



International Journal of PharmTech Research

CODEN (USA): IJPRIF, ISSN: 0974-4304, ISSN(Online): 2455-9563 Vol.9, No.12, pp 476-481, 2016

Estimation of Oxidative stress states in DM type1 in Iraqi patients

Mohammed Ramadhan Abd Ali

University of Babylon, college of Nursing, Iraq

Abstract : The present study was carried out to investigate redox modulation in diabetes mellitus type 1 in Iraqi patients, social demographic, total antioxidant and reactive oxygen species were using to estimate oxidative stress states in DM patient, the results show that there was high prevalence at over 50 years, the total antioxidant level was significant elevation in patients than control while ROS level was no-significant variation between study groups. **Keywords:** Oxidative stress, DM type1, Iraqi patients.

Introduction

The review of literature clarified the role of oxidative stress (OS) states in many disease, the unbalance between reactive oxygen species (ROS), reactive nitrogen species(RNS) and antioxidant molecules causes incidence, progressive disease and complication, in DM it is believed that the OS has been important factors contributed of cell disorders in DM, beginning by hyperglycemia¹⁻³in spite of some authors improved that indirect link between hyperglycemia and OS⁴. The pathogenesis of diabetic complications is associated with increment of free radicals levels⁵ and decreased antioxidants activity of molecules⁶.

Type 1 diabetes (T1D) is an autoimmune disorder involving immune-mediated recognition of islet β cells by auto reactive T cells, which leads to the elevated of ROS and pro-inflammatory cytokines, resulting in the destruction of pancreatic β -cells and loss of insulin secretion. the cause of T1D have been unknown despite of a large investigations deal with T1D, studies clarified that there are many factors have role in this disease like genetic susceptibility, environmental factors, and dietary deficiencies are known to contribute to disease, however, the impact of oxidative stress in a genetically susceptible individual is of particular interest. Oxidative stress becomes harmful when the production of free radicals overcomes antioxidants activities. oxidative stress has been linked to β -cell cytotoxicity⁷⁻⁹ and has been suggested to play a role in T1D pathology¹⁰⁻¹³.

Materials and methods

Study subjects

The present study is case –control study included 30 patients and 30 control, samples were collected from diabetic center in al-sadder hospital. Data were collected from patients and control in questioner then 5 ml of Venus blood were collected in gel tube to separation serum, it centrifugation at 4000 rpm\min for 10 min, serum were stored in -20 c to detected total antioxidants and ROS levels. Total antioxidant and ROS were estimation in serum using ELIZA kits (elabscience),the data were analyzed using spss version 21.

Results

The results of present study show there is significant variation between patients and control in ROS and TAO levels in diabetic patients type 1. First of the all the demographic study showthat the weight mean of study population was 78.76 for patients and 75.00 for control, and significant differences in BMI and gender, male were more frequent than female in patients while it less in control.

Mean of age was convergent in patients and control. according to family history; more than half population have family history of this disease and large percentage was no smoker habitats and passive smoking, other disease related with patients ware hypertension, it was 50% of population study. BMI was classification to 4 categories, the high percentage was in obesity, and overweight respectively in patient while in control over weight was the higher. Age was classification in to four categories, (51-60) was more frequent than others in patient and control (table-1).

Categories	Patients	Control	Statics	p-value	
Weight kg	78.76±11.95	75.00±8.514	1.3544	0.1814	
BMI	31.09±6.218	26.307±3.307	3.6245	0.0006*	
Gender					
Male	69.23	48.27	2.474*	0.1157	
Female	30.769	51.72			
Age	49.03±12.43	50.52±6.500	0.5654	0.5742	
Duration	11.63±6.88				
Family history					
no	57.67	0			
yes	42.30	0			
Smoking					
no	84.615	68.96	0.187*	0.6656	
yes	15.38	17.24			
Passive smoking					
No	88.46	100	3.065*	0.0800	
yes	11.54	0			
Hypertension					
No	50	0			
Yes	50	0			
BMI categories					
Under weight	3.8%	0			
Normal	7.69%	16%			
Over weight	38.46%	72%			
Obesity	50%	12%			
Age categories					
<30	7.69	0			
31-40	15.38	0			
51-60	11.53	40			
>50	65.38	60			

Table (1) distribution	studysubjects a	ccording to Demog	raphic

The mean differences of TAO show significant differences (p value 0.0075,t = 2.7836) figure (1), the difference between ROS in patients and control was non-significant (p value 0.9438, t=0.0709) (figure 2).

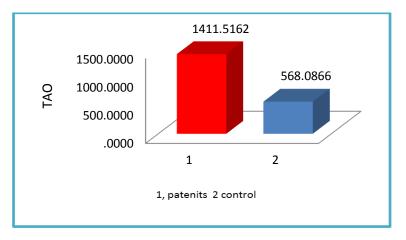
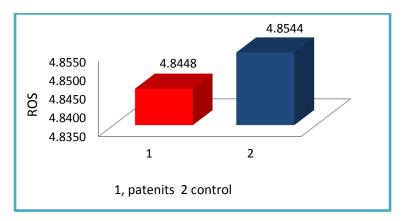


Figure (1) Mean differences of TOA level of patients and control.



Figure(2) Mean differences of ROS level of patients and control

Table (2) show mean differences of ROS according to demographic classification, all results indicated that there were no significant differences between patient and control except in gender, male in patients hadsignificant decrease in level of ROS, also ROS level was increased with body mass index in non-significant increment, significant differences was shown in age categories it recoded high level in (31-40).

Categories	Patients	Control	Statics	p-value		
Gender						
Male	4.831±0.139	5.027±0.062	4.8972	0.0001*		
Female	4.87±0.073	4.716±0.899	0.4784	0.6373		
Duration						
1-10	4.8517±0.168	0	0.0461	0.9636		
11-20	4.84±0.09	0				
Family history	Family history					
no	4.83±0.148	0	0.4028	0.6907		
yes	4.85±0.083	0				
Smoking						
no	4.855±0.08	4.788±0.805	0.3888	0.6995		
yes	4.78±0.27	5.051±0.03	2.2670	0.0577		
Passive smoking						
No	4.84±0.12	4.83±0.736	0.1418**	0.8884		
yes	4.85±0.002	0				
Hypertension						
No	4.84 ± 0.088	0	0.0000**	1.0000		

Yes	4.84±0.153	0			
BMI categories					
Under weight	4.56±0.23	0	1.130**	0.347	
Normal	4.78±0	4.62±1.099			
Over weight	4.84±0.099	4.99±0.09			
Obesity	4.89±0.057	5.132±0.03			
Age categories					
<30	4.59±0.27	0	4.76**	0.010*	
31-40	4.90±0.36	0			
51-50	4.88±0.056	4.65±0.99			
>50	4.85±0.94	5.011±0.142			

The mean differences and statically analysis of TOA level in patients and control according to demographic was clarified in table (3), there were significant increment in TOA in male and female of patients and its level increment also with duration of disease and in the patienthadn't family history, smoking behavior show high level of TAO, and passive smoker have major role in TAO elevation in patients, the patients suffer from hypertension recorded high level of TAO, and it had positive association with BMI. According to age categories there is no association between age and TAO level.

Table (3) total antioxidant	level in study groups	s according to dem	ographic study

Categories	Patients	Control	Statics	p-value	
Gender					
Male	1535.78±1467.85	722.2±132.7	2.0598	0.0482*	
Female	1131.92±713.97	444.79±270.7	3.3556	0.0030*	
Duration					
1-10	1366.69±796.28	0	0.1518**	0.8806	
11-20	1439.52±1535.37		-		
Family history					
no	1590.83±1361.71	0	0.8275**	0.4161	
yes	1166.98±1183.20	0			
Smoking					
no	1440.88±1335.58	425.83±254.14	3.3407	0.0018*	
yes	1249.99±1082.74	270.12±175.17	2.0257	0.0824	
Passive smoking	j				
No	1293.49±1142.89	398.75±246.31	1.3187**	0.1997	
yes	2316.37±2189.35	0			
Hypertension					
No	1396.76±1242.85	0	0.0575	0.9546	
Yes	1426.27±1371.06	0			
BMI categories					
Under weight	2147.34±170.6	0	F=1.486**	0.231	
Normal	2084.45±0	365.26±214.97			
Over weight	1489.78±1667.56	374.72±189.48			
Obesity	1186.29±1071.85	377.68±239.93			
Age categories					
<30	217.56±129.4	0	0.479**	0.70	
31-40	1167.64±865.97	0			
51-50	4826.4±478.8	641.75±1326.23			
>50	1482.1±1496.9	429.154±256.41			

Discussion

The present study investigate to estimate oxidative stress state in DM type1 in Iraqi patients, oxidative stress has been the most important factors which studied in different scientific researches because of its role in progression and complication of disease. DM prevalence in Iraq recorded elevation in the last years, in Basra Mansour *et al.*, $(2015)^{14}$. Show that the prevalence of diabetes is very high, affecting one in five adults. The epidemic of diabetes will result in strain on the financial resources of health care systems.

The elevation of total antioxidant level in patients resulted from increment of free radicals which are formed disproportionately in diabetes by glucose oxidation, non-enzymatic glycation of proteins, and the subsequent oxidative degradation of glycated proteins. Thislead to damage of cellular organelles and enzymes activity, increased lipid peroxidation, and development of insulin resistance¹⁷.

In diabetes mellitus, main sources of oxidative stress are mitochondria. During oxidative metabolism in mitochondria, a component of the utilized oxygen is reduced

to water, and the remaining oxygen is transformed to oxygen free radical (O⁻) which is an important ROS that is converted to other RS such as ONOO⁻, OH and H2O2¹⁶.Insulin signaling is modulated by ROS/RNS by two ways. On one side, in response to insulin, the ROS/RNS are produced to exert its full physiological function and on the other side, the ROS and RNS have got negative regulation on insulin signaling, interpreting them to develop insulin resistance which is a risk factor for diabetes type 2¹⁷.

The life style of third world contributed in oxidative stress balance in DM patients, like nutrition, sleeping inadequate, food supplements, sport playing and smoking, all these factors can be effect in free radicals producing and scavenger by antioxidant enzymes and molecules^{18,19}.

Some genetic polymorphism contributed in the oxidative stress state like catalase, super oxide dismutase, and TNF- alfa some of these gene were association with DM and other no association with this diseases²⁰ The present study need more investigation like study antioxidant gene polymorphisms to evaluation oxidative states

References:

- 1. Ceriello A. Hyperglycaemia and the vessel wall: the pathophysiological aspects on the atherosclerotic burden in patients with diabetes. Eur J Cardiovasc PrevRehabil 2010; 17:S15-S19.
- 2. 4.Gaede P, Poulsen HE, Parving HH, Pedersen O. Double-blind, randomised study of the effect of combined treatment with vitamin C and E on albuminuria in Type 2 diabetic patients. Diabet Med 2001; 18:756-760.
- 5.Wang X, Li YL, Wu H, Liu JZ, Hu JX, Liao N, Peng J, Cao PP, Liang X, Hai CX. Antidiabetic effect of oleanolic acid: a promising use of a traditional pharmacological agent. Phytother Res 2011; 25:1031-1040.
- 4. 6.Choi SW, Benzie IF, Ma SW, Strain JJ, Hannigan BM. Acute hyperglycemia and oxidative stress: direct cause and effect? Free RadicBiol Med 2008; 44:1217-1231.
- 5. 7.Porte D, Jr. Clinical importance of insulin secretion and its interaction with insulin resistance in the treatment of type 2 diabetes mellitus and its complications. Diabetes Metab Res Rev 2001; 17:181-188.
- 6. 8.Kashiwagi A, Asahina T, Nishio Y, Ikebuchi M, Tanaka Y, Kikkawa R, Shigeta Y. Glycation, oxidative stress, and scavenger activity: glucose metabolism and radical scavenger dysfunction in endothelial cells. Diabetes 1996; 45:S84-S86.
- 7. R. Bottino, A. N. Balamurugan, H. Tse et al.(2004), "Response of human islets to isolation stress and the effect of antioxidant treatment," Diabetes, 53(10): 2559–2568.
- A. Rabinovitch, (1992) "Free radicals as mediators of pancreatic islet β-cell injury in autoimmune diabetes," Journal of Laboratory and Clinical Medicine, 119(5): 455–456
- 9. P. O. T. Tran, S. M. Parker, E. LeRoy et al., (2004) "Adenoviral overexpression of the glutamylcysteine ligase catalytic subunit protects pancreatic islets against oxidative stress," Journal of Biological Chemistry, (279) 52: 53988–53993.
- 10. I. C. West, (2000) "Radicals and oxidative stress in diabetes," Diabetic Medicine, 17(3): 171–180.

- 11. E. Horio, M. Fukuda, H. Katoh et al., "Reactive oxygen intermediates in autoimmune islet cell destruction of the NOD mouse induced by peritoneal exudate cells (rich in macrophages) but not T cells," Diabetologia, vol. 37, no. 1, pp. 22–31, 1994.
- 12. J. Nerup, T. Mandrup-Poulsen, J. Molvig, S. Helqvist, L. Wogensen, and J. Egeberg, (1988) "Mechanisms of pancreatic β -cell destruction in type I diabetes," Diabetes Care, 11(1)16–23.
- 13. W. L. Suarez-Pinzon, C. Szabó, and A. Rabinovitch, (1997) "Development of autoimmune diabetes in NOD mice is associated with the formation of peroxynitrite in pancreatic islet β -cells," Diabetes, 46(5): 907–911.
- 14. Mansour AA, Al-Maliky AA, Kasem B, Jabar A, Mosbeh KA. Prevalence of diagnosed and undiagnosed diabetes mellitus in adults aged 19 years and older in Basrah, Iraq. *Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy.* 2014;7:139-144. doi:10.2147/DMSO.S59652.
- 15. Maritime A.C., Sanders R.A. Watkin J.B. (2003) Diabetes, oxidative stress, and antioxidants: A reviewJournal of Biochemical and Molecular Toxicology, 17(1): 24–38
- 16. Moussa, S.A., 2008. Oxidative stress in diabetes mellitus. Romanian J.Biophys. 18 (3), 225–236.
- 17. Erejuwa, O.O., 2012. Oxidative stress in diabetes mellitus: is there arole for hypoglycemic drugs and/or antioxidants. Oxid. Stress Dis., 2(3): 217–246.
- Al-Terehil, M. al-kilabi2, L.H., AL–Mamooril, A., Al-Jboori, M.J., Al-Saadil, A H. Zaidan H.K. (2016) Some Heavy Metals Concentrations in Tumor Tissue, International Journal of ChemTech Research CODEN (USA): IJCRGG, 9(3):407-411.
- 19. Al-Terehil M., Haider K. Zaidan, Ayad M.J. AL –Mamoori; Ali Hmood Al-Saadi, Israa Harjan(2015) Effective of different factors on trace elements concentrations in Iraqi lactating mother's milk, International Journal of Pharm Tech Research,8(10): 151-157.
- Al-Terehi M., RanaGhaleb, Shaimaa A. Al-Oubaidy, Ali H. Al-Saadi, Haider K. Zaidan, (2015) Study TNF-α gene polymorphism in Type 1 Diabetic Patients Using Amplification Refectory Mutation System (ARMS) technique, JCPS Volume 9 Issue 3. 1107-1111.
