https://ijrps.com

ISSN: 0975-7538
Research Article

Evaluation of homocysteine levels in metabolic syndrome and its relationship between homocysteine and cardiovascular events

Sai Ravi Kiran B[1], Mohanalakshmi T[2], Srikumar R[3], Prabhakar Reddy E*[1]

[1]Department of Biochemistry, Sri Lakshmi Narayana Institute of Medical Sciences, Puducherry-605502, India
[2]Department of Microbiology, Sri Lakshmi Narayana Institute of Medical Sciences, Puducherry-605502, India
[3]Centre for Research, Sri Lakshmi Narayana Institute of Medical Sciences, Puducherry-605502, India

ABSTRACT

Metabolic syndrome is portrayed by a group of cardiovascular (CV) causing factors including raised triglycerides, lessened HDL cholesterol, central obesity, hypertension, expanded fasting glucose and hyper insulinemia. The metabolic syndrome is related with expanded cardiovascular risk. Expanded homocysteine may leads from insulin resistance, and may demonstrate cardio vascular scatters or be associated with atherogenesis. Our point is asses the evaluate the Hcy in Metabolic syndrome. The study was conducted at SLIMS, Puducherry. The study included 200 MetS patients and 200 Controls. Homocysteine (Hcy) estimated by ELISA method. To sum up, a significant ($p<0.001$) increase in Homocysteine was observed in MetS patients when compared to controls. However, further studies are required to determine whether genetic, nutritional deficiencies, or diseases related to Hcy metabolism account for hyperhomocysteinemia observed in patients of type 2 DM with and without cardiovascular complications.

Keywords: Homocysteine; Metabolic syndrome; cardiovascular disorders; Hyperinsulinemia.

INTRODUCTION

The metabolic syndrome (MetS) is considered as the most essential general wellbeing risk of the 21st century, influencing between 10 & 15% of grown-up populaces around the world. This disorder is portrayed by a bunch of cardiovascular (CV) chance components including abdominal obesity, raised triglycerides, lessened HDL cholesterol, hypertension, expanded fasting glucose and hyper insulinemia (Taskinen MR.2007).

MetS is a state of chronic low grade inflammation as a consequence of complex interplay between genetic and environmental factors which induce the several factors such as insulin resistance, visceral adiposity, atherogenic dyslipidemia, endothelial dysfunction, genetic susceptibility, elevated blood pressure, hypercoagulable state and chronic stress (H, Abel ED.2008.Nicolson GL.2007.Grundy SM, Brewer HB, et al., 2004).

Metabolic Syndrome is otherwise called Insulin protection disorder. This disorder is a group of scatters like Insulin resistance, impaired glucose intolerance and hyper insulinemia. Insulin protection has all the earmarks of being the essential mediator person of metabolic syndrome.

Homocysteine (Hcy) is a sulfur containing amino acid in the body, which is created by transformation of methionine. The normal value ranges from 5-15 µmol/L. (Hajer GR, van der Graaf Y,et al.,2007. LinksHajer GR, et al., 2007).

As of late, mellow rises of plasma Hcy have been recognized as an autonomous hazard factor for early atherosclerotic vascular disorder and thromboembolic disease (Hajer GR, van der Graaf Y,et al., 2007. Links Hajer GR, et al., 2007).

Objective of the Study

Our aim is to evaluate homocysteine levels in metabolic syndrome and to distinguish the connection amongst homocysteine and the frequency of new cardiovascular complications in patients with show vascular complications with the metabolic syndrome.

MATERIALS AND METHODS

The present study was conducted at SLIMS, Puducherry. The study included 200 MetS patients and 200 Controls. Homocysteine (Hcy) estimated by ELISA method. FBS, lipid profile assessed by using standard method using commercial kits. 5 ml of venous blood samples were collected from patients and controls and these samples were collected overnight fasting of 12 hrs. Collected samples centrifuged under 2000 rpm for 20 min and after centrifugation of samples (plasma) used

* Corresponding Author
Email: drpebyreddy@yahoo.com
Contact: +91-9159186879
Received on: 24-08-2017
Revised on: 07-10-2017
Accepted on: 11-10-2017

Table 1: Homocysteine levels in metabolic syndrome

S.No.	Parameters	MetS(n-200) Mean± SEM	Controls(n-200) Mean±SEM	p Value
1	Homocysteine µmol/lit)	19.21±1.32	10.85±0.76	p<0.001
2	FBS(mg/dl)	156.55±4.82	106.12±1.68	p<0.001
3	Cholesterol (mg/dl)	245± 30.51	188.5 ±27.3	p<0.001
4	TGL (mg/dl)	243±28.62	169.2±28.4	P<0.001
5	HDL (mg/dl)	30± 5.52	50± 8.25	p<0.001
6	LDL (mg/dl)	123.8±31.4	68.3±13.2	P<0.002

to assess the Hcy levels.

The diagnosis of metabolic syndrome was based waist, BMI, waist-hip ratio, systolic and diastolic blood pressure, Blood glucose levels.

NCEP ATP III 2001 Criteria for Metabolic Syndrome

The agenda of ATP III was to recognize the people with long term risk of cardiovascular disorders (CVD's) who merited clinical way of life mediation to lessen chance.

Existence of three of the below mentioned five elements is required for conclusion of metabolic disorder. Central obesity: Abdominal waist circumference: Men >102 cm, women >88 cm. Fasting plasma glucose >110mg/dl or identified type 2 diabetes mellitus (T2DM). Fasting plasma triglyceride >150 mg/dl or medication. Fasting plasma HDL cholesterol: Men <40 mg/dl, women <50 mg/dl or medication. Blood pressure ≥120/80 mmHg or medication.

Statistical analysis

All results were summarized as mean ± SEM. The statistical analysis were done using SPSS software 11.5 version (SPSS, Inc., Chicago) and the comparison between patients and control was done by using ANOVA. A p value < 0.05 were considered as statistically significant. The p value was kept of <0.001 is comparatively highly significant.

RESULTS AND DISCUSSION

FBS, lipid profile significantly increased in the studied subjects such that DM (p<0.05) and MetS (p<0.05) were observed, when compared with control group. Increase FBS, lipid profile were observed, when compared with MetS group. FBS, lipid profile were done rule out the DM, MetS patients.

As of late, gentle rises of plasma Hcy have been distinguished as an autonomous risk factor for early atherosclerotic vascular disorders and thromboembolic disease. (Hajer GR, van der Graaf Y, et al., 2007. LinksHajer GR, et al., 2007). A significant (p<0.001) increase in Homocysteine was observed in MetS patients when compared to controls. This findings suggests it is thought to be a risk factor for the improvement of cardiovascular complications. A few perceptions propose that there may be connects between hyper homocysteinemia and insulin protection, its clinical surrogate of metabolic syndrome. Lifted homocysteine levels were connected to the metabolic syndrome in patients with earlier myocardial localized necrosis.

In Framingham Offspring Study appeared, expanded tHcy levels were appeared to build the danger of cardiovascular disease just within the sight of unusual proteinuria in patients with insulin protection (Buysschaert M, Dramais AS, et al., 2000).

Since hyperhomocysteinemia and microalbuminuria additionally reflect endothelial damage, these perceptions likewise bolster the theory that endothelial dysfunction is related with articulation of the metabolic syndrome. In exhibit contemplate metabolic variations from the norm to be more pervasive in subjects with higher serum Hcy levels. It was corresponded with MetS segments, particularly through SBP. As an earlier report recommends, Hcy and SBP stack on a similar factor, which had the best consistency for CHD. Which has been connected to different MetS segments including insulin protection, Obesity, and waist circumference too. In present study comes about is well concurrence with grouping of Hcy with MetS components, including waist circumference (Links Hajer GR, van der Graaf Y,2007.Pacana T, Cazanave S, et al., 2015).

On one hand, increased plasma homocysteine levels might be a reason for insulin protection and might be effectively associated with atherogenesis. On the other hand, raised levels of homocysteine could be viewed as a pointer of vascular risk and just be utilized for chance estimation. In this study hoisted homocysteine levels were related with an expanded danger of future cardiovascular complications in patients with show vascular infection (Hajer GR, van der Graaf Y, et al., 2007. Lyon CJ, Law RE, et al., 2003. Banecka-Majkutewicz Z, et al., 2014).

High circulating Hcy concentrations may increase the risk of CVDs when present with other cardiovascular risk factors like hypercholesterolemia and diabetes. Hyperhomocysteinemia is an independent risk factor of CVD incidence in type 2 diabetic patients. This finding was consistent with the findings of Soinio et al., (Coppola A, Astarita C, et al.,2004) and Coppola et al., (A. M. McNeill, W. D. Rosamond. et al., 2006) .

Hyperhomocysteinemia causes endothelial dysfunction by expanding oxidant stress and declines the arrival of nitric oxide, weakening vasodilation and abundance of Hcy fortifies smooth muscle cell proliferation and col-

lagen combination advancing intima- media thickening (Steed MM, Tyagi SC.2011. Hayden MR, Tyagi SC.2004). It is likewise considered to have thrombogenic movement by expanding platelet accumulation and causing variations from the norm in the coagulation framework. High plasma Hcy level is likewise appeared to be related with expanded lipid peroxidation and dyslipidemia in patients of type 2 diabetes with cardiovascular complications (Domagala TB, Undas A, et al.,1998).

CONCLUSION

To sum up, a significant (p<0.001) increase in Homocysteine was observed in MetS patients when compared to controls. Raised levels of homocysteine could be viewed as a marker of vascular risk and just be utilized for chance estimation. In this study lifted homocysteine levels were related with an expanded danger of future cardiovascular occasions in patients with show vascular disorders.

However, further studies are required to determine whether genetic, nutritional deficiencies, or diseases related to Hcy metabolism account for hyperhomocysteinemia observed in patients of type 2 DM with and without cardiovascular complications.

REFERENCES

A. M. McNeill, W. D. Rosamond, C. J. Girman et al.The metabolic syndrome and 11-year risk of incident cardiovascular disease in the atherosclerosis risk in communities study,"Diabetes Care, vol. 28, no. 2, pp. 385–390, 2005.

Banecka-Majkutewicz Z, Sawuła W, Kadziński L, Węgrzyn A, Banecki B. Homocysteine, heat shock proteins, genistein and vitamins in ischemic stroke--pathogenic and therapeutic implications. Acta Biochimica Polonica. 2012 Dec 19;59(4).

Buysschaert M, Dramais AS, Wallemacq PE, Hermans MP. Hyperhomocysteinemia in type 2 diabetes: relationship to macroangiopathy, nephropathy, and insulin resistance. Diabetes care. 2000 Dec 1;23(12):1816-22.

Coppola A, Astarita C, Oliviero M, Fontana D, Picardi G, Esposito K, et al. Impairment of coronary circulation by acute hyperhomocysteinemiain type 2 diabetic patients. Diabetes Care 2004;27:2055-6.

Domagala TB, Undas A, Libura M, Szczeklik A. Pathogenesis of vascular disease in hyperhomocysteinaemia. Journal of cardiovascular risk. 1998 Aug;5(4):239-47.

Executive Summary of the Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). JAMA2001; 285: 2486–2497.

Grundy SM, Brewer HB Jr, Cleeman JI, et al: Definition of metabolic syndrome. Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. Arterioscler Thromb Vasc Biol 24: e13-e18, 2004.

H, Abel ED. Molecular mechanisms for myocardial mitochondrial dysfunction in the metabolic syndrome. 2008 Clin Sci (Lond). 114(3):195–210.

Hajer GR, van der Graaf Y, Olijhoek JK, Verhaar MC, Visseren FL. Levels of homocysteine are increased in metabolic syndrome patients but are not associated with an increased cardiovascular risk, in contrast to patients without the metabolic syndrome. Heart. 2007 Feb 1;93(2):216-20.

Hajer GR, van der Graaf Y, Olijhoek JK, Verhaar MC, Visseren FL. Levels of homocysteine are increased in metabolic syndrome patients but are not associated with an increased cardiovascular risk, in contrast to patients without the metabolic syndrome. Heart. 2007 Feb 1;93(2):216-20.

Hayden MR, Tyagi SC. Homocysteine and reactive oxygen species in metabolic syndrome, type 2 diabetes mellitus, and atheroscleropathy: the pleiotropic effects of folate supplementation. Nutrition Journal. 2004 May 10;3(1):4.

LinksHajer GR, van der Graaf Y, Olijhoek JK, SMART Study Group. Levels of homocysteine are increased inmetabolic syndrome patients but are not associated with an increased cardiovascular risk, in contrast to patients without the metabolic syndrome. Heart. 2007;93:216-0.

LinksHajer GR, van der Graaf Y, Olijhoek JK, SMART Study Group. Levels of homocysteine are increased inmetabolic syndrome patients but are not associated with an increased cardiovascular risk, in contrast to patients without the metabolic syndrome. Heart. 2007;93:216-0.

LinksHajer GR, van der Graaf Y, Olijhoek JK, SMART Study Group. Levels of homocysteine are increased inmetabolic syndrome patients but are not associated with an increased cardiovascular risk, in contrast to patients without the metabolic syndrome. Heart. 2007;93:216-0.

Lyon CJ, Law RE, Hsueh WA. Minireview: adiposity, inflammation, and atherogenesis. Endocrinology. 2003 Jun 1;144(6):2195-200.

Nicolson GL. Metabolic syndrome and mitochondrial function: molecular replacement and antioxidant supplements to prevent membrane peroxidation and restore mitochondrial function. J Cell Biochem. 2007; 100(6):1352–69.

Pacana T, Cazanave S, Verdianelli A, Patel V, Min HK, Mirshahi F, Quinlivan E, Sanyal AJ. Dysregulated hepatic methionine metabolism drives homocysteine

elevation in diet-induced nonalcoholic fatty liver disease. PLoS One. 2015 Aug 31;10(8):e0136822.

Steed MM, Tyagi SC. Mechanisms of cardiovascular remodeling in hyperhomocysteinemia. Antioxidants & redox signaling. 2011 Oct 1;15(7):1927-43.

Taskinen MR. Is metabolic syndrome the main threat to human health in the twenty-first century 2007 Arterioscler Thromb Vasc Biol; 27: 2275.