

Comparative Study of Sympathetic Activity in Normotensive Obese and Nonobese Adults

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Abstract

Aim: The aim of the study is to assess sympathetic activity in Obese Normotensive subjects

Objectives: To investigate any changes in sympathetic activity in Normotensive obese subjects using Cold Pressor test

Method: 50 Obese subjects of both genders between age group 18-25 years with BMI>25kg/m² were selected as study group and 50 age matched subjects of both the genders with BMI<25kg/m² were selected as control group. Sympathetic activity was assessed using Cold pressor test. In this test, After recording the resting blood pressure (BP), the subject was asked to immerse his hand in cold water (temperature maintained between 5°–9°C). BP measurement from other arm was done at 30 sec interval for two minutes using continuous ambulatory Blood pressure monitoring, after which the subject was asked to remove the hand from cold water. Maximum increase in Diastolic Blood Pressure (DBP) was taken as test response.

Results: Statistical analysis was done using unpaired t test. Increase in diastolic blood pressure with Cold pressor test in study group was significantly increased compared to control group.

Conclusion: Increased sympathetic activity is seen in obese normotensive subjects, which is a risk factor for future development of hypertension and other complications associated with increased sympathetic activity. Weight loss and maintenance of healthy lifestyle is suggested to the subjects to prevent the future complications.

Keywords: Normotensive Obese, Cold pressor test, sympathetic activity, Diastolic blood pressure.

Introduction

Obesity, considered a worldwide epidemic, is characterized by the excessive accumulation of fat tissue in the body and its causes are multifactorial, such as genetic susceptibility, sex, age, occupation, diet and others¹. It is well recognized that obesity is associated with reduced quality of life and increased risk

of premature death and predisposes individuals to the development of a number of chronic illnesses including cardiovascular disease, type 2 diabetes, dyslipidemia, insulin resistance, hyperglycemia, hypertension, degenerative joint diseases, obstructive sleep apnea (OSA), gastroesophageal reflux disease, nonalcoholic fatty liver, and various forms of cancer².

Obesity and its early complications (i.e. insulin resistance and impaired fasting glucose) are associated with overstimulation of the sympathetic nervous system (SNS) and decreased tone of the parasympathetic nervous system (PNS)³

Studies using norepinephrine urinary excretion and plasma concentration measurements from hypothalamic

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models of obesity show that sympathetic nervous activity in obesity is low⁴. Young and Macdonald⁵ found that there were numerous studies proposing that Sympathetic nervous system (SNS) in subjects with obesity was either low, normal, or elevated. The heterogeneity of the results most likely occurred because of inadequacy of the method used (in particular, venous or urinary norepinephrine concentrations) and because SNS activity is typically regionalized, where the efferent outflow throughout the body is not uniform.

Studies have also shown that SNS activation may be responsible for development of obesity. Increased plasma norepinephrine concentration and a hyperkinetic circulation in young adulthood have been shown to predict future weight gain and the development of insulin resistance⁶.

The present study is carried out to evaluate the sympathetic activity in Obese subjects.

Materials and Method

The study was carried out at Great Eastern Medical school, Srikakulam, Andhra Pradesh

Study Group: 50 Obese subjects of both genders between age group 18-25 years with BMI>25kg/m² were selected as study group

Control Group: 50 Nonobese subjects of both genders between age group 18-25 years with BMI<25kg/m² were selected as control group

Inclusion criteria for study group:

1. BMI>25kg/m²
2. Normotensive subjects

Inclusion criteria for control group:

1. BMI<25Kg/m²
2. Normotensive subjects

Exclusion criteria (common for study and control group):

1. Hypertensive subjects
2. Diabetic subjects
3. Any history of chronic illness

All the subjects were explained about the test to assess sympathetic function and an informed consent was taken.

Sympathetic activity was assessed by cold pressor test.

Cold pressor test (CPT): After recording the resting blood pressure (BP), the subject was asked to immerse his hand in cold water (temperature maintained between 5°–9°C). BP measurement from other arm was done at 30 sec interval for two minutes using continuous ambulatory Blood pressure monitoring, after which the subject was asked to remove the hand from cold water. Maximum increase in Diastolic Blood Pressure (DBP) was taken as test response.

Somatosensory stimulation induced by the cold stimulus increases blood pressure; impulses from receptors in the skin relay via afferent pathways to C1 cells in the rostral ventrolateral (RVL) reticular nucleus and are transmitted via efferent sympathetic neurons to peripheral blood vessels from thoracic spinal cord. Thus, somatosensory stimulation produces the pressor response during the cold pressor test.

Statistical analysis:

Statistical analysis was done using unpaired t test

P value < 0.05 was considered as statistically significant

The results were expressed as Mean ± standard deviation

Finding:

Variable	Study Group		Control Group		P Value
	Mean	SD	Mean	SD	
Increase in DBP (mmHg) with Cold Pressor Test	15.7	3.1	13.2	2.3	<0.0001*

DBP-Diastolic blood pressure

On Analysis,

Increase in diastolic blood pressure with cold pressor test in study group was significantly increased compared to control group.

Discussion

In our study, we found that increase in Diastolic blood pressure with cold pressure in obese normotensive subjects was significantly increased compared to Nonobese normotensive subjects.

The findings of our study were in accordance with previous studies⁷ which showed increased sympathetic activity in obese normotensive subjects.

However some studies reported no change and even reduction in sympathetic activity in obese subjects⁸.

Obesity is characterized by excessive accumulation of fat, a highly dynamic endocrine and paracrine organ that releases many cytokines and bioactive mediators which may influence sympathetic nervous system activity⁹. While there exists a large body of evidence indicating that sympathetic nervous activity is evident in obesity, it is important to recognize that many factors may be involved in the genesis of obesity-related sympathetic activation¹⁰.

It is well established that obesity is one of the major determinants in the development of hypertension in the general population¹¹. The mechanisms contributing to the development of higher blood pressure in humans with obesity include many factors such as hyperinsulinemia, activation of the renin-angiotensin-aldosterone system, abnormal levels of certain adipokines such as leptin, and an altered spectrum of cytokines acting at the vascular endothelial level¹².

Sympathetic nervous stimulation is certainly a key factor in the development of hypertension. It was demonstrated that when weight gain develops in young men, increased Muscle Sympathetic Nerve Activity (MSNA) occurs early, together with increased blood pressure¹³. It was shown that sympathetic activation to the kidneys occurs as early as 1 wk after exposure to a high-fat diet in rabbits¹⁴.

Conclusion

Increased sympathetic activity is seen in obese

normotensive subjects, which is a risk factor for future development of hypertension and other complications associated with increased sympathetic activity. Weight loss and maintenance of healthy lifestyle is suggested to the subjects to prevent the future complications.

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Ethical Clearance: Taken from Scientific Ethical committee, Great Eastern Medical school, Srikakulam, Andhra Pradesh.

Conflict of Interest: Nil

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