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General Medicine

"EVALUATION ON SERUM HOMOCYSTEINE LEVELS IN CEREBROVASCULAR ACCIDENTS: A TERTIARY CARE TEACHING HOSPITAL STUDY"

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ABSTRACT Homocysteine is a risk factor for atherosclerotic vascular disease, with adverse influence on endothelial cells, vascular smooth muscle cells, connective tissue, interactions with plasma lipoproteins and platelets. Aim of the study was to detect the serum homocysteine levels in Cerebrovascular accidents as a risk factor for stroke. These findings suggest that the total homocysteine level is significantly higher in patients with stroke (cerebral infarction) than healthy controls. Now a day, hyperhomocysteinemia is considered an independent risk factor for the incidence of stroke, even though there is lack of convincing explanation till date.

KEYWORDS : Homocysteine, Cerebrovascular accident, risk factors & Hyperhomocysteinemia.

Introduction:

Stroke is the second commonest cause of mortality and the fourth leading cause of disease burden, Globally.[1] it remains a major cause of mortality and morbidity worldwide.[2] The risk factors are varied, which include both modifiable and non-modifiable ones.[3] Among these serum homocysteine concentrations are emergingas risk factors for ischaemic cerebrovascular accidents[4], although there are studies that showed no increase in risk.[5] Aim of the study was to detect the serum homocysteine levels in Cerebrovascular accidents as a risk factor for stroke.

Material and methods:

This Prospective observational case control study was carried on 35 cerebrovascular accidents patients with 35 control match persons over a period from August 2015 to July 2017, with following criteria's: All the patients admitted to Medicine, IQ City medical College & hospital, Durgapur, India with focal neurological deficit due to suspected Cerebrovascular accident. And patients other than the study diagnosis are excluded.

Clinical information including age, sex and history of current evidence of hypertension (HTN): systolic blood pressure (SBP) - 140mmHg and diastolic BP - 90mmHg, Diabetes Mellitus (DM): fasting blood glucose mmol/L/126 mg, cardiac disease, life style, diet pattern, family history of vascular diseases was recorded for all subjects. Serum total cholesterol (CH), HDL cholesterol, LDL cholesterol VLDL cholesterol and triglycerides were measured by using standard enzymatic procedures.[6]

Borderline for normal values were: total cholesterol 200-239 mg, HDL-C < 60 mg, LDL-C <130-159 mg, VLDL< 1.1 mmol/L and triglyceride < 150 mg.[6]

Serum homocysteine was estimated by Chemilunescent Immuno assay method, variant of standard ELISA.

The upper limit of the manufacturer and the laboratory was 15 µmol/L. Values above 15 µmol/L were accepted as high.[7]

Analyzers used:

Immulite 1000 Chemiluminescent Technology of SIEMEN'S Company.

Normal levels of homocysteine:

- Adult male: 06-15 µmol/L
- Adult female: 03-12µmol/L
- Elderly >65 years: 15-20µmol/L.

In order to compare these parameters between patients and controls, student's t-test was applied and the results were presented in tabular form.[7,8]

Results and Discussion:

A case-control study consisting of 35 patients and 35 age and sex matched controls is undertaken to investigate the relationship of homocysteine with Cerebrovascular accidents as a risk factor for stroke. Table-1 shows that the maximum number of cases is in the age group 56-65 years with homocysteine levels of 16.06 ± 3.31 mmol/L. The highest homocysteine level was observed in the age group 66 years and above. This implies that the level of homocysteine increases as age advances.

Table 1: Age wise distribution of Homocysteine level (µmol/L) in cases and controls group:

Parameters (Age in yrs.)	Cases(N=35)		Controls(N=35)	
	No.	Mean ± Sd	No.	Mean ± Sd
26-35	3	12.5 ± 2.05	2	10.00 ± 3.41
36-45	5	13.20 ± 1.43	4	11.20 ± 1.42
46-55	7	15.50 ± 5.02	3	12.00 ± 4.00
56-65	12	16.06 ± 3.31	10	12.70 ± 3.37
66 and Above	3	16.78 ± 2.03	11	12.14 ± 2.2

P < 0.05 (Significant)

Table 2 : Blood sugar, lipid profile, Creatinine & Homocysteine level in study and control group:

Parameters	Cases (N=35)	Controls (N=35)
Blood Sugar (R) (mg/dl)	117.5±45.8	85.03±4.8
Total Chol (mg/dl)	187.4±24.2	155.4±14.03
Triglycerides (mg/dl)	112.8±40.5	96.3±20.16
HDL (mg/dl)	41.61±5.01	47.638.4
Creatinine (mg/dl)	1.12±0.2	0.9±0.03
Homocysteine level (µmol/L)	14.8± 5.82	11.6 ± 2.35

P < 0.05 (Significant)

In Table-2, we observed that blood sugar levels are higher in cases (117.5±45.8mg/dl) than control (85.03±4.8 mg/dl) which is significant (P < 0.05). HDL cholesterol level is significantly low in study group when compared with control group (P<0.05). Total cholesterol level is significantly higher in patients (187.4±24.2 mg/dl) than in controls (155.4±14.03mg/dl) whereas no significant difference could be observed in serum trglycerides and creatinine levels. When the levels of total homocysteine is compared between the cases and control, the study group has 14.8± 5.82 µmol/L whereas in the control, it is 11.6 ± 2.35 µmol/L which is statistically significant (P < 0.05).

Increasing age and male sex is associated with higher total homocysteine concentrations.[9] The difference between the sexes could be due to large muscle mass in men since the formation of muscle is associated with the simultaneous formation of homocysteine in connection with creatine/creatinine synthesis.[10] The increase in plasma homocysteine may also be due to the influence of sex hormones.[11] The concentrations of total homocysteine was significantly higher in study group than in controls. Further, patients with cerebrovasular accident had elevated levels of blood sugar and serum cholesterol with low

HDL-cholesterol. These observations are consistent with that of Tanne et al [12]. Kam et al [13] also found a strong association between plasma total homocysteine and atherothrombotic disease, which is consistent with our findings. Epidemiological studies have demonstrated that hyperhomocysteinemia is an independent risk factor for stroke.[14] However, the molecular mechanism by which homocysteine promotes atherothrombosis is unknown. Experimental evidence suggests that atherogenic propensity associated with hyperhomocysteinemia results from endothelial dysfunction. Endothelial cell injury, platelet activation, deleterious effect on thrombomodulin expression, protein C activation, and an increased oxidizability of LDL have been described as a few possible mechanism by which homocysteine provokes arteriosclerosis and thrombosis.[15] As far as the prognostic value of hyperhomocysteinaemia on cerebrovasular accident is concerned, the present study failed to reveal any significant correlation, although the patients were followed up for only three months, and the cases were only monitored clinically. A long term follow-up is necessary to highlight the matter further.

Conclusion:

These findings suggest that the total homocysteine level is significantly higher in patients with stroke (cerebral infarction) than healthy controls. Now a day, hyperhomocysteinemia is considered an independent risk factor for the incidence of stroke, even though there is lack of convincing explanation till date. It is concluded therefore that fasting homocysteine levels should be assessed in patients with first episode of ischaemic cerebrovascular accidents, and also in those who are at risk of developing ischaemic cerebrovascular accidents.

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