ORIGINAL ARTICLE

A Comparative Study of Hypertension and Peripheral Insulin Resistance among Obese and Non-Obese Individuals

KAMRAN ARZOO[1], AMNA AKRAM [2], FAROOQ AHMAD MALIK [3]
[1]*Assistant professor Department of Physiology Rai Medical College Sargodha Pakistan.*
[2]*Department of Biochemistry, Shahida Islam Medical and Dental College, Lodhran -Pakistan.*
[3]*Assistant professor Department of Biochemistry Bakhtawar Amin Medical and Dental College, Multan-Pakistan.*
Correspondence to Dr. Farooq Ahmad Malik Email: malik.farooq6975@ gmail.com cell.03336456975

ABSTRACT

Aim: To test the hypertension and peripheral insulin resistance in Obese and non-Obese individuals.
Place of study: Medical and Diabetological units of Nishtar Hospital Multan.
Methods: In this study 250 individuals were selected and divided them into three different groups. In Group A which was Control all the 50 individuals were normal and in Group B 100, diabetic obese individuals were selected while in Group C 100, non-obese diabetic individuals were selected.
Results: The findings of current study presented different Biomarkers such as systolic and di-systolic blood pressure, serum insulin levels with fasting and postprandial conditions and serum glucose levels (120±11.30, 80±10.10, 25.10±11.22, 18±20.30, 135±5.10), (170±23.20, 100±15.30, 27.3±25.28, 2±11.10, 290±25.30) (140±13.10, 90±10.20, 24.20±25.28, 10.11±11.10, 200±20.10) of Group A, Group B and Group C respectively. Individuals of each group have significant changes (**<**0.005) regarding their results.
Keywords: Hypertension, obese, insulin resistant

INTRODUCTION

Obesity is a physiological, biochemical, anatomical and pathological abnormality in which individual presented high range of weight of given height than the normal levels *Gregor and Hotamisligil, (2015).* If the given weight of any individual in kilograms divided by the square of height in meters it is called body mass index (BMI) which is correlated to body fat and varies with age and sex in children more than it does in adults. But BMI does not measure body fat directly *Nakamura et al, (2010).*

Different researchers concluded through their studies that hypertension, obesity and glucose intolerance are biological mechanisms which are correlated with each other regarding all metabolic changes *Hummasti et al, (2010).* It is also found that insulin resistant state and Noninsulin-dependent diabetes are same biological abnormalities in the biological system *Fronzo,(1997)*. Researchers stated that in obese individuals renal sodium retention is higher than normal individuals therefore they have hypertension. In many cases reduced intracellular and elevated plasma potassium concentrations was seen. In obese individuals transportation through membranes become reduced because of threshold of receptors *Chapman and Sposito, (2008).*

Glucose intolerance, obesity and hypertension are common pathophysiological mechanisms in which normal metabolic state showed different variations and changes. As a result of these changes many life threatening syndromes developed in the body *Arruda et al, (2014)*. Hypertension and insulin response in single or combined with obesity showed increased sodium and potassium concentrations in the plasma and erythrocytes and their correlation with metabolic changes is significant *Wolfs et al, (2015).*

Received on 03-05-2019
Accepted on 15-08-2019

MATERIALS AND METHODS

The present research was conducted in medical and Diabetological units of Nishtar Hospital Multan . In this study 250 individuals were selected and divided them into three different groups. In Group A which was Control all the 50 individuals were normal and in Group B 100, diabetic obese individuals were selected while in Group C 100, non-obese diabetic individuals were selected. In current study systolic and di-systolic blood pressure and basal insulin secretion levels in fasting and postprandial conditions were measured. Glucose levels were calculated by colorimeter kit method. Basal insulin secretion levels were measured by employing spectrophotometric method. The raw data was interoperated with model SSPS.

RESULTS

In the current study different parameters such as systolic and di-systolic blood pressure, serum insulin levels with fasting and postprandial conditions and serum glucose levels (120±11.30, 80±10.10, 25.10±11.22, 18± 20.30, 135±5.10), (170±23.20, 100±15.30, 27.3±25.28, 2± 11.10, 290±25.30) (140±13.10, 90±10.20, 24.20±25.28, 10.11± 11.10, 200±20.10) of Group A, Group B and Group C respectively. Individuals of each group have significant changes (**<**0.005) regarding their results.

Group A, control n= 50

Parameters	**Units**	**Mean± SD**	**P value**
Systolic (B.P)	mmHg	120±11.30	0.00
Di-systolic (B.P)	mmHg	80±10.10	0.00
Serum insulin levels (fasting)	mIU/L	25.10±11. 22	0.00
Serum insulin levels(postprandial)	mIU/L	18± 20.30	0.00
Glucose levels (random)	mg/dl	135±5.10	0.00

<0.005

Group B, Diabetic obese individuals n= 100

Parameters	Units	Mean± SD	P value
Systolic (B.P)	mmHg	170±23.20	0.00
Di-systolic (B.P)	mmHg	100±15.30	0.00
Serum insulin levels (fasting)	mIU/L	27.3±25.28	0.00
Serum insulin levels (postprandial)	mIU/L	2± 11.10	0.00
Glucose levels (random)	mg/dl	290±25.30	0.00

<0.005

Group C, Diabetic non-obese individuals n= 100

Parameters	Units	Mean±SD	P value
Systolic (B.P)	mmHg	140±13.10	0.00
Di-systolic (B.P)	mmHg	90±10.20	0.00
Serum insulin levels (fasting)	mIU/L	24.20±25. 28	0.00
Serum insulin levels (postprandial)	mIU/L	10.11±11.10	0.00
Glucose levels (random)	mg/dl	200±20.10	0.00

<0.005

DISCUSSION

In the current study the findings are identifying a close association of obesity with hypertension, hyperinsulinemia and hyperglycemia. This study also explained that insulin resistance is a common pathophysiologic feature in obesity. *Locke et al, (2015)* stated the same findings that glucose intolerance, and hypertension is closely correlated to the peripheral insulin resistance. *Croteau-Chonka et al, (2015)* claimed in their research that insulin resistance demonstrates a considerable degree of overlap of hypertensive and glucose intolerance in the population. The results of this study was significant (**<**0.005).

REFERENCES

1. Gregor, M.F.; Hotamisligil, G.S. Inflammatory mechanisms in obesity. Ann. Rev. Immunol. 2011, 29, 415–445. Healthcare 2015, 3 413.
2. Nakamura, T.; Furuhashi, M.; Li, P.; Cao, H.; Tuncman, G.; Sonenberg, N.; Gorgun, C.Z.; Hotamisligil, G.S. Double-stranded RNA-dependent protein kinase links pathogen sensing with stress and metabolic homeostasis. Cell 2010, 140, 338–348.
3. Hummasti, S.; Hotamisligil, G.S. Endoplasmic reticulum stress and inflammation in obesity and diabetes. Circ. Res. 2010, 107, 579–591.
4. De Fronzo, R.A. Insulin resistance: A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidaemia and atherosclerosis. Neth. J. Med. 1997, 50, 191–197.
5. Chapman, M.J.; Sposito, A.C. Hypertension and dyslipidaemia in obesity and insulin resistance: Pathophysiology, impact on atherosclerotic disease and pharmacotherapy. Pharmacol. Ther. 2008, 117, 354–373.
6. Arruda, A.P.; Pers, B.M.; Parlakgül, G.; Güney, E.; Inouye, K.; Hotamisligil, G.S. Chronic enrichment of hepatic endoplasmic reticulum-mitochondria contact leads to mitochondrial dysfunction in obesity. Nat. Med. 2014, 20, 1427–1435.
7. Wolfs, M.G.M.; Gruben, N.; Rensen, S.S.; Verdam, F.J.; Greve, J.W.; Driessen, A.; Wijmenga, C.; Buurman, W.A.; Franke, L.; Scheja, L.; et al. Determining the association between adipokine expression in multiple tissues and phenotypic features of non-alcoholic fatty liver disease in obesity. Diabetes 2015.43-50.
8. Locke, A.E.; Kahali, B.; Berndt, S.I.; Justice, A.E.; Pers, T.H.; Day, F.R.; Powell, C.; Vedantam, S.; Buchkovich, M.L.; Yang, J.; et al. Genetic studies of body mass index yield new insights for obesity biology. Nature 2015, 518, 197–206.
9. Croteau-Chonka, D.C.; Ferreira, T.; Locke, A.E.; Mägi, R.; Strawbridge, R.J.; Pers, T.H.; Fischer, K.; Justice, A.E.; Workalemahu, T.; Wu, J.M.; et al. New genetic loci link adipose and insulin biology to body fat distribution. Nature 2015, 518, 187–196.
10. Goossens, G.H. The role of adipose tissue dysfunction in the pathogenesis of obesity-related insulin resistance. Nat. Med. 2014, 20, 1427–1435.
11. Roberts, C.K.; Hevener, A.L.; Barnard, R.J. Metabolic syndrome and insulin resistance: Underlying causes and modification by exercise training. Compr. Physiol. 2013, 3, 1–58.