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Threat of Heart Diseases and Physic on Account of Blood Sugar (Glucose) and The Fluid Rest of the Blood Clotting Concentration of an Endogenous methylated amino acid (EMAA) Derived from Arginine

Dr. Pramod Kumar, Dept. of Chemistry GDC Manikpur, Chitrakoot (UP)

Dr. Hemant Baghel , Dept. of Physics GTD GPG College, Karwi, (UP)

Dr. Shiv Jee Malviya, Deputy Secretary UPHESC Prayagraj (UP)

Shiv Kumar Upadhyay ,(M-EB,IJMREAD) IIT Delhi

Abstract

Endogenous methylated amino acid (EMAA) is a nonselective nitric oxide (NO) syntheses' inhibitor associated with heart diseases and metabolic disorders. EMMA plays an important role in the regulation of vascular tone by acting as an endogenous inhibitor of NO synthesis. This study aimed to investigate EMMA with respect to diabetes and its clinical relevance as an independent predictor of heart diseases and physic. The current folder manage learning includes twenty blood sugar (glucose) suffering people chosen indiscriminately. The fluid rest of the blood clotting concentration of an endogenous methylated amino acid (EMAA) derived from Arginine analyzed by using enzyme immunoassay for the quantitative determination of endogenous EMAA, and blood serum nitric oxide was determined.

Keywords - EMAA, Arginine, Clotting etc.

Introduction-

Heart diseases (HD) is the major cause of morbidity and mortality in patients with Blood Sugar (BS). Diabetes is at high risk for several cardiovascular disorders: coronary heart disease, stroke, peripheral arterial disease, cardiomyopathy, and congestive heart failure . Endothelial dysfunction is a common feature in diabetic patients and may contribute to diabetes-induced endothelial dysfunction include the production of prostanoid vasoconstrictors and the increased oxidative degradation of Nitric oxide.

Smaller quantity of NO increases vascular resistance and promotes atherogenesis Nitric oxide is a very active but short lived, a molecule that is released into the circulation from endothe- lial cells. Hence, endothelial dysfunction due to reduced Nitric oxide availability is an early step in the course of atherosclerotic vascular disease.Nitric oxide is synthesized by stereospecific oxidation of the terminal guanidino nitrogen of the amino acid, L-arginine, by the action of a family of NOS. The synthesis of NO can be blocked, however, by inhibition of the NOS active site with guanidino-substituted analogs of L-arginine, such as EMAA.

EMAA is a naturally occurring amino acid that has the interesting property of competitively inhibiting the activity of nitric oxide synthase (NOS).EMAA is produced by methylation of arginine residues in intracellular proteins via protein arginine N-methyltransferases (PRMT).After hydrolyzation

The primary route of EMAA clearance, however, is by enzymatic degradation. Dimethylamine dimethylaminohy- drolase (DDAH) converts EMAA to citrulline and dimethy lamine. By regulating the plasma and tissue concentrations of ADMA, NOS activity is protected by DDAH .Recently, it has been demonstrated that plasma levels of EMAA are elevated in patients with Blood Sugar. These findings suggest that the elevated EMAA in diabetes could contribute to accelerated atherosclerosis in this population. Further, as EMAA is mainly metabolized by dimethylarginine dimethylaminohydrolase (DDAH), it is conceivable that the inhibition of EMAA via upregulation of EMAA may be a novel therapeutic target for the prevention of HD in patients with diabetes. EMAA is elevated to a level that can significantly inhibit NOS activity in individuals with hypercholesterolemia, hypertension, and hyperhomocysteinemia ,tobacco exposure, and hyperglycemia in each of these conditions; the elevation in EMAA level is a result of oxidative stress. Further oxidative stress impairs the ability of Endogenous methylated amino acid EMAA to metabolize EMAA Hyperglycemia can elevate intracellular oxidative stress through multiple mechanisms.This makes NO synthases a superoxide ($O2^-$) radical-producing enzyme .The endothelium-derived relaxation factor, nitric oxide, provides a unifying mechanism for the action of many cardio- vascular disease risk factors. Nitric oxide cannot be measured directly, but the inhibitor of its formation and EMAA can be measured. The function of EMAA in modulating nitric oxide production has a significant impact on heart diseases and physic. Endogenous methylated amino acid (EMAA) is a nonselective nitric oxide (NO) synthase inhibitor associated with cardiovascular and metabolic disorders. This study aimed to investigate EMAA with respect to diabetes and its clinical relevance as an independent predictor of EMAA.

Materials and Methods-

The present folder manage learning includes twenty patients, selected randomly. into two groups. Group I, patients presenting HD (BS = 80); Group II, patients with type II HD (BS = 80). All patients selected underwent medical examination by a physician. Inclusion criteria and patients clinically diagnosed having angiographically proven HD including or excuding type II BS. Prevalence of diabetes was assessed either by fasting blood glucose level ≥127.0 mg/DL or HbA1C levels more than 7%. People for study matched seventy eighty subjects as be in command of, and group II was randomly selected from patients attending diabetic Outpatient department(Opd). Controls have a negative history of HD, Type II BS and had a normal resting Electrocardiogram (Ecg). Patients in group I and II were recruited from inpatient and outpatients departments of the hospital. The diagnosis of HD was made on the basis of the clinical history (typical angina, history of myocardial infarction) and lead stan- dard ECG before subjecting them to coronary angiography. The presence of any stenosis > Twenty Nine % according to coronary angiography by visual assessment of coronary artery was included in the trial consideration.

ADMA was analysed by enzyme linked immunoassay for the quantitative determination of endogenous asymmetric dimethylarginine in serum, manufactured by DLD diagnos- tics GMBH, which is based on principal, the competitive EMAA-ELISA uses the microtiter plate format. EMAA is bound to the solid phase of the microtiter plate.EMAA in the samples is acetylated and competes with solid phase bound EMAA for a fixed number of rabbit anti-EMAA antiserum binding stanzas.

Experimental and Discussion -

The antibody bounded to the solid phase EMAA is detected by the anti-rabbit/peroxidase. The substrate TMB/peroxidase reaction is monitored at 448 nm. The amount of antibody bound to the solid phase EMAA is inversely proportional to the EMAA concentration of the sample. Statistical analysis was carried by student's test and chisquare test. Results were expressed as mean ± HD, feasibility

TABLe A : Fundamental data of the learning of patients

Era (years)	45.29 ± 09.21	55.21 ± 05.73	<0.10
Ancestors record	11	22	—
Tobacco Users	03	19	—
Body M Index	27 (>23)	29 (>23)	—
Blood Stress systolic(mm/Hg	118.00 ± 2.98	123.00 ± 5.23	<0.0.98
Blood Stress diastolic(mm/H	78.24 ± 5.53	83.54 ± 6.54	<0..0.18
Entire cholesterol	160.78 ± 24.98	278.26 ± 26	<0.0.98
Highdens.lip. (Mg/dl)	43.18 ± 8.35	27.16 ± 8.89	.96 <0.0
Lowdens.lip. (Mg/dL)	99.10 ± 25.78	148.56 ± 29.65	<0.04
VLowDenslip. (Mg/dl)	32.98 ± 12.58	42.37 ± 16.28	<0.03

Patients who had no Blood Sugar and Heart Diseases were grouped as control; diabetes with HD and nondiabetes with HD are categorized as diseased group and others are categorized as nondiseased group.Baseline characteristics of the participants are presented in Table A. The overall mean age for control was 45.0 years, and diseased group was 53.0 years; 70% were men. Diabetes with HD had higher systolic blood stress and hyper lipidemia than normal healthy subjects. Table B shows that type II diabetes with HD patients have significantly higher EMAA concentration than normal healthy subjects and other study groups HD <0.001. NO concentration was lower lowest 23.50±, In a multiple logistic regression analysis adjusting for hypertension, hypercholesterolemia, low HDL cholesterol and diabetes or fasting glucose, EMAA remained a strong and independent predictor for the presence of HD. Elevated No level was a strong predictor and significantly associated with occurrence of HD. Increased EMAA level was found to be another strong predictor and associated significantly with heart diseasesThe univariate logistic regression analysis for risk factors versus HD (as a dependent variable) was done to assess relative risk of development of HD with each risk factor in the entire study population that includes diseased and non diseased subjects. Age, family history, obesity, and SBS and DBS are highly significant and associated with HD pathogenesis.

Conclusion-

Associations between increased levels of EMAA and many cardiovascular risk factors such as age, hypertension, dia- betes, insulin resistance, hypercholesterolemia, hypertriglyc- eridemia, and hyperhomocysteinemia have been documented. Evidence for a causal relationship between increased ADMA levels and endothelial vasodilator dysfuntion has been demonstrated in many of these conditions. Hyperglycaemia is associated with endothelial

dysfunction both and therefore, endothelial dysfunction is an early feature in the development of vascular levels are capable of inhibiting activity in cultured endothelial cells . Clinical investigations in patients also indicate that ADMA is directly related to blood glucose levels . As demonstrated in a recent study, strict glycemic control may exert antiathero- genic effects by reducing EMAA levels in patients with typeII BS . Consistent with these studies, the present study provide evidence that elevated serum EMAA and reduced NO independently associated with cardiovascular risk in diabetes patients with coronary artery disease.EMAA might act as pathophysiologically relevant factor for diabetes associated complications.Nevertheless, hyperglycemia remains a major cause for both increased EMAA and the development of diabetic complications which makes the interpretation of the data more complex.

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