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Evaluation of Anti-GAD, IAA Presence and Measurement of C- Peptide Level in T2DM Patients, 60 and above 60 years old in Nasiriyah City / South of Iraq

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Dr. Talib Hassan Ali

Abstract

***Background**: The study aimed to estimate the prevalence of positive autoantibodies (Anti-GAD, IAA) among (T2DM) as T1DM especially in elderly DM patients in a sample of the Iraqi population, and to compare some metabolic and inflammatory markers to those of patients having negative autoantibodies. **Methods**. 65 Patients with T2DM and 21 control participants 60 or older than 60 years were enrolled in this study. Antiglutamic acid decarboxylase GAD, and insulin autoantibody IAA, and C peptide, were evaluated by ELISA. Fasting blood sugar, Random blood sugar, BMI, cholesterol concentration, triglyceride concentration systolic and diastolic blood pressure were assessed, in addition to considering demographic characteristics. **Results**. 3% of patients with T2DM had both anti-GAD and anti-IAA autoantibodies, 10(15%) and 7(10.8%) of the patient were positive for two antibodies respectively, however, none of healthy control group have one of them. The mean duration of initiation insulin up taking by bearing autoantibody group, was (60±38.7 months) and in -ve antibody, group was (48 ±53 months) but these differences were not significant (P=0.56) . there are no significant differences in the median age (P =0.80), BMI (p=0.4218), main cholesterol concentration. (p=0.57) and C peptide (p=0.091) between +ve and -ve autoantibody bearing T2DM. +ve patients had lower body mass index (p=0.014) than those in -ve autoantibodies cases. **Conclusion**. Prevalence of having positive autoantibodies within Iraqi T2DM patients 60 and over 60 years old was 3%. They shared common clinical and metabolic features with subjects with non antiGAD, and IAA autoantibody bearing subjects.*

Keywords: T2DM, IAA, anti-GAD, C peptide, Nasiriyah, Iraq

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Introduction

In contrast to type 1 diabetes mellitus (T1DM) which most commonly diagnosed in children, type 2 diabetes mellitus (T2DM) predominantly affects adults after 40 years of age in overweight people but it is hard to establish any age boundaries between both forms of the disease [1]. Circulating autoantibodies insulin autoantibody IAA, glutamic acid decarboxylase (GAD) can be detected years prior to clinical diagnosis of type 1 diabetes [2]. In elderly patients, the pathogenesis of (T2DM) encompasses autoimmune aspects is recognized increasingly, based on the presence of circulating autoantibodies with slowly progressive manifestation of diabetes with slowly progressive β-cell failure, they are not often overweight described as latent autoimmune diabetes in adults (LADA). Auto-antibodies to GAD and IAA are found in 5–10% of patients classified with type 2 diabetes [3-5]. LADA may have therapeutic consequences, they are generally defined by age of diagnosis >30 years, presence of circulating islet autoantibodies, and lack of insulin requirement for 6 months after diagnosis [6], need insulin earlier during disease
progression are likely to respond poorly to oral antidiabetic mediation, but they could respond favorably to immunomodulator therapy [7]. In 1997 Turner et al., identified the glutamic acid decarboxylase (GAD) and islet cytoplasm autoantibodies (ICA) in 12% of over 3,000 T2DM patients aged between 25 and 65 years [8]. Prospective studies of β-cell function show that LADA patients with multiple islet antibodies develop β-cell failure within 5 years, whereas those with only (GAD) antibodies or only islet cell antibodies (ICAs) mostly develop β-cell failure after 5 years [9]. Beside autoantibody attacking of β-cell, elevated glucose and free fatty acids levels, undergo apoptosis of β cells [10]. Stressed adipocytes secrete pro-inflammatory adipokines and cytokines, which
activate B cells, T cells and macrophages that eventually secrete pro-inflammatory cytokines and chemokines, contributing to the persistence of inflammatory reactions within the tissue and subsequently cause autoimmune-mediated β-cell destruction [11]. The aim of the study is to analyze the prevalence of unrecognized cases with positive diabetes-associated autoantibodies among patients initially diagnosed with T2DM, to describe their metabolic features and characterize the alterations GAD, IAA and C peptide in comparison with healthy control group at the same age because correct diagnosis of LADA patients allows an early and accurate therapeutic intervention.

Material and Methods

This study was achieved by collecting blood samples from (65) patients (45 male and 20 female) and 22 healthy control group, onset at the age of 60 or more, and (22) subjects as Control group (12 male and 11 female) clinically healthy volunteers. patient's serum specimen taken from Diabetes and Endocrine center in Dhi-Qar (Nasiriyah) governorate, while control specimen taken from Imam Al Hassan Health center in Nasiriyah governorate. Inclusion criteria were age equal or more than 60 years old, T2DM diagnosed according to the criteria of the American Diabetes Association. Exclusion criteria were age lower than 60 years, DMT1, concomitant decompensated endocrine disorder, acute or chronic inflammatory disease, malignancies, hepatic diseases. The sample collection was achieved from 15th August 2019 to 15th January 2020. A full medical history was taken, and a physical examination was performed, including anthropometric data (name, age, gender, duration of disease, duration of treatment with insulin, history of

disease (only patients), weight, height, body mass index—BMI and systolic and diastolic blood pressure from patients and control cohorts). body mass index BMI was calculated as the equation:

$$\text{BMI} = \frac{\text{weight}}{(\text{Height})^2}$$

Triglyceride concentrations, cholesterol, Random blood sugar, fasting blood sugar profile, in the enrolled subjects were assessed by standard techniques in the Central Laboratory of Al-Hussain Teaching hospital. All patients and control subjects were assayed for anti-GAD, anti IAA. and C peptide that were measured in 65 cases with T2DM and 21 control participants.

Serum samples processing

Approximately 5 ml of human blood was collected from each subject (patients and control), transferred into sterilized test tubes (Gel tube) and allowed for 30 minutes to clot at room temperature. the sample was centrifuged for 15 minutes at 3000 rpm and the serum was immediately separated, added to Eppendorf tubes (1.5 ml) and stored at (-48^0 C) till used for Anti glutamic acid decarboxylase (Anti GAD65), Insulinautoantibody (IAA), C peptide and another biochemical analysis.

- **Determination of blood glucose**

Blood glucose was determinate by enzymatic colorimetric method using Randox diagnostic kits (UK).

- **Serum total cholesterol (TC)**

Cholesterol determinated by enzymatic colorimetric method using Biolabo diagnostic kits (France).

- **Serum triglycerides (TG):**

Triglycerides determinates by enzymatic colorimetric method using Biolabo diagnostic kits (France).

- **Anti-glutamic acid decarboxylase (Anti GAD65) ELISA kit (Medizym, German)** the analysis achieved according to company instructions

Cut off (negative < 5.0 IU/ml, positive ≥ 5.0 IU/ml)

- **Insulin autoantibody (IAA) ELISA kit (Medizym, German)** the analysis achieved according to company instructions

Cut off (negative < 2.4 U / ml, positive ≥2.4 U / ml)

- **peptide ELISA kit (Novatec, German)** the analysis achieved according to company instructions

Cut off

1. Fasting (ng/ml)

normal (0.78 – 1.9), increase (>1.9), decrease (<0.78), decline (>0.5)

2. Random (ng/ml)

normal (3 – 9), increase (>9), decrease (<3), decline (>1.1)

Statistical Analysis:

All data were presented as a mean and standard deviation SD. Statistically analyzed by GraphPad prism version 8 software. Unpaired non parametric Mann whitney test was used to compare between the mean of different variables . The P value> 0.05 was considered statistically significant.

Results

Prevalence of Autoantibodies. Among 65 patients with T2DM, 2 cases (3%) had positive for both autoantibodies. ten (15%) were positive for anti-GAD only, eight (12.3%) for anti IAA only. None of the control subjects (n = 21) was in positive range of anti-GAD and anti-IAA. The level of anti-GAD and anti-IAA, were varied widely from 0.282 IU/ml to 629.2 IU/ml and from 0.172 IU/ml to 13.471IU/ml in patient and control groups respectively. In contrast of patient, the control group was very stable and the range (from 0.573 to 0.213) for anti-GAD and from (0.763 to 0.113) for Anti-IAA within negative values range (see table 1). The mean duration

of initiation insulin up taking by bearing autoantibody group, was (60 ±38.7 months) and in -ve antibody group was (48 ±53 months) but these differences were not significant (P=0.56). Also we got lower amount of C peptide produced by + ve than - ve autoantibodies bearing patients (see table 2) As shown in the table (3), none of the patients who have a positive of autoantibody (Anti-GAD & IAA) concentration had an increase in the concentration of c-peptide in the results of this study , despite the presence of hyperglycemia(which presence lead to an increase in proinsuline secretion and this lead to an increase in the concentration of c-peptide). This means the presence and increasing the concentration of autoantibody may lead to a decrease in the concentration of c-peptide.

Metabolic Features. Differences between age of patients with T2DM, autoantibody-negative and autoantibody-positive cases of the group were 65± 3.41and 65.7 ±4.32 respectively (P =0.80) while was (p=0.53) when compared difference between patient and control group age (66.7±7.6) (see figure 1A). The gender distribution between groups (autoantibody-negative 69.39% male versus 30.61%, female newly found autoantibody-positive—68.75% male versus 31.25 female and control subjects—52.38% male versus 47.61% for) and the difference was non-significant statistically (P < 0.05). The comparative analysis between patients and control subjects showed non-significance concerning difference in BMI (p=0.4218) (figure 1B), duration of disease (p=0.221) and cholesterol conc. (p=0.5708) (figure 1C).

Analysis systolic and diastolic blood pressure values between patients' group who were found to have positive autoantibodies showed non- significant difference in in comparison with negative autoantibody bearing patients (see figure1D). 75% of both antibodies positive group (11 of 16), and antibody negative (37 of 49) also had diagnosed high blood pressure. 31% (5 of 16) of positive autoantibody were have positive history with DM, while there are 20% (9 of 44), who had DM family history in negative antibody group Cutoff values of body mass index (BMI) for association with type 2 diabetes mellitus was 25.4 kg/m2 in men and 26.1 kg/m2 in women [23]. so, the vast majority of our T2DM patients are obese (93% of +ve and 77.35.45% of -ve autoantibody bearing patients) in consensus with fact the risk of T2DM increases with the BMI.

We got clear significant differences between patients and healthy control cohorts when compared of triglyceride concentration (p=0.0018), total anti-GAD(p<0.0001), IAA(p<0.0001), random blood sugar (p<0.0001) and C peptide (p=0.000) see figure 2 A,B,C,D and figure (3).

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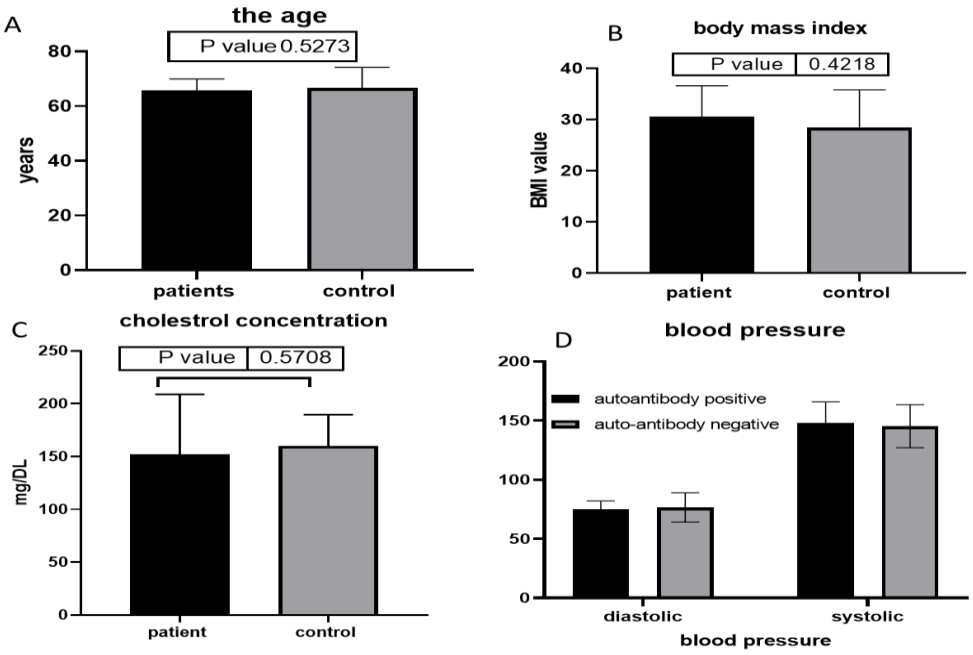

Figure 1: some of non-significant demographic and clinical parameters when compared between T2DM patients (black color column) and healthy (grey color column) control groups: A-age, B-BMI, C- cholesterol concentration and D- the comparison of systolic and diastolic blood pressure between +ve (black color column) and -ve (grey color column) autoantibody bearing patients.

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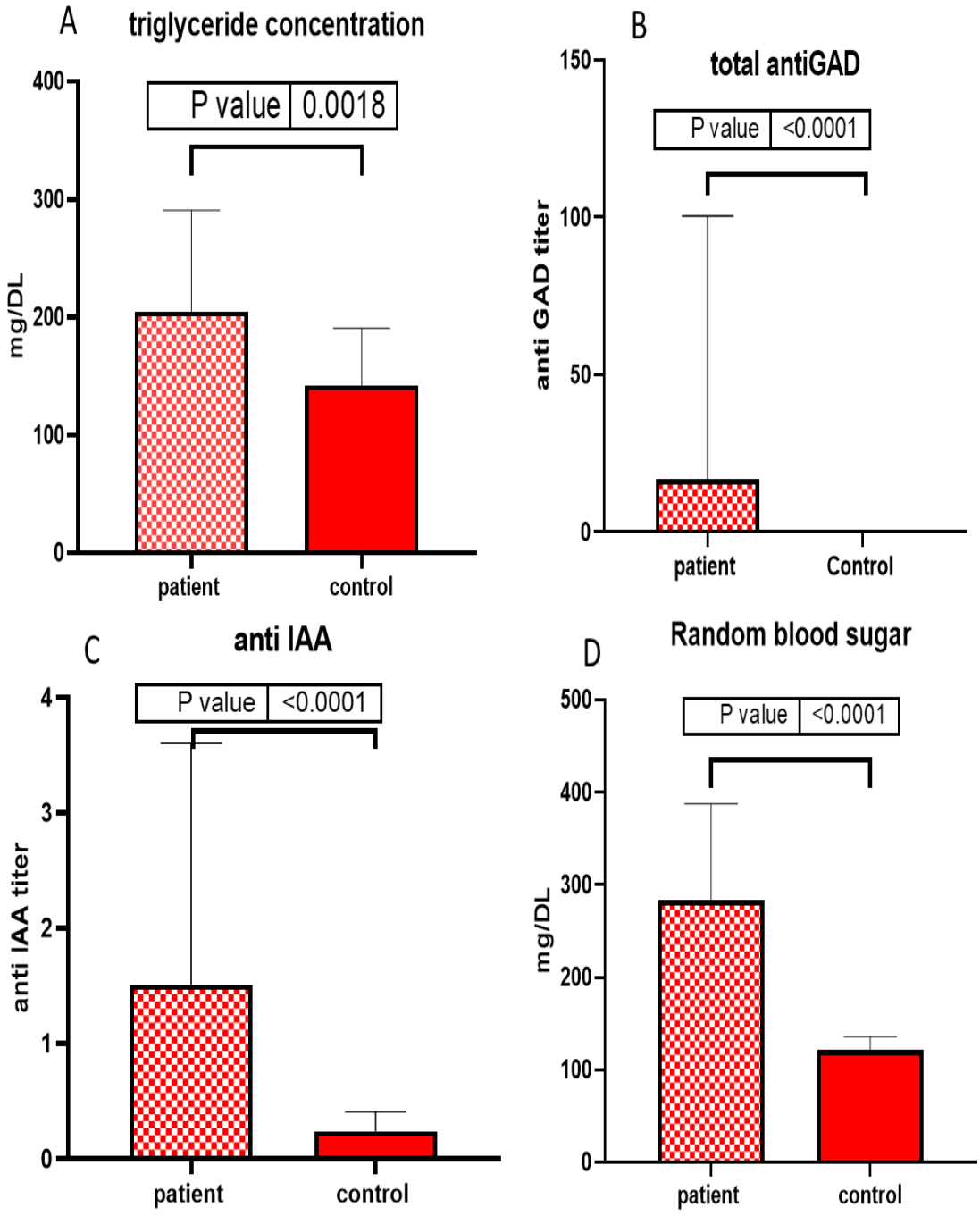

Figure 2: the significant clinical parameters of studied T2DM patients (dotted red color column) in comparison with healthy control (red color column) groups. A-triglyceride conc., B- total anti-GAD, C- cholesterol concentration and D- random blood sugar.

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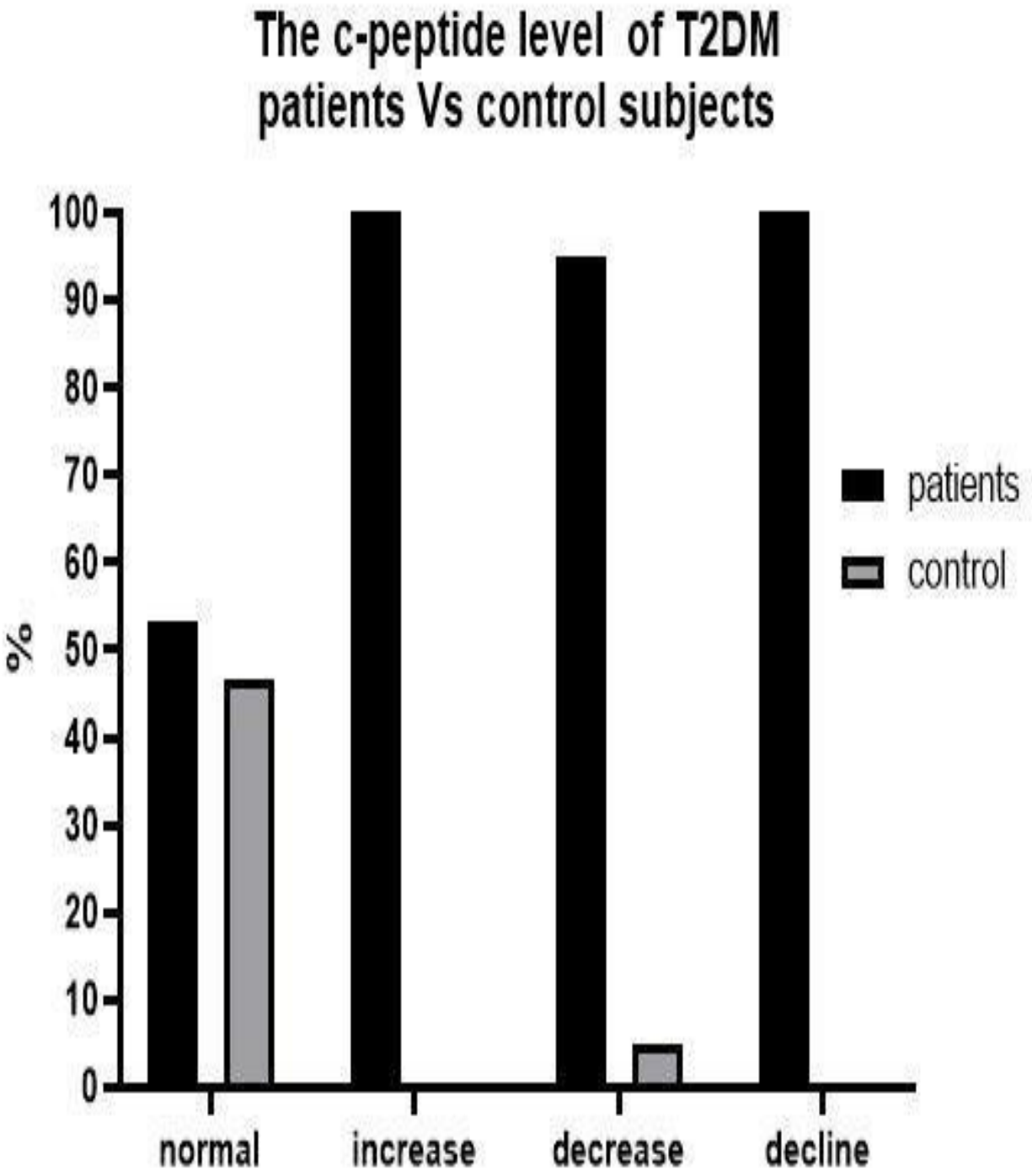

Figure (3) the c-peptide level 0f T2DM patients Vs control subjects.

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Table (1) Anthropometric and clinical data of patients and control cohorts

	Age (years)	Gender	initiation of insulin therapy (Months)	duration of disease (years)	BMI	cholesterol conc.	triglyceride conc.	diastolic	systolic	R.B.S	F.B.S	Anti-GAD	Anti IAA
Positive autoantibody patients	66.06±3.41	11M(68.75%)	66±38.7	15.1±6.4	31.32±4.34	137.19±62.6	223.5±82.7	74.87±7.2	148±17.8	(No.8)276.13±66.6	No.8)182.88±56.23	66.26±162	3.48±3.5
	63-75	5F(31.25%)	12-180	3.0-29	24.22-40	56-255	113-400	63-83	121-181	174-372	85-230	0.355-629	0.172-13.5
Negative autoantibody patients	65.77±4.32	35M (69.38%	57.61±53	13.7±7.3	30.35±6.5	156.86±54.6	199±86.7	76.59±12.4	145.28±18.2	No.35)284.97±112.7	(No.14)228.64±114	0.642±0.44	0.869±0.59
	60-77	15F (30.61%)	1-228	3.0-36	19.83-59.79	60-300	65-450	46-101	108-183	65-512	50-485	0.282-2.262	0.19-2.3
Control group	66.67±7.6	12M(52.4%)	ND	ND	28.4±7.37	160±29.57	142.19±48.3	ND	ND	(N.12)121.47±14.3	(N.10)91.9±9.7	0.39±0.08	0.24±0.16
	60-87	10F(47.6%)		ND	16.14-44.58	112-212	66-240	ND	ND	90-150	77-113	0.123-0.573	0.113-0.763

Table (2) c-peptide level with anti-GAD & IAA in T2DM patients

	Normal	Increase	Decrease	Decline	Control	Sig
N.	24	7	19	15	22	
Anti-GAD +ve\-ve	4\20	0\7	0\19	6\9	0\22	0.005
IAA +ve\-ve	4\20	0\7	1\18	2\13	0\22	0.867

Discussion

A minority of people diagnosed with T2DM also have evidence of autoantibody production [12,13]. Therefore, the number of positive autoantibodies titer has been considered a major risk factor for disease progression in LADA [14]. Agardh CD et al found that the early introduction of insulin in LADA patients with high titer GAD autoantibodies may limit the rate of loss of insulin secretory capacity as determined by C-peptide secretion.[15]. In present study, the prevalence of positive diabetes-associated autoantibodies (anti-GAD and anti-IAA) among T2DM studied patients is 3%, and Anti-GAD was the main single positive autoantibody and followed by IAA. previously documented that individuals with single autoantibody positivity frequently revert to negative, but reversion is rare in people with multiple autoantibodies [16].

Our results of antibody titer were lower than the range cited in studies in European countries [17]. Our study has some limitation such as low number of patients who were double positive for both anti-GAD and anti-IAA. This could be a possible reason why there is not significant differences between two countered groups in several aspects. The amount of C-peptide in the blood can indicate the presence or absence of insulin secretion, so we got here significantly lower mean C peptide production by +ve autoantibodies patient in comparison with -ve counterpart. This finding may interpret reduced insulin production by autoantibody mediated attacking of beta cell in double positive antibodies patients. the opposed correlation between anti-GAD and C. peptide concentration that we got in agreement with Borg H, et al study finding of GAD autoantibodies tend to remain high even when C-peptide secretion becomes undetectable [18]

Obesity is a major risk factor for type 2 diabetes [19,20] with complex genetic and environmental etiology, and its pathological potential lies in obesity-associated insulin resistance, activation of innate immunity and chronic low-grade inflammation [21]. Obesity is found in approximately 55% of patients diagnosed with type 2 diabetes [22]. In our study BMI of +ve auto antibodies was higher than -ve patients (30.75 ±31.3 and 29.74 ±30.3 respectively) but without significant difference. This finding is opposed to the observation of positive diabetes-associated autoantibodies usually have lower body mass index (BMI), better hypertensive control, and better lipid status compared to those of subjects without autoantibodies as found by [23,24]. The obesity-associated chronic adipose tissue inflammation and β-cell stress induced by gluco- and lipotoxicity continuously provide danger signals which cause a local or generalized immune response [25]. Cutoff values of body mass index (BMI) for association with type 2 diabetes mellitus was 25.4 kg/m2 in men and 26.1 kg/m2 in women [26], while we got a higher BMI than that even in control cohort rather than +ve and -ve antibodies cohorts.

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تقييم وجود اضداد Anti-GAD و IAA وقياس مستوى الببتيد سي في مرضى السكري من النوع الثاني ممن هم بعمر 60 سنة وما فوق في مدينة الناصرية \ جنوب العراق

د. حسين علي كريم د. مهدي مرشد ثويني
د. طالب حسن علي

الخلفية:

هدفت الدراسة إلى تقدير انتشار الأجسام المضادة الذاتية Anti-GAD، IAA في مرضى السكري من النوع الثاني المسنين في عينة من العراقيين ومقارنة بعض العلامات الأيضية والالتهابية بتلك الخاصة بالأشخاص غير المصابين.

طرق العمل: تم تسجيل 65 مريضًا يعانون من السكري من النوع الثاني و 21 مشاركًا في المجموعة السيطرة ممن هم بعمر 60 أو أكبر في هذه الدراسة. تم تقييم حامض أنتيجلوتاميك ديكاربوكسيلاز GAD ، والأجسام المضادة للأنسولين IAA ، الببتيد سي بواسطة تقنية الاليزا ELISAتم تقدير سكر الدم الصائم ، سكر الدم العشوائي ، مؤشر كتلة الجسم ، تركيز الكوليسترول ، تركيز الدهون الثلاثية ، ضغط الدم الانبساطي والانقباضي ، بالإضافة إلى جمع الخصائص الديموغرافية لكلا المجموعتين .

النتائج: 3 ٪ من المرضى الذين يعانون داء السكري من النوع الثاني لديهم أجسام مضادة لـ GADوالمضادة لـIAA ، بينما كان 10 (15 ٪) و 7 (10.8 ٪) من المرضى كانوا إيجابيين لاي من الأجسام المضادة السابقة على التوالي ، ولكن لم يكن أي منهم لدى مجموعة السيطرة . كان متوسط مدة بدء تناول الأنسولين عن طريق مجموعة الحاملين للأضداد الذاتية (60 ± 38.7 شهرًا) بينما كانت المدة لمجموعة غير الحاملين للأجسام المضادة من مرضى السكري هي (48 ± 53 شهرًا) ولكن هذه الاختلافات لم تكن معنوية .(P = 0.56) لا توجد فروق ذات دلالة إحصائية في متوسط العمر (P = 0.80) ، مؤشر كتلة الجسم (P =0.4218) ، تركيز الكوليسترول الرئيسي. (P =0.57) و الببتيد سي (P =0.091) بين الحاملين وغير الحامليناللاجسام المضادة الذاتية من مرضى النوع الثاني من السكري . كان لدى خمسة مرضى مؤشر كتلة جسم أقل(p = 0.014) ، من تلك الموجودة في حالات الأجسام المضادة الذاتية.

الاستنتاج. ان معدل انتشار الأجسام المضادة الذاتية بين مرضى النوع الثاني من السكري بين العراقيين ممن هم بسن60 وما فوق هو 3 ٪. لقد كانت هنالك الكثير من الميزات السريرية والايضية المشتركة بين الأشخاص ممن يحملون اولا يحملون الأجسام المضادة الذاتية لـ GAD و.IAA