

## A REVIEW ON CEROPLASMIN ACTIVITY IN PLASMA OF DIABETIC PATIENTS

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### ABSTRACT

Type II Diabetes Mellitus (Type II DM) is a metabolic disorder characterized by glucotoxicity and lipotoxicity. Markers of glycemic control are HbA1c, fasting blood glucose and postprandial blood glucose. There are altered lipid parameters in Type II DM which possess significant cardiovascular risk to the patient. In the present study we are investigating about ceroplasmin activity on diabetic patients.

**Keywords:** Vincristine, Cancer, Ceruplasmin, Chemotherapy.

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No: of References: 12

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## INTRODUCTION

Type II diabetes mellitus (Type II DM) is a chronic progressive disease primarily characterized by hyperglycaemia and dyslipidemia that later leads to cardiovascular complications. Other complications affecting kidney, eye and nervous system can also occur. Hyperglycaemia impairs glucose metabolic pathways and increases auto-oxidative glycosylation and free radical production. Deranged lipid status favours free radical-induced lipid peroxidation and leads to deposition of oxidized low-density lipoprotein (LDL) cholesterol resulting in atherosclerosis of blood vessels.<sup>1</sup> Hyperglycaemia in long run causes toxic effects on macro as well as microvascular structure and affects proper functioning of organs. The present study is designed to know the status of the serum ceruloplasmin in the type II diabetes mellitus and to study the correlation of the ceruloplasmin with glycemic status and lipid profile. This study tries to find the association of ceruloplasmin with lipid.

## DISCUSSION

Ceruloplasmin was found to be higher in diabetic group than non-diabetic group. Similar findings have found in various studies conducted in by Jung Lee M et al 2015 in Korea and Nasif ZN et al 2010 in Iraq.<sup>8</sup> the cut off value of serum ceruloplasmin for distinguishing diabetic and non-diabetic group was in our study 46.5 mg/dL which has sensitivity of 87.5% and specificity of 62%. Similarly, serum glucose level was high in participants with high serum ceruloplasmin level. Overall, considering both groups; hyperglycaemia correlates with serum ceruloplasmin.

Surprisingly hyperglycaemia does not correlate with serum ceruloplasmin in diabetic group. The probable explanation may be that ceruloplasmin increases in diabetic group but does not fluctuate considerably in the diabetic range. Similarly, considering both groups ceruloplasmin correlates with age, fasting glucose, post prandial glucose, glycated haemoglobin, triglycerides and TG/HDL-C ratio. Thus, it implies that serum triglycerides and TG/HDL-C ratio which is substitute marker of insulin resistance could be reflected by ceruloplasmin. Ceruloplasmin could be used as substitute to mark the insulin resistance. In the diabetic group HbA1c is correlated with ceruloplasmin. Thus, glycemic index correlates with inflammatory status, as ceruloplasmin indicates the inflammatory status of the body. Further, LDL-C, TC/HDL-C ratio and LDL-C/HDL-C ratio correlates with ceruloplasmin. The importance of this correlation is highlighted by the fact that LDL-C is one of the key factors for development of atherosclerosis. Epidemiological data suggests that serum ceruloplasmin may be an important risk factor predicting myocardial infarction and cardiovascular disease. This is because ceruloplasmin is a potent catalyst of LDL oxidation. Several studies have observed that LDL-C/HDL-C ratio predicts carotid intima-media thickness progression and it predicts better than other lipid ratios. Similarly, TC/HDL-C ratio variation is associated with more substantial alterations in metabolic indices predictive of ischemic heart disease risk and related to the insulin resistance syndrome. TC/HDL-C ratio is the substitute for metabolic index and its correlation with ceruloplasmin verifies the importance of serum

ceruloplasmin in monitoring total cholesterol and manage accordingly TC, TG and TG/HDL-C correlation with ceruloplasmin is seen in female diabetic group. For male in diabetic group HbA1C and LDL-C/HDL-C ratio correlated significantly with ceruloplasmin. The probable reason may be due to that the cut off for TG/HDL-C ratio is lower for female. The clinical implication may be that, the female population should get benefit by controlling their lipid level. We could assume that the response of inflammatory status differs between male and female in the diabetic group. It also indicates our approach and priorities during treatment of hyperlipidemia in diabetic population.

## REFERENCES

- Bambolkar S and Sainani GS.** Evaluation of oxidative stress in diabetics with or without vascular complications. The Journal of the Association of Physicians of India 1995 Jan;43(1):10-12.
- Robertson RP.** Chronic oxidative stress as a central mechanism for glucose toxicity in pancreatic islet beta cells in diabetes. Journal of Biological Chemistry 2004; 279(41):42351-42354.
- Zozulinska D and Wierusz-Wysocka B.** Type 2 diabetes mellitus as inflammatory disease. Diabetes Research and Clinical Practice 2006;74(2): S12-S16.
- Ryu TY, Park J and Scherer PE.** Hyperglycaemia as a risk factor for cancer progression. Diabetes & metabolism journal 2014;38(5):330-336.
- Lee MJ, Jung CH, Kang YM, Jang JE, Leem J, Park JY,** et al. Serum Ceruloplasmin Level as a Predictor for the Progression of Diabetic Nephropathy in Korean Men with Type 2 Diabetes Mellitus. Diabetes & metabolism journal 2015;39(3):230-239.
- Shukla N, Maher J, Masters J, Angelini GD and Jeremy JY.** Does oxidative stress change ceruloplasmin from a protective to a vasculopathic factor? Atherosclerosis 2006;187(2):238-250.
- Varela AS, Saez JB and Senra DQ.** Serum ceruloplasmin as a diagnostic marker of cancer. Cancer letters 1997; 121(2):139-145.
- Nasif ZN.** Evaluation of Ceruloplasmin Oxidase Activity and C - reactive protein in the Sera of Patients with Diabetes Mellitus. National Journal of Chemistry 2010; 37: 175-185.
- Enomoto M, Adachi H, Hirai Y, Fukami A, Satoh A, Otsuka M,** et al. LDL-C/HDL-C Ratio Predicts Carotid Intima-Media Thickness Progression Better Than HDL-C or LDL-C Alone. J Lipids 2011;2011(1):1-6.
- Fox PL, Mazumder B, Ehrenwald E and Mukhopadhyay CK.** Ceruloplasmin and cardiovascular disease. Free Radical Biology and Medicine 2000;28(12):1735-1744.
- Lemieux I, Lamarche B, Couillard C, Pascot A, Cantin B, Bergeron J,** et al. Total cholesterol/HDL cholesterol ratio vs LDL cholesterol/HDL cholesterol ratio as indices of ischemic heart disease risk in men: the Quebec Cardiovascular Study. Archives of internal medicine 2001; 161(22):2685-2692.
- Banha J, Marques L, Oliveira R, de Fátima Martins M, Paixão E,** et al. Ceruloplasmin expression by human peripheral blood lymphocytes: a new link between

immunity and iron metabolism. Free  
Radical Biology and Medicine 2008;  
44(3):483-492.

