(e) ISSN Online: 2321-9599

(p) ISSN Print: 2348-6805

ORIGINAL ARTICLE

Assessment of Adiponectin Levels in Type 2 Diabetes patients

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ABSTRACT:
Background: The present study was conducted by for assessing adiponectin Levels in Type 2 Diabetes patients. **Materials & methods:**A total of 50 diabetic patients and 50 healthy controls were enrolled. Complete demographic and clinical details were obtained. A Performa was made and detailed medical history was reported. All the patients were recalled in the morning and blood samples were obtained. Serum adiponectin levels were recorded separately. All the results were recorded and analyzed using SPSS software. **Results:**Mean age of the patients of the diabetic group and control group was 45.6 years and 48.1 years respectively. Majority proportion of patients of both the study groups were males. Mean serum adiponectin levels among diabetic patients and healthy controls was 1.9 µg/ml and 2.9 µg/ml respectively. While comparing the results statistically, significant results were obtained. **Conclusion:** Adiponectin levels are significantly altered in diabetic patients.
Key words: Diabetes, Adiponectin

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This article may be cited as: Ramrao TD, Verma AK. Assessment of Adiponectin Levels in Type 2 Diabetes patients. J Adv Med Dent Scie Res 2014;2(4):275-277.

INTRODUCTION

Diabetes is a chronic illness that requires continuing medical care and ongoing patient self-management education and support to prevent acute complications and to reduce the risk of long-term complications. Diabetes care is complex and requires that many issues, beyond glycemic control, be addressed. A large body of evidence exists that supports a range of interventions to improve diabetes outcomes.These standards of care are intended to provide clinicians, patients, researchers, payors, and other interested individuals with the components of diabetes care, general treatment goals, and tools to evaluate the quality of care. While individual preferences, comorbidities, and other patient factors may require modification of goals, targets that are desirable for most patients with diabetes are provided. These standards are not intended to preclude clinical judgment or more extensive evaluation and management of the patient by other specialists as needed.[1-3]

Several pathogenic processes are involved in the development of diabetes. These range from autoimmune destruction of the β-cells of the pancreas with consequent insulin deficiency to abnormalities that result in resistance to insulin action. The basis of the abnormalities in carbohydrate, fat, and protein metabolism in diabetes is deficient action of insulin on target tissues. Deficient insulin action results from inadequate insulin secretion and/or diminished tissue responses to insulin at one or more points in the complex pathways of hormone action. Impairment of insulin secretion and defects in insulin action frequently coexist in the same patient, and it is often unclear which abnormality, if either alone, is the primary cause of the hyperglycemia.[4, 5]Symptoms of marked hyperglycemia include polyuria, polydipsia, weight loss, sometimes with polyphagia, and blurred vision. Impairment of growth and susceptibility to certain infections may also accompany chronic hyperglycemia. Acute, life-threatening consequences of uncontrolled diabetes are hyperglycemia with ketoacidosis or the nonketotic hyperosmolar syndrome.[6-9]

Adiponectin is a 244–amino acid collagen-like protein that is solely secreted by adipocytes and acts as a hormone with anti-inflammatory and insulin-sensitizing properties. Findings from animal studies and metabolic studies in humans suggest several mechanisms through which adiponectin may decrease the risk of type 2 diabetes, including suppression of hepatic gluconeogenesis, stimulation of fatty acid oxidation in the liver, stimulation of fatty acid oxidation and glucose uptake in skeletal muscle, and stimulation of insulin secretion.[10] Hence; the present study was conducted by for assessing adiponectin Levels in Type 2 Diabetes patients.

MATERIALS & METHODS

The present study was conducted for assessing adiponectin Levels in Type 2 Diabetes patients. A total of 50 diabetic patients and 50 healthy controls were enrolled. Complete demographic and clinical details were obtained. A Performa was made and detailed medical history was reported. All the patients were recalled in the morning and blood samples were

obtained. Serum adiponectin levels were recorded separately. All the results were recorded and analyzed using SPSS software. Student t test was used for evaluation of level of significance.

RESULTS

Mean age of the patients of the diabetic group and control group was 45.6 years and 48.1 years respectively. Majority proportion of patients of both the study groups were males. Mean serum adiponectin levels among diabetic patients and healthy controls was 1.9 µg/ml and 2.9 µg/ml respectively. While comparing the results statistically, significant results were obtained.

Table 1: Comparison of serum adiponectin levels

Adiponectin levels (µg/ml)	Diabetic patients	Controls
Mean	2.9	1.9
SD	1.7	1.1
p-value	0.001 (Significant)	

DISCUSSION

Diabetes mellitus (DM) is a clinical syndrome associated with deficiency of insulin secretion or action. It is considered one of the largest emerging threats to health in the 21st century. It is estimated that there will be 380 million persons with DM in 2025. Besides the classical complications of the disease, DM has been associated with reduced response of T cells, neutrophil function, and disorders of humoral immunity. Consequently, DM increases the susceptibility to infections, both the most common ones as well as those that almost always affect only people with DM (e.g. rhinocerebral mucormycosis). Such infections, in addition to the repercussions associated with its infectivity, may trigger DM complications such as hypoglycemia and ketoacidosis.[10- 12]

Mean age of the patients of the diabetic group and control group was 45.6 years and 48.1 years respectively. Majority proportion of patients of both the study groups were males. Mean serum adiponectin levels among diabetic patients and healthy controls was 1.9 µg/ml and 2.9 µg/ml respectively. While comparing the results statistically, significant results were obtained. Tschritter O et al genotyped single nucleotide polymorphisms (SNPs) in ADIPOQ to evaluate the association of common variants with adiponectin levels and risk of diabetes.Participants in the Framingham Offspring Study (n = 2,543, 53% women) were measured for glycemic phenotypes and incident diabetes over 28 years of follow-up; adiponectin levels were quantified at exam 7. We genotyped 22 tag SNPs that captured common (minor allele frequency >0.05) variation at r(2) > 0.8 across ADIPOQ plus 20 kb 5' and 10 kb 3' of the gene. We used linear mixed effects models to test additive associations of each SNP with adiponectin levels and glycemic phenotypes. Hazard ratios (HRs) for incident diabetes were estimated using an adjusted Cox proportional hazards model.Two promoter SNPs in strong linkage disequilibrium with each other (r(2) = 0.80) were associated with adiponectin levels (rs17300539; P(nominal) [P(n)] = 2.6 x 10(-8); P(empiric) [P(e)] = 0.0005 and rs822387; P(n) = 3.8 x 10(-5); P(e) = 0.001). A 3'-untranslated region (3'UTR) SNP (rs6773957) was associated with adiponectin levels (P(n) = 4.4 x 10(-4); P(e) = 0.005). A nonsynonymous coding SNP (rs17366743, Y111H) was confirmed to be associated with diabetes incidence (HR 1.94 [95% CI 1.16-3.25] for the minor C allele; P(n) = 0.01) and with higher mean fasting glucose over 28 years of follow-up (P(n) = 0.0004; P(e) = 0.004). No other significant associations were found with other adiposity and metabolic phenotypes.[13]

Menzaghi C et al assessed the Genetic influences of adiponectin on insulin resistance, type 2 diabetes, and cardiovascular disease. Two independent effects, corresponding to the two linkage disequilibrium blocks that can be identified at the adiponectin locus, appear to be present. In the 5' block, the g.-11391G-->A variant has a modest but significant effect on adiponectinemia, with a mean difference between genotypes of 1.64 ng/ml (95% CI 0.88-2.41). In the 3' block, the g.+276G-->T variant is a strong determinant of insulin resistance and CAD, with minor allele homozygotes having a lower homeostasis model assessment of insulin resistance (HOMA(IR)) index (-0.36 units, 95% CI 0.24-0.47) and a lower cardiovascular risk (odds ratio 0.55, 95% CI 0.38-0.80) than carriers of other genotypes. No consistent effect on BMI or risk of type 2 diabetes is evident. Polymorphisms in the genes coding for the adiponectin receptors may also influence the risk of insulin resistance and CAD, but data on these genes are still too sparse to draw firm conclusions.[14]

CONCLUSION

Adiponectin levels are significantly altered in diabetic patients.

REFERENCES

1. American Diabetes Association Medical Management of Type 1 Diabetes. Alexandria, VA, American Diabetes Association, 2008
2. American Diabetes Association Medical Management of Type 2 Diabetes. Alexandria, VA, American Diabetes Association, 2008
3. American Diabetes Association Intensive Diabetes Management. Alexandria, VA, American Diabetes Association, 2009
4. Expert Committee on the Diagnosis and Classification of Diabetes Mellitus Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Diabetes Care 1997; 20: 1183– 1197
5. Nathan DM, Davidson MB, DeFronzo RA, Heine RJ, Henry RR, Pratley R, Zinman B: American Diabetes Association Impaired fasting glucose and impaired glucose tolerance: implications for care. Diabetes Care 2007; 30: 753– 759

6. American Diabetes Association: Consensus statement on self-monitoring of blood glucose. Diabetes Care 1987; 10: 95– 99
7. American Diabetes Association: Self-monitoring of blood glucose. Diabetes Care 1994; 17: 81– 86
8. Welschen LM, Bloemendal E, Nijpels G, Dekker JM, Heine RJ, Stalman WA, Bouter LM: Self-monitoring of blood glucose in patients with type 2 diabetes who are not using insulin: a systematic review. Diabetes Care 2005; 28: 1510– 1517
9. Farmer A, Wade A, Goyder E, Yudkin P, French D, Craven A, Holman R, Kinmonth AL, Neil A: Impact of self monitoring of blood glucose in the management of patients with non-insulin treated diabetes: open parallel group randomised trial. BMJ 2007; 335: 132.
10. Carpenter MW, Coustan DR: Criteria for screening tests for gestational diabetes. Am J ObstetGynecol 1982; 144: 768– 773
11. O'Sullivan JB, Mahan CM: Criteria for the oral glucose tolerance test in pregnancy. Diabetes 1964; 13: 278.
12. HAPO Study Cooperative Research Group. Metzger BE, Lowe LP, Dyer AR, Trimble ER, Chaovarindr U, Coustan DR, Hadden DR, McCance DR, Hod M, McIntyre HD, Oats JJ, Persson B, Rogers MS, Sacks DA: Hyperglycemia and adverse pregnancy outcomes. N Engl J Med 2008; 358: 1991– 2002
13. Tschritter O, Fritsche A, Thamer C, et al. Plasma adiponectin concentrations predict insulin sensitivity of both glucose and lipid metabolism. Diabetes. 2003;52(2):239-243
14. Menzaghi C, Trischitta V, Doria A. Genetic influences of adiponectin on insulin resistance, type 2 diabetes, and cardiovascular disease. Diabetes. 2007;56(5):1198-1209