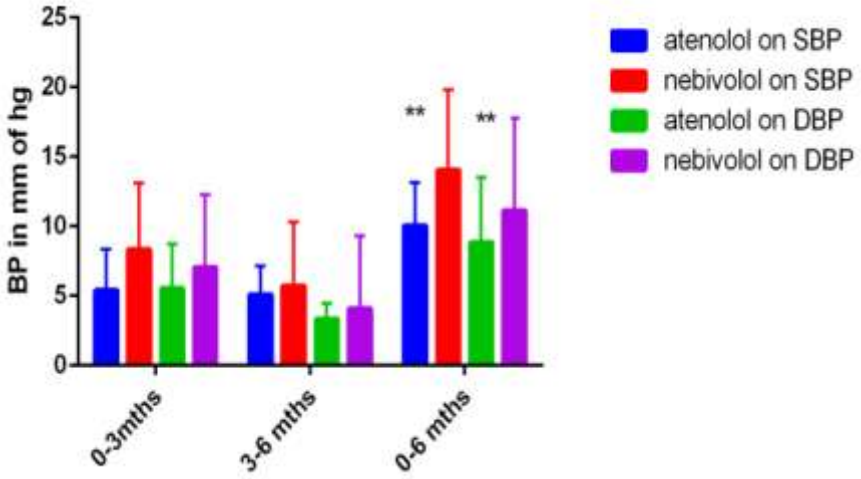


**Fig 6:** Comparison of effect of atenolol and nebivolol on mean reduction of blood pressure.

**Table 6:** Effect of Atenolol on SBP, DBP and heart rate at 0 and 6 months

| Atenolol | SBP ( mm of hg) | DBP (mm of hg) | Heart rate (beats/min) |
|---|---|---|---|
| Base line | 142.48±4.9 | 89.07±5.6 | 85.6±2.4 |
| Six months | 132.32±4.12 | 80.25±2.12 | 67.2±2.14 |
| p value | ** | ** | ** |

p value < 0.001- ** significant**;** p value > 0.001 - * not significant**,** Test – paired student's t test

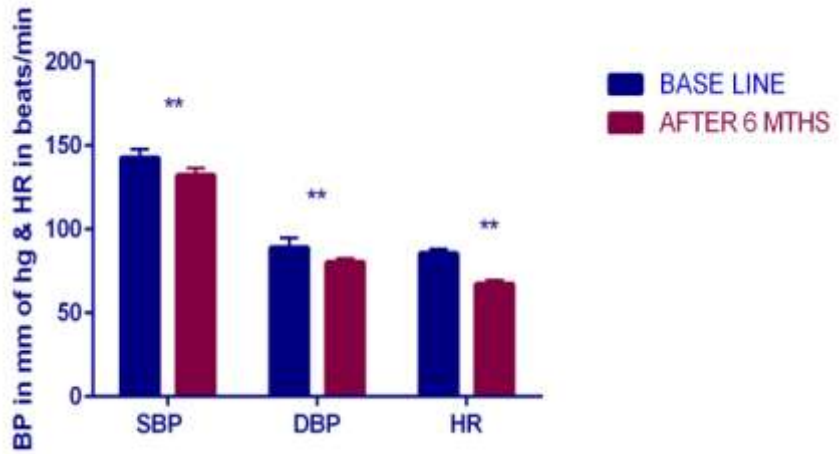


Fig 7: Effect of Atenolol on SBP, DBP and heart rate at 0 and 6 months

**Table7 -** Effect of Nebivolol on SBP, DBP and heart rate at 0 and after 6 months.

| Nebivolol | SBP( mm of hg) | DBP(mm of hg) | Heart rate (beats/min) |
|---|---|---|---|
| Base line | 145.16±8.7 | 88.2±5.73 | 84.65±2.4 |
| Six months | 131.12±6.5 | 77.08±4.5 | 70.1±3.1 |
| p value | ** | ** | ** |

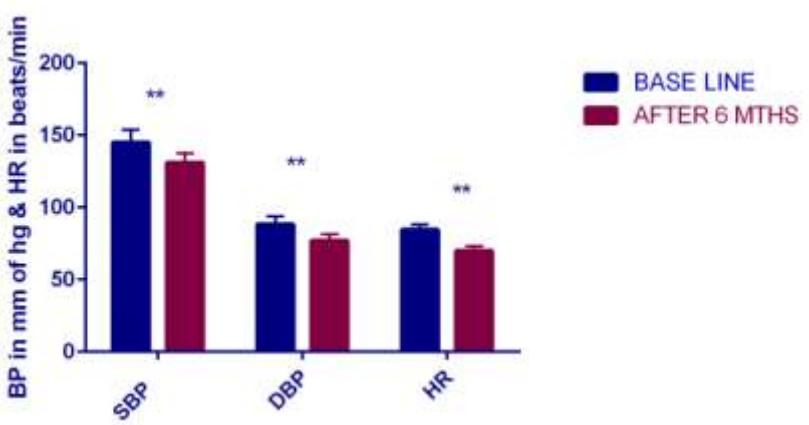


**Fig 8-** Effect of Nebivolol on SBP, DBP and heart rate at 0 and after 6 months.

## III. Discussion

Hypertension, if not detected or treated earlier, very often leads to lethal complications with involvement of target organs resulting in the enhancement of mortality and morbidity. Cardiovascular morbidity and mortality increases as both SBP and DBP rise [1]. Essential hypertension is associated with endothelial dysfunction, which is caused mainly by the production of oxygen free radicals that destroy NO and impair protective effects on the vessel wall. This impairment of protective mechanism of NO on endothelium is known to cause atherosclerosis. It is now considered as an important target for cardiovascular treatment [14]. Nebivolol is a selective $\beta_1$-adrenoreceptor blocker that possesses vasodilatation property. Nebivolol also provided significant dose-dependent BP reduction and was safe and well tolerated [15]. Antihypertensive response rates were higher with nebivolol than with other anti-hypertensive drugs.

**Following parameters were studied:**

Effect of atenolol and nebivolol on heart rate. Effect of atenolol and nebivolol on blood pressure. 4.1) Effect on heart rate: Beta blockers decrease the sympathetic activity and accelerate the parasympathetic activity by a reflex phenomenon which results in decreased heart rate. It has been observed in the previous studies that nebivolol attenuates the sympathetic tone, but does not promote vagal activity more than atenolol. Hence, fall in the heart rate with nebivolol is less when compared to atenolol. Nebivolol causes lesser beta 1 blockade compared to atenolol, and thus contributes to its lesser bradycardia response when compared to atenolol [16].

In the present study, the mean baseline values of heart rate in atenolol group was 85.6±2.41 bpm, 76.23±2.32 bpm at three months and 67.21±2.14 bpm at the end of six months. The mean reduction in heart rate in the atenolol group at the end of three months is 9.35 ± 2.32 bpm and at the end of six months is 19.05 ± 3.04 bpm.

The mean baseline value of heart rate in nebivolol group is 84.65±3.61 bpm, 77.43 ± 3.31 bpm at the end of three months and 70.12 ± 3.12 bpm at the end of six months.The mean reduction in heart rate in nebivolol group at the end of three months is 7.25 ± 1.8 bpm and at the end of six months is 14.51 ± 3.19 bpm.

When compared between the two groups the mean reduction of heart rate in atenolol is 19.05±3.04 bpm which is more than the nebivolol group which is 14.51±3.19 bpm. The p value is determined by unpaired t test is < 0.001 which is considered to be statistically significant.

**V A Badar et al.,** conducted a similar study in about 60 patients and showed the significant reduction of heart rate at the end six months. The reduction in heart rate in atenolol group was from 74.13±1 to 60.8±0.61. And in nebivolol group it was from 73.33±1.03 to 65.33±0.79. The reduction in heart rate was significant ($p < 0.001$) [17].The mean reduction of heart rate was 13.33±0.84 in atenolol group. And 8±0.73 in nebivolol group which was considered to be statistically significant ($p< 0.001$). This study supports our observation, that the reduction in heart rate atenolol group was more when compared to nebivolol group.

**G N Sahana et al.,** conducted a similar study which, when compared both drugs showed significant decrease in heart rate. The mean reduction in heart rate in nebivolol group is 14.51 ± 4.69 and in atenolol group was 17.55 ± 5.6 which was considered to be statistically significant ($p<0.0001$) [18].The study supported our observation that atenolol has significantly higher bradycardia response ($p<0.001$).

In a study conducted by **Poirier et al.,** in diabetic hypertensive patients, heart rate was reduced to a greater extent in the atenolol group when compared to nebivolol group. p value is < 0.05 which is considered to be statistically significant. Though this study was conducted in hypertensive patients with impaired glucose tolerance, this study supports our observation that nebivolol may have a better tolerability profile than atenolol in impaired glucose tolerance [19].

In a multicentre study conducted by **Grassi et al**., the efficacy of atenolol and nebivolol are compared in mild and moderate hypertensive patients. The study was conducted for three months. Heart rate was significantly ($p<0.05$) reduced by both drugs, the bradycardia response induced by nebivolol treatment being significantly ($p<0.005$) less than atenolol. The study supports our study in showing that the bradycardia response of atenolol and nebivolol was comparable and the difference was statistically significant even after 12 weeks of treatment.

**Vivek V Bhosle et al.,** conducted a study in which nebivolol and atenolol reduced heart rate significantly. The study supported our observation, that the mean reduction in heart rate in nebivolol group was 14.51±4.69 bpm while in atenolol group was 17.55±5.06 bpm. At the end of 12 weeks of treatment both the drugs reduced heart rate but the reduction in heart rate was more with atenolol, which was considered to be statistically significant ($p<0.005$). The study supports the present study in concluding, that the bradycardia response of both drugs is comparable at the end of three months of treatment [21].

4.2) Effect on blood pressure: The present study states that both the drugs have shown significant reduction in blood pressure (both systolic and diastolic) at the end of three months and at the end of six months. The mean reduction in SBP and DBP by both the drugs when compared is not statistically significant at the end of three months. But by the end of six months it is considered to be statistically significant ($p<0.001$).

In the present study, the reduction in systolic blood pressure by atenolol by the end of three months was from 142.78±4.91mm of Hg (baseline value) to 137.39 ± 4.71mm of Hg. And it was reduced to 132.32±4.12mm of Hg by the end of six months. The reduction in systolic blood pressure is considered to be statistically significant ($p<0.001$). The reduction in diastolic blood pressure by atenolol by the end of three months was from 89.07±5.61mm of Hg (baseline value) to 83.57±4.36. mm of Hg and it was reduced to 80.25±2.12mm of Hg by the end of six months. The reduction in diastolic blood pressure is considered to be statistically significant ($p<0.001$).

The reduction in systolic blood pressure by nebivolol by the end of three months was from 145.16±8.71mm of Hg (baseline value) to 136.83±7.10mm of Hg. And it was reduced to 131.12±6.52mm of Hg by the end of six months. The reduction in systolic blood pressure is considered to be statistically significant ($p<0.001$).The reduction in diastolic blood pressure by nebivolol by the end of three months was from 88.20±5.73mm of Hg (baseline value) to 81.16±4.18mm of Hg. And it was reduced to 77.08±4.52 mm of Hg by the end of six months. The reduction in diastolic blood pressure is considered to be statistically significant ($p<0.001$).

The mean reduction in blood pressure, when compared between both the groups, it was found to be statistically insignificant at the end of three months. But at the end of six months it is considered to be statistically significant ($p<0.001$). The anti hypertensive effect of nebivolol is more when compared to atenolol.

**VA Badar et al.,** conducted a prospective randomised trial for six months in which they compared the effect of atenolol and nebivolol on blood pressure. The reduction in SBP was 160.13±1.62 mm of Hg to 118.93±0.97 mm of Hg in the atenolol group. And in the nebivolol group, the reduction in SBP was from 159.93±1.62 mm of Hg to 116.73±0.91 mm of Hg. And reduction in DBP was 97.13±1.06 mm of Hg to 81.13±0.78 is mm of Hg in the atenolol group. And in the nebivolol group it was 98.27±1.09 mm of Hg to 79.66±0.53 mm of Hg. The study supported our observation that both the drugs showed statistically significant reduction in SBP and DBP .

In a study conducted by **Vivek B Bhosle et al.** the reduction in SBP in nebivolol group was 151.53±10.4 mm of Hg to 134.25±4.6 mm of Hg. And in atenolol group it was from 153.63±8.4 mm of Hg to 136.73±6.08 mm of Hg after three months .The reduction in DBP in nebivolol group was from 97.53±2.4 mm of Hg to 86.76±2.64 mm of Hg. And in atenolol group it was from 97.89±3.47 mm of Hg to 87.84±4.06 mm of Hg after three months. The study supports our observation that both the drugs had significant anti hypertensive effect at the end of three months. But when the antihypertensive effect of two drugs is compared, there is no statistical significance ($p>0.001$).

**G N Sahana et al.,** conducted a similar study for three months in which they showed that both atenolol and nebivolol had significant anti hypertensive effect but when compared there is no statistical significance between the two drugs ($p> 0.001$).The reduction in SBP in nebivolol group was from 158±17 mm of Hg to 118±8 mm of Hg and in atenolol group was from 160±16 mm of Hg to 115±7 mm of Hg after three months. The reduction in DBP in nebivolol was from 97±10 mm of Hg to 71±3 mm of Hg and in atenolol group was from 99±10 mm of Hg to 71±3.The present study also proves that there is no statistical significance in the anti hypertensive effect of the two drugs at the end of three months.

In a multicentre study conducted by **Grassi et al.,** the efficacy of atenolol and nebivolol are compared in mild and moderate hypertensive patients. The study was conducted for three months. The study supports our study by saying that the two drugs show significant anti hypertensive effect at the end of three months. The reduction in SBP and DBP was 18.12±14.10 mm of Hg and 14.6±7.9 mm of Hg in atenolol group. And it was 19.1 ±12.9 mm of Hg and 14.8±7.1 mm of Hg for nebivolol group [20].

The present study is supported by the finding of **Porrier et al.,** who compared the anti hypertensive effect of atenolol and nebivolol. In their study nebivolol showed significant anti hypertensive effect. The reduction in SBP was about 15 mm of Hg and the reduction in DBP was about 10 mm of Hg .
The reduction in blood pressure was considered to be significant ($p<0.001$). And nebivolol can be efficiently used in patients with heart failure. The study supports our study in showing significant anti hypertensive effect of nebivolol and atenolol.

Nebivolol is a cardio selective beta 1 blocker. It achieves blood pressure control by β1 blockade and stimulation of nitric oxide release, which leads to vasodilatation and is associated with reduction in peripheral vascular resistance. It has been hypothesized that nebivolol and its metabolites increase the activity of NO synthase enzyme III (NOS III) which increases the synthesis of NO. The improved secretion of NO and antioxidant property of nebivolol helps in the maintenance of normal endothelial functions [20]. Thus nebivolol has important therapeutic implications in protecting the cardiovascular system from atherosclerotic complications such as hypertension. Selectivity to beta 1 receptors and NO release makes nebivolol an effective anti hypertensive agent. Atenolol has also been proved to be an effective cardio selective beta blocker, but lacks the NO releasing property and vaso dilatory property as that of nebivolol. In this regard nebivolol offers higher advantage over atenolol as antihypertensive agent.

Nebivolol binds to the β receptor on cell membrane leading to activation of adenyl cyclase resulting in accumulation secondary messenger cAMP. Nebivolol induces nitric oxide production via activation of β3 adrenergic receptors. This activates phospholipase C, which breaks down the membrane phospholipid PIP (Phosphotidyl 2 inositol bisphosphate) to IP (Inositol triphosphate) and DAG 3 (Diacyl-glycerol) releases calcium from endoplasmic reticulum producing an increase in free cytoplasmic calcium which binds to calmodulin, this calcium-calmodulin complex is responsible for stimulating nitric oxide synthase (NOS), which acts as a catalyst.

- It has a protective effect on left ventricular function. It reduces preload, afterload and increases stroke volume. It decreases pre-ejection period and lengthens left ventricular ejection time. It reduces cardiac output and total peripheral resistance when given at the dose of 5mg once daily.
- Decreases resting heart rate and reduces exercise induced tachycardia.
- Reduces total cholesterol and low density lipoprotein levels.
- Reduces plasma renin and aldosterone levels.

## IV. Summary

The baseline value of heart rate in group A was 85.6±2.4 bpm. After 3 months it was 76.23±2.3 bpm. And after 6 months it was 67.2±2.14 bpm. The baseline value of heart rate in group B was 84.65±3.6 bpm. After 3 months it was 77.4±3.3 bpm. And after 6 months it was 70.1±3.1 bpm.

When compared between the two groups by student's(unpaired )t test, the reduction in heart rate in group A was more than group B and it was statistically significant ($p<0.001$).

The baseline value of SBP in group A was 142.48±4.9 mm of Hg. After 3 months it was 137.39±4.7 mm of Hg. And after 6 months it was 132.32±4.2 mm of Hg. The baseline value of DBP in group A was 89.07±5.6 mm of Hg. It was 83.57±4.36 mm of Hg at the end of 3 months, and it was 80.25±2.12 mm of Hg at the end of 6 months.The baseline value of SBP in group B was 145.16±8.7 mm of hg. After 3 months it was 136.83±7.1 mm of Hg. And after 6 months it was 131.12±6.5 mm of Hg. The baseline value of DBP in group B was 88.2±5.73 mm of Hg. It was 81.16±4.18 mm of Hg at the end of 3 months, and it was 77.08±4.5 mm of Hg at the end of 6 months.

On comparison the reduction in blood pressure in group B is more than group A. The reduction is statistically significant at the end of 6 months ($p<0.001$).
From the above observations it can be proved that nebivolol (5 mg) is better than atenolol (50 mg) in terms of lesser reduction in heart rate.

## V. Conclusion

The present study concludes that nebivolol which is a newer beta blocker, is an effective anti hypertensive than atenolol with favourable profile on heart rate. The present study included the age group of 45 to 60 years which is considered to be the potential target of essential hypertension. And included only newly diagnosed hypertension cases, so that the effect of drugs can be seen without any interactions with other anti hypertensive drugs. The present study has been done for six months which provides sufficient time to evaluate the anti hypertensive effect. It helped to compare the drugs, unlike the previous studies mentioned, which were conducted for a shorter period. In present study blinding was not done in the study so as to avoid the observer bias. Sample size was small due to which the extrapolation of the results obtained to the general population becomes difficult. The study included the patients of mild and moderate hypertension and did not include severe hypertension patients, so the results obtained cannot be extrapolated to severe hypertension cases. The sample

size in the present study was small to extrapolate the results to a larger population. Therefore studies with larger sample sizes are needed to be done to bring out the effectiveness of nebivolol as a better antihypertensive agent.

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