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Original Research Article

A study of role of Insulin resistance in recently detected hypertensives in a rural population of Kishanganj, Bihar-A Cross sectional observational study

Authors
Dr Sourav Chattopadhyay[1], Dr Aniket Sinha[2], Dr Suman Kumar Singh[3], Dr Sumit Singh Kaushal[4], Dr Abhinav Chaudhary[5], Dr Sandeep Bharadwaj[6]
[1]Associate Professor, Dept. of General Medicine, M.G.M. Medical College & L.S.K. Hospital, Kishanganj, Bihar
[2]Resident, Dept. of General Medicine, M.G.M. Medical College & L.S.K. Hospital, Kishanganj, Bihar
[3]Resident, Dept. of General Medicine, M.G.M., Medical College & L.S.K. Hospital, Kishanganj, Bihar
[4]Resident, Dept. of General Medicine, M.G.M. Medical College & L.S.K. Hospital, Kishanganj, Bihar
[5]Resident, Dept. of General Medicine, M.G.M. Medical College & L.S.K Hospital, Kishanganj, Biha
[6]Dept. of General Medicine, Medical College & L.S.K. Hospital, Kishanganj, Bihar

Introduction

Insulin resistance and compensatory hyperinsulinaemia commonly occur in patients with untreated essential hypertension. The coexistence of insulin resistance and hypertension can be viewed as a cause-effect relationship (insulin resistance as a cause of hypertension or vice versa) or as a noncausal association. Insulin can increase blood pressure via several mechanisms: increased renal sodium reabsorption, activation of the sympathetic nervous system, alteration of transmembrane ion transport, and hypertrophy of resistance vessels. Conversely, hypertension can cause insulin resistance by altering the delivery of insulin and glucose to skeletal muscle cells, resulting in impaired glucose uptake.(1)

Aims &Objectives

1. To measure the serum Insulin level and Insulin Resistance in newly detected Hypertensive patients.
2. To coorelate the serum Insulin level and Insulin Resistance with grading of Hypertension

Study Design

A cross sectional observational study

Study Place

The study was conducted at Dept. of General Medicine, M.G.M. Medical College & L.S.K. Hospital, Kishanganj, Bihar.

Study Population

The target population consisted of patients with the diagnosis of newly detected untreated hypertension, either admitted as In-patient (IPD) or visiting the Out Patient Department (OPD) between December 2018 and November 2020. 100 consecutive patients fulfilling all inclusion and exclusion criteria were included in the study as cases.

100 healthy age and sex-matched controls, all of who were non-hypertensive and fulfilling the exclusion criteria were included as controls in the study.

Inclusion Criteria

The inclusion criteria are newly diagnosed hypertension (Systolic blood pressures>140 mm hg or diastolic blood pressure >90 mm hg
Adult of age <60 years and >18 years
Age-matched and sex matched normal healthy individuals.

Exclusion Criteria

Patients who had a BMI of more than 25 kg/m2 and known case of
Type 2 diabetes mellitus
Dyslipidemia
hypertension
Congestive heart failure
Pregnant women
Chronic kidney disease
Thyroid disorder
Rheumatological disease
Chronic liver disease

Results

Our study was a hospital-based case–control study that included 100 newly detected hypertensive patients and 100 matched controls. Serum fasting insulin levels were measured in cases and controls both. Statistical association of hypertension with serum fasting insulin levels and insulin resistance was studied. The finding of the study are placed below –
The mean value of serum fasting insulin in our study was 15.31 ± 3.65 µIU/mL in
hypertensive patients and 7.99 ± 1.24 µIU/mL in control.

This is well supported by study done by Rayees Tarray et al where the mean serum fasting insulin values were 15.32±13.76 µIU/mL in patients and 8.01±4.08 µIU/mL in controls. Serum fasting insulin levels in our study were found to be statistically significantly related to the hypertension and this was well in accordance with the study done by Rayees Tarray et al.[2] The positive association between Serum fasting insulin levels and hypertension was also well supported by study done by Ferrannini E et al[3]
In addition, our patients were first-time detected hypertensive and had not received
any antihypertensive treatment. We hypothesize that these parameters may have a confounding effect on the overall results. In addition, ethnicity may also have a role. We also report a significant difference in insulin resistance between the two groups.
The mean HOMA2-IR was 3.22 ± 1.19 in the hypertensive group as compared with
1.73 ± 0.48 among the controls. This difference was statistically significant
(P=0.0001). The mean value of HOMA2– IR level observed in our study in well accordance with those observed by
Rayees Tarray et al[2] where the mean value of HOMA2- IR in cases was 3.81±3.42 and controls was 1.76±0.93.
HOMA2 -IR in our study were found to be statistically significantly related to the
hypertension and this was well in accordance with the study done by Rayees Tarray et al[2]. The positive association between insulin resistance and hypertension was also well supported by study done by Ferrannini E et al[3]. They conclude that insulin resistance is directly correlated with the severity of hypertension which further supports our study.
In our study insulin resistance and fasting insulin was significantly associated with
hypertension but ethnic differences were not analyzed separately.
In our study we did find statistical significant correlation of serum fasting insulin levels and insulin resistance values with stages of hypertension. The study done by Rayees Tarray et al did not mention it in terms of stages of hypertension. There is no reference of study for correlation of serum insulin resistance with grading of hypertension.

Review of Literature

Insulin resistance and reactive hyperinsulinemia occur not only with obesity, impaired glucose tolerance or non-insulin-dependent (type 2) diabetes mellitus, but also in many non-obese, non-diabetic patients with essential hypertension and their currently normotensive, lean young offspring and in some other conditions known to promote hypertension. Insulin resistance impairs glucose tolerance, while insulin resistance and/or hyperinsulinemia promote dyslipidemia, body fat deposition and probably atherogenesis. Therefore, the common coexistence of a genetic predisposition for hypertension with insulin resistance helps to explain the frequent, although temporally often dissociated, occurrence of hypertension as well as dyslipidemia, obesity and type 2 diabetes in a given subject. Pathogenetic mechanisms: In the pathogenesis of hypertension, inappropriate vasoconstriction (due to dysbalance of vasoactive substances and/or raised cytosolic Ca2+) and/or a structural vasculopathy is a very important ultimate causative event. In the presumed mosaic of participating pressor mechanisms, distinct Na+ retention is almost obligatory with diabetes mellitus, while essential and particularly obesity-associated hypertension probably involves a tendency for sympathetic activation. Development of insulin resistance: Insulin resistance may develop as a consequence of an intracellular excess of Ca2+ or decrease in Mg2+, an impaired insulin-mediated rise in skeletal muscle blood flow, increased sympathetic activity or being overweight. Acute hyperinsulinemia on the one hand causes arterial vasodilation and on the other hand enhances renal sodium reabsorption and sympathetic activity. Chronically, hyperinsulinemia may promote cardiovascular muscle cell proliferation and atherogenesis, and it has been proposed that insulin resistance in certain transmembranous cation exchange systems may elevate cytosolic Ca2+. Nevertheless, whether insulin resistance and/or hyperinsulinemia itself contribute to the pathogenesis of hypertension is still unclear.[4]

Resistance to the action of insulin on glucose metabolism, with the ensuing compensatory hyperinsulinaemia, is closely linked to essential hypertension. The decreased insulin sensitivity observed in hypertensive patients is independent of obesity. Hyperinsulinaemia is likely to promote the dyslipidaemia that frequently accompanies the hypertensive state, and often presents as increased total and very low density lipoprotein (VLDL)-triglycerides, low high density lipoprotein (HDL)-cholesterol and, in some studies, elevated levels of low density lipoprotein (LDL)-cholesterol. Lipid abnormalities, hypertension and possibly hyperinsulinaemia act together to increase the risk of atherosclerotic disease manifestations in hypertensive patients. Acutely, insulin has been shown to stimulate sympathetic nervous system activity and transmembrane electrolyte transport, to promote sodium retention and to cause vascular wall changes, including increased cholesterol biosynthesis and smooth muscle proliferation. If these mechanisms operate on a chronic basis, the continuous exposure to elevated plasma insulin levels may play a pathogenetic role in the development of high blood pressure, and also of a predisposition toward atherosclerosis in patients with hypertension. Further studies are necessary to establish these hypothetical cause-effect relationship which, if shown to be true, will contribute to a more wide-ranging view of essential hypertension and the optimum strategy for antihypertensive treatment.[5]

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