International Journal of Research in Health Sciences
ISSN: 2321-7251
Available online at: http://www.ijrhs.org/
Original Article

Influence of obesity On Antihypertensive Therapy

NS Neki[1], Neeraj Joshi[2], Jaswinder Singh[2], Pratibha Singla[3]

[1]Professor &Head, [2]Junior Resident and [3]Intern, Department of Internal Medicine, Government Medical College & Guru Nanak Dev Hospital, Amritsar, India 143001.

Received: 12-11-2017 / Revised Accepted: 04-01-2018 / Published: 10-01-2018

ABSTRACT

Aim: To study the effect of obesity in the management of hypertension with ramipril and hydrochlorthiazide as compared to non-obese objects.

Methodology: The study was undertaken in the department of Medicine, Govt. Medical College, Amritsar, India after taking permission from the ethical committee. This was an open study where the patient, the observer and the supervisor had clear knowledge of an anti-hypertensive drug used, which will avoid any physiological trauma to the patient as to what therapeutical trial is being done on him/her by using an unknown drug. 100 age and sex matched patients, 50 obese and 50 non obese, with both stage 1 and stage 2 hypertension (according to JNC 8 classification of hypertension), were selected at random from the outpatients clinics and in-door ward.

Data for the above mentioned parameters were compiled, tabulated and statistically analysed for their significance. Utilising the student 't' test, 'p' values were determined to finally evaluate the levels of significance. 'p' value of <0.05 were considered significant. The relevance of the results in the light of stastical analysis are displayed and discussed.

Results: Baseline mean systolic BP in obese patients was 172.4± 7.08 .At the end of 6th week fall in systolic blood pressure was 135.5±9.31with a mean fall 36.96±10.2 (p<0.001: highly significant). Baseline mean systolic BP in non-obese patients was 165.2± 14.22. At the end of 6th week fall in systolic blood pressure was 111.8±5.22 with a mean fall 53.40±11.8 (p<0.001: highly significant). Baseline mean diastolic BP in obese patients was 100.1± 4.54. At the end of 6th week fall in diastolic blood pressure was 70.08± 3.92 with a mean fall 22.04± 5.83 (p<0.001: highly significant). Baseline mean diastolic BP in non-obese patients was 98.04± 8.14. At the end of 6th week fall in diastolic blood pressure was 62.76± 3.89 with a mean fall 35.28± 6.72 (p<0.001: highly significant). Baseline MAP (mean arterial pressure) in obese patients was 124.2± 4.44 (in supine position). At the end of 6th week fall in MAP was 97.21± 4.82 with a mean fall in MAP 27.01 ± 6.39 (p<0.001: highly significant). Baseline MAP (mean arterial pressure) in non-obese patients was 120.4± 9.90 (in supine position). At the end of 6th week fall in MAP was 79.12± 3.96 with a mean fall in MAP 41.32 ± 7.88 (p<0.001: highly significant).

Conclusion: There was fall in systolic, diastolic and mean arterial blood pressure in both age groups at the end of 6 weeks with a mean fall observed more in non-obese hypertensives than obese hypertensives. Average dose of ramipril required is more in obese hypertensives than in non-obese hypertensives to control the blood pressure. Average dose of hydrochlorthiazide required is more in obese hypertensives than in non-obese hypertensives to control the blood pressure. Treatment with angiotensin-converting enzyme inhibitors may show greater efficacy as monotherapy at lower doses compared with thiazide diuretics.

Key Words: Obesity, Hypertension, Ramipril, Hydrochlorthiazide.

Address for Correspondence: Dr. Neeraj Joshi, Junior Resident, Dept. of Medicine, Govt. Medical College, Amritsar, India; E-mail: joshineeraj941@gmail.com

How to Cite this Article: N.S Neki, Neeraj Joshi, Jaswinder Singh, Pratibha Singla. Influence of obesity On Antihypertensive Therapy. Int J Res Health Sci 2018; 6(1): 23-30.

INTRODUCTION

Hypertension is currently among the leading causes of morbidity and mortality in the world and is expected to have an even greater impact on health of the public as more of the world becomes developed. High blood pressure is a powerful risk factor for cardiovascular, cerebrovascular and renal diseases.[1]

Despite progress in hypertension control that has been noted in the United States over the years, the goal of Healthy People 2020 (61.2% by 2020) has not been met.[2] currently, just less than one-half of adults with hypertension have their hypertension under control (48.3%).

Obesity defined as a body mass index (BMI) greater than 30kg/m^2 ,is an important risk factor for hypertension, and excess adiposites believed to account for 70%; of hypertension in men and 60%; in women.[3,4] There is now accumulating evidence that adipose tissue itself be involved directly in the pathogenesis of hypertension, not only by contributing to an increase in sympathetic activity[5], but also to sodium and volume retention.[6]

Current guidelines for the management of hypertension provide a specific recommendations for a variety of special patient populations, including diabetes mellitus, coronary artery disease, renal dysfunction and the elderly. Surprisingly, however, these guidelines do not provide specific recommendations for managing the obese hypertensive patients that go beyond recommending weight loss. Thus for example, the JNC-VIII guidelines[7] include only one paragraph on the specific management of hypertension in obese patients, with a focus on non-pharmacological weight reduction.

MATERIAL AND METHODS

The patients under the study were started with a dose of Ramipril 2.5 mg and Hydrochlorthiazide 12.5 mg and the blood pressure recording was done at weekly intervals after which an increment of Ramipril 2.5 mg and Hydrochlorthiazide 12.5 mg was made at an interval of one week wherever required to attain the desired blood pressure.

After washout period of 2 weeks, antihypertensive drugs were given and blood pressure recording was done at weekly intervals for 6 weeks. Levels of blood urea, serum creatinine, serum cholesterol, fasting blood sugar and ECG were determined in the beginning of the study and again at the end of 6 weeks trial.

Inclusion Criteria

1. Hypertension defined as a mean seated diastolic blood pressure of >80 mmHg/or systolic pressure of >130 mmHg, measured by manual cuff.
2. Persons 18-65 years of age.
3. Ability to stop current anti-hypertensive therapy and other disallowed medications without risk to the patient.
4. Overweight or obese as defined by a BMI of ≥25 Kg/m^2 in Asians as per the Asia specific guidelines.

Exclusion Criteria

1. Pregnant women
2. Known or suspected secondary hypertension, hepatic and/or renal dysfunction.
3. Patients on dialysis or post renal transplant.
4. Congestive heart failure.
5. Sustained ventricular tachycardia, atrial finrillation, atrial flutter or other clinically relevant cardiac arrhythmias.
6. Patients who have previously experienced symptoms of angioedema during angiotensin converting enzyme inhibitor treatment.
7. Chronic administration of any medication allowed by the protocol.
8. Any investigational drug therapy within the past month.
9. Known hypertensive to any component of the study drug.
10. Concurrent use of corticosteroid, colestipol or cholestyramine resins.
11. Any clinical condition which would not allow safe completion of the protocol.
12. Inability to comply with the protocol.
13. Any surgery that is, at the time of screening, planned to take place during the study period.

RESULTS

TABLE 1: COMPARISON OF BASELINE SBP WITH ALL TIMES OF OBESE

	MEAN SBP(mm/Hg)	SD	FALL IN MEAN SBP (mm/Hg)	't' VALUE	'p'VALUE
Baseline	172.4	7.08			
1st week	167.9	6.76	4.48±2.44	12.96	<0.001
2nd week	162.1	7.41	10.32±4.52	16.13	<0.001
3rd week	156.1	9.28	16.36±7.44	15.56	<0.001
4th week	149.7	9.39	22.72±8.67	18.52	<0.001
5th week	141.9	10.17	30.52±10.2	21.09	<0.001
6th week	135.5	9.31	36.96±10.2	25.58	<0.001

P < 0.001: Highly significant

Table 1 shows that the baseline mean SBP in mm Hg was 172.4±7.08, at the end of 1st week 167.9 ±6.76, at the end of 2nd week 162.1± 7.41, at the end of 3rd week 156.1± 9.28, at the end of 4th week 149.7± 9.39, at the end of 5th week 141.9± 10.17, at the end of 6th week 135.5 ± 9.31 with a mean fall in SBP mm Hg 4.48± 2.44, 10.32 ± 4.52, 16.36± 7.44, 22.72± 8.67, 30.52± 10.2,36.96± 10.2 at the end of 1st week, 2nd week, 3rd week, 4th week, 5th week, 6th week respectively.

TABLE 2: COMPARISON OF BASELINE SBP WITH ALL TIMES OF Non-OBESE

	MEAN SBP(mm/Hg)	SD	FALL IN MEAN SBP (mm/Hg)	't' VALUE	'p'VALUE
Baseline	165.2	14.22			
1st week	149.6	12.12	15.6±5.20	21.16	<0.001
2nd week	137.2	11.18	28.0±7.60	25.86	<0.001
3rd week	127.5	10.25	37.72±7.8	34.13	<0.001
4th week	118.8	7.28	46.48±10.4	31.74	<0.001
5th week	114.9	5.32	50.28±10.8	32.66	<0.001
6th week	111.8	5.22	53.40±11.8	31.94	<0.001

P< 0.001 Highly significant

Table 2 shows that the baseline mean SBP in mm Hg was 165.2±14.22, at the end of 1st week 149.6 ±12.12, at the end of 2nd week 137.2± 11.18, at the end of 3rd week 127.5± 10.25, at the end of 4th week 118.8± 7.28, at the end of 5th week 114.9± 5.32, at the end of 6th week 111.8± 5.22 with a mean fall in SBP mm Hg 15.6± 5.20, 28.0 ± 7.60, 37.72± 7.80, 46.48± 10.4, 50.28± 10.8, 53.40± 11.8 at the end of 1st week, 2nd week, 3rd week, 4th week, 5th week, 6th week respectively.

TABLE 3: COMPARISON OF BASELINE DBP WITH ALL TIMES OF OBESE

	MEAN DBP(mm/Hg)	SD	FALL IN MEAN DBP (mm/Hg)	't' VALUE	'p'VALUE
Baseline	100.1	4.54			
1st week	96.40	4.66	3.72±2.06	12.77	<0.001
2nd week	91.76	4.67	8.36±3.32	17.78	<0.001
3rd week	87.60	4.24	12.5±4.37	17.78	<0.001
4th week	84.04	4.27	16.08±4.83	23.54	<0.001
5th week	80.60	4.07	19.52±5.81	23.77	<0.001
6th week	70.08	3.92	22.04±5.83	26.71	<0.001

P< 0.001 Highly significant

Table 3 shows that the baseline mean DBP in mm Hg was 100.1±4.54, at the end of 1st week 96.40 ±4.66, at the end of 2nd week 91.76± 4.67, at the end of 3rd week 87.60± 4.24, at the end of 4th week 84.04± 4.27, at the end of 5th week 80.60± 4.07, at the end of 6th week 70.08 ± 3.92 with a mean fall in DBP mm Hg 3.72±2.06, 17.78, 17.78, 23.54, 23.77, 26.71 at the end of 1st week, 2nd week, 3rd week, 4th week, 5th week, 6th week respectively.

TABLE 4: COMPARISON OF BASELINE DBP WITH ALL TIMES OF Non-OBESE

	MEAN DBP(mm/Hg)	SD	FALL IN MEAN DBP (mm/Hg)	't' VALUE	'p'VALUE
Baseline	98.04	8.14			
1st week	85.96	7.54	12.08±4.4	19.46	<0.001
2nd week	78.28	6.93	19.76±5.58	25.04	<0.001
3rd week	72.22	6.50	25.82±5.87	31.11	<0.001
4th week	67.32	5.68	30.72±6.44	33.75	<0.001
5th week	64.64	4.42	33.40±6.14	38.44	<0.001
6th week	62.76	3.89	35.28±6.72	37.11	<0.001

P< 0.001 Highly significant

Table 4 shows that the baseline mean DBP in mm Hg was 98.04±8.14, at the end of 1st week 85.96 ±7.54, at the end of 2nd week 78.28± 6.93, at the end of 3rd week 72.22± 6.50, at the end of 4th week 67.32± 5.68, at the end of 5th week 64.64± 4.42, at the end of 6th week 62.76 ± 3.89 with a mean fall in DBP mm Hg 12.08±4.4, 19.76±5.58, 25.82±5.87, 30.72±6.44, 33.40±6.14, 35.28±6.72 at the end of 1st week, 2nd week, 3rd week, 4th week, 5th week, 6th week respectively.

TABLE 5: COMPARISON OF BASELINE MEAN MAP WITH ALL TIMES OF OBESE

	MEAN MAP(mm/Hg)	SD	FALL IN MEAN MAP (mm/Hg)	't' VALUE	'p'VALUE
Baseline	124.2	4.44			
1st week	120.2	4.46	3.97± 1.79	15.77	<0.001
2nd week	115.2	4.48	9.01± 2.93	21.75	<0.001
3rd week	110.4	4.80	13.80± 4.66	20.94	<0.001
4th week	105.9	5.01	18.29±5.32	24.31	<0.001
5th week	101.0	5.20	23.19±6.42	25.53	<0.001
6th week	97.21	4.82	27.01±6.39	29.90	<0.001

P< 0.001 Highly significant

Table 5 shows that the baseline mean MAP in mm Hg was 124.2±4.44, at the end of 1st week 120.2 ±4.46, at the end of 2nd week 115.2± 4.48, at the end of 3rd week 110.4± 4.8, at the end of 4th week 105.9± 5.01, at the end of 5th week 101.0± 5.20, at the end of 6th week 97.21 ± 4.82 with a mean fall in MAP mm Hg 3.97± 1.79, 9.01± 2.93, 13.80± 4.66, 18.29±5.32, 23.19±6.42, 27.01±6.39 at the end of 1st week, 2nd week, 3rd week, 4th week, 5th week, 6th week respectively.

TABLE 6: COMPARISON OF BASELINE MEAN MAP WITH ALL TIMES OF Non-OBESE

	MEAN MAP(mm/Hg)	SD	FALL IN MEAN MAP (mm/Hg)	't' VALUE	'p'VALUE
Baseline	120.4	9.90			
1st week	107.2	8.67	13.26± 3.89	24.05	<0.001
2nd week	97.93	7.75	22.5± 5.35	29.71	<0.001
3rd week	90.65	7.09	29.79± 5.4	38.68	<0.001
4th week	84.47	5.79	35.97±7.0	36.30	<0.001
5th week	81.41	4.38	39.03±7.1	38.67	<0.001
6th week	79.12	3.96	41.32± 7.88	37.02	<0.001

P< 0.001 Highly significant

Table 6 shows that the baseline mean MAP in mm Hg was 120.4±9.90, at the end of 1st week 107.2 ± 8.67, at the end of 2nd week97.93 ± 7.75, at the end of 3rd week 90.65 ± 7.09, at the end of 4th week 84.47 ± 5.79, at the end of 5th week 81.41 ± 4.38 , at the end of 6th week 79.12 ± 3.96 with a mean fall in MAP mm Hg 13.26± 3.89, 22.5± 5.35, 29.79± 5.4, 35.97±7.0, 39.03±7.1, 41.32± 7.88 at the end of 1st week, 2nd week, 3rd week, 4th week, 5th week, 6th week respectively.

TABLE 7|: COMPARISON OF DOSE A (RAMIPRIL) IN BOTH GROUPS AT ALL TIMES

	GROUP 1	GROUP 2	't' VALUE	'p'VALUE
Baseline	2.5± 0	2.5 ± 0	-	-
1st week	5.0± 0	5.0± 0	16.80	-
2nd week	7.40 ±0.49	5.25 ±.76	29.69	<0.001
3rd week	9.75 ±0.76	5.25 ± .76	31.50	<0.001
4th week	9.80± 0.69	5.25 ± .76	17.47	<0.001
5th week	9.80± 0.69	6.25 ± 1.26	17.47	<0.001
6th week	9.80 ±0.69	6.26 ± 1.26		<0.001

P< 0.001: Highly significant

Table 7 showing comparison of mean dose of ramipril in mg given in group 1 and group 2 is 2.5± 0, 2.5± 0 at baseline, 5.0± 0, 5.0± 0 at the end of 1st week, 7.40 ±0.49, 5.25 ±.76 at the end of 2nd week, 9.75 ±0.76, 5.25 ± .76 at the end of 3rd week, 9.80± 0.69, 5.25 ± .76 at the end of 4th week, 9.80± 0.69, 6.25 ± 1.26 at the end of 5th week, 9.80 ±0.69, 6.26 ± 1.26 at the end of 6th week respectively and the comparison was statistically significant as more dose of ramipril was needed in obese hypertensives as compared to non obese hypertensives to control the blood pressure.

TABLE 8: COMPARISON OF DOSE B (HCTZ) IN BOTH GROUPS AT ALL TIMES

	GROUP 1	GROUP 2	't' VALUE	'p'VALUE
Baseline	12.5± 0	12.5 ± 0	-	-
1st week	25.0± 0	25.0± 0	-	-
2nd week	37.40 ± 2.47	26.5 ±.3.79	16.80	<0.001
3rd week	48.75 ± 3.79	26.5± 3.79	29.69	<0.001
4th week	49.0± 3.43	26.5 ± 3.79	31.50	<0.001
5th week	26.5 ± 3.79	31.25 ± 6.31	17.47	<0.001
6th week	9.80 ±0.69	31.25 ± 6.31	17.47	<0.001

P< 0.001 Highly significant

Table 8 showing comparison of mean dose of HCTZ in mg given in group 1 and group 2 is 12.5± 0, 12.5± 0 at baseline, 25.0± 0, 25.0± 0 at the end of 1st week, 37.40 ± 2.47, 26.5 ±.3.79 at the end of 2nd week, 48.75 ± 3.79 , 26.5± 3.79 at the end of 3rd week, 49.0± 3.43 , 26.5 ± 3.79 at the end of 4th week, 26.5 ± 3.79, 31.25 ± 6.31 at the end of 5th week, 9.80 ±0.69, 31.25 ± 6.31at the end of 6th week respectively and the comparison was statistically significantas more dose of hydrochlorthiazide was needed in obese hypertensives as compared to non-obese hypertensives to control the blood pressure.

TABLE 9: COMPARISON OF MEAN % FALL IN SBP FROM BASELINE AT ALL TIMES IN BOTH GROUPS

	GROUP 1	GROUP 2	't' VALUE	'p'VALUE
1st week	2.58± 1.37	9.38± 2.73	15.73	<0.001
2nd week	5.97 ± 2.59	16.81 ± 3.85	16.52	<0.001
3rd week	9.47 ± 4.27	22.70± 3.57	16.81	<0.001
4th week	13.13± 4.89	27.86 ± 4.38	15.87	<0.001
5th week	17.64 ± 5.71	30.11 ± 4.33	12.30	<0.001
6th week	21.35 ± 5.58	31.97 ± 4.78	10.22	<0.001

P< 0.001 Highly significant

Table 9 shows the mean fall in SBP in group 1 and group 2 is 2.58± 1.37, 9.38± 2.73 at the end of 1st week , 5.97 ± 2.59, 16.81 ± 3.85 at the end of 2nd week, 9.47 ± 4.27, 22.70± 3.57 at the end of 3rd week, 13.13± 4.89, 27.86 ± 4.38 at the end of 4th week, 17.64 ± 5.71, 30.11 ± 4.33 at the end of 5th week , 21.35 ± 5.58, 31.97 ± 4.78 at the end of 6th week respectively and the fall was statistically significant and this shows that fall in systolic blood pressure in non-obese hypertensives is more than obese hypertensives.

TABLE 10: COMPARISON OF MEAN % FALL IN DBP FROM BASELINE AT ALL TIMES IN BOTH GROUPS

	GROUP 1	GROUP 2	't' VALUE	'p'VALUE
1st week	3.70± 1.99	12.25± 4.15	13.11	<0.001
2nd week	8.32 ± 3.14	20.03 ± 5.06	13.90	<0.001
3rd week	12.43 ± 3.91	26.21± 4.93	15.49	<0.001
4th week	15.97± 4.31	31.16 ± 4.91	16.44	<0.001
5th week	19.36 ± 5.07	33.86 ± 4.11	15.71	<0.001
6th week	21.87 ± 5.05	35.72 ± 4.50	14.49	<0.001

P< 0.001 Highly significant

Table 10 shows the mean % fall in DBP in group 1 and group 2 is 3.70± 1.99, 12.25± 4.15 at the end of 1st week , 8.32 ± 3.14, 20.03 ± 5.06 at the end of 2nd week, 12.43 ± 3.91, 26.21± 4.93 at the end of 3rd week, 15.97± 4.31, 31.16 ± 4.91 at the end of 4th week, 19.36 ± 5.07, 33.86 ± 4.11 at the end of 5th week , 21.87 ± 5.05, 35.72 ± 4.50 at the end of 6th week respectively and the fall was statistically significant and this shows that fall in diastolic blood pressure in non-obese hypertensives is more than obese hypertensives.

TABLE 11: COMPARISON OF MEAN % FALL IN MAP FROM BASELINE AT ALL TIMES IN BOTH GROUPS

	GROUP 1	GROUP 2	't' VALUE	'p'VALUE
1st week	3.19± 1.40	10.95± 2.9	17.05	<0.001
2nd week	7.24 ± 2.25	18.58 ± 3.73	18.40	<0.001
3rd week	11.07 ± 3.51	24.63± 3.39	19.65	<0.001
4th week	14.67± 3.99	29.67 ± 4.06	18.63	<0.001
5th week	18.58 ± 4.72	32.17 ± 3.70	16.02	<0.001
6th week	21.66 ± 4.62	34.03 ± 4.17	14.06	<0.001

P< 0.001 Highly significant

Table 11 shows the mean % fall in DBP in group 1 and group 2 is 3.19± 1.40, 10.95± 2.9 at the end of 1st week , 7.24 ± 2.25, 18.58 ± 3.73 at the end of 2nd week, 11.07 ± 3.51, 24.63± 3.39 at the end of 3rd week, 14.67± 3.99, 29.67 ± 4.06 at the end of 4th week, 18.58 ± 4.72, 32.17 ± 3.70 at the end of 5th week , 21.66 ± 4.62, 34.03 ± 4.17 at the end of 6th week respectively and the fall was statistically significant and this shows fall in mean arterial pressure in non-obese hypertensives is more than obese hypertensives.

DISCUSSION

Obesity is an increasingly observed pathologic entity in the industrialized world and causally linked to the development of hypertension. Consequently, not only the prevalence of obesity but also the prevalence of obesity hypertension is increasing worldwide. However, current guidelines still do not include recommendations for the treatment of obese patients with hypertension.[8]

While in theory, weight loss may indeed be the most effective measure for the treatment of hypertension in obese patients, in practice, long term maintenance of weight loss is notoriously unsuccessful, even in context of controlled clinical trials.[9] Thus, as most obese patients will either not lose weight or fail to maintain any relevant weight loss in long run, we have no option but to use antihypertensive medication in the majority of obese patients.

A Meta-Analysis of Randomized Trials by Messerli FH. Fourteen studies of HCTZ dose 12.5 to 25 mg with 1,234 patients and 5 studies of HCTZ dose 50 mg with 229 patients fulfilled the inclusion criteria. The decrease in 24-h BP with HCTZ dose 12.5 to 25 mg was systolic 6.5 mm Hg (95% confidence interval: 5.3 to 7.7 mm Hg) and diastolic 4.5 mm Hg (95% confidence interval: 3.1 to 6.0 mm Hg) and was inferior compared with the 24-h BP reduction of angiotensin-converting enzyme inhibitors (mean BP reduction 12.9/7.7 mm Hg; p 0.003), angiotensin-receptor blockers (mean BP reduction 13.3/7.8 mm Hg; p 0.001).[10] Our study results were comparable with this study (Table 7 and Table 8).

Study by Reisin E compared the efficacy and safety of the angiotensin-converting enzyme inhibitor lisinopril and the diuretic hydrochlorothiazide in a 12-week, multicenter, double-blind trial in 232 obese patients with hypertension. Patients with an office diastolic pressure between 90 and 109 mm Hg were randomized to treatment with daily doses of lisinopril (10, 20, or 40 mg), hydrochlorothiazide (12.5, 25, or 50 mg), or placebo. At week 12, lisinopril and hydrochlorothiazide effectively lowered office diastolic (-8.3 and -7.7 versus -3.3 mm Hg, respectively; P<.005) and systolic (-9.2 and -10.0 versus -4.6 mm Hg, respectively; P<.05) pressures compared with placebo. Significant dose-response differences were observed between treatments. Sixty percent of patients treated with lisinopril had

an office diastolic pressure <90 mm Hg compared with 43% of patients treated with hydrochlorothiazide (P<.05). Treatment with angiotensin-converting enzyme inhibitors may show greater efficacy as monotherapy at lower doses compared with thiazide diuretics.[11] Results of our study was in similar aspect as with above mentioned study. We used ramipril which reduced bood pressure more effectively than hydrochlorthiazide in obese patients as well as in non-obese. (Table 7 and Table 8).

One reason for this glaring lack of specific recommendations for obese patients is perhaps the even more glaring lack of antihypertensives trials in such patients.The lack of specific recommendations for the obese hypertensive patient is far from trivial. Firstly, compared to hypertension in non-obese patients, obesity related hypertension is characterized by volume expansion, increased cardiac output, and a decreased rather than increase in total peripheral resistance.[12,13] Thirdly, obesity per se often is associated with end-organ damage, including eccentric left ventricular hypertrophy[12], glomerular hyperfiltration and microalbuminuria.[14]

An increase in adipose mass may also be important as a potential determinant of response to treatment, possibly reducing the effectiveness of antihypertensive therapy. Thus, patients surveys clearly indicate that overweight hypertensives are less likely to achieve normal blood pressure on treatment than non-obese patients. Other issues, related to pharmacokinetics of lipophilic drugs[15,16] or increased prevalence of liver abnormalities[17,18] in obese may also warrant attention.

Indirect measure of adiposity is BMI = weight(Kg)/height^2(m^2). Advantage of this index rather than the use of weight alone is that it is height independent, such that tall and short people of similar proportions have a similar BMI.[19]
Waist circumference is correlated closely to BMI and abdominal fat mass, and is used as a surrogate marker for (upper body) obesity. Waist circumference (greater than measurement of hip circumference or more than 38 inches in a man or 34 inches in a woman) is positively correlated with increased risk of diabetes, hypertension, dyslipedemia and ischemic heart disease. The BMI and the waist circumference should be measured in all hypertensive subjects.[20]

CONCLUSION

Thus our study concluded that obese patients even when presenting with apparently uncomplicated hypertension, require more aggressive blood pressure control with higher dose of similar drug than in non-obese patients. Inspite of guidelines for management of hypertension in other comorbid conditions, there must be an initiative to set a particular target range to reduce hypertension in obese patients after dietary and life style modification.

References

1. Black HR, Bakris GL, Elliot WJ,Hypertension: Epidemiology, Pathyphysiology, Diagnosis and Treatment. In: Fuster V, Alexander RW, O Rourke RA (editors). Hurst's The Heart.. 11th Ed. New York: McGraw Hill Publication 2005; 2: 1553-1606.
2. U.S. Department of Health and Human Services, Office of Disease Prevention and Health Promotion. Healthy People 2020. Available from: https://www.healthypeople.gov/2020/default.aspx.
3. Kannel WB, Garrison RJ, Dannenberg AL. Secular blood preeure trends in normotensive persons: the Farmingham Study. Am Heart j. 1993; 125: 1154-58.
4. MacMahon SW, Blacket RB, Macdonald GJ, Hall W. Obesity, alcohol consumption and blood pressure in Australian men and women. The National Mark AL Correia M, Morgan DA, Shaffer RA, Haynes WG. Obesity-induced hypertension: new concepts from the emerging biology of obesity. Hypertension 1999; 33: 537-41.
5. Engeli S, Sharma AM. Role of adipose tissue for cardiovascular-renal regulation in health and disease. Hormone Metab Res. 2001; 32: 485-99.
6. The sixth report of the Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure. Arch Intern Med 1997; 157: 2413-46.
7. JNC 8 Guidelines for the Management of Hypertension in Adults. Am Fam Physician. 2014; 90:503-04.
8. Wenzel UO, Benndorf R, Lange S. Treatment of arterial hypertension in obese patients. Semin Nephrol. 2013;33 :66-74.
9. Stevens VJ, Obarzanek E, Cook NR et al. Long-term weight loss and changes in blood pressure: results of the Trials of Hypertension Prevention, Phase II. Ann Intern Med. 2001; 134: 1-11.
10. Messerli FH, Makani M, Benjo A. Antihypertensive Efficacy of Hydrochlorothiazide as Evaluated by Ambulatory Blood Pressure Monitoring. Journal of the American College of Cardiology 2011; 57:5.

11. Reisin E, Weir MR, Falkner B, Hutchinson HG, Anzalone DA, Tuck ML. Lisinopril versus hydrochlorothiazide in obese hypertensive patients: a multicenter placebo-controlled trial. Treatment in Obese Patients With Hypertension (TROPHY) Study Group. Hypertension 1997 ;30: 140-5.
12. Messerli FH, Sundgaard-Riise K, Reisin ED. Dimorphic cardiac adaptation to obesity and arterial hypertension.Ann Intern Med. 1983; 99: 757-61.
13. Rocchini AP. Cardiovascular regulation in obesity-induced hypertension. Hypertension 1992; 19: 156-60.
14. Valensi P, Assayag M, Busby M Paries J, Lormeau B, Attali JR. Microalbuminuria in obese patients with or without hypertension. Int J Obes. 1996; 20: 574-79.
15. Cheymol G. Effects of obesity on pharmacokinetics: implications for drug therapy. Clin Pharmacokinet. 2001; 39: 215-31.
16. Cheymol G, Poirier JM, Carrupt PA. Pharmmacokinetics of beta-adrenoceptor blockers in obese and normal volunteers. Br J Clin Pharmacol. 1997; 43: 563-70.
17. Ratziu V, Giral P, Charlotte F. Liver fibrosis in overweight patients. Gastroenterology 2000; 118: 1117-23.
18. Luyckx Fh, Lefebvre PJ, Scheen AJ. Non-alcoholic steatohepatitis: association with obesity and insulin resistance, and influence of weight loss. Diabetes Metab. 2000; 26: 98-106.
19. Fried BM, Truswell AS, Shephard J,Jung R. Obesity In: Christopher Haslett, Edwin R, Chilver S, John A Hunter, Nicholas A Boon, Eds. Davidson's Principles and practice of Medicine. 18th Edn. London: Churchill Livingstone 1999; 7: 526-28.
20. Baron RB. Nutrition. In: Lawrence M. Tierney, Jr. Stephen j. Mcphee, Maxine A. Papadakis Eds. CMDT 41st Edn. London: Churchill Livingstone 2002;1275.