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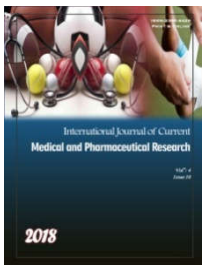

ASSOCIATION BETWEEN GLYCEMIC CONTROL AND SERUM LIPID PROFILE IN TYPE 2 DIABETES MELLITUS PATIENTS

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ABSTRACT

Introduction: Diabetes mellitus (DM) patients with dyslipidemia are soft targets of cardiovascular deaths. Very few studies have previously tried to find correlation between HbA1c levels and lipid profile. In present study primary objective was compare dyslipidaemia between good glycemic control and poor glycemic control groups whereas secondary objective was to find correlation between fasting serum glucose (FBS), two hours post prandial serum glucose (PPBS) and HbA1c and serum lipid profile in type 2 DM patients.

Material and Methods: One hundred ninety seven patients age ≥30 years were included in study. Investigations included were FBS, two hours PPBS, HbA1c, serum total cholesterol(TC), serum triglycerides (TG), serum high density lipoprotein (HDL) cholesterol, serum low density lipoprotein(LDL) cholesterol, very low density lipoprotein(VLDL) cholesterol, Apo lipoprotein A and Apo lipoprotein B (Apo A & Apo B). Atherogenic index of plasma (AIP) = log (TG/HDLc) and Atherogenic coefficient (AC) = (TC– HDLc)/HDLc were calculated). In the present study, we have defined dyslipidaemis when any one parameter of lipid profile was abnormal. Primary outcome measures were to compare dyslipidaemia between good glycemic control and poor glycemic control groups and study correlation between lipid profile and FBS, PPBS and HbA1c. Chi-square test, Mann-Whitney U test and Spearman correlation were used for statistical analysis.

Results: In poor glycemic control group (HbA1c >7) dyslipdemia was observed in all patients which was statistically significant. FBS had significant direct positive correlation with TC ($r = 0.21$, $P = 0.004$), HDL cholesterol ($r = 0.25$, $P = 0.001$), LDL cholesterol ($r = 0.30$, $P = 0.001$) and APO lipoprotein A($r = 0.35$, $P = 0.001$) whereas PPBS and HbA1c did not show significant correlation with lipid profile.

Conclusion: Glycemic control is important for preventing dyslipidaemia in DM patients.

INTRODUCTION

Diabetes Mellitus (DM) is a global endemic with rapidly increasing prevalence in both developing and developed countries.[1] WHO has declared India as "Diabetic Capital of the world".[2] Although the prevalence of both type 1 and type 2 DM is going to increase, type 2 DM is expected to rise more rapidly in future because of increased obesity and reduced activity levels.

Glycated hemoglobin (HbA1c) is routinely used as a diagnostic tool for measuring long term glycemic control. The United Kingdom Prospective Diabetes Study (UKPDS) has shown that in patients with type 2 DM, the risk of diabetic complications were strongly associated with previous hyperglycemia. Glycemic control with decreased level of HbA1c is likely to reduce the risk of complications.[3]

Apart from classical risk factors like dyslipidemia, elevated HbA1c has now been regarded as an independent risk factor for cardio vascular diseases (CVD) in subjects with or without DM .Estimated risk of CVD has shown to be increased by 18% for each 1% increase in absolute HbA1c value in DM patients.[4]

DM patients with unnoticed dyslipidemia are soft targets of cardiovascular deaths. Patients with type 2 DM often exhibit an atherogenic lipid profile, which greatly increases their risk of CVD compared with people without DM. An early intervention to normalize circulating lipids has been shown to reduce cardiovascular complications and mortality.[5,6]

Very few studies have previously tried to find the correlation between HbA1c levels and lipid profile. Some of these have shown that all the parameters of lipid profile have significant correlation with glycemic control.[7-10] On the other hand, some

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studies do not report significant correlation between glycemic control and all parameters of lipid profile. [11-13] In the present study primary objective was to find association between glycemic control and serum lipid profile in type 2 DM patients.

MATERIAL AND METHODS

All the patients age ≥30 years who attended medicine outpatient department, diabetes mellitus outpatient department or admitted in Poona Hospital and Research Centre, Pune between May 2015 and November 2016 and ready to participate were included in study. Permission was obtained from ethics committee and scientific advisory committee of the institution.

Based on previously published study [14], setting an alpha error at 0.05, and power at 80%, sample size of 138 was calculated for the present cross sectional study by formula.[15] We included 197 patients for better validation of results. Written informed consent was obtained from all patients after explaining them study in detail. Each patient was subjected to detailed clinical history, clinical examination and investigated as per the study proforma. Exclusion criteria were patients with known case of type-1 DM, patients already on lipid lowering drugs, patients with functional thyroid disorder, chronic renal failure, nephrotic syndrome, liver disease, chronic alcoholic, familial hypercholesteremic syndromes, patients with anemia, patients using corticosteroid therapy, oral contraceptives, pregnant

Table 1 Lipid profile and HbA1c

Parameters		HbA1c <=7 N= 37 (%)	HbA1c >7 N= 160 (%)	Total N= 197 (%)	p-value
TC	Normal (<200 mg/dL)	25(67.6)	98(61.3)	123(62.4)	0.573 *
	Abnormal (≥ 200 mg/dL)	12(32.4)	62(38.7)	74(37.6)	
	Mean(SD)	179.3(54.2)	194.3(57.2)		0.129 **
HDL	Normal (≥ 40 mg/dL)	11(29.7)	50(31.3)	61(31.0)	0.999 *
	Abnormal (< 40 mg/dL)	26(70.3)	110(68.7)	136(69.0)	
	Mean(SD)	41.4(14.1)	43.1(15.8)		0.742**
LDL	Normal (< 100 mg/dL)	17(45.9)	65(40.6)	82(41.6)	0.582 *
	Abnormal (≥ 100 mg/dL)	20(54.1)	95(59.4)	115(58.4)	
	Mean(SD)	106.4(38.4)	114.1(43.8)		0.315**
VLDL	Normal (≤ 51 mg/dL)	35(94.6)	141(88.1)	176(89.3)	0.377 *
	Abnormal (> 51 mg/dL)	2(5.4)	19(11.9)	21(10.7)	
	Mean(SD)	41.3(11.4)	43.4(11.6)		0.403**
TG	Normal (<150 mg/dL)	21(56.8)	84(52.5)	105(53.3)	0.716 *
	Abnormal (≥ 150 mg/dL)	16(43.2)	76(47.5)	92(46.7)	
	Mean(SD)	143.0(58.5)	167.6(73.8)		0.059**
APO A	Normal (110-205 mg/dL)	22(59.5)	66(41.3)	88(44.7)	0.066 *
	Abnormal (>205 mg/dL)	15(40.5)	94(58.7)	109(55.3)	
	Mean(SD)	140.8(55.8)	129.3(58.6)		0.121**
APO B	Normal (55-125 mg/dL)	23(62.2)	97(60.6)	120(60.9)	0.999 *
	Abnormal (>125 mg/dL)	14(37.8)	63(39.4)	77(29.1)	
	Mean(SD)	101.7(38.4)	103.9(45.1)		0.915**
APO B/ APO A ratio	Normal (0.3-0.9)	27(73.0)	95(59.4)	122(61.9)	0.137 *
	Abnormal (>0.9)	10(27.0)	65(40.6)	75(38.1)	
	Mean(SD)	0.93(0.9)	0.96(0.6)		0.143**
TC/ HDL ratio	Normal (≤ 5.0)	26(70.3)	98(61.3)	124(62.9)	0.349 *
	Abnormal (>5.0)	11(29.7)	62(38.7)	73(37.1)	
	Mean(SD)	4.7(1.7)	4.9(1.9)		0.430**
LDL /HDL ratio	Normal (≤ 3.6)	30(81.1)	125(78.1)	155(78.7)	0.825 *
	Abnormal (>3.6)	7(18.9)	35(21.9)	42(21.3)	
	Mean(SD)	2.8(1.2)	2.9(1.3)		0.713**
AIP	Mean(SD)	0.54(0.26)	0.58(0.25)		0.357**
Non HDL cholesterol	Normal (≤ 130 mg/dL)	19(51.4)	58(36.3)	77(39.1)	0.096 *
	Abnormal (>130 mg/dL)	18(48.6)	102(63.7)	120(60.9)	
	Mean(SD)	138.0(49.2)	150.6(53.7)		0.108**

* X^2 or Fisher's exact test was used
** Mann-Whitney U test was used

women and absence or withdrawal of consent. Investigations included fasting serum glucose (FBS), two hours post prandial serum glucose (PPBS), HbA1c, serum total cholesterol(TC), serum triglycerides (TG), serum high density lipoprotein (HDL) cholesterol, serum low density lipoprotein(LDL) cholesterol, very low density lipoprotein(VLDL) cholesterol, Apo lipoprotein A and Apo lipoprotein B (Apo A & Apo B). Atherogenic index of plasma (AIP) = log (TG/HDLc) and Atherogenic coefficient (AC) = (TC– HDLc)/HDLc were calculated.

The American diabetes association (ADA) has designated HbA1c level of <7% as a goal of optimal blood glucose control [16] whereas the American association of clinical endocrinologist has recommended HbA1c level of <6.5%. [17] We have used ADA goal for optimal blood glucose control (HbA1c level ≤ 7 good control , and HbA1c level > 7 poor control).National cholesterol education programme (NCEP) guidelines [18] were used for definition of dyslipidemia, hypercholesterolemia(serum TC levels ≥200 mg/dl), hypertriglyceridemia (serum TG levels ≥150 mg/dl), low HDL cholesterol (HDL cholesterol levels <40 mg/dl), high LDL cholesterol (LDL cholesterol levels ≥100 mg/dl calculated using the Friedewald equation). In the present study, we have defined dyslipidaemis when any one parameter of lipid profile was abnormal. Patients with dyslipidemia were further subdivided into those with mixed dyslipidemia (all parameters outside the recommended targets), combined dyslipidemia (two parameters outside the recommended targets) and those with isolated dyslipidemia (any one parameter outside the recommended targets).

Primary outcome measures were to compare dyslipidaemia between good glycemic control and poor glycemic control groups and study correlation between lipid profile and FBS, PPBS and HbA1c.

Data analysis was done by using SPSS (Statistical package for social sciences) Version 20:0 (IBM, USA).Qualitative data are expressed by using frequency and percentage (%). Quantitative data are expressed by using mean and SD. Chi-square test or Fisher's exact test was used to compare qualitative data variables. Mann-Whitney U test was used to find the significant difference between quantitative variables. Spearman correlation coefficient used to find the correlation between two quantitative data variables, lipid profile and FBS, PPBS and HbA1c. P –value <0.05 was considered significant.

RESULTS

The study included 126 male (64 %) and 71 female (36%) patients. Ninety one (46.2%), 62/197 (31.5%), and 31 (15.7%) patients were between the age group of 41-50, 51-60 and 61-70 respectively. Eight patients (4.1%) were above 70 years whereas 5/197 (2.5%) were below 41 years of age. Mean age of the patients was 62 ± 11.2 years. One hundred seventy (86.3%) patients had hypertension and 86/197 (43.7%) patients were smokers. One hundred forty (71.1%), 30/197(15.2%), 26(13.2%0) and 1/197(0.5%) patients had body mass index of > 25, 23.0 ≤ 25, 18.5- < 23, and < 18.5 respectively. Duration of diabetes mellitus was one to five years, six to ten years and more than 10 years in 5/197(2.6%), 96/197(48.7%) and 96/197(48.7%) patients respectively. One hundred sixty seven (84.8%) patients had dyslipidaemia. Mixed dyslipidaemia, combined dyslipidaemia and isolated dyslipidaemia was observed in 60/197(30.5%), 54 (27.4%), and 53(26.9%) patients respectively whereas 30 (15.2%) patients had normal lipid levels. One hundred sixty (81.2%) patients had HbA1c >7 whereas 37/ 197(18.8%) had HbA1c ≤ 7. The atherogenic lipid profile (high TG, high LDL and low HDL) was seen in 10 (5.1%) patients.

There was no statistically significant difference between isolated dyslipidaemia and age group, gender, obesity, socioeconomic status and duration of DM. All the patients whose HbA1c > 7 had dyslipidaemia whereas 7/37(19.9%) patients had dyslipidaemia whose HbA1c ≤ 7 which was statistically significant (p <0.001).

As depicted in table 1, there was no statistically significant difference in lipid profile of patients whose HbA1C was ≤ 7 and those HbA1C was > 7.

FBS appeared to have a direct correlation with TC, HDL, LDL and APO A. A significant positive correlation (correlation coefficient $r = 0.21$, $P = 0.004$) was seen between the TC and FBS, (correlation coefficient $r = 0.25$, $P = 0.001$) between HDL and FBS, (correlation coefficient $r = 0.30$, $P = 0.001$) between LDL and FBSL (correlation coefficient $r = 0.35$, $P = 0.001$) between APO A and FBS concentrations. There was no significant correlation between PPBS, HBA1C and lipid profile parameters.

DISCUSSION

In the present study conducted on 197 patients, we evaluated the pattern of lipid profile parameters in DM patients and it's correlation with glycemic control. Patient's characteristics was compared with prevalence of dyslipidemia, only glycemic control showed association with dyslipidemia ,other factors such as age, gender, duration of DM ,BMI & socioeconomic status did not show association. In our study 37 patients had HbA1c level of ≤ 7 indicating a good glycemic control and 160 patients had HbA1c of > 7 indicating a poor glycemic control. This suggest higher number of DM patients still do not achieve glycemic targets. The levels of TC, LDL, VLDL, TG, Non-HDL, Lipid ratios were statistically not significant, as observed in other studies. [10, 19-23] As elevated HbA1c and dyslipidemia are independent risk factors of CVD, DM patients with elevated HbA1c and dyslipidemia can be considered as a very high risk group for CVD. Improving glycemic control can substantially reduce the risk of cardiovascular events in DM patients. [24]

Patient with poor glycemic control (HBa1C >7) had more dyslipidemia. The prevalence of dyslipidemia in our study was 84.8%. Parikh *et al* reported prevalence of dyslipidemia among DM males and females 85.5% and 97.8% respectively. [14] M Agarwal *et al* reported 80.7% prevalence of dyslipidemia among DM patients who were not on lipid lowering drugs in tertiary care hospital in Ahemdabad. [21] Study of prevalence and pattern of dyslipidemia in type 2 DM patients attending rural health training centre of medical college in Bhopal, Madhya Pradesh, India reported high (86%) prevalence of dyslipidemia and most common pattern observed was mixed type dyslipidemia. [25] In our study isolated single parameter dyslipidemia was seen in 26.9% patients, combined dyslipdemia in 27.4% patients and mixed dyslipidemia in 30.5 % patients. The most common lipid abnormality seen was low serum HDL (69.0%), increased LDL (58.4%) and hypertriglyceridemia (46.7%) which was similar to western studies reporting hypertriglyceridemia and low serum HDL as major abnormality. [26, 27] The most common pattern of

dyslipidemia in study by Agarwal *et al*[21] in Gujarati population was high LDL and hypertriglyceridemia.

In the present study, 46.7 % had hypertriglyceridemia and this would have resulted in unreliable LDL calculation. So, relying on LDL targets alone can be misleading in such patients hence, a new parameter on lipid profile, the non-HDL-C can be used as a marker of dyslipidemia since it reflects the sum of serum cholesterol carried by all of the potentially atherogenic lipoproteins LDL, VLDL, IDL, and other remnant lipoproteins. The measurement of Non-HDL-C is simple which can be conducted even in non-fasting state of patients and can be determined regardless of TG concentration. Hence, Non-HDL cholesterol can be of great value in determining dyslipidemia in diabetic subjects.

In our study HbA1c did not show correlation with lipid profile parameters which was contrary to previous studies. [20-23] HbA1c showed no significant correlation with TC in our study ,this was also seen in a similar study conducted in Turkey. [28] In the present study, FBS showed direct correlation with TC, LDL, HDL, and APO A and no correlation was found between PPBSL with any lipid profile parameters. This was seen in some studies.[13,19] Most of the previous studies reported that PPBSL contributed maximum to the glycemic control and fluctuations in which leads to macro and micro vascular complication,[29, 30] nonetheless multiple studies have linked FBS to increased cardiovascular risk.[31-34] Our study did not show significant correlation between FBS and non-HDL-C. Non-HDL-C was shown to be the stronger predictor of CVD in diabetic population by 'The Strong Heart Study' with hazard ratios of 2.23 and 1.80 respectively in male and female.[35] This study reported that increased Non-HDL cholesterol concentrations had significant, curvilinear relationships with CVD and CHD risk (P <0.001). Moreover, NCEP ATPIII has recommended using Non-HDL cholesterol in assessing CVD risk in patients with diabetes.

Experimental studies show that abnormal glucose metabolism impairs normal endothelial function, accelerates atherosclerotic plaque formation, and contributes to plaque rupture and thrombosis. Epidemiological studies provide complementary evidence. In the Rotterdam study, among elderly participants with a FBS <110 mg/dL and without diabetes, those with higher blood glucose levels had higher levels of arterial stiffness.[36] The CATHAY study reported that higher levels of fasting glycemia (102–124 mg/dL) were associated with arterial endothelial dysfunction and intima-media thickening. [37] In a biomarker study in Italy, a number of CVD biomarkers showed positive dose-response relationships with fasting glucose across three strata: <100; 100–109; and 110–125 mg/dL. [38]

This was a cross sectional study conducted in a single tertiary care hospital and represents only a small population of patients. Patients had other risk factors like hypertension, smoking and high body mass index which could have affected lipid profile. Randomized controlled studies may be conducted to compare lipid profile parameters in various anti diabetic regimen.

CONCLUSIONS

In poor glycemic control group dyslipdemia was observed in all patients which was statistically significant. FBS had significant direct positive correlation with TC, HDL, LDL and APO whereas PPBS and HbA1c did not show significant correlation with lipid profile.

References

1. Berry C, Tardif JC, Bourassa MG. Coronary heart disease in patients with diabetes: part I: recent advances in prevention and noninvasive management. Journal of the American College of Cardiology. 2007;49(6):631-42.
2. Gupta V, Suri P. Diabetes in elderly patients. JK practitioner. 2002;91(4):258-9.
3. Stratton IM, Adler AI, Neil HA, Matthews DR, Manley SE, Cull CA *et al*. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. Bmj. 2000;321(7258):405-12.
4. Selvin E, Marinopoulos S, Berkenblit G, Rami T, Brancati FL, Powe NR *et al*. Meta-analysis: glycosylated hemoglobin and cardiovascular disease in diabetes mellitus. Annals of internal medicine. 2004;141(6):421-31.
5. Chambless LE, Heiss G, Folsom AR, Rosamond W, Szklo M, Sharrett AR *et al*. Association of coronary heart disease incidence with carotid arterial wall thickness and major risk factors: the Atherosclerosis Risk in Communities (ARIC) Study, 1987–1993. American journal of epidemiology. 1997;146(6):483-94.
6. Burtis CA, Ashwood ER, Bruns DE. Tietz textbook of clinical chemistry and molecular diagnostics: Elsevier Health Sciences; 2012.
7. Ramachandran A, Snehalatha C, Latha E, Vijay V, Viswanathan M. Rising prevalence of NIDDM in an urban population in India. Diabetologia. 1997;40(2):232-7.
8. Mehta SR, Kashyap AS, Das S. Diabetes mellitus in India: The modern scourge. Medical journal armed forces India. 2009;65(1):50-4.
9. Rani HS, Madhavi G, Rao VR, Sahay B, Jyothy A. Risk factors for coronary heart disease in type II diabetes mellitus. Indian journal of clinical biochemistry. 2005;20(2):75-80.
10. VinodMahato R, Gyawali P, Raut PP, Regmi P, Singh KP, Raj Pandeya D *et al*. Association between glycaemic control and serum lipid profile in type 2 diabetic patients: Glycated haemoglobin as a dual biomarker. Biomedical Research. 2011;22(3):375-80.
11. Zachary T Bloomgarden ZT. Cardiovascular disease, neuropathy, and retinopathy. Diabetes care. 2009;32(6):e64-e8.
12. Yan Z, Liu Y, Huang H. Association of glycosylated hemoglobin level with lipid ratio and individual lipids in type 2 diabetic patients. Asian Pacific journal of tropical medicine. 2012;5(6):469-71.
13. Khan H, Sobki SH, Khan SA. Association between glycaemic control and serum lipids profile in type 2 diabetic patients: HbA1c predicts dyslipidaemia. Clinical and experimental medicine. 2007;7(1):24-9.
14. Parikh RM, Joshi SR, Menon PS, Shah NS. Prevalence and pattern of diabetic dyslipidemia in Indian type 2 diabetic patients. Diabetes & Metabolic Syndrome: Clinical Research & Reviews. 2010;4(1):10-2.

15. Charan J, Biswas T. How to calculate sample size for different study designs in medical research?. Indian Journal of Psychological Medicine. 2013;35(2):121-26.
16. Haffner SM. American Diabetes Association. Dyslipidemia management in adults with diabetes.Diabetes care. 2004;27(suppl 1):S68-71
17. Lebovitz HE, Austin MM, Blonde L, Davidson JA, Prato SD, Gavin III JR *et al*. ACE/AACE consensus conference on the implementation of outpatient management of diabetes mellitus: consensus conference recommendations. Endocrine Practice. 2006;12(Supplement 1):6-12.
18. Executive summary of the Third Report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). JAMA 2001;285(19):2486-97.
19. Reddy S, Meera S, William E. Correlation between glycemic control and lipid profile in type 2 diabetic patients: HbA1c as an indirect indicator of dyslipidemia. Asian Journal of Pharmaceutical and Clinical Research. 2014;7(2):153-5.
20. Meenu J, Jadeja Jayendrasinh M, Neeta M. Correlation between HbA1c values and lipid profile in type 2 diabetes mellitus. International journal of basic and applied physiology. 2013;2(1):47-50.
21. Agarwal M, Patel JP, Lala MK. Association between glycemic control and serum lipid profile in known diabetic patients of civil hospital, Ahmedabad. International Journal of Medical Science and Public Health. 2016;5(2):356-60.
22. Samatha P, Siva Prabodh V, Chowdary NV, Shekhar R. Glycated hemoglobin and serum lipid profile associations in type 2 diabetes mellitus patients. J Pharmac Biomed Sci. 2012;17:1-4.
23. Dshmukh S, Singh VB, Hb CK, Meena B, Beniwal S, Saini VK. Can Hba1c be a marker for cardiovascular risk in type 2 Diabetes Mellitus. International Journal of Medical Research and Review. 2015;3(04).
24. Selvin E, Wattanakit K, Steffes MW, Coresh J, Sharrett AR. HbA1c and peripheral arterial disease in diabetes the Atherosclerosis Risk in Communities study. Diabetes care. 2006;29(4):877-82.
25. Borle AL, Chhari N, Gupta G, Bathma V. Study of prevalence and pattern of dyslipidaemia in type 2 diabetes mellitus patients attending rural health training centre of medical college in Bhopal, Madhya Pradesh, India. International Journal Of Community Medicine and Public Health. 2017;3(1):140-4.
26. Cowie CC, Howard BV, Harris MI. Serum lipoproteins in African Americans and whites with non-insulin-dependent diabetes in the US population. Circulation. 1994;90(3):1185-93.
27. UK Prospective Diabetes Study Group. UK Prospective Diabetes Study 27: plasma lipids and lipoproteins at diagnosis of NIDDM by age and sex. Diabetes Care. 1997;20(11):1683-7.
28. Sert M, Morgul G, Tetiker BT. Diabetic dyslipidemia is a well-known issue, but what about lipoprotein a levels in Type 2 diabetics. Int J Diabetes Metabol. 2010;18:81-7.
29. Masram SW, Bimanpalli MV. Assessment of contribution of fasting and post meal plasma glucose to increased HbA1C in diabetes mellitus-comparative study. Int J Biol Med Res. 2012;3(3):2020-4.
30. Ning F, Zhang L, Dekker JM, Onat A, Stehouwer CD, Yudkin JS *et al*. Development of coronary heart disease and ischemic stroke in relation to fasting and 2-hour plasma glucose levels in the normal range. Cardiovascular diabetology. 2012;11(1):76.
31. Boden-Albala B, Sacco RL, Lee HS, Grahame-Clarke C, Rundek T, Elkind MV *et al*. Metabolic syndrome and ischemic stroke risk. Stroke. 2008;39(1):30-5.
32. Park C, Guallar E, Linton JA, Lee DC, Jang Y, Son DK *et al*. Fasting glucose level and the risk of incident atherosclerotic cardiovascular diseases. Diabetes Care. 2013;36(7):1988-93.
33. Sorkin JD, Muller DC, Fleg JL, Andres R. The Relation of Fasting and 2-h Postchallenge Plasma Glucose Concentrations to Mortality Data from the Baltimore Longitudinal Study of Aging with a critical review of the literature. Diabetes Care. 2005;28(11):2626-32.
34. Emerging Risk Factors Collaboration. Diabetes mellitus, fasting glucose, and risk of cause-specific death. N Engl J Med. 2011;2011(364):829-41.
35. Lu W, Resnick HE, Jablonski KA, Jones KL, Jain AK, Howard WJ *et al*. Non-HDL cholesterol as a predictor of cardiovascular disease in type 2 diabetes the strong heart study. Diabetes care. 2003;26(1):16-23.
36. Van Popele NM, Elizabeth Hak A, Mattace-Raso FU, Bots ML, Van Der Kuip DA, Reneman RS *et al*. Impaired fasting glucose is associated with increased arterial stiffness in elderly people without diabetes mellitus: the Rotterdam Study. Journal of the American Geriatrics Society. 2006;54(3):397-404.
37. Thomas GN, Chook P, Qiao M, Huang XS, Leong HC, Celermajer DS *et al*. Deleterious impact of "high normal" glucose levels and other metabolic syndrome components on arterial endothelial function and intima-media thickness in apparently healthy Chinese subjects: the CATHAY study. Arteriosclerosis, thrombosis, and vascular biology. 2004;24(4):739-43.
38. Andreozzi F, Succurro E, Mancuso MR, Perticone M, Sciacqua A, Perticone F *et al*. Metabolic and cardiovascular risk factors in subjects with impaired fasting glucose: the 100 versus 110 mg/dL threshold. Diabetes/metabolism research and reviews. 2007;23(7):547-50.

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