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Original Article

Effectiveness of Atenolol on the Basis of Pattern of Side Effects in Hypertensive Patients

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ABSTRACT

Hypertension plays a significant role in changing Insulin parameter. They are taken as valid diagnostic markers in determining side effects of anti-hypertensive drugs. These parameters are grossly affected by the use of beta blockers which are used to control hypertension. **Objective:** To determine the effects of atenolol, a beta blocker on insulin when taking alone in hypertensive patients. **Methods:** A total of 80 patients participated in this study of which 43 patients were taking atenolol alone while 37 were taking atenolol in combination with other anti-hypertensive drugs and 20 healthy controls were included of all age groups population to make a comparison and to find variation in values of insulin levels, in patients experiencing hypertension. Medical efficacy was evaluated on the basis of variations in insulin levels upon use of anti-hypertensive medications. ELISA technique was used for conducting insulin levels. **Results:** We found significant results of insulin values because atenolol cause hyperglycemia Atenolol was non-significant in both groups of patients having low insulin levels due to use of atenolol alone or use of atenolol in combination therapy. Other anti-hypertensive drugs did not affect the insulin levels therefore variation is basically because of atenolol so the main focus of our study was atenolol. **Conclusions:** Atenolol prove efficacy but it also causes the disturbance in insulin levels therefore we recommend use of any other drug in conjunction with atenolol to avoid insulin variation due to atenolol. Further these results may be employed on large patient population for strengthening our evidences.

INTRODUCTION

According to Chobanian, hypertension (HTN) is a chronic medical disorder marked by elevated blood pressure in the arteries [1]. The heart has to work harder to pump blood through the blood arteries, which puts more strain on it. Systolic and diastolic readings, which represent the heart's contraction and relaxation stages between beats, are used to calculate blood pressure. The average range of normal resting blood pressure is 60-90mmHg diastolic and 100-140mmHg systolic. Hypertension is characterized by a blood pressure level that is consistently at or higher than 140/90 mmHg [2]. However, rather than being completely attributable to high blood pressure itself, these symptoms are more likely linked to concomitant worry [3]. Numerous pieces of evidence suggest that genetic factors have a role in controlling blood pressure [4]. Blood pressure between related people is more similar than it is between unrelated people, indicating that there may be some type of heredity [5]. Mendelian variations of high and low blood pressure have occasionally been linked to a single gene mutation [6]. According to Lifton *et al.*, and Wilson *et al.*, these types of hypertensions are caused by about 10 genes [7, 8]. By altering renal salt processing, these mutations interfere with the control of blood pressure [9]. The control of pressure, volume, and chemoreceptor signals is another

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important function of the autonomic nervous system in preserving cardiovascular homeostasis [10]. The peripheral vasculature and kidneys are modulated to do this, which results in an increase in cardiac output, vascular resistance, and fluid retention [11]. Blood pressure elevation and the onset and maintenance of hypertension are caused by sympathetic nervous system dysfunction, which is characterized by overactivity [12]. Renin-angiotensin-aldosterone regulation of blood pressure is another function of this system. Renin is an enzyme that contributes to the maintenance of arterial vasoconstriction and extracellular volume. It does this by converting the liver's angiotensinogen into the peptide angiotensin I. Angiotensin-converting enzyme (ACE), which is largely found in the pulmonary circulation and is linked to endothelium, further cleaves angiotensin I to create angiotensin II, the most powerful vasoactive peptide [13]. According to Singh and Haldar, angiotensin II is a powerful blood artery constrictor that raises blood pressure and increases peripheral resistance [14]. Additionally, angiotensin II stimulates the adrenal glands to generate aldosterone, which causes kidney epithelial cells to improve salt and water reabsorption, resulting in an increase in blood volume and a rise in blood pressure. As a result, high blood levels of renin, which typically range from 1.98 to 24.6 ng/L in adults sitting erect, can cause hypertension [15]

METHODS

In our case-control study, a total of 80 cases were selected with hypertension taking atenolol alone or in combination and 20 with blood pressure within normal range as control. Patients were selected after taking complete history and medical as well as physical examination who visited Punjab Institute of Cardiology Lahore for the evaluation of hypertension status. Those with hypertension taking atenolol alone or in combination were selected and their insulin levels were checked. All positive hypertensive patients were included and those on multidrug therapy and with concomitant disorder were excluded in this study. Those with normal blood pressure were taken as control for comparison. After collection of 5 cc blood from these hypertensive patients, blood was centrifuged and serum were separated at 3000 rpm for 5 minutes at room temperature and then insulin test was done and effects of medicines were recorded on hypertension profiling. Simple random sampling technique was employed to collect the sample. Informed consent was filled and signed by the subject on the consent form for the collection of blood sample. Relevant history and general physical examination were recorded on the performa (Annexure- B). The lab reports of insulin report indicate whether to include them in the study or take them as controls. The cases that were within our criteria of study, blood was drawn from these patients through 5cc disposable syringe by random sampling. Blood was drawn from cubical vein of the fore-arm and a period of 6 month was employed to collect samples and conduct the study. A sample of 80 individuals of hypertension taking atenolol and 20 with normal blood pressure was taken as a control. Insulin microplate ELISA test is intended to be used for the quantitative determination of insulin levels in human serum. SPSS version-20 was used for result analysis.

RESULTS

The assessment of insulin levels was done on the hypertensive people who made up the control group. Each participant's gender, age, and insulin levels (measured in U/L) are included in the data (Table 1).

Table 1: Evaluation of Insulin in control population

Sr. No.	Gender	Age	Insulin (0.7-9µU/L)
1	Male	35	2.6
2	Male	45	4.7
3	Male	37	3.9
4	Male	48	6.4
5	Male	51	7.5
6	Male	36	5
7	Male	42	4.3
8	Male	41	4.5
9	Male	55	5.4
10	Male	34	3.2
11	Female	35	0.9
12	Female	44	3.6
13	Female	45	5.6
14	Female	46	4.5
15	Female	44	5.6
16	Female	55	6.8
17	Female	35	1.6
18	Female	33	2.6
19	Female	57	6.2
20	Female	42	5.6

An analysis of insulin levels in hypertensive individuals using just atenolol is shown in Table 2. The table contains information about the subjects' gender, age, administration period, atenolol dosage (in milligrammes), and insulin concentrations (measured in U/L).

Table 2: Evaluation of insulin in hypertensive patients taking atenolol alone

Sr. No.	Gender	Age	Duration of Administration	Dose (mg)	Insulin (0.7-9µU/L)
1	Female	34	2 years 2 months	100	-0.48
2	Female	35	1 week	50	0.03
3	Female	38	8 years	50	4.84
4	Female	38	1 years	50	11.42
5	Male	39	4 months	100	-0.23
6	Female	42	1 year 1 month	50	8
7	Female	43	7 months	50	-0.35
8	Female	43	7 years	100	2.05

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Sr. No.	Gender	Age	Duration of Administration	Dose (mg)	Insulin (0.7-9µU/L)
9	Female	45	1 week	100	2.94
10	Male	45	9 months	100	4.84
11	Female	45	4 months	100	-3.27
12	Male	47	6 months	25	56.99
13	Male	47	1 year	100	16.99
14	Female	47	1 year 3 months	100	-0.35
15	Female	48	2 years 10 months	100	17.87
16	Female	48	1 year 1 month	50	4.46
17	Male	48	7 months	25	-1.37
18	Male	50	1 year 3 months	100	5.22
19	Male	50	1 year 1 month	50	-1.37
20	Female	50	3 years	100	14.33
21	Female	50	1 year 2 months	50	4.58
22	Female	50	6 months	100	6.73
23	Female	50	5 months	50	4.71
24	Female	51	7 months	20	9.77
25	Female	52	9 months	100	14.58
26	Female	54	1 year 3 months	50	2.3
27	Male	56	2 years	50	3.19
28	Male	60	1 month	20	7.49
29	Male	60	1 month	25	7.49
30	Female	60	6 years 1 months	50	1.42
31	Female	60	10 months	100	1.42
32	Female	60	2 years 2 months	100	5.97
33	Female	60	1 year 9 months	50	8.51
34	Female	60	5 months	100	13.82
35	Female	60	7 years	50	5.09
36	Male	61	7 years	100	10.28
37	Female	62	4 months	100	4.46
38	Male	65	2 years 7 months	50	1.67
39	Female	67	6 months	50	-0.61
40	Male	70	1 year	50	1.67
41	Female	70	5 months	100	3.19
42	Female	70	1 year 2 months	50	22.3
43	Male	77	2 weeks	100	10.91

In figure 1 20% control and remaining diseased population is shown with 42 % patients with normal insulin values, 22 % with higher insulin values and 16% with lower insulin values.

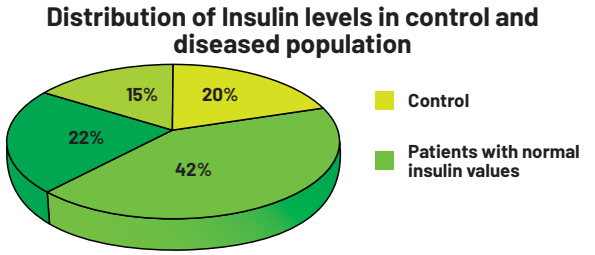

Figure 1: Distribution of Insulin levels in control and diseased population

DISCUSSION

The results of this study and its comparison with earlier studies in the area are the focus of the discussion on the effects of atenolol, a beta blocker, on insulin when taken alone in hypertensive individuals. We can better understand the effect of atenolol on insulin regulation and its implications for controlling hypertension by analyzing and contrasting the data. According to the results of the current study, hypertensive patients who received atenolol as a stand-alone therapy saw considerable changes in their insulin levels, mostly as a result of the onset of hyperglycemia [15]. These results are consistent with other studies that suggested beta blockers, such as atenolol, may contribute to abnormalities in insulin control and glucose metabolism. The impact of beta blockers on insulin and glucose metabolism has been investigated in a number of research. For instance, research by Palatini and Julius found that nonselective beta blockers like propranolol can cause reduced glucose tolerance and insulin resistance [16]. Comparably, research by Bühler *et al.*, found that beta-blocker-treated hypertension individuals had higher insulin levels and worse insulin sensitivity [17, 18]. These findings and others support the idea that beta blockers may have a negative impact on insulin control. In contrast to other research, the current study paid particular attention to atenolol's impact on insulin levels in hypertensive individuals. According to the study, atenolol alone was linked to considerable fluctuations in insulin levels, which is consistent with beta blockers' overall effects on the control of insulin. However, it is notable that regardless of whether patients were receiving atenolol alone or in combination treatment, the effects of atenolol on insulin were non-significant among those who already had low insulin levels. It is important to recognize its limitations and take into account the larger body of research in this field since the current study offers insightful information on the effects of atenolol on insulin in hypertensive individuals. First off, the study only evaluated insulin levels; it did not evaluate other indicators of glucose metabolism, such as insulin resistance or glucose tolerance [19, 20]. Future research might look into these areas to have a more complete knowledge of atenolol's impact on glucose control. Second, with just 80 participants, the sample size of the current study was somewhat small. This restricts the findings' applicability to other contexts and necessitates further extensive research to confirm and support the findings. Furthermore, the study did not take into account variables that can affect how atenolol affects insulin levels, such as the length of atenolol administration, dose fluctuations, or unique patient features.

CONCLUSIONS

Atenolol prove efficacy but it also causes the disturbance in insulin levels therefore we recommend use of any other drug in conjunction with atenolol to avoid insulin variation due to atenolol. Further these results may be employed on

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large patient population for strengthening our evidences.

Authors Contribution

Conceptualization: MFS
Methodology: MR, MFS
Formal analysis: SS
Writing-review and editing: MFS, SS

All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

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