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Bilirubin, Uric acid level changes in Metabolic syndrome and Diabetes Mellitus: A Comparative study

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Abstract
Metabolic syndrome (MetS) represents a cluster of several risk factors that includes dyslipidemia, obesity, increased blood pressure and hyperglycemia, proinflammatory state, prothrombiotic state. Diabetes Mellitus (DM) is chronic hyperglycemia which includes most common complications i.e. atherosclerosis, nerve damage, renal damage, retinopathy. Our aim is to evaluate Bilirubin and Uric acid in patients with MetS and DM controls. The study included 50 MetS patients, 50 diabetic patients and 50 Controls 5 ml of venous blood samples were collected from patients and controls. All the samples were collected overnight fasting of 12 hrs. Collected samples centrifuged under 2000 rpm for 20 min and after centrifugation of samples used for the determination of Cholesterol, Bilirubin and Uric acid. Blood glucose, Uric acid, Bilirubin significantly increased in diabetic patients comparing with Mets and Controls.
Keywords: *Chronic hyperglycemia, Obesity, Atherosclerosis, Antioxidant acitivity.*

Introduction

Metabolic syndrome (MetS) first depicted by Reaven in 1988. It speaks to a group of a few risk factors that incorporates dyslipidimia, weight, expanded pulse and hyperglycemia[1]. MetS is an accumulation of cardiometabolic risk factors that incorporates dyslipidimia, hypertension, insulin resistance, Proinflammatory state, Prothrombiotic state. Obesity in charge of rising prevalence of metabolic syndrome[2]. Obesity is the central and casual components of MetS particularly abdominal obesity, physical inertia and atherogenic diet. MetS is highly significant for type 2 DM and CVD[3].75-80% of adults diabetic patients death caused by CVD incorporates increased level of fasting TG and depletion of (HDL-C) cholesterol levels, abnormal state of VLDL levels, unusual postprandial lipemia, apolipo protein B and HbA1C[4]. MetS have a five –fold higher risk of type 2 DM and a two to

three –fold higher risk factor of atherosclerotic CVD[5].

The etiology of metabolic syndrome patients with CVD involves coronary atherosclerotic diseases, artery hypertension, left ventricular hypertrophy, diastolic dysfunction, endothelial dysfunction, coronary macro-vascular disease and autoimmune dysfunction diseases occurred[6]. MetS individuals seemingly are susceptible to other conditions notably polycystic ovary syndrome, fatty liver, cholesterol gallstones, asthma, sleep disturbance and some forms of cancer[7].

Abdominal obesity is strongly associated with the MetS. It present clinically as increased waist circumference (Men->40 inch-women>35inch) Obesity strongly associated with increased blood pressure in insulin–resistant persons. Increased CRP level, clinically observed in proinflammatory persons with MetS[8]. Obesity is one of the causes for elevation of CRP levels due to excess adipose tissues release, inflammatory cytokines that may elicit higher CRP levels. Increased Plasma plasminogen Activator Inhibitor (PAI) and fibrinogen associated with the metabolic syndrome[9].

Diabetes mellitus (DM) is a group of metabolic issue which incorporates hyperglycemia because of deformities in insulin emission, insulin activity (or) both. Chronic hyperglycemia of diabetics is related with long term complications, dysfunction, retinopathy, nephropathy and neuropathy[10]. Diabetic patients are categorised into type I and Type II. Type I (insulin dependent) Type II (insulin independent) as a result of autosomal insusceptible demolition of β cells of pancreas with subsequent insulin lack. 90-95% diabetes causes represents type II. The vast majority of the type II diabetic patients are obese and weight itself causes some level of insulin resistance[11].

If diabetes isn't diagnosed early and managed properly, patients are at upgraded danger of microvascular and macrovascular inconveniences. Uricacid and bilirubin act as a non- enzymatic antioxidant biomarkers as they prevent free radical reactions. Provides the primary extracellular defence against the oxidative stress sequestering the transition metal ions by chelating plasma [12-13]. The present study focused on Uric acid, Bilirubin levels in DM patients and Metabolic syndrome.

Objective of the Study

Our aim is assess the Uric acid, Bilirubin levels which consists antioxidant capacity in patients with MetS and DM.

Materials and Methods

The study was conducted at SLIMS, Puducherry. The study included 50 diabetic patients, 50 MetS patients and 50 Controls. 5 ml of venous blood samples were collected from patients and controls. All the samples, and this separated serum were collected overnight fasting of 12 hrs. Collected samples centrifuged under 2000 rpm for 20 min and after centrifugation of samples used for the determination of FBS, Cholesterol, Bilirubin and Uric acid and were estimated by using enzymatic kits on Siemens TM auto analyzer

Results

Table. No.1: The Mean ± SD values of UA, Bilirubin, FBS, Cholesterol in DM, MetS patients and Controls

S.No.	Parameter	MetS	DM	Controls	P value
1	UA	6.5± 0.42	8.2 ± 0.06	5.22± 0.23	p<0.001
2	Bilirubin	1.0± 0.04	1.2 ± 0.07	0.07 ± 0.02	p<0.001
3	FBS	156.55± 4.82	168.55± 4.92	106.12± 1.68	p<0.001
4	Cholesterol	245± 30.51	263± 32.62	188.5 ±27.3	p<0.001

Discussion

Uric acid is the end product of purine metabolism serum uric acid is a diprotic acid produced by the enzyme xanthine oxidase from xanthine and hypoxanthine. Xanthine oxidase uses molecular oxygen as electron acceptor and

generates superoxide anion and other reactive oxygen oxygen species(ROS)[14]. Uric acid is chain breaking antioxidant, which is of great importance in plasma. Increased serum uric acid results gout, lesch nyhan's syndrome, and uric acid stones hyper ureacemic also found to be associated with insulin resistance and components of the Mets[15-17].

In olden days bilirubin was believed only waste product of Heme catabolic pathway, a potentially toxic compound and have various biological functions. Recent studies showed mildly increased serum bilirubin levels are strongly associated with low prevalence of oxidative stress –mediated diseases and it's shows antioxidant property. Total bilirubin (TB) concentration was inversly related with coronary artery diseases, hyper tension and Mets[18-20]. Bilirubin shows negative reaction ship with DM and Mets, abdominal obesity.

Uric acid is the final product of purine metabolism serum uric acid is a diprotic acid delivered by the chemical xanthine oxidase from xanthine and hypoxanthine. Xanthine oxidase utilizes sub-atomic oxygen as electron acceptor and produces superoxide anion and other responsive oxygen species (ROS)[21]. Uric acid is chain breaking cell reinforcement, which is of incredible significance in plasma. Expanded serum uric acid outcomes gout, lesch nyhan's disorder, and uric corrosive stones hyper ureacemic likewise observed to be related with insulin obstruction and segments of the Mets[22-24].

In long time past day's bilirubin was accepted just waste result of Heme catabolic pathway, a conceivably poisonous compound and have different organic capacities. Recent studies demonstrated somewhat expanded serum bilirubin levels are emphatically connected with low commonness of oxidative stress – mediated diseases and it's shows antioxidant property [24]. Add up to bilirubin (Total Bilirubin) fixation was inversely related with coronary vein diseases, hyper tension and Mets. Some studies shown low levels of birlirubin, Uric acid

Conclusion

Uric acid as clinical effective levels of drug treatments along with aimed to reduce cardiovascular risk in diabetic patients and MetS should be considered. Bilirubin indicates negative response dispatch with DM and Mets, stomach obesity.

Antioxidants are important for the prevention of MetS and its complications, so more research is needed to differentiate the effects of major serum antioxidant on MetS and complications.

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