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Is low adiponectin concentration linked to the development of type 2 diabetes in Sudan

Abstract

Objectives: It was previously thought that adiponectin influenced insulin activity in tissues. Insulin resistance caused by obesity is associated to reduced plasma adiponectin levels. Researchers may be able to better understand the role of adiponectin in insulin resistance and type 2 diabetes by comparing adiponectin levels in T2DM patients to non-diabetic patients, as well as its connection with BMI and WC.

Method: A case-control study was conducted at the Abu A'gla Health Care Center for diabetes care in Wad Madani, Gezira State, Sudan, between April 2012 and March 2013. The study involved a total of 181 participants. To measure adiponectin, FPG, and HbA_{1C} levels, patients were divided into diabetes and non-diabetic groups. The body mass index (BMI) was calculated, and the waist circumference (WC) was measured. Personal information (age and gender) were obtained. Samples were analyzed for many biochemical parameters using the A15, a random-access auto-analyzer bio system. To quantify adiponectin, ELIZA employed the techniques of a human adiponectin ELISA kit. A statistical software for social sciences was used to conduct the statistical analysis (SPSS version 16, Chicago, IL, USA).

Result: The mean BMI (29.007) increased significantly between diabetic and non-diabetic groups (p=0.001) indicating that the study participants were overweight. There was significant increased (p<0.0001) in FPG (160.10) and HbA_{1C} (6.9813) and non- significant decreased in adiponectin mean (1.567) concentration. SBP and DBP mean (116.52) and (75.51) were significantly low (p=0.006) and (0.054), respectively.

Conclusion: Adiponectin levels were lower in diabetic and non-diabetic patients. Only two diabetics had excessive quantities. Adiponectin and BMI were thought to have an inverse relationship, with no association between adiponectin and WC.

Keywords: adiponectin, type 2 diabetes mellitus, BMI, WC, Sudan.

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Abbreviations

T2DM, type2 diabetes mellitus; FPG, fasting plasma glucose; HbA_{1C}, glycosylated hemoglobin; HT, hypertension; WC, waist circumferance; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; p, probability; F, degree of freedom.

Introduction

White adipose tissue releases a variety of regulating chemicals known as adipocytokines, such as leptin and adiponectin.[1] since adipocytokines have multi-potent impacts on health and illness; over 700 distinct adipocytokines have been found up to 2012. This fact offers adipose tissue an extra role as a significant and biggest endocrine organ.[2] Beside their basic role on store fat, provide energy, and provide insulation.[3] Protein molecules produced by adipose tissue have a role in autocrine and paracrine control inside adipose tissue, as well as affecting the activities of distant organs such muscle, the pancreas, the liver, and the central nervous system.[4] The adipocytokines modulate hemostasis, blood pressure, lipid and glucose metabolism, inflammation, and atherosclerosis.[5] Adiponectin is a protein identified in 1995 and exclusively produced from adipocytes, known as (the gene product of the adipose most abundant gene transcript-1 (apM1).[6] Its structure consists of 224 amino acid-long polypeptide of 30 kDa.[7] The N-terminal region consists of a 20-residue signal sequence without homology to any known proteins, beside a collagen-like region, and a C-terminal globular domain. The three-dimensional structure of its C-terminal globular domain is similar to that of tumor necrosis factor-alpha (TNF-α).[8] It account for 0.01% of total plasma protein.[9] Moreover, adiponectin has two distinct isoform, a high molecular weight form, and a more bioactive low molecular weight form[10] and two specific receptors AdipoR1 and R2. AdipoR1 is abundantly expressed in skeletal muscle, whereas AdipoR2 is predominantly expressed in the liver.[11]

Adiponectin possesses an effective insulin-sensitizing with anti-diabetic effect, anti-inflammatory, and anti-atherogenic properties[12] by inhibition of monocytic cell adhesion to endothelial cells,[13] suppression of vascular smooth muscle cell proliferation,[14] and inhibition of foam cell formation from macrophages.[15] It exerts its function through phosphorylation of tyrosine kinase which signaling insulin receptors[16] in liver and skeletal muscle,[17] this action is mediated by AMP kinase that phosphorylate several target protein thus effect metabolic pathways. adiponectin has obvious effect on both carbohydrate and lipid metabolism because it promote the uptake and oxidation of fatty acids by myocytes, but blocks synthesis of fatty acids and gluconeogenesis by hepatocyte. At the same time uptake and metabolism of glucose by muscle and liver are favored.[18] Low circulating adiponectin is associated with increasing adipose tissue mass and obesity[19] resulting in insulin resistance, type 2 diabetes mellitus (T2DM) and dyslipidemia.[20]

Induction of insulin sensitivity by administration of recombinant adiponectin predominantly in the blood stream result in increased insulin secretion,[21] hypoglycemia and hyper-insulinemia without inducing weight gain or even inducing weight loss.[22] However, in response to weight loss circulating total or high molecular weight adiponectin levels lead to improvements in insulin sensitivity.[23] Weight loss is achieved by adiponectin in the brain which increase energy expenditure and may thereby promote weight loss.[24]

Adiponectin is now recognized as component of a novel signaling network among adipocytes,[25] insulin-sensitive tissues, and vascular function that has important consequences for cardiovascular risk.[26] An adipocyte-endothelium interaction might be an important mechanism of inflammation and vascular dysfunction.[27] Insulin action is affected in various tissues by the release of different adipocytokines.[28] The interacting of insulin with adiponectin cause obesity-induced insulin resistance, DM, and diabetic complications.[29] Investigating adipocytokine levels in T2DM and their association with anthropometric measurements BMI and WC may help understanding the role of adipocytokines in type 2 diabetes patients in Sudan.

Material and methods

Study Subject, design and area: A total of 181 people were involved in a cross-sectional study with a case-control design between April 2012 and March 2013, with 100 participants having diabetes and 81 serving as the control group. All patients who came to the Abu A'gla Health Care Center from the Wad Madani city district and nearby rural and urban areas were taken care of. The participants represented many Sudanese tribes.

Inclusion and exclusion criteria: The participants in this study were between the ages of 18 and 60, had no current infection, and had no diabetes complications. The non-diabetic group was made up of healthy people who volunteered to take part in the study. A subject was removed from the study if they did not meet any of the inclusion criteria.

Ethical approval: The study received ethical approval from the Ministry of Health's Ethics Committee.

Study procedure: after receiving informed consent all participants provided bio data and anthropometric measures (weights was measured in kilograms (kg), heights was measured in meters (m), and the body mass index (BMI) was calculated using the formula: BMI = (weight in kg)/ (height in m)^2(Ng M, 2014). Plasma samples were assessed for different biochemical parameters using the A15, a random-access auto-analyzer bio system. To quantify adiponectin, ELIZA used techniques from a human adiponectin ELISA kit. This test can be used to measure the amount of adiponectin present in human serum, plasma, and other bodily fluids.

Statistical analysis: The statistical analysis was done with the help of a statistical software for social sciences (SPSS version 16, Chicago, IL, USA). All the numerical data were expressed as mean ± Standard Error of Mean. The proportion of distribution of study participants was calculated using the Chi-square test. Analysis of variance was used to compare differences in the means of continuous variables between the research groups (ANOVA). P-values of <=0.05 were considered significant.

Results

Tables 1 and 2 show the study participants' distribution as well as the results of a one-way ANOVA, which show the significant and non-significant relationship between adiponectin mean concentration and the mean of research variables.

Table 1 Distribution of participants with in study variables according to group and adiponectin concentration

Variables	Characteristic	Group		Adiponectin concentration n=181		Total
		Diabetic n=100	Non-diabetic n=81	Low < 6.4	High=>8.4	
Gender	Male	26	27	53	0	**53**
	Female	74	54	125	3	**128**
Age group\yrs.	20-29	1	2	3	0	**3**
	30-39	3	4	7	0	**7**
	40-49	40	47	87	0	**87**
	50-59	49	22	68	3	**71**
	60-69	7	6	13	0	**13**
WC(Cm)*	Male<=102	14	23	37	0	**37**
	Male>102	12	4	16	0	**16**
	Female<=88	9	7	16	0	**16**
	Female>88	65	47	109	3	**112**
BMI (kg/m2) *	under weight (BMI < 18.50)	0	1	1	0	**1**
	Normal (BMI 18.50 - 24.99)	15	30	44	1	**45**
	Overweight (BMI 25 - 29.99)	40	30	68	2	**70**
	Obese class I (BMI 30 - 34.99)	27	9	36	0	**36**
	Obese class II (BMI 35-39.99)	14	8	22	0	**22**
	Obese class III (BMI =>40.00)	4	3	7	0	**7**
HT Group**	Normal (SBP\DBP less than 120\ 80)	28	43	69	2	**71**
	Pre-hypertension (SBP\DBP 120\80)	64	33	96	1	**97**
	Stage 1 HT (SBP 140 -159 or DBP 90 -99)	8	4	12	0	**12**
	Stage 2 HT (SBP\ DBP 160\100 and above)	0	0	1	0	**0**
Physical activity	Low	7	22	29	0	**29**
	Moderate	57	52	107	2	**109**
	High	36	7	42	1	**43**
Medications	Hypoglycemic drug	93	-	90	3	**93**
	Dietary control	7	-	7	0	**7**
	Metformin	21	-	-	-	**21**
Hypoglycemic drug	glyburide	37	-	-	-	**37**

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Table Continued...

Variables	Characteristic	Group		Adiponectin concentration n=181		Total
		Diabetic n=100	Non-diabetic n=81	Low < 6.4	High=>8.4	
	Metformin+ glyburide	34	-	-	-	**34**
	Others	8	-	-	-	**8**
Additional drugs	Yes	4	-	4	0	**4**
	No	96	-	93	3	**96**
Dietary restriction	Yes	44	7	51	0	**51**
	No	56	73	126	3	**129**
	Low < 75	2	12	14	0	**14**
FPG (mg/dl)	Normal 75 - 115	19	60	79	0	**79**
	High > 115	79	9	85	3	**88**
	Excellent <6.5	30	75	105	0	**105**
HbA1C (%)	Good 6.5-7.5	6	6	11	1	**12**
	Moderate 7.5-8.9	20	0	19	1	**20**
	Poor >=9	44	0	43	1	**44**
Adiponectin (ug/L)* **	Low < 6.4	98	81	-	-	**179**
	Normal 6.5-8.4	0	0	-	-	**0**
	High >8.4	2	1	-	3	**3**

Source NHLBI Obesity Education Initiative (2000) *; American heart association**; (Wang, Y et al.) [47]***;

FPG, fasting plasma glucose; HbA_{1C}, glycosylated hemoglobin; HT, hypertension; WC, waist circumferance; BMI, body mass index; Cm, centimeter; Kg, kilogram; m, meter

Table 2 Comparison of mean of adiponectin concentration with mean of anthropometric and biochemical measurements of study variables

Variables	Minimum	Maximum	Mean in Diabetic	Mean in Non-diabetic	Total Mean	SEM	F	Sig.
Age (years)	22	65	49.67±0.71	46.42±0.88	48.22	0.567	8.42	0.004
Weight (kg)	40	171	79.95±1.69	71.80±1.60	76.3	1.211	11.9	0.001
Height (m)	1.4	1.9	1.62±0.01	1.62±0.01	1.6236	0.007	0.03	0.861
WC (Cm)	52	127	98.69±1.15	97.27±1.27	98.06	0.851	0.69	0.409
BMI (kg/m2)	17.31	55.2	30.36±0.58	27.33±0.64	29.007	0.446	12.2	0.001
SBP (mmHg)	80	170	118.60±0.80	113.95±1.59	116.52	0.853	7.62	0.006
DBP (mmHg)	30	100	76.70±0.71	74.07±1.25	75.51	0.687	3.77	0.054
FPG (mg/dL)	46	442	215.33±9.93	93.42±2.12	160.1	7.199	121	<0.0001
HbA1C (%)	3.1	15	8.32±0.29	5.33±0.09	6.9813	0.196	83	<0.0001
Adiponectin (ug/L)	0	8.55	1.73±0.12	1.37±0.15	1.567	0.096	3.46	0.064

SBP, systolic blood pressure; DBP, diastolic blood pressure; p, probability; F, degree of freedom; µg, microgram; mg, milligram; dL, deciliter; mmHg, millimeter of mercury; SEM, standard error of the mean

The difference in mean BMI (29.007) between the diabetic (30.36) and non-diabetic (27.33) groups was significant (p=0.001) therefore the study population was overweight or obese.

The difference between diabetes and non-diabetic groups in mean FPG (160.10) and HbA_{1C} (6.9813) was significant (p<0.0001). The diabetes and non-diabetic groups had significantly reduced mean SBP and DBP by (p=0.006) and (0.054), respectively, while only 8 diabetic and 4 non-diabetic participants had Stage 1 HT.

Discussion

Diabetic patients had significant increase in FPG and they can't achieve the goal for HbA_{1C}. So, study patients had uncontrolled plasma glucose. With increased in BMI, the diabetic participants were at high risk for developing diabetes complications.[30] Dietary control and regular exercise are the first steps in treating diabetes; these two strategies appear to lower HbA_{1C} but not FPG.[31] Additionally, prevention of diabetes can be achieved by engaging in 30 minutes of moderate-intensity physical activity; however, this eventually leads to a chronic increase in the concentration of inflammatory molecules like IL-6 and TNF.[32] Participants in the current study showed moderate physical activity with no regular exercise; this may have contributed to the significant rise in BMI, which may lead to obesity and metabolic abnormalities such as insulin resistance and decreased fatty acid oxidation.[33,34] The dietary plan was ignored by both the diabetic patients and Abu A'gla diabetic health care center.[35] showed that dietary restriction is effective in reducing adipose mass and central or visceral adiposity by diminished the biomarkers of inflammation, and increase the adiponectin which is associated with insulin resistance.

In terms of adipocytokines, adiponectin levels were lower in all individuals in the current investigation, with high concentrations in only three diabetic female. There was no correlation with WC, which exhibited a non-significant rise when compared to adiponectin concentration. The previous studies showed that low adiponectin concentration is associated with high risk of developing T2DM[36]

and that adiponectin is a marker of metabolic control associated with a high risk of cardiovascular complications and atherosclerosis. These finding were in agreement with the study of[37] which also found individuals with T2DM had lower adiponectin values and that subjects with well controlled diabetes mellitus had higher values than those with uncontrolled diabetes. These findings matched those of recent research we conducted. Furthermore, lower adiponectin levels have been reported in African-American children compared to their European-American counterparts[38] and have been linked to a variety of phenotypic traits. As a result, adiponectin has been proposed as a metabolic syndrome marker.[39]

Three diabetic female (whom had similar characteristics to most diabetic participants included in this study) reveal unexpected higher adiponectin level. The characteristics of those participants as follow: the age between 50-59 years with WC above 88, two were overweight and one had a normal BMI, two with moderate physical activity, and one involved in vigorous physical activity, all three female take hypoglycemic medications and did not follow any dietary restrictions. Those female have high blood glucose with good, moderate and poor glycemic control (table 3). In a cross-sectional study that has been conducted in Jordanian subjects diagnosed with type 2 diabetes, female diabetic patients had a statistically significant higher adiponectin level than male diabetic patients which may indicate a gender effect, which is in line with current study. Adiponectin levels, in contrast to our findings, were inversely associated to abdominal obesity.[40]

Table 3 The characteristic of study participants with high adiponectin concentration

Variable	Characteristics	High Adiponectin concentration =>8.4
Group	Diabetic	3
Gender	Female	3
Age group\yrs.	50-59(57, 56, 50)	3
WC(Cm)*	Female>88(89, 95,99)	3
BMI (kg/m2) *	Normal (BMI 18.50 - 24.99) (24.97)	1
	Overweight (BMI 25 - 29.99) (29.86, 28.89)	2
HT Group**	Normal (SBP\DBP less than 120\ 80)	2
	Pre-hypertension (SBP\DBP 120\80)	1
Physical activity	Moderate	2
	High	1
Medications	Hypoglycemic drug	3
Dietary restriction	No	3
FPG (mg/dl)	High > 115(224, 181, 409)	3
	Good 6.5-7.5(7.10)	1
HbA1C (%)	Moderate 7.5-8.9(8.80)	1
	Poor >=9(10.90)	1
Adiponectin (ug/L) ***	High >8.4(8.55, 8.72, 8.43)	3

Source NHLBI Obesity Education Initiative (2000) *; American heart association**; (Wang, Y et al. 2018) ***; FPG, fasting plasma glucose; HbA_{1C}, glycosylated hemoglobin; HT, hypertension; WC, waist circumferance; BMI, body mass index; Cm, centimeter; Kg, kilogram; m, meter

The current study discovered an inverse relationship between adiponectin and BMI, demonstrating that a reduction in BMI or weight loss will result in an increase in plasma adiponectin. This finding was consistent with that of,[41] who suggested that adiponectin could be a marker of cardiovascular disease associated with T2DM, and that the lower the level of adiponectin concentration, the higher the risk of cardiovascular disease.[42] found that a higher BMI is linked to a worse insulin response, as evidenced by higher HbA_{1C} levels and worse accomplishment of the goal value. The distribution of body fats and visceral adiposity in diabetics is correlated with the development of insulin resistance and T2DM, according to observations obtained using BMI and WC.[43] Metformin, glyburide, or both were used as hypoglycemic medications on diabetic individuals in the current investigation. The main one, metformin, works by decreasing the liver's ability to produce glucose and by enhancing insulin receptor binding by 20% in circulating cells (erythrocytes and monocytes), resulting in lowering blood glucose levels.[44] The K_{ATP} channel blocker glyburide, a second-generation sulfonylurea, increases intracellular potassium and calcium ion concentrations in beta cells.[45] Our findings were consistent with those of the[46] study which showed that the co-administration of metformin or glyburide had no impact on the increase in adiponectin concentration.

Conclusion

Adiponectin levels were lower in diabetic and non-diabetic patients. Only two diabetics had excessive quantities. Adiponectin and BMI were thought to have an inverse relationship, with no association between adiponectin and WC.

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Disclosure of conflict of interest

None.

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