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Insulin Resistance and Systemic Hypertension with Aging

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Abstract

This work was conducted on 18 normotensive, 7 hypertensive elderly subjects above the age of 60 and 10 normal young adults below the age of 40. They were subjected to blood pressure determination, body mass index estimation, estimation of fasting and 2 hour postprandial plasma glucose and serum insulin. Age and blood pressure both significanlty correlated with postprandial serum glucose and insulin levels. We thus concluded that there was an insulin resistant state and glucose intolerance in elderly poeple. Hypertension in elderly individuals was associated with and aggrevated insulin resistance and glucose intolerance. Postprandial serum insulin was a better predictor of insulin resistance and glucose intolerance in elderly individuals than the fasting levels.

Introduction

HYPERINSULINEMIA and insulin resistance are common in patients with essential hypertension even in the absence of diabetes mellitus or overt obesity [1,2].

It was found that the mean systemic arterial pressure and the incidence of non insulin dependent diabetes mellitus (NIDDM) increased significantly with aging [3,4].

Recent studies reported that hyperinsulinemia is a pathogenic mechanism rather than purely an association with hypertension [5,6].

The aim of this paper is to study the relationship between serum insulin level, plasma glucose and systemic blood pressure in normal elderly subjects.

Material and Methods

The study was conducted on 25 healthy elderly subjects above the age of 60 years with no history of hypertension or diabetes and 10 normal control group below the age of 40 years.

Patients were chosen from outpatient clinic of Ahmed Maher Hospital. They were subjected to the following:

1. Complete clinical assessment.
2. Estimation of plasma glucose fasting and 2 hours after 75g oral glucose using the glucose oxidase method on the Beckman ASTRA-4 (automated state routine analyser) [7].
3. Estimation of serun insulin by radioimmunoassay method [8].

Results

Out of the 25 elderly subjects studied, 18 were normotensive and 7 were accidentaly found to be hypertensive and acordingly were classified to 2 groups; a normotensive and a hypertensive group. The results of the work are shown in tables 1-5 and Figs. 1-3.

Table (1): Results of the Work.

	Control		T. Elderly		Normoten.		Hyperten.	
	Mean	S.D	Mean	S.D	Mean	S.D	Mean	S.D
SPP	122.0	7.9	141.2	24.5	127.8	10.0	175.7	14.0
DBP	75.0	5.3	81.8	10.1	76.6	4.9	95.0	7.6
MBP	99.0	5.7	111.7	16.5	102.5	6.0	135.4	7.6
FBG	85.9	7.5	103.1	23.6	96.2	18.4	120.0	27.5
PPG	98.6	9.3	153.3	40.4	137.8	32.9	193.3	29.7
FSI	13.0	6.5	14.8	6.9	12.6	6.2	20.3	5.4
PPSI	22.2	5.4	69.5	39.3	49.1	12.8	122.1	35.1

SBP, DBP, MBP: systolic, diastolic and mean blood pressure (mm Hg).
FBG, PPG: fasting and 2hr postprandial serum glucose (mg / dl).
FSI, PSI: fasting and 2 hr postprandial serum insulin (μIU / ml)

Table (2): Correlation Obtained between Age and each of the Following.

Parameter	r	p
Systolic blood pressure	0.34	< 0.05
Diastolic blood pressure	0.39	< 0.05
Fasting blood glucose	0.42	< 0.02
2 hrs blood glucose	0.48	< 0.01
Fasting plasma insulin	0.1	> 0.05
2 hrs plasma insulin	0.5	< 0.01

Discussion

Insulin resistance can be physiological, but most often is associated with various metabolic disorders, such as obesity, NIDDM, hypertension and dyslipidemia [9].

Insulin resistance and hyperinsulinemia is common in patients with essential hypertension, even in the absence of diabetes mellitus or overt obesity [2]. Recent studies reported that this hyperinsulinemia is a pathogenic mechanism rather than

Table (3): Correlation Obtained between SBP, DBP and each of the Following:

Parameter	r	p	r	p
Fasting blood glucose	0.37	< 0.05	0.63	< 0.001
2 hrs blood glucose	0.69	< 0.001	0.92	< 0.001
Fasting plasma insulin	0.47	< 0.01	0.34	< 0.05
2 hrs plasma inslin	0.85	< 0.01	0.84	< 0.001

Table (4): Correlation Obtained between waist/ Hip Ratio and each of the Following:

Parameter	r	p
Fasting blood glucose	0.13	> 0.05
2 hrs blood glucose	0.48	< 0.01
Fasting plasma insulin	0.19	> 0.05
2 hrs plasma inslin	0.35	< 0.05

Table (5): Correlation Obtained between Serum Insulin and Bloood Glucose:

Parameter	r	p
Fasting blood glucose vs 2 hrs blood glucose	0.43	< 0.05
Fasting plasma insulin vs2 hrs plasma inslin	0.76	< 0.001

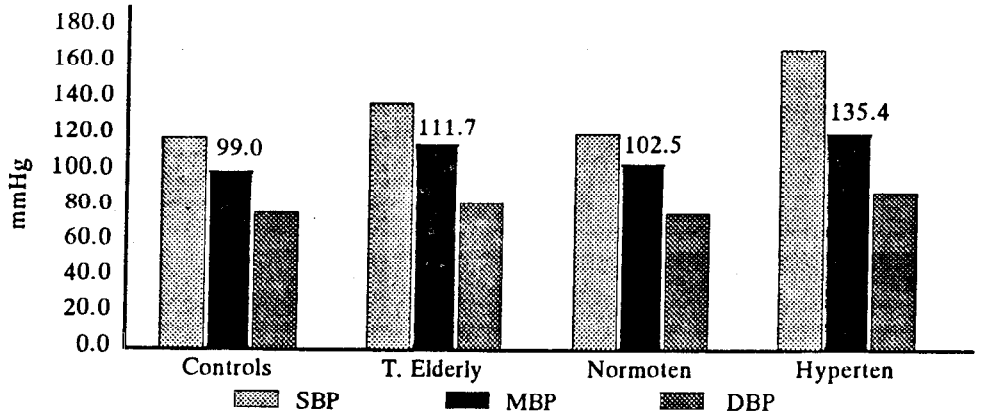

Fig. (1): Systolic, mean and diastolic arterial blood pressure in all groups

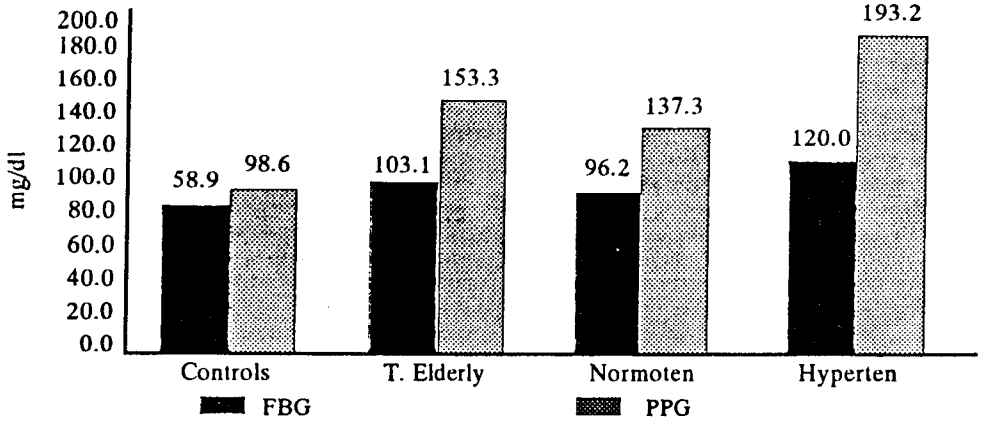

Fig. (2): Mean fasting and postprandial serum Glucose in all groups

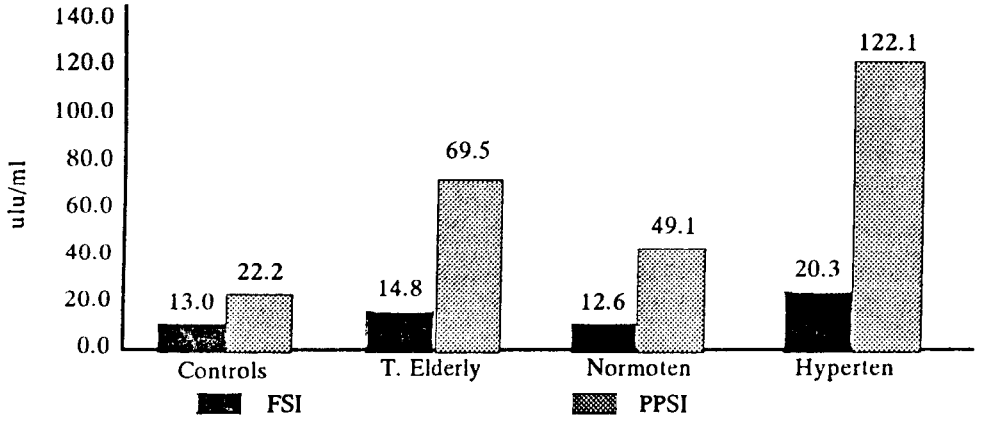

Fig. (3): Mean fasting and posprandial serum insulin in all groups

purely an association with hypertension or obesity [5,6].

Diabetes mellitus and hypertension are common diseases that coexist at a greater frequency than chance alone would predict [10]. It was found that the prevalence of hypertension among diabetic subjects was twice that of non diabetics and that the prevalence of diabetes significantly increased as the mean arterial pressure rose [11].

Kitzman and colleagues [3] reported that the mean systemic arterial pressure increased significantly with normal aging independent of gender. Croxson and colleagues [4] found that the incidence of NIDDM increased with age.

The aim of our work was to study the relationship between plasma glucose, serum insulin and systemic blood pressure in normal elderly subjects.

The systolic, diastolic and mean blood pressure were shown to be significantly higher in the total elderly versus control group ($p < 0.02$; $p < 0.01$; $p < 0.01$ respectively). There was a significant positive correlation between age and systolic and diastolic blood pressure ($p < 0.05$ in each). These results are in agreement with McFate [12] who stated that the observational changes in all Western sociaties studied have shown an increase in systolic and diastolic blood pressure with age. Wilking and colleagues [13] supported this conclusion and added that this increase occurs until at least the sixth decade, then the systolic blood pressure continues to increase almost linearly with advancing age.

The reason for this rise of blood pressure is not well established. It appears that the major hemodynamic abnormality is an increased peripheral resistance [14] which is a consequence of reduction in connective tissue elasticity of large blood vessels and an increase in aortic impedence or a decrease in aortic wall compliance [15]. Shimamoto and colleagues [16] concluded that the response of terminal aortic vascular bed to sodium loading contributes to the change in total peripheral resistance in elderly subjects.

Imoka and colleagues [17], found that elderly hypertensive patients compared to normotensive elderly and young controls had an increased level of basal serum parathyroid hormone and vitamin D with excessive loss of calcium and phosphorus in urine. This may participate in the abnormality of calcium metabolism in low renin hypertensive elderly. Abrass [18] suggested that there is a diminished beta adrenergic mediated vasodilatation with age whereas alpha adrenergic mediated vasoconstriction continues unabated.

Obesity, arteriosclerosis, atherosclerosis, baroreceptor insensitivity, decline in renal function, physical inactivity and insomnia are factors that can lead to or

aggrevate hypertension in old patients [19].

In our work, the total elderly group had a central type of obesity with a significantly higher waist/hip ratio than controls (p <0.001). Their blood pressure values were significantly higher than the controls. This is in accordance with Landen and colleagues [20] who found that the blood pressure was significantly higher in those with central fat distribution (waist hip ratio > 0.8).

The fasting blood glucose was significantly higher in the total elderly group and in the hypertensive elderly group than the control group (p < 0.002 in each). Fasting blood glucose was also significantly higher in the normotensive elderly group than both the control group and the hypertenive elderly group (p < 0.02 for each).

The postprandial blood glucose was significantly higher in the total elderly, the normotensive elderly and the hypertensive elderly groups than the control group (p < 0.001 in all). Also, the postprandial blood glucose was significantly higher in the normotensive elderly group than the hypertensive elderly group (p < 0.001).

A positive correlation was obtained between age and both fasting blood glucose and postprandial blood glucose (p 0.02 and 0.01 respectively).

Our results are in accord with others [21, 22, 23] who reported that glucose intolerance increased with age and that the greater effect of age was on the two hours postprandial glucose level.

The importance of peripheral insulin resistance caused be deficiency of post receptor mechanisms in the pathogenesis of glucose intolerance occurring with advancing age was reported by others [21]. Broughton and colleagues [24] added that the late insulin secretion may also be responsible. Meneilly and coworkers [25] found that with normal aging there is an impairment in non insulin mediated glucose disposal only in the basal state. This may explain in part the increase in fasting glucose with age.

Normal aging is associated with significantly delayed but overall equal post hepatic delivery of oral glucose in men [26], also much of the abnormal glucose metabolism that occurs with aging is postulated to be a result of changes in body composition and body fat distribution [27]. Our results showed that the waist hip ratio was positively correlated with both postprandial serum insulin and blood glucose levels (p < 0.05 and < 0.01 respectively). This is in agreement with others [28, 29].

The fasting and postprandial serum insulin were significantly higher in the hypertensive elderly group than the control and the normotensive elderly groups (p < 0.01 and <0.001 in both groups respectively).

There was a positive correlation between systolic blood pressure and both fasting and postprandial serum insulin ($p < 0.01$ and 0.001 for each respectively). Also, diastolic blood pressure positively correlated with both fasting and postprandial serum insulin levels ($p < 0.05$ and 0.001 for each respectively). These results agree with others [28, 30].

The postprandial serum insulin was significantly higher in the normotensive elderly group than the control group ($p < 0.001$). There was a highly significant correlation between postprandial serum insulin and age in normotensive patients ($p < 0.001$).

There was a significantly positive correlation between age and postprandial serum insulin ($p < 0.01$), while there was no statistically significant correlation between age and fasting serum insulin. These findings agree with those of Reaven and colleagues [30] who explained the associated age related postprandial hyperinsulinemia by increased secretion rather than decreased insulin clearance.

The insulin resistance in elderly subjects is suggested by our results as serum insulin level positively correlated with blood glucose both in the fasting and postprandial states ($p < 0.05$ and < 0.001 respectively). Smith [31] suggested insulin resistance as a cause of this hyperinsulinemia in the elderly. Broughton and colleagues [24] suggested that insulin resistance with aging might be due to inadequate increase of calcium in adipocytes.

Conclusions:

1. There is an insulin resistant state and glucose intolerance in elderly poeple.

2. Hypertension in the elderly individuals is associated with an insulin resistant state.

3. Hypertension in elderly individuals aggrevates insulin resistant state and glucose intolerance.

4. Postprandial serum insulin is a better predictor of insulin resistance and glucose intolerance in elderly individuals rather than the fasting levels.

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