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Study of C - reactive protein, D-dimer in correlation with HbA1C levels in patients with diabetes mellitus type 2

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ABSTRACT

Background: Diabetes Mellitus is a chronic metabolic disease presented with chronic hyperglycemia, which causes abnormalities in the endothelial cells, proinflammatory state, altered platelet function & plasma coagulation factors that lead to a hypercoagulable state & thrombotic incidents. D-dimer is an indirect marker of thrombotic activity. C-reactive protein (CRP) is an important risk factor for systemic inflammation, so the present study aimed to determine the relationship between CRP, D-dimer in correlation with HbA1C in patients with Diabetes Mellitus.
Aim: The present study is undertaken to evaluate the study of D-dimer, C-reactive protein in correlation with HbA1C levels in Type 2 Diabetes Mellitus.
Materials and Methods: A total of one hundred & twenty subjects aged 30-70 years were included. The patients were selected from those who attended the medical OPD at Gandhi Medical College & hospital, Secunderabad, & their samples are analyzed by Beckmann Coulter Fully Automatic Analyzer AU5800. CRP is estimated by rate turbidimetry method & D-dimer is estimated by rate turbidimetry method. HbA1C by HPLC in Bio-rad-D-10
Conclusion: The prospective of elevated D-dimer, CRP levels are in correlation with increased HbA1C levels. So screening of inflammatory & hemostatic markers must be done in patients with uncontrolled Diabetes Mellitus.

1. Introduction

Diabetes Mellitus is a persistent metabolic illness introduced through ongoing hyperglycemia, which causes irregularities in the endothelial cells, proinflammatory state, modify platelet capability & plasma coagulation factors that lead to hypercoagulable state & apoplexy.[1] Hyperglycemia was a significant element to incite endothelial brokenness, expanding the prothrombin part 1+2 & D-dimer. A backhanded marker of thrombotic movement, d-dimer is the last section of corruption of cross-connected fibrin. D-dimer is a marker for fibrinolytic/coagulation processes related with illness conditions like profound vein apoplexy & atherosclerosis. Expanded D-dimer values have been accounted for diabetes mellitus, yet the inquiry is whether there is variety in D-dimer level related with the movement of diabetes mellitus & it is conjectured that plasma D-dimer levels might vary in individuals at various phases of diabetes mellitus.[2] CRP is a pentameric protein orchestrated by the liver, whose level ascents because of irritation. There are various reasons for a raised C-responsive protein. These incorporate intense & persistent circumstances, & these can be irresistible or non-irresistible in etiology. C-receptive protein (CRP), a marker of fundamental irritation, is a free gamble factor for cardiovascular infection. The

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C-receptive protein is one of the intense stage reactants which have a place with the pentraxin bunch. Raised CRP levels have likewise been connected to an expanded gamble of later improvement of diabetes.[3,4] So, the current review is embraced to assess the investigation of D-dimer, C-receptive protein in relationship with HbA1C levels in Type 2 Diabetes Mellitus.

2. Materials and Methods

2.1. Subject

The study was carried out in the Department of Biochemistry Gandhi Medical College & Hospital, Secunderabad. The cases were selected from those attended, the medicine OPD Gandhi Hospital, Secunderabad. The Investigations were carried out in Bio-Chemistry laboratory, Gandhi Medical College & Hospital, Secunderabad.

2.2. Design

The study is a cross-sectional observational prospective study.

2.3. Criteria for selection

The subjects were selected based on the medical history obtained from a health questionnaire & from recent lab reports, established diagnosed cases of diabetes.

Total 120 subjects divide into three groups. Group-1 consisted of 40 healthy controls, whose HbA1C is less than 6%. Group-2 consisted of 40 patients of newly detected type 2, Diabetes Mellitus whose HbA1C is 6-7%. Group-3 consisted of 40 patients having diabetes, whose HbA1C is more than 7%. Patients were considered to be diabetic based on ADA criteria for the diagnosis of Diabetes Mellitus.

2.4. Inclusion criteria

Age – 30 to 70 years (both male & female), Non diabetic (HbA1C < 6%), Controlled Diabetic (HbA1C 6%-7 %), Uncontrolled Diabetic (HbA1C >7%) subjects are included in the study.

2.5. Exclusion criteria

Patients with Type 1 diabetes, Gestational diabetes, Hypertension, bleeding disorders medications -antiplatelet drugs & HMG-CoA reductase inhibitors (statins) & steroids subjects are excluded from the study.

2.6. Blood sample collection

The subjects were divided into three groups. After informed consent from patients, a venous blood sample was obtained from every volunteer into citrate tubes (light blue cap BD vacutainer system) for D-dimer, plain tube (red cap BD vacutainer system) for CRP, & EDTA tube (lavender cap BD vacutainer system) for HbA1C.

2.7. Procedure

All the blood samples were immediately carried to the biochemistry laboratory in a crushed ice block with cold chain maintenance. Blood samples for D-dimer were centrifuged at 3000 RPM for 20 minutes. Whereas Blood samples for CRP were centrifuged at 3000 RPM for 10 minutes. Blood samples were analyzed by Beckmann Coulter Fully Automatic Analyzer AU5800. CRP is estimated by rate turbidimetry method, D-dimer estimated by rate turbidimetry method, HbA1C by HPLC in Bio-Rad D-10.

2.8. Ethics approval

The study was approved by the Ethics in Human Research Committee review at Gandhi Medical College & Hospital, Musheerabad, Secunderabad as part of a biochemistry research project. The study is a cross-sectional observational prospective study. The study is carried out at Gandhi Medical College & Hospital, Musheerabad, Secunderabad.

2.8.1. Reference Range

HbA1C

Normal - 4% to 5.6%.
Prediabetes - 5.7% to 6.4%
Diabetic - > 6.5%.

CRP

Normal - < 5 mg/L.
High - > 5 mg/L.

D-dimer

Normal - < 0.5 μ g/mL
High - < 0.5 μ g/mL

3. Results

The Patients enrolled for the study were in the age group of 30 to 70 years & divided into 3 groups.

Table 1 shows mean & SD of HbA1C, CRP & D-dimer in non-diabetic, controlled diabetic & uncontrolled diabetic subjects of age < 50 years & 50-70 years. The mean age & HbA1C of non-diabetic age are (30 no), controlled diabetes (30 no), & uncontrolled diabetic (25 no) is 39.9±5.3, 39.9±5.4, & 42.0±9.07 respectively. The mean age & HbA1C of non-diabetic age are more than 50-70 years (10 no), controlled diabetes (10) & uncontrolled diabetes (15) is 57.6±5.3, 59.3±6.3, & 59.30±9.41 respectively. There is no significant difference in age in the <50 & 50-70 years non-diabetic group. In the controlled diabetic group, the CRP & D-dimer are high in 50-70 years age when compared with the <50 years age group. In the uncontrolled diabetic group, the CRP is high in <50 years age when compared with 50-70 years age.

Table 1:

Non-Diabetic					Controlled Diabetic				Uncontrolled Diabetic			
Age	No of Cases	HbA1C	CRP mg/L	D-dimer (FEU) μg/ml	No of Cases	HbA1C	CRP mg/L	D-dimer (FEU) μg/ml	No of Cases	HbA1C	CRP mg/L	D-dimer (FEU) μg/ml
<50	30	5.3	3.5	0.14	30	5.4	3.5	0.14	25	9.07	19.86	0.35
		0.60	3.5	0.18		0.6	3.5	0.18		2.41	33.99	0.72
50-70	10	5.3	3.0	0.21	10	6.3	7.9	0.30	15	9.41	5.98	0.54
		0.7	2.8	0.26		2.0	5.9	0.30		3.27	5.79	0.61

Table 2: Shows the comparison of CRP, D-dimer in three groups of patients in correlation with HbA1C

Non-Diabetic					Controlled Diabetic				Uncontrolled Diabetic			
Particulars	No. of cases	HbA1C	CRP mg/L	D-dimer (FEU) μg/ml	No. of cases	HbA1C	CRP mg/L	D-dimer (FEU) μg/ml	No of cases	HbA1C	CRP mg/L	D-dimer (FEU) μg/ml
Mean	40	5.4	3.3	0.15	40	6.3	7.7	0.30	40	9.2	15.4	0.61
SD		0.6	3.3	0.20		1.0	6.3	0.29		8.4	5.3	0.30
'Pearson Coefficient			0.30	0.32			0.32	0.35			0.35	0.32
P-Value			0.05	0.04			0.04	0.03			0.03	0.04

Table 2 shows, increase in CRP & D-dimer along with increase in HbA1C levels.

Table 2 shows, mean, SD, Pearson Coefficient & P-value of HbA1C, CRP & Di-dimer in three groups of uncontrolled diabetic, controlled diabetic & uncontrolled diabetic. The P – value is significant (P=<0.05) in three groups. Mean & standard deviation are high in uncontrolled diabetic when compare with non-diabetic & controlled diabetic groups

1. Mean CRP (mg/L) is statistically significantly higher in uncontrolled diabetic patients.
2. Mean D-dimer (μg/ml) is statistically significantly higher in uncontrolled diabetic patients compared with the controlled diabetic patients (P=<0.05).

Table 3 shows the patients enrolled for the study were in the age group of 30 to 70 years. The mean age & HbA1C of non-diabetic in male patients (18 no), controlled diabetes (21), & uncontrolled diabetes (19) are 47.1±5.3, 46.8±6.2, & 48.6±9.58 respectively. The Patients enrolled for the study were in the age group of 30 to 70 years. The mean age & HbA1C of non-diabetic in female patients (22), controlled diabetes (19), & uncontrolled diabetes (21) are 42.7±5.4, 46.5±6.3, & 48.0±8.74 respectively. There is no significant difference in men & women in the Non-diabetic & controlled diabetic groups. In the uncontrolled diabetic group, the CRP & D-dimer is high in men when compared with women

1. Non diabetic men has less HbA1C.
2. Where as Uncontrolled men has more HbA1C.
3. CRP is high in Uncontrolled diabetic in men & women when compared with non-diabetic & controlled diabetic group.

4. Discussion

Diabetes mellitus, is a gathering of metabolic problems described by a high glucose level (hyperglycemia) over a delayed time frame. Diabetes mellitus leads after some time to serious harm to the heart, veins, eyes, kidneys & nerves. The most well-known is type 2 diabetes, which happens when the body becomes impervious to insulin or doesn't make sufficient insulin. Type 2 diabetes has deceptive beginning where an irregularity between insulin levels & insulin responsiveness causes a useful shortage of insulin.[5,6] Insulin obstruction is multi factorial yet ordinarily creates from corpulence & maturing. Diabetes Mellitus (DM) is a with miniature & large scale vascular entanglements like intense myocardial dead tissue, stroke, & fringe vascular infection.[7] In diabetes patients because of, there is dis-capability of endothelial cells, irregularities with the inclination for thrombotic occasions[8] the centralization of glycated hemoglobin (HbA1C) a standard proportion of constant glycemia that is accustomed to recognizing high-risk people who will foster diabetes, control of diabetes & as an indicator of diabetes.

Hyperglycemia alone can hinder pancreatic beta-cell capability & adds to debilitated insulin emission. Insulin obstruction is inferable from abundance unsaturated fats & proinflammatory cytokines, which prompts impeded glucose transport & increments fat breakdown. Since there is a lacking reaction or creation of insulin, the body answers by improperly expanding glucagon, in this manner further adding to hyperglycemia.[9] While insulin opposition is a part

Table 3: Shows the gender Base - Mean

	Non-Diabetic				Controlled Diabetic				Uncontrolled Diabetic			
	No of cases	HbA1C	CRP mg/L	D-dimer (FEU) μg/ml	No of cases	HbA1C	CRP mg/L	D-dimer (FEU) μg/ml	No of cases	HbA1C	CRP mg/L	D-dimer (FEU) μg/ml
Men	18	5.3	2.8	0.14	21	6.2	7.6	0.34	19	9.58	16.38	0.62
		0.5	3.5	0.16		1.4	6.6	0.3		8.6	3.34	0.20
Women	22	5.4	3.8	0.17	19	6.3	7.7	0.25	21	8.74	13.94	0.59
		0.7	3.1	0.23		1.4	6.3	0.30		8.2	7.78	0.35

of T2DM, the full degree of the illness results when the patient has lacking creation of insulin to make up for their insulin obstruction.

Constant hyperglycemia likewise causes nonenzymatic glycation of proteins & lipids. The degree of this is quantifiable through the glycation hemoglobin (HbA1c) test. Glycation prompts harm in little veins in the retina, kidney, & fringe nerves. Higher glucose levels rush the cycle. This harm prompts the exemplary diabetic complexities of diabetic retinopathy, nephropathy, & neuropathy & the preventable results of visual impairment, dialysis, & removal, individually.[10] The prothrombotic state in diabetes is contributed by the rising degrees of thickening elements, essential hemostasis changes, & disabled fibrinolysis, & poor quality aggravation. One of the main pathways related with thromboembolism is platelet reactivity, this will speed up platelet collection.[11]

The raised CRP levels have likewise been connected to an expanded gamble of later advancement of diabetes. The unit for estimating CRP in milligrams per liter (mg/L). Results for a standard CRP test are generally given as keeps: Normal: Less than 3 mg/L. High: Equal to or more prominent than 3 mg/L. Kervinen H, Palosuo at al[12,13] in their review CRP levels are higher in individuals with diabetes contrasted & those without diabetes. CRP has both proinflammatory & calming properties. It assumes a part in the acknowledgment & freedom of unfamiliar microbes & harmed cells by restricting to phosphocholine, phospholipids, histone, chromatin, & fibronectin. There are various reasons for a raised C-receptive protein. These incorporate intense & constant circumstances, & these can be irresistible or non-irresistible in etiology. CRP fixations somewhere in the range of 2 & 10 mg/L are considered as metabolic irritation: metabolic pathways that cause arteriosclerosis & type II diabetes mellitus. Barzilay JI, Abraham L[14–18] in their review CRP is a protein made by the liver & sent into the circulatory system. Blood levels might be higher when you have aggravation or a contamination.

Coagulation, the development of a blood coagulation or clots, happens when the proteins of the coagulation overflow are enacted, either by contact with a harmed vein wall & openness to collagen in the tissue space (characteristic pathway) or by initiation of variable VII by tissue enacting factors (extraneous pathway). The two pathways lead to the age of thrombin, a compound that transforms the dissolvable blood protein fibrinogen into fibrin, which totals into proteofibrils. Another thrombin-produced catalyst, factor XIII, then, at that point, cross links the fibrin proteofibrils at the D part site, prompting the arrangement of an insoluble gel which fills in as a platform for blood clump development. Herren T, Stricker H, Haeberli[18–22] review showed D-dimers are not ordinarily present in human blood plasma, with the exception of when the coagulation framework has been actuated in hypercoagulable states like apoplexy.

Smith FB, Lowe GDO[23–25] review showed the raised d-dimer level was identified at the beginning of apoplexy, & the negative level was utilized to prohibit the venous thromboembolism. D-dimer was expanded in diabetic patients under, & when there is a hypercoagulable state, hyperfibrinolysis will happen, in this manner height. Typical D-dimer range is under 0.5 μg/mL feu. A typical D-dimer is under 0.50. A positive D-dimer is 0.50 or more prominent. Since this is a screening test, a positive D-Dimer is a positive screen.

Zafar et al.[26] Study showed HbA1C levels are expanded in uncontrolled Diabetic Mellitus 9.07 which is associated with the investigation of where their HbA1C levels are 10 ±_So, the raised HbA1C, & raised D-dimer are expanded in the uncontrolled Diabetic Mellitus ± 0.3μg/ml which is connected. In the current review, the mean HbA1C esteem in non-diabetes was 8 is 44.7±5.4, .24±1.74. The mean age & HbA1C of controlled diabetic patients were 46.7±6.3. The mean age & HbA1C of uncontrolled diabetic patients & 48.3±9.19 separately. There was no genuinely tremendous distinction in HbA1c values. The HbA1C is viable in checking long haul glycaemic control in patients with Diabetes Mellitus. The entanglements of diabetes not just rely upon the length of Diabetes Mellitus yet in addition on the dependable poor glycaemic control as demonstrated by an elevated degree of HbA1C. Subsequently, there is a prerequisite for a marker for checking long haul diabetic difficulties. In my investigation of non-diabetes patients, the mean & coefficient of CRP are 3.3 ±0.30 mg/L which is inside the ordinary reach (P = 0.05). Controlled diabetes

patients, the mean & coefficient of CRP is 7.7 ±0.32 mg/L (P = 0.04). In Uncontrolled diabetes patients, the mean & coefficient of CRP is 15.4 ±0.35 mg/L (P = 0.03) which is essentially high when contrasted & non-diabetic & controlled diabetic. In my investigation of non-diabetes patients, the mean & coefficient of D-dimer are 0.15 ±0.32 μg/ml which is inside the ordinary reach (P = 0.04). In Controlled diabetes patients, the mean & coefficient of D-dimer is 0.30 ±0.35 μg/ml which is inside the typical reach (P = 0.03). In Uncontrolled diabetes patients, the mean & coefficient of D-dimer is 0.61±0.32 μg/ml which is inside the ordinary reach (P = 0.04) which altogether high when contrasted & non-diabetic & controlled diabetics. Mean show that CRP & D-dimer are expanded in connection with the expansion in HbA1C. The p-esteem is low (<than 0.05), in all situations, connection is genuinely critical, utilizing the determined Pearson coefficient.

5. Conclusion

The present study consisted of 120 patients with type 2 DM in the age of 30-70 years & the complication started in uncontrolled diabetes. The study showed equal distribution of diabetes & its complication in males & females. If DM patients present with high HbA1C the CRP & D-Dimer can serve as a novel marker for the prediction of the risk of diseases. From the perspective of elevated D-dimer, CRP levels are in correlation with increased HbA1C levels. So screening of inflammatory & hemostatic markers must be done in patients with uncontrolled Diabetes Mellitus to decrease the incidence of systemic Inflammation & Cardiovascular diseases

6. Source of Funding

None.

7. Conflict of Interest

None.

References

1. Kaur R, Kaur M, Singh J. Endothelial dysfunction and platelet hyperactivity in type 2 diabetes mellitus: molecular insights and therapeutic strategies. *Cardiovasc Diabetol*. 2018;17(1):121. doi:10.1186/s12933-018-0763-3.
2. Behnam-Rassouli M, Ghayour MB, Ghayour N. Microvascular complications of diabetes. *J Biol Sci*. 2010;10(5):411–23. doi:10.3923/jbs.2010.411.423.
3. Grandl G, Wolfrum C. Hemostasis, endothelial stress, inflammation, and the metabolic syndrome. *Semin Immunopathol*. 2017;40(2):215–24. doi:10.1007/s00281-017-0666-5.
4. Goldberg RJ, Nakagawa T, Johnson RJ, Thurman JM. The role of endothelial cell injury in thrombotic microangiopathy. *Am J Kidney Dis Elsevier Inc*. 2010;56(6):1168–74. doi:10.1053/j.ajkd.2010.06.006.
5. Lagrand WK, Visser CA, Hermens WT, Niessen HW, Verheugt FW, Wolbink GJ, et al. C-reactive protein as a cardiovascular risk factor: more than an epiphenomenon? *Circulation*. 1999;100(1):96–102. doi:10.1161/01.cir.100.1.96.
6. King D, Mainous AG, Buchanan TA, Pearson WS. C-reactive protein and glycemic control in adults with diabetes. *Diabetes Care*. 2003;26(5):1535–9. doi:10.2337/diacare.26.5.1535.
7. Abdelmouttaleb I, Danchin N, Ilardo C, Aimone-Gastin I, Angioï M, Lozniewski A, et al. C-Reactive protein and coronary artery disease: additional evidence of the implication of an inflammatory process in acute coronary syndromes. *Am Heart J*. 1999;137(2):346–51. doi:10.1053/hj.1999.v137.92052.
8. Ijar D, Kanani. Association Of D-Dimer In Type 2 Diabetes Mellitus. *Gmers Medical College*;p. 28–30.
9. Abdelmouttaleb I, Danchin N, Ilardo C, Aimone-Gastin I, Angioi M, Lozniewski A, et al. C-reactive protein & coronary artery disease: additional evidence of the implication of an inflammatory process in acute coronary syndromes. *Am Heart J*. 1999;137:346–351.
10. Ridker PM, Rifai N, Lowenthal SP. Rapid reduction in C-reactive protein with cerivastatin among 785 patients with primary hypercholesterolemia. *Circulation*. 2001;103(9):1191–3. doi:10.1161/01.cir.103.9.1191.
11. Ridker PM, Glynn RJ, Hennekens CH. Hennekens CH: C-reactive protein adds to the predictive value of total & HDL cholesterol in determining the risk of first myocardial infarction. *Circulation*. 1998;97(20):2007–11. doi:10.1161/01.cir.97.20.2007.
12. Kervinen H, Palosuo T, Manninen V, Tenkanen L, Vaarala O, Manttari M, et al. Joint effects of C-reactive protein & other risk factors. *Am Heart J*. 2001;141(4):580–5. doi:10.1067/mhj.2001.113572.
13. Ridker PM, Stampfer MJ, Rifai N. Novel risk factors for systemic atherosclerosis: a comparison of C-reactive protein. *JAMA*. 2001;285(19):2481–5. doi:10.1001/jama.285.19.2481.
14. Pradhan AD, Manson JE, Rifai N, Buring JE. Ridker PM: C-reactive protein, interleukin, and risk of developing type 2 Diabetes Mellitus. *JAMA*. 2001;286(3):327–34. doi:10.1001/jama.286.3.327.
15. Barzilay JI, Abraham L, Heckbert SR, Cushman M, Kuller LH, Resnick HE, et al. The relation of markers of inflammation to the development of glucose disorders in the elderly: the Cardiovascular Health Study. *Diabetes*. 2001;50(10):2384–9. doi:10.2337/diabetes.50.10.2384.
16. Ford ES. Body mass index, diabetes, & C-reactive protein among adults. *Diabetes Care*. 19999;22(12):1971–7. doi:10.2337/diacare.22.12.1971.
17. Grau AJ, Buggle F, Becher H, Werle E, Hacke W. The association of leukocyte count, fibrinogen & C-reactive protein with vascular risk factors. *Thromb Res*. 1996;82(3):245–55. doi:10.1016/0049-3848(96)00071-0.
18. Goldberg RB. Cardiovascular disease in diabetic patients. *Med Clin North Am*. 2000;84(1):81–93. doi:10.1016/s0025-7125(05)70208-x.
19. Wu T, Dorn JP, Donahue RP, Sempos CT, Trevisan M. Associations of serum C-reactive protein with fasting insulin, glucose, and glycosylated hemoglobin: the Third National Health and Nutrition Examination Survey, 1988-1994. *Am J Epidemiol*. 2002;155(1):65–71. doi:10.1093/aje/155.1.65.
20. Miller JL. Blood coagulation & fibrinolysis. In: Henry J, editor. Clinical Diagnosis and Management by Lab Methods. Philadelphia: WB Saunders; 2001. p. 767–72.
21. Al-Zahrani H, Lowe GDO, Douglas JT, Cuschieri R, Pollock JG, Smith WCS, et al. Increased fibrin turnover in peripheral arterial disease: comparison with a population study. *Clin Hemorheol*. 1992;12(6):867–72. doi:10.3233/CH-1992-12609.
22. Fowkes FGR, Lowe GDO, Housley E, Rattray A, Rumley A, Elton RA, et al. Cross-linked fibrin degradation products, progression of peripheral arterial disease, & risk of coronary heart disease. *Lancet*. 1993;342(8863):84–90. doi:10.1016/0140-6736(93)91288-w.
23. Herren T, Stricker H, Haeberli A, Do DD, Straub PW. Fibrin formation and degradation in patients with arteriosclerotic disease. *Circulation*. 1994;90(6):2679–86. doi:10.1161/01.cir.90.6.2679.
24. Ridker PM, Hennekens CH, Cerskus A, Stampfer MJ. Plasma concentrations of cross-linked fibrin degradation product (D-dimer) & the risk of future myocardial infarction. *Circulation*. 1994;90(5):2236–40. doi:10.1161/01.cir.90.5.2236.

25. Smith FB, Lowe GD, Fowkes FG, Rumley A, Rumley AG, Donnan PT, et al. Smoking, hemostatic factors & lipid peroxides in a population case-control study of peripheral arterial disease. *Atherosclerosis*. 1993;102(2):155–62. doi:10.1016/0021-9150(93)90157-p.
26. Koenig W, Rothenbacher D, Hoffmeister A, Griesshammer M, Brenner H. Plasma fibrin D-dimer levels and risk of stable coronary artery disease: results of a large case-control study. *Arterioscler Thromb Vasc Biol*. 2021;21(10):1701–5. doi:10.1161/hq1001.097020.

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