

PRE AND POST-PRANDIAL CHANGES IN BP IN OBESE AND NON OBESE POPULATION: A COMPARATIVE STUDY AT A TERTIARY CARE HOSPITAL

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ABSTRACT:

Background: A higher or lower sympathetic tone having higher or lower catecholamine levels along with varied abnormalities in autonomic nervous system associated with obesity which finally leads to change in the function of heart such as arrhythmia along with involvement of other systems, ultimately lead to sudden death. This study planned to find pre and post-prandial changes in BP in obese and non obese persons in the age group of 15 to 45 years. **Materials and Methods:** 40 obese and 40 non obese patients admitted to our hospital over a period of two months were included in the study. Random selection of patients was made from Department of Medicine, JNU Medical College and Hospital, Jaipur. Both systolic and diastolic blood pressure was measured in supine position of the right arm using sphygmomanometer 30 minutes before and 30 minutes after meals. Then fall in BP was recorded. BMI of patients was measured to identify grade of obesity. **Results:** Mean systolic (pre-prandial 126.28, post-prandial 117.9) and diastolic blood pressure (pre-prandial 79.2 and post-prandial 74.6) was found, which was higher in obese than non obese patients. This study was statistically significant (p value <0.05). **Conclusion:** Fall in BP was significantly higher in obese (14 patients out of 50) as compared to non obese (3 patients out of 40) patients. Reduced cardiac sympathetic tone leads to post-prandial hypotension were significantly associated in obese patients.

Key Words: Systolic BP, Diastolic BP, Post-prandial, Obese, Non-obese.

INTRODUCTION:

Obesity, a major emerging health problem worldwide, cause of morbidity and mortality in developing as well as developed countries, have an association with sudden death as per the involvement of multiple organs. Due to adoption of western culture in food habits shifting the

nutritional status of Indian population gradually from malnutrition to obesity is the main reason for development of hypertension. Many studies have shown the relationship between obesity, hypertension, diabetes mellitus and sudden deaths. (1)

A higher or lower sympathetic tone having higher or lower catecholamine levels along with varied abnormalities in autonomic nervous system associated with obesity which finally leads to change in the function of heart such as arrhythmia along with involvement of other systems, ultimately lead to sudden death.(1) In healthy, non-obese and elderly individuals as the food intake is associated with increase in the HR, prevent significant fall in BP but in patients with post-prandial hypotension this phenomenon does not occur.(2)

After consumption of food, peptides are released in gastro intestinal tract (GIT), causes local vasodilatation and post-prandial redistribution of blood, leading to shifting of large volume of blood in GIT leads to post-prandial hypotension as compared to normal individuals in which it doesn't lead to post-prandial hypotension because of compensation mechanisms.(3) Post-prandial hypotension is defined as the fall in the Systolic blood pressure (SBP) of $> 20\text{mmHg}$ or reduction in the post-prandial SBP $< 90\text{mmHg}$ when pre-prandial BP is $> 100\text{mmHg}$ within two hours of meal.(2)

Our hypothesis is based on the fact that food intake may result in a transient decrease in sympathetic activity and impaired parasympathetic activity associated with post-prandial hypotension.

MATERIAL AND METHODS

40 obese and 40 non obese patients admitted to our hospital over a period of two months were included in the study. Random selection of patients was made from Department of Medicine, JNU Medical College and Hospital, Jaipur. This study is a randomized cross sectional study.

Inclusion Criteria

- Age matched non obese and obese adults between the age group 15 to 45 years were included in the study.

Exclusion Criteria

- Diabetes, Alcohol abuse, asthma, endocrine disease, surgery
- Patient on Antihypertensive drugs, other drug affecting ANS
- Disease associated with hypertension
- Autonomic neuropathies, neuromuscular disorder

The BMI was calculated as

$$\text{BMI} = \text{Weight in kg} / (\text{Height in meters})^2$$

Subjects were classified into two groups based on BMI as follows:

Normal weight: BMI – $18.5 - 24.99 \text{ kg/m}^2$

Obese: BMI $> 30 \text{ kg/m}^2$

Category	BMI in Kg/m ²
Underweight	<18.5
Normal weight	18.5-24.9
Over weight	25-29.9
Obesity (class 1)	30-34.9
Obesity (class2)	35-39.9
Extreme obesity (class 3)	>40

Both systolic and diastolic blood pressure was measured in supine position of the right arm using sphygmomanometer 30 minutes before and 30 minutes after meals. Then fall in BP was recorded. BMI of patients was measured to identify grade of obesity.

RESULT

Out of 40 obese patients 18 were males and 22 were females and in non obese patients (40 in no.) 29 were males and 11 were females.

Mean systolic (pre-prandial 126.28, post-prandial 117.9) and diastolic blood pressure (pre-prandial 79.2 and post-prandial 74.6) was found, which was higher in obese than non obese patients. This study was statistically significant (p value <0.05).

Table 1: Distribution of patients according to Sex

	Male	Female
Obese	18	22
Non obese	29	11

Table 2: Distribution of groups according to Systolic blood pressure

	Group	Mean	SD	p-value
SBP pre-prandial	Non obese	114.3	13.96	<0.05
	Obese	126.28	13.26	
SBP post-prandial	Non obese	112.6	14.21	<0.05
	Obese	117.9	16.12	

Table 3: Distribution of groups according to Diastolic blood pressure

	Group	Mean	SD	p-value
DBP pre-prandial	Non obese	74.6	9.23	<0.05
	Obese	79.2	10.99	
DBP post-prandial	Non obese	73.4	10.34	<0.05
	Obese	74.6	9.27	

Table 4: Distribution of groups according to fall in blood pressure

	Total patients	No. of patients with fall in BP
Obese	40	14
Non obese	40	3

Fall in BP was significantly higher in obese (14 patients out of 50) as compared to non obese (3 patients out of 40) patients. Reduced cardiac sympathetic tone and impaired parasympathetic activity leads to post-prandial hypotension were significantly associated in obese patients.

DISCUSSION

Traditionally, after ingestion of food, sympathetic tone was thought to be activated over the entire human body, responsible for approximately 30% of the thermic effect of food (4, 5) and also helps to maintain blood pressure post-prandially.(6) Both the mean systolic and diastolic blood pressure is found to be higher in obese compared to non obese patients, as depicted by p value showing it as statistically significant. Fall in BP was significantly higher in obese than non obese patients.

There may be a transient decrease in sympathetic activity and impaired parasympathetic activity after meals to compensate for the change in blood distribution thereby causing post-prandial hypotension. Usha et al and Ambarish et al showed that post-prandial hypotension is significantly higher among obese individuals when compared to non-obese individuals and statistically significant. (1,3)

Frenco et al (7) and Nagai et al (8) have showed that in obese individual, a significant degree of autonomic dysfunction occurs, cardiac sympathetic tone is reduced, caused post-prandial hypotension more in comparison to non obese patients.(9) Later on, imbalance in cardiac autonomic activity may predispose to arrhythmia along with involvement of other systems, ultimately lead to sudden death which is a common phenomenon in obese individuals as complications such as syncope, falls, weakness, angina, dizziness, visual disturbance and cerebro-vascular accident (CVA) are associated more. A strong association between falls and syncope in elderly subjects with post-prandial hypotension has been established. (10)

The mechanism involved in the changes of parasympathetic and sympathetic nervous activities in overweight is unknown till date. As far as the parasympathetic activity is considered there is not much difference among the studies, as almost all of the studies showed a significant reduction in the parasympathetic activity associated with increased body weight.(11) Several hormonal signals have been postulated. These include insulin, increase muscle sympathetic nerve activity during euglycemic condition; free fatty acids, increase BP by stimulation of excitatory hepatic afferent nerves in rats; and leptin, increase sympathetic

discharge to several tissues in rats, and elevated during rapid weight gain in humans. (12)

Orthostatic hypotension, effect of posture on post-prandial fall in BP commonly caused by autonomic function, additive to post-prandial hypotension rather than synergistic, (13) so that in elderly patients, orthostatic and post-prandial hypotension should be taken care of.

Sympathovagal imbalance can explain the increased incidence of sudden cardiac deaths associated with obesity. Early interventional programs like weight reduction, life style modification and physical exercises, which reduce fat content, can be advised to reduce the chances of subsequent cardiac arrhythmia.

Limitations-To understand the underlying mechanism and patho-physiology, for the occurrence of post-prandial hypotension among obese individuals, further studies are required with larger number of patients, as not more studies are available till date on post-prandial hypotension among obese and non obese individuals.

CONCLUSION

Post-prandial hypotension was significantly higher in obese than non obese patients. Post-prandial hypotension has received less attention, as there are very few studies done regarding

association of it's with obesity, although it is a cause of morbidity and mortality. Hence there is a need for further studies on post-prandial hypotension in various groups.

REFERENCES

1. Usha RYS, Ramakrishna MR, Manjunath P, Trupti RR, Rangaswamy R. Comparative study of pre-prandial and post-prandial autonomic nervous system response between obese and non obese young women aged 18-25 years. *Int J Pharm Bio Sci* 2013; 4(3):239-58.
2. Fisher AA, Davis MW, Srikusalanukul W, Budge MM. Postprandial hypotension predicts all cause mortality in older, low level care residents. *J Am Geriatr Soc* 2005; 53(8):1313-20.
3. Ambarish V, Barde P, Vyasa A, Deepak K. Comparison between pre-prandial and post-prandial heart rate variability (hrv). *Indian J Physiol Pharmacol* 2005; 49(4):436-42.
4. Acheson KJ, Ravussin E, Wahren J, Jequier E: Thermic effect of glucose in man. Obligatory and facultative thermogenesis. *J Clin Invest* 1984; 74:1572-80.
5. Schwarz RS, Jaeger LF, Veith RC: Effect of clonidine on the thermic effect

- of feeding in humans. *Am J Physiol* 1988; 254:R90-4.
6. Mathias CJ: Postprandial hypotension. Pathophysiological mechanisms and clinical implications in different disorders. *Hypertension* 18:694-704, 1991.
 7. Frenco R, Bernard S, Andrea C, Tiziana G, Barbara DV, Ivana R, et al. Assessment of cardiac autonomic modulation during adolescent obesity. *Obes Res.* 2003; 11(4):541-48.
 8. Nagai N, Matsumoto T, Kita H, Moritani T. Autonomic nervous system activity and the state and development of obesity in Japanese school children. *Obese Res.* 2003; 11(1):25-32.
 9. Vloet LCM, Pel-Little RE, Jansen PAF, Jansen RWMM. High prevalence of post-prandial and orthostatic hypotension among geriatric patients admitted to Dutch hospitals. *J Gerontolo A* 2005; 60(10):1271–77.
 10. Puisieux F, Bulckaen H, Fauchais AL, Drumez S, Salomez-Granier F, Dewailly P. Ambulatory blood pressure monitoring and post-prandial hypotension in elderly persons with falls or syncope. *J Gerontol A Biol Sci Med Sci* 2001; 55(9):535–40.
 11. Hrushesky WJ, Fader D, Schmitt O, Gilbertsen V. The respiratory sinus arrhythmia: a measure of cardiac age. *Science* 1984; 224:1001–4.
 12. Rabbia F, Silke B, Conterno A, Grosso T, Vito BD, Rabbone I et al. Assessment of cardiac autonomic modulation during adolescent obesity. *Obes Res* 2003; 11:541-548.
 13. Tanakaya M, Takahashi N, Takeuchi K, Katayama Y, Yumoto A, Kohno K, et al. Postprandial hypotension due to a lack of sympathetic compensation in patients with diabetes mellitus. *Acta Med.Okayama* 2007; 61(4):191-17.