



INTERNAL CAROTID PEAK-SYSTOLIC AND END-DIASTOLIC VELOCITIES ARE THE SENSITIVE PARAMETERS IN PREDICTING PATIENTS CONDITIONS IN TYPE 2 DIABETES

AL-EQABI D.A.M.^{1*}, AL-SIAIDY W.F.¹, AL-SABBAGH A.A.J.² AND AL-KIRWI I.N.²

¹Department of Physics, College of Medicine, University of Baghdad, Baghdad, Iraq.

²National Diabetes Center, Al-Mustansiriah University, Baghdad, Iraq.

*Corresponding Author: Email- dalya1900@yahoo.com

Received: October 21, 2013; Accepted: November 11, 2013

Abstract- This study carried out to examine the possible relations between the common carotid intima media thickness (CCIMT), peak- systolic velocity (PSV) and end-diastolic velocity (EDV) of the internal carotid artery in type 2 diabetic mellitus (T2DM) patients, with exemption of any cardiovascular diseases.

B-mode ultrasound is used on 100 patients suffering from type 2 diabetics, and 40 normal volunteers. Patients group was divided into four groups: 1- diabetic patients, 2- diabetic with hypertension, 3- diabetic with dyslipidemia, and 4- diabetic with hypertension and dyslipidemia.

Generally, the mean of CCIMT on both sides in patients groups is higher than control. The left PSV result in the T2DM patients with hypertension, Group-2 and Group-4 show the highest value (39.9 - 38.4 cm/s) than those with dyslipidemia. The EDV results of left side demonstrate a higher data (13.9 - 134 cm/s) in patient groups suffering from hypertension, and the lowest (10.8 - 10.5 cm/s) on the right side for patient groups suffering from dyslipidemia.

PSV and EDV can be considered as the most sensitive parameters to predict patients conditions in type 2 diabetes.

Keywords- Peak-systolic velocity, end-diastolic velocity, intima- media thickness, type 2 diabetic, resistive index, pulsatility index

Citation: Al-Eqabi D.A.M., et al. (2013) Internal Carotid Peak-systolic and End-diastolic Velocities are the Sensitive Parameters in Predicting Patients Conditions in Type 2 Diabetes. Journal of Biomedical and Bioengineering, ISSN: 0976-8084 & E-ISSN: 0976-8092, Volume 4, Issue 1, pp.-90-92.

Copyright: Copyright©2013 Al-Eqabi D.A.M., et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

Introduction

Patients with type 2 diabetes mellitus (T2DM) are at high risk for developing atherosclerosis, and cardiovascular disease [1]. Atherosclerosis is associated with changes in vascular structure, and function; by accelerating the cortex progression, and impairing the endothelial function [2]. These changes increasing the thickness, and the stiffness of arterial walls.

The intima - media thickness (IMT) of common carotid artery (CCA) measured by ultrasonography is widely used as a useful marker confirms early stage of atherosclerosis in large arteries [3]. While the pulsatility index (PI), and the resistive index (RI) which firstly introduced by Gosling, et al [4] and Porcelot, et al [5] can reflect the severity of arterial stiffness.

The resistive and pulsatility indices are calculated from the ultrasound Doppler spectrum according to flowing relations.

$$\text{Resistive Index (RI)} = \frac{\text{PSV} - \text{EDV}}{\text{PSV}}$$

$$\text{Pulsatility Index (PI)} = \frac{\text{PSV} - \text{EDV}}{mV}$$

where PSV = Peak systolic velocity; EDV = End diastolic velocity; mV = Mean velocity

Manabel, et al [6] reported that carotid hemodynamic such as PI, RI can be used as a useful indicator of atherosclerosis in hypertensive Patients. While Staub, et al [7] showed that RI of the internal carotid artery (ICA) had a higher risk of cardiovascular events. A good correlation was reported between RI of ICA and IMT of CCA in patients with known vascular disease [8].

Bai, et al [9] revealed that RI, PSV, and EDV of CCA were associated with ischemic stroke (IS), but the EDV is the potential indicator of internal resistance. Recently Chuage, et al [10] concluded that the common carotid IMT, and EDV jointly and independently predicted future IS.

Subjects and Methods

A hundred patients with type 2 diabetes, without any evidence of cardiovascular disease, were involved in this work.

The study performed at the National Diabetes center of Al- Mustansiriah University (Baghdad-Iraq).

Diabetic patients with duration of 1-20 years on diet only or on drug therapy were included in the study.

The control group includes 40 normal volunteers approximately matched for age, and sex with patients group.

All subjects provided a full medical history, received physical exami-

nation, and routine laboratory tests. The history status for hypertension (BP), and dyslipidemia before the day of the measurements, was reviewed for all patients.

Patients with dyslipidemia taking antilipid drug for more than 6 months were excluded.

Body mass index (BMI) and waist circumference (WC) measured on the day of examination. After the subject had rest for at least 5 min. in supine position, the ECG, and blood pressure were monitored. The blood pressure measured manually, and checked again by Doppler ultrasound. Hypertension was diagnosed when systolic (SBP), and diastolic- blood pressure (DBP) \geq 140, and 90 mm Hg respectively, based on the average of 2 readings, plus on pressure reported by the patient with a known history of hypertension. Diabetic patients with hypertension had already controlled by medications.

The laboratory investigations were done including: Fasting plasma glucose (FPG), lipid profile (Ch, TG, LDL, VLDL, HDL), creatinine (Creat.) and blood urea (B.urea).

From the medical history, the physical examination, and the laboratory tests results, the patients group was divided into four sub-groups according to the existence of dyslipidemia, and on the history of hypertension:

Group-1: 23% diabetic patients.

Group-2: 40% diabetic patients with dyslipidemia (2DM +Ch).

Group-3: 20% diabetic patients with hypertension (2DM+BP).

Group-4: 17% diabetic patients suffering from hypertension and dyslipidemia simultaneously (2DM+BP+Ch).

Ultrasound machine, (FUKUDA DENSHI, Japan) with (6-9) MHz linear probe (Fut-LD386-9A), was used to scan and measure both left and right IMT, RI, and PI.

Ultrasound beam was adjusted to detect the common carotid artery (CCA), and then the IMT of the CCA was measured.

The stiffness was examined by the blood flow in the internal carotid artery (ICA) depending on the resistive and pulsatility indices.

Doppler evaluation of RI and PI for the ICA were performed with guidance of color blood flow mapping. Once the imaging positioned and the gain were selected the peak systolic, and the end-diastolic velocities were recorded.

The IMT, RI, and PI measurements were done by the same radiologist, ultrasonographic unit, and at one center.

The statistical analysis was done to evaluate the mean and standard deviation for each parameter in each group.

Results

The mean and the standard deviation (\pm SD) for all the clinical, blood sample results, and Sonographic data are presented in [Table -1].

All patient groups are matched in height, and weight. But because it is difficult to get aged volunteers (normal) without any chronic disease, so the mean age of the control group is lower than that of the patient groups.

The average blood urea (B.urea) and creatinine for all groups are within the normal range, but the mean value of the creatinine in normal subjects (control group) is lower than the patient groups.

The mean of the common carotid artery IMT on both right, and left sides in patient groups are higher than the control (0.9-0.82 mm VS 0.71 mm). While the T2DM patients with hypertension (Group-2, and Group-4) show that, the left (L) carotid IMT is thicker than the right (R) side.

Table 1- The mean and standard deviation of the clinical and ultrasonographic parameters for the patients groups and control

Examined parameters	Patient Groups				Control group
	Group-1 T2DM -23%	Group-2 T2DM+BP -20%	Group-3 T2DM+Ch-40%	Group-4 T2DM+BP+Ch-17%	Normal
Age (years)	54.15 \pm 7.87	56.5 \pm 5.7	56.9 \pm 7.31	56.88 \pm 7.13	48 \pm 7.9
2DM Duration (years)	7.02 \pm 6.12	6.85 \pm 4.43	4.47 \pm 3.47	5.82 \pm 5.8	
Height(m)	161.28 \pm 9.82	159.15 \pm 9.42	160.73 \pm 9.01	159.64 \pm 6.07	162.18 \pm 6.8
Weight(kg)	77.71 \pm 14.74	79.07 \pm 11.78	79.65 \pm 14.39	78 \pm 12.79	75.22 \pm 14
BMI(kg/m2)	29.88 \pm 5.89	31.2 \pm 5.35	30.734 \pm 5.078	29.69 \pm 4.333	28.53 \pm 4.11
WC (cm)	102.52 \pm 14.32	104.55 \pm 8.49	106.43 \pm 22.01	105.17 \pm 11.24	93.79 \pm 9.38
BPS(mm Hg)	138.26 \pm 28.361	149.5 \pm 17.61	146.52 \pm 16.12	160.58 \pm 23.57	116.7 \pm 17.74
BPD(mm Hg)	79.21 \pm 9.69	82. \pm 12.39	80.434 \pm 10.215	89.411 \pm 12.48	81.477 \pm 9.05
B urea	31.39 \pm 7.20	31.3 \pm 5.93	29.69 \pm 5.91	30.47 \pm 6.43	29.56 \pm 8.01
FPG(mg/dL)	197.94 \pm 64.37	159.3 \pm 38.99	187.26 \pm 58.45	174.70 \pm 49.83	98.88 \pm 8.93
Cholesterol(mg/dL)	144.26 \pm 26.27	141.2 \pm 30.92	254.69 \pm 43.43	255.47 \pm 48.36	153.9 \pm 32.36
Triglyceride(mg/dL)	118.23 \pm 43.89	125.55 \pm 71.73	217.26 \pm 121.69	194.58 \pm 105.19	100.3 \pm 20.69
Low-density lipoprotein (mg/dL)	73.31 \pm 28.22	69.7 \pm 30.47	174.60 \pm 31.72	178.47 \pm 44.38	88.93 \pm 28.58
Very low-density lipoprotein (mg/dL)	25.71 \pm 13.83	26.25 \pm 14.09	40.43 \pm 26.37	35.35 \pm 16.94	20.25 \pm 4.21
Creatinine(mg/dL)	0.72 \pm 0.17	0.72 \pm 0.18	0.68 \pm 0.17	0.71 \pm 0.19	0.52 \pm 0.23
R intima media thickness (mm)	0.847 \pm 0.12	0.84 \pm 0.09	0.83 \pm 0.13	0.829 \pm 0.09	0.71 \pm 0.10
L intima media thickness (mm)	0.831 \pm 0.11	0.905 \pm 0.17	0.821 \pm 0.12	0.852 \pm 0.10	0.71 \pm 0.09
R Peak Systolic Velocity, R PSV(cm/s)	34.64 \pm 12.19	35.93 \pm 9.77	32.90 \pm 9.42	32.14 \pm 4.82	34.6 \pm 8.09
L Peak Systolic Velocity, L PSV(cm/s)	34.28 \pm 8.99	39.95 \pm 11.8	36.64 \pm 12.76	38.41 \pm 8.19	34.42 \pm 6.85
R End diastolic velocity, R ESV(m/s)	12.41 \pm 5.12	13.12 \pm 4.56	10.87 \pm 3.94	10.58 \pm 2.49	14.9 \pm 5.1
L End diastolic velocity, L ESV(m/s)	12.57 \pm 3.75	13.47 \pm 4.43	12.99 \pm 5.46	13.96 \pm 3.58	15.2 \pm 4.30
R Resistive index (RI)	0.67 \pm 0.14	0.63 \pm 0.06	0.69 \pm 0.26	0.67 \pm 0.06	0.58 \pm 0.06
L Resistive index (RI)	0.66 \pm 0.14	0.66 \pm 0.07	0.71 \pm 0.33	0.63 \pm 0.07	0.57 \pm 0.04
R Pulsatility index (PI)	1.17 \pm 0.31	1.61 \pm 2.17	1.54 \pm 1.85	1.27 \pm 0.29	0.98 \pm 0.22
L Pulsatility index (PI)	1.08 \pm 0.26	1.24 \pm 0.30	1.18 \pm 0.45	1.1 \pm 0.22	0.92 \pm 0.15

Generally the patient groups demonstrate a rise in the hemodynamic parameters RI, and PI values on both sides relative to the control. The left PSV data in patient Group-2, Group-3 and Group-4 reveal higher results relative to the control, [Table-1]. But the T2DM patients with hypertension, Group-2 and Group-4 show the highest value (39.9 - 38.4 cm/s) than those with dyslipidemia, Group-3 (36.6 cm/s). In addition the EDV of both sides in patient groups demonstrate lower values than control. But the EDV results of left side still show a higher data (13.4, and 13.9 cm/s) in patient groups (Group-2 and Group-3) suffering from hypertension, and the lowest (10.8, and 10.5 cm/s) on the right side for patient groups suffering from dyslipidemia (Group-3 and Group-4).

Discussion

Several studies have shown that the IMT of the CCA is a useful indicator of atherosclerosis in large arteries [3], while RI, and PI are regarded as a valid indicator of arterial stiffness [4, 5], and angiopathy [11].

The mean of all results, presented in [Table-1] showed a high IMT of CCA in diabetic patient groups compared to the normal (control group). These results are in agreement with many workers [12-14]. But a thicker IMT was recorded on the left side of the CCA among the T2DM patients suffering from hypertension (Group-2 and Group-4), which are in agreement with Vicenzini, et al [15] result. These finding may be due to the higher hemodynamic stress [16], and then the frictional force excited by blood flow which may cause an injury or damaging to the endothelial surface vessel wall. Any endothelial injury or inflammations are the key in developing atherosclerosis [17]. Furthermore patients with hypercholesterolemia accelerate atherosclerosis in CCA more than normal subjects [18].

The diabetic patient groups demonstrated a rise in the means of hemodynamic parameters (RI and PI of the ICA) than the control for both sides. While the mean of the PI for the right side in patient groups recorded higher values than the left side, [Table-1]. But the differences between the right and left sides not behave equally, because of the variations in the means of the peak-systolic velocity (PSV), and the end-diastolic velocity (EDV).

The elevated PSV on the left side (L PSV) of the ICA for T2DM patients with hypertension (Group-2 and Group-4) may come from the rise in the IMT and the stiffness in arteries. The thicker the arterial wall, the smaller the arterial diameter (cross sectional area), the higher the blood velocity in stiff tube. This high figure may reflect the effect of the elevated blood pressure (high hemodynamic stress).

The mean of the PSV data on the right side indicated lower values for the T2DM patients suffering simultaneously from dyslipidemia (Group-3 and Group-4), [Table-1]. This low figure reflects the effect of the blood viscosity on the flow. Carollo, et al [19] reported that blood viscosity is strongly influenced by the lipid profile. This phenomenon appears clearly on the EDV at the right for these groups.

Generally, the EDV results showed lower values in patient groups than control. The greater the differences between the PSV and EDV increase the RI and the PI values, according to their formula. But the R EDV data were higher in T2DM patients with hypertension who have thicker IMT of the CCA, but not those with dyslipidemia.

Conclusion

The PSV and EDV did not reflect arterial stiffness only but also atherosclerosis process, hemodynamic stress, and blood viscosity.

So we think that the EDV and PSV are more sensitive parameters to predict patient conditions.

Conflicts of Interest: None declared.

References

- [1] Ahmed K.A., Muniandy S. and Ismail I.K. (2010) *Biomedical Research*, 21(2), 147-155.
- [2] Van popele N.M., Grobbee D.E., Bots M.L., Asmar R., Topouchian J. and Reneman R.S. (2001) *Stroke*, 32, 454-460.
- [3] Grobbee D.E. and Bots M.L. (1994) *J. Intern Med.*, 236, 567-573.
- [4] Gosling R.G., Dunbar G., King D.H., Newman D.L., Side C.D., Woodcock J.P., Fitzgerald D.E., Keates J.S. and MacMillan D. (1971) *Angiology*, 22, 52-55.
- [5] Pourcelot L. (1975) *La Revue du Praticien*, 25(59), 4671-4680.
- [6] Manabe S., Okura T., Watanabe S. and Higaki J. (2005) *Journal of Human Hypertension*, 19, 787-791.
- [7] Staub D., Meyerhans A., Bundi B., Schmid H.P. and Frauchiger B. (2006) *Stroke*, 37, 800-850.
- [8] Frauchiger B., Schmid H.D., Roedel C., Moosmann P. and Staub D. (2001) *Stroke*, 32, 836-841.
- [9] Bai C.H., Chen J.R., Chiu H.C. and Pan W.H. (2007) *J. Clin. Ultrasound*, 35, 322-330.
- [10] Chuang S.Y., Bai C.H., Chen J.R., Yeh W.T., Chen H.J., Chiu H.C., Shiu R.S. and Pan W.H. (2011) *Stroke*, 42, 1338-1344.
- [11] Fukuhara T. and Hida K. (2006) *J. Ultrasound Med.*, 25(5), 599-605.
- [12] Amer M.S., Maher M.M., Omar O., Reda R., Elawam E.A. and Sweed H.S. (2010) *Eur. J. Gen. Med.*, 7(3), 245-249.
- [13] Christensen L.L., Almdal P.L., Carstensen B., Tarnow L. and Wiinberg N. (2010) *Cardiovasc. Diabetol.*, 10, 9-40.
- [14] Al-Siaidy W.F., Al-Sabbagh A.A., Al-Karawi I.N., and Mohammed D.A. (2013) *J. Fac. Med. Baghdad*, 55(1), 77-82.
- [15] Vicenzini E., Ricciardi M.C., and Puccinelli F. (2007) *J. Ultrasound Med.*, 26(4), 427-432.
- [16] Jiang Y.N., Kohara K. and Hiwada K. (1999) *Hypertens. Res.*, 22, 203-207.
- [17] Libby P., Ridker P.M. and Maseri A. (2002) *Circulation*, 105, 1135-1143.
- [18] Montecchi F.R., Menzinger G. and Lala A. (2001) *Diabetes Nutr. Metab.*, 14(1), 58-61.
- [19] Carallo C., Spagnuolo V., Siclari D., Talarico R., Pujia A. and Gnasso A. (1996) *Minerva Cardioangiol.*, 44(1-2), 53-57.