



Correlation of Lipid Profiles, Electrolytes and Antioxidant Status In Type 2 Diabetic Patients With Hypertension In Sikkimese Population: A Case Controlled Study.

Chhunthang Thanpari M Sc^{*1}, Roshan Takhelmayum M Sc², Robby Kumar M.Phil³

1. Lecturer, Department of Biochemistry, Nepalgunj Medical College, Chisapani, Banke, Nepal
2. Lecturer, Department of Biochemistry, College of Medical Sciences, Bharatpur, Nepal
3. Lecturer, Department of Biochemistry, SSR Medical College, Mauritius

Abstract

The purpose of the present study was to analyze antioxidant status with Lipid Profile & electrolytes in diabetic patients with hypertension among Sikkimese population in Gangtok, Sikkim, India. Type 2 diabetic patients with hypertension were selected from the subjects attending OPD Central Referral Hospital, Gangtok, Sikkim, India for medical checkup. The blood from the patients was taken by normal venipuncture and all biochemical parameters were done using Kit methods. Along with increased Blood Sugar level the lipid profile was also altered with a significant increase in serum electrolytes. Erythrocyte GSH, SOD and TBARS were estimated and compared with healthy control subjects. A significant decrease of erythrocyte GSH and TBARS and Increase in SOD was found

Key Words

Diabetes Mellitus, Hypertension, Lipid Profile, Electrolytes, Antioxidants

*Corresponding Author: Lecturer, Department of Biochemistry, Nepalgunj Medical College, Chisapani, Banke, Nepal. Email: cthanpari@yahoo.co.in

Received: Feb 22, 2012 Accepted: May 05, 2012.

Published: May 20, 2012

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:

Diabetes mellitus (DM) is one of the most common metabolic disorders characterized by hyperglycemia due to defects in insulin secretion, insulin action or

both. Its global prevalence is increasing every year and expected to rise from 171 million cases in 2000 to 366 million cases in 2030. Likewise, urban population in developing countries is said to double between 2000 and 2030 [1]. There are approximately 30 million diabetic in India at present and the number is expected to rise to 80.9 million by 2032. India leads the world with the largest number of diabetics [2]. Diabetes and hypertension are deadly duo. The pathogenesis of hypertension is thought to be multifactorial: Peripheral insulin resistance and hyperinsulinemia causing impairment of insulin mediated renal sodium reabsorption. Hyperinsulinemia leads to sympathetic activation causing vascular over activity. Insulin resistant state causing increased erythrocyte Na^+/Li^+ counter transport, leading in turn to hypertension [3]. Strong

and complex relationship exist between obesity, glucose tolerance and hypertension. Every 3 kg increase in weight results in 1mmHg increase in mean BP. Weight gain and central adiposity have positive correlation for the excess prevalence of hypertension in people with diabetes. The systolic BP is a significance predictor of diabetes regardless of age or obesity. Once people with diabetes develop incipient or over nephropathy there is marked increase in exchangeable sodium and fluid retention, often manifesting as edema [4]. There are accumulating evidences that changes in lipid metabolism in diabetes has specific role in pathogenesis and complication of the disease. Various studies have addressed relation between lipid profile and diabetes with sometimes conflicting results. American Diabetes Association (ADA) showed that lipid level in diabetes mellitus has effect on cardiovascular disease [5]. Studies have revealed that dyslipidaemia in diabetes mellitus with hypertension is higher in African Americans than Whites or Hispanics [6]. A negative relationship between plasma concentration of insulin and its resistance with HDL indicating influence of insulin on HDL levels in plasma. It was concluded that error in lipid profile is strongly associated with diabetes [7]. Diabetes is usually accompanied by increased free radical production or impaired antioxidant defences. Increased free radicals causes damage to cellular proteins, membrane lipids and nucleic acid and finally cell death [10]. Some studies suggest enhanced free radicals due to elevated glucose concentrations. While others on reduced antioxidant status in diabetes [15,16]. In Sikkim such survey has not been conducted and therefore prevalence of diabetes mellitus with hypertension and Antioxidant status is unknown. This prompted me to study and determine Lipid profile, Antioxidant status & electrolytes level in diabetic patients in Gangtok, Sikkim. It is hoped that the findings of this study would be useful in formulating national strategies for diabetes and hypertension control and conducting nationwide prevalence survey in future.

MATERIALS AND METHODS:

1. Sample collection

A total of 100 diabetic patients with hypertension from Gangtok, Sikkim who visited Outpatient

department (OPD) of Central Referral Hospital, Gangtok, Sikkim, India from October, 2007 to April, 2008 were included in this study. All these patients were referred to clinical Biochemistry laboratory for their blood glucose, lipid profile, electrolytes, erythrocytes GSH, SOD & TBARS to be estimated. Approximately 5 ml of venous blood was collected from each patient in a plain glass vial and processed for the test.

2. Estimation of serum glucose, lipid profile and Antioxidants and Lipid peroxidation

Blood glucose estimation was carried out by glucose oxidase(GOD) / peroxidase (POD) method, lipid profile estimation by CHOD-POD method and electrolytes level by using Kit supplied by Beacon Diagnostic Ltd. Tests were performed according to manufacturer's protocol using a semi-autoanalyzer. The concentrations of serum glucose and lipid profile were expressed in milligram/ deciliter (mg/dl) and electrolytes in mmol/L. The Red Blood Cells left were washed in cold saline and diluted hemolysates were prepared for estimation of GSH, SOD, and TBARS. Erythrocyte GSH was estimated by method of Beutler E et.al[11]. Erythrocyte SOD was estimated by method of Beauchamp and Fridovic[12]. Along with this erythrocytic lipid peroxidation products were measured by assaying TBARS[13].

RESULTS:

Blood Sugar along with Serum Electrolytes and Lipid profile was increase in diabetic subjects when compared with control. The antioxidant status is done by measuring TBARS and GSH which was normal for control but TBARS and GSH were decreased in diabetic study subjects which were statistically significant. Enzyme SOD was slightly increased which is not statically significant (Table 1).

DISCUSSION:

It has been observed that among the diabetic patients serum level of TG is elevated most. This means that most of the patients have diabetic dyslipidemia. Dyslipidemia has been strongly associated with coronary artery disease (CAD). Elevated serum total cholesterol, TG, LDL cholesterol and reduced HDL cholesterol concentrations have been the markers of CAD which occur most commonly in type2 diabetic subjects [8, 9].

Parameters	Control	Type2 Diabetic Subjects with Hypertension
Blood Glucose (mg/dL)	84±12	244±28
Serum-Na ⁺ (mmol/L)	141±5	150±9
Serum-K ⁺ (mmol/L)	5±1	5.4±1.2
TC (mg/dL)	167±22	290±38
TG (mg/dL)	110±17	210±31
HDL (mg/dL)	48±8	37±5
LDL (mg/dL)	118±11	138±26
SOD(U/gHb)	860±109	1060±421
GSH (mg/gHb)	17.6±4	15.6±2 ^a
TBARS (nM/gHb)	4.8±2	3.4±1.5 ^b

^ap=0.0011 ^bp=0.0005

Table 1. Comparison of different serum parameters in control and diabetic subjects.

Diabetic patients with elevated levels lipid and electrolyte parameters show that the patients suffered from diabetic nephropathy, this means that in these patients their blood glucose level and blood pressure are poorly controlled. These observations suggest that diabetes is a sodium retaining state with the magnitude of sodium retention increasing with increasing severity of nephropathy [4]. An increased production of SOD has been found in erythrocyte membrane of diabetic with hypertension patients together with depressed erythrocyte content of GSH & TBARS. Some studies suggested that hyperglycemia results in the generation of reactive oxygen species which leads to increased oxidative

stress in a variety of tissues and impaired endogenous antioxidant compensatory response, which will lead to cellular damage and ultimately contribute to development of diabetic complications.. [14,17]

There are no metabolic alterations in those patients whose sugar level only is increased. Since it is mainly characterized by hyperglycemia, if not controlled or treated that may lead to several long term complications. The etiology of serum lipids in diabetes is multifactorial. However, insulin deficiency with end organ resistance and impaired lipid metabolism are implicated in majority of the case.

ACKNOWLEDGEMENT:

We are thankful to Mr. Prakash Mothay and Mr. Jai Karki, laboratory technicians who had help us in running the tests and in collection of the data during the course of the study. We are also thankful to our respected faculty and friends of Sikkim Manipal Institute of Medical Sciences, Gangtok, Sikkim for their support.

References:

1. Wild S, Bchir MB, Roglic P, et al. Global prevalence of diabetes. Estimates for the year 2000 and projections for 2030. *Diabetes Care* 2004; 27: 1047-53.
2. King H, Aubert RE, Herman WH. Global burden of diabetes, 1995-2025. Prevalence, numerical estimates and projections. *Diabetes Care* 1998; 2: 1414-31.
3. Deepa R, Shanthirani CS, Premlatha G, et al. Prevalence of insulin resistance in a selected South Indian Population-The Chennai Urban Population Study (CUPS No.7). *Indian .J. Med. Res.* 2002; 115: 118-27.
4. Gres TW, Nieto J, Shahar E, et al. For the Atherosclerosis Risk in Community (ARIC) study. Hypertension and antihypertensive therapy as risk factors for type 2 diabetes mellitus. *N. Engl. J. Med.* 2000; 342: 905-12.
5. Management of dyslipidaemia in adults with diabetes. Diabetes association: clinical practice recommendation 1998. *Diabetes care.* 1998; 21 (suppl 1) s36-s39.
6. Shama M.D., Pavlik V.N. Diabetes, Obesity & Metabolism. February 2001; 3(1): 41-45.
7. Cowei C, Howard B V, Haris M I: Serum

- lipoproteins in African Americans and Whites with non insulin dependent diabetes in the US population. *Circulation.* 1994; 90: 1185-93.
8. Kulkarni KR, Markovitz JH, Nanda NC, et al. Increased Prevalence of smaller and dense LDL particles in Asians Indians. *Arterioscler. Thromb. Vasc. Biol.* 1999; 19: 2749-55.
 9. Mohan V, Deepa R, Shanthirani S. and Premlatha G. Prevalence of coronary artery disease and its relationship to lipids in a selected population in South India- The Chennai Urban Population Study (CUPS No.5). *J. Am. Coll. Cardiol.* 2001; 38: 682-87.
 10. Kumar R ,Ahmed S. Antioxidant and lipid peroxidation level in type2 diabetes mellitus. *Int J Cur Bio Med Sci.* 2011; 1(4): 147 – 148
 11. Beutler E, Duron O, Kelly BM. Impaired method for the determination of blood Glutathione. *J lab Clin Med.* 1963;61:882-88.
 12. Beauchamp C, Fridovich I. Superoxide dismutase: Improved assay and as assay applicable to acrylamide gel. *Anal Biochem.* 1971;44:276-87.
 13. Jain SK, Mcvie R, Duett J, Herbst JJ. Erythrocyte membrane lipid peroxidation and glycosylated hemoglobin in diabetes. *Diabetes.* 1989;38:1539-42.
 14. Kumar R, Kumar AN, Ahmed S. Changes in Erythrocyte Membrane in Type-2 Diabetes Mellitus with and without Dyslipidemia. *J Diabetes Metab.* 2011; 2:141
 15. Maxwell SR, Thomson H, Sandler D, et al. Antioxidant Status in Patients with Uncomplicated Diabetes Mellitus. *Eur J Clin Invest* 1997; 27:484-90.
 16. Rocic B, Vucic M, Knezevic-Cuca J, et al. Total Plasma Antioxidants in First Degree Relatives of Patients with Insulin Dependents. *Exp Clin Endocrinol Diabetes* 1997;105:213-7.
 17. Evans JL, Goldfine ID, Maddux BA, et al. Oxidative Stress and Stress Activated Signaling Pathways: A Unifying Hypothesis of Type 2 Diabetes. *Endocrine Reviews* 2002; 23:599-622