

## Assessment of amino acid in hypertensive patients.

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### Abstract

**Introduction:** Hypertension is a global disease and in 95% of cases the cause is unidentified. Its early diagnosis and management can block severe complications such as ischemic heart disease (IHD). Moreover the combination of lipids with IHD is important fact. Despite that abnormally high levels of amino acid (homocysteine) were detect to be strongly connected to a raise hazard of coronary artery disease.

**Objectives:** To assess plasma homocysteine strengths level in patients having hypertension was on treatment.

**Methods:** Plasma homocysteine strengthens were calculated using High Presentation Liquid Chromatography (HPLC) with Ultraviolet finder in 60 hypertensive patients samples (27 male and 33 female) aged 35 years and more. VLDL-cholesterol, LDL-cholesterol, HDL-cholesterol, Cholesterol and triglyceride were resolved. The frequency of high total homocysteine rates were resolved by contrast with usual indications.

**Results:** Plasma homocysteine grade were greatly higher in sick than in typical population. Serum cholesterol and triglyceride strengths were also greatly above in patients than in typical population with no link to the grade of homocysteine which is considered as a certain detached heart risk factor.

**Conclusions:** The study concludes that plasma homocysteine grades were noticeably uppermost in patients than in control groups. No significant variation observed among male and female patients.

**Keywords:** Homocysteine, LDL, Vascular disease, Atherosclerosis, Hypertension.

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### Introduction

The determination of hypertension in adulthood is built when the middle of or extra systolic and diastolic blood pressure (BP) assessments on at least two following measurements are above 90 and 140 mmHg [1]. Its early identification and management may inhibit many critical complexities such as Angina pectoris, MI, heart failure and kidney diseases [2]. With the most significant risk elements for hypertension is elevated serum lipids specially serum cholesterol [3]. It is founded that unusual higher status of homocysteine in the blood is intensely connected to a raise hazard of vascular heart diseases as it may damage the lining of the vascular and afford to clotting of the blood by producing endothelial damage accompanied by platelet stimulation and thrombus generations [4,5]. Homocysteine was a sulphur-holding amino acid created throughout the metabolic process of methionine whom may be detected in meats and milk products, thus high dietary intake of such milk may lead to the over generation of homocysteine [6]. Raising in plasma homocysteine is whether result from genetic imperfections in the enzyme engage in its metabolic process or because of dietary insufficiency of vitamin (B6, B12 or folic acid) due to these dietary vitamins are vital co-factors for enzymes include in the metabolic process of homocysteine such as methionine synthase and 5,10-methylene-tetrahydro folic-acid reductase [7]. Hyperhomocysteinemia can be related with various illness condition and drugs. It can be raise in chronic renal failure frequently impending strength that are more than four times the usual rate which can be describe the noticed increase of atherosclerosis in renal failure [8]. In

addition there is some relation among hyperhomocysteinemia and patients with rheumatoid arthritis and hypothyroidism, which indicate a possible mechanism for the great prevalence of coronary disease noticed in those clients. Many drugs such as methotrexate and phenytoin interact with foliate metabolic process and can lead to kind hyperhomocysteinemia [9,10].

### Methods

The study was directed on 60 hypertensive samples with 27 male and 33 female whom attend to Azady Teaching Hospital over 6 months interval with a usual age of 35-85-years old. As well as another 20 healthy samples were included as a control group. A complete personal history was taken from these patients. A sample of blood of 7 ml was drawn into disposable syringe. About 5 ml from the blood sample were permitted to be clotted after 30 minutes in a plain tube and serum was isolated by centrifugation at 3000 rpm at room temperature for about 5 minutes. The serum then analysed for lipid profile. The left over 2 ml were placed in a K3 EDTA tube and directly placed in a crashed ice. The plasma was isolated from RBC by centrifugation at 3000 rpm at 4°C for about 5 minutes then banked at 20°C until examines. Total serum cholesterol was set by Cholesterol Enzymate PAP Kits (BioMeriux). Triglyceride was set using enzymatic procedure provided by BioMeriux Lab. HDL-Cholesterol serum level was determined by precipitate of LDL-Cholesterol and VLDL-Cholesterol by the adding of phosphotungastic-acid in the existence of magnesium ions. The superimposed acquired a later centrifugation consist only HDL-Cholesterol as the cholesterol segment was finished

by the same enzymatic method discussed in cholesterol. Serum level of Low density lipoprotein (LDL) cholesterol was measured by Friedwald rule. Resolution of the biologically active homocysteine (THCY) was done by using HPLC with Shimodzu SPD-6AV UV observable finder within a wave length of 195-700 nm [11].

## Results

The study revealed an important significant higher total plasma homocysteine strength in the hypertensive samples patients ( $56 \pm 40.15 \mu\text{mol/l}$ ) than in the control group samples ( $14 \pm 3.9 \mu\text{mol/l}$ ) with  $P < 0.05$  but the study displayed no significant differences in homocysteine levels among male and female as manifested in Table 1. Serum triglyceride strength in patient samples were also more than in control samples as manifested in Table 4. The table also displaced that there is significant differences among patient. The severity of homocysteine is shown in Table 2 where intermediate hyper-homocysteine was representing 55% of all cases in compare to moderate and sever degrees which were express 30% and 15% of all samples respectively [11].

**Table 1.** Patients and control group total plasma homocysteine at  $P < 0.05$ .

Groups		Homocysteine (mmol/l)		
		N	Mean	SD
Patients	Male	27	58.15	3.66
	Female	23	55.45	4.36
	Total	60	56.8	4.014
Control		20	14	3.9

**Table 2.** Male and female classification of plasma homocysteine according to concentrated level.

Concentrated level	Male			Female		
	N	%	Mean $\pm$ SD	N	%	Mean $\pm$ SD
Moderate (15-30) mmol/l	8	29.6	22.9 $\pm$ 7.8	10	30.3	20.3 $\pm$ 5.2
Intermediate (30-100) mmol/l	15	55.6	55 $\pm$ 20.6	18	54.5	45.3 $\pm$ 15.8
Sever >100 mmol/l	4	14.8	102 $\pm$ 40.1	5	15.2	117.4 $\pm$ 50.5

Table 3 demonstrated that controlled group of hypertensive patients express 73.33% of all samples with a mean homocysteine grade of  $50.7 \pm 29.2 \mu\text{mol/l}$ , while 26.66% were uncontrolled hypertensive samples with a mean homocysteine level of  $62.6 \pm 27.4 \mu\text{mol/l}$  [12].

**Table 3.** Controlled and uncontrolled hypertension plasma homocysteine.

Groups	N	Homocysteine (mmol/l)		
		%	Mean	SD
Controlled Hypertension	44	73.33	50.7	29.2
Uncontrolled Hypertension	16	26.66	62.6	27.4

**Table 4.** Patient and control group lipid profile.

Type of cholesterol	Patient		Control		P<0.05
	Mean	SD	Mean	SD	
Cholesterol (mg/dl)	221.09	45.82	173.2	18.57	Significant
Triglyceride (mg/dl)	182.09	111.04	117.89	43.22	Significant
HDL (mg/dl)	41.95	14.31	52.72	12.41	Significant
VLDL (mg/dl)	36.13	22.41	19.11	8.81	Significant
LDL (mg/dl)	138.19	49.7	83.55	18.31	Significant

The study also showed that total serum cholesterol strength in patient group were significantly higher than the concentration in control group. It is also obvious from the study that 32 samples patients who had hypercholesterolemia more than 220 mg/dl and 21 patients who held hypertriglyceridemia greater than 160 mg/dl had no relation to the grade of homocysteine which is considered as a distinct risk factor independent from other risk factors, other risk factors for raised homocysteine ex. smoking, life style excluded as it is showed in Table 5.

**Table 5.** Hypercholesterolemia and hypertriglyceridemia plasma homocysteine.

	Homocysteine (mmol/l)			
	N	%	Mean	SD
Hypercholesterolemia >220 mg/dl	32	53.3	53.9	33.74
Hypercholesterolemia >160 mg/dl	21	35	56.47	31.9

## Discussion

The study had displayed an important increase in plasma homocysteine grade in hypertensive samples and a primary relationship among homocysteine grade and heart risk which is also founded by Graeme et al. [13]. There were no important difference in plasma homocysteine strength among male and female patients with moderate and intermediate hyperhomocysteine grade. This difference rise in clients with severe hyperhomocystinemia that is similar to the conclusion of Stamler et al. [14]. Hyperhomocystinemia were as founded in 73.33% of all samples which may be due to along period of disease which was greater than 10 years. The elevated concentrations of homocysteine in uncontrolled hypertensive samples may be result from sever hypertension due to ignoring of management by antihypertensive drugs [15]. Serum cholesterol and triglyceride grade were significantly greater

than the concentration in control group and this finding accepted with Neaton and co-workers who have mentioned that rises in cardiac outcome incident with raising cholesterol levels [16]. Such result accepts with previous conclusions in additional study which concluded a significant triglyceride-coronary disease relationship [17]. High density lipoprotein (HDL-Cholesterol) in patients and control groups were significantly various and a latest study demonstrated a basic relationship of high HDL and low risk of Ischemic heart diseases. A similar conclusion for LDL and VLDL were gain. The study declared that raising in cholesterol and triglyceride grade have no relation to the levels of homocysteine which is considered as a specific independent risk factor [18]. These results accept with Konecky et al. who mentioned that hyperhomocysteinemia is a free risk element for aortic diseases [19].

## References

1. Lee Goldman, Dennis A. Cardiovascular disease. Cecil Text book of medicine, 22nd Edition. 2004:242-485.
2. Eikelboom JW, Lonn E, Genest J, et al. Homocysteine and Cardiovascular disease; A critical review of the epidemiologic evidence. *Ann Intern Med.* 1999;131(5): 363-75.
3. AF Smith, GJ Beckett, SW Walker, et al. clinical Biochemistry, 6th Edition .1998:110-23.
4. Austin RC, Lentz SR, Werstuck GH. Role of Hyperhomocysteinemia in endothelial dysfunction and atherothrombotic disease. *Cell Death Differ.* 2004;11(S1):S56-S64.
5. Harker LA , Slicher SJ , Scott CR. et al. Homocysteinemia. *N Engl J Med.* 1974;291(11):537-43.
6. Mudd SH, Skovby F, Levy HL, et al. The Natural History of Homocystinuria Due to Cystathionine beta-Synthase deficiency. *Am J Hum Genet.* 1985;37(1):1-31.
7. Silaste ML, Rantala M, Sämpi M, et al. Polymorphisms of key Enzymes in Homocysteine Metabolism Affect Diet Responsiveness of Plasma Homocysteine In Healthy Women. *J Nutr.* 2001;131(10):2643-7.
8. Robinson K. Renal disease, Homocysteine and Cardiovascular complications. *Circulation.* 2004;109(3): 294-5.
9. Allon NF, Andrew GB, Jacob S, et al. The kidney and Homocysteine Metabolism. *J Am Soc Nephrol.* 2001;12(10):2181-9.
10. Kang SS, Wong PW, Norusis M. Homocysteinemia due to folate deficiency. *Metab.* 1987;36(5):458-62.
11. Lentz SR, Haynes WG. Homocysteine, Is it a clinically important Cardiovascular risk factor. *Cleve Clin J Med.* 2004;71(9):729-34.
12. Boushey CJ, Beresford SA, Omenn GS, et al. A quantitative assessment of plasma homocysteine as a risk factor for vascular disease: probable benefits of increasing folic acid intake. *JAMA.* 1995;274(13):1049-57.
13. Hankey GJ, Graeme JH , Eikelboom JW. Homocysteine and vascular disease. *Lancet.* 1999;354(9176):407-13.
14. Stamler J, Wentworth D, Neaton J. Is Relationship between serum cholesterol and risk of premature death from Coronary Heart Disease , continuous and Graded. *JAMA.* 1986;256(20):2823-8.
15. Graham IM, Daly LE, Refsum HM. Plasma Homocysteine as a risk factor for vascular disease. *JAMA.* 1997;277(22): 1775-81.
16. Castelli WP, Garrison RJ, Wilson PW, et al. Incidence of coronary Heart disease and lipoprotein cholesterol levels. *JAMA.* 1986;256(20): 2835-8.
17. Gauthier GM, Keevil JG, McBride PE, et al. The association of homocysteine and coronary Artery Disease. *Clin Cardiol.* 2003;26(12):563-8.
18. Kenket P, Reunanen A, Alfthan G, et al. Hyperhomocysteinemia; A risk factor or consequence of coronary heart disease. *Arch Inter Med.* 2001;161(13): 1589-94.
19. Konecky N, Malinow R, Tunick PA, et al. Correlation between plasma homocysteine and aortic atherosclerosis. *Am Heart J.* 1997;133(5):534-40.

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