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Different comparison of mean SR. homocystene in serum 25 - hydroxyvitamin D in acute stroke

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***Abstract*---**The active form of vitamin D is 1,25 dihydroxy vitamin D. Renal and many extra renal tissues are able to synthesize 1,25 dihydroxy vitamin D on a local and intracellular level. Local tissue level of 1,25 di-hydroxyvitamin D is determined by concentration of circulating 25(OH) vitamin D level. Stroke is a common worldwide health problem. It is a major cause of morbidity, mortality and disability in developed as well as developing countries. Both vitamin D deficiency and stroke are very common in urban and rural India. Hence this study was designed to evaluate the association of vitamin D deficiency in acute stroke. This study shows the different comparison of MEAN SR. HOMOCYSTENE in serum 25 – hydroxyvitamin D in acute stroke and found to be in total serum homocysteine levels were 19.64 micro mole/l in cases than 10.1 in control However the difference was statistically highly significant (p<.0002).

***Keywords*---**serum 25, hydroxyvitamin D, homocysteine, stroke.

Introduction

The active form of vitamin D is 1, 25 dihydroxy vitamin D. Renal and many extra renal tissues are able to synthesize 1,25 dihydroxy vitamin D on a local and

intracellular level. Local tissue level of 1,25 dihydroxy vitamin D is determined by concentration of circulating 25(OH) vitamin D level. That is why serum level of 25(OH) vitamin D is measured to assess and classify vitamin D status. [1] In some Indian studies by Marwaha et al (2005), Zargar et al (2007), Goswami et al (2008), Sahu et al (2008), vitamin D deficiency is epidemic in India despite plenty of sunshine. All Indian study point to low 25(OH)D levels in the populations [2]Both vitamin D deficiency and stroke are very common in urban and rural India. Hence this study was designed to evaluate the association of vitamin D deficiency in acute stroke. Stroke is a common worldwide health problem. It is a major cause of morbidity, mortality and disability in developed as well as developing countries. [3]

Stroke is the 2nd leading cause of death after ischaemic heart disease and major cause of disability worldwide (world health statistics 20ll). Worldwide, about 20 million people suffer from stroke each year, 5 million die and 5 million more are left with chronic disability. In 2011, a study published in the Annals of Neurology revealed that between 1995-1996 and 2007-2008, the incidence of stroke increased by at least 23% for males and female aged between *5* and 44years. [3] Stroke incidence is rising among young adult and decreasing among elderly (according to American Stroke Association International Conference 2010; Science Daily, March 1, 2010 and American Academy of Neurology published 14 August 2013)

Of all stroke, up to 85% are ischaemic, and of these up to l2% occur in young individuals. Ischaemic stroke in young adult constitute about *5* % of all stroke. The incidence varies from 11 to 20/100,000 in different studies. Stroke in young adults (18-44years) is an important cause of morbidity and mortality throughout the world, especially in developing country. [4] In INDIA, the latest available estimates from Indian Council Of Medical Research (ICMR) indicate that in 2004 there were 930,985 cases of stroke with 639,455 deaths and 6.4 million disability adjusted life year (DALY)lost [4]

A Stroke (previously known as cerebrovascular accident) is rapidly developing clinical symptoms and/or signs of focal and at times global (applied to patients in deep coma and those with subarachnoid hemorrhage) loss of brain function, with symptoms lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin. There is a wide range of severity, fromrecovery in few days, through persistent disability, to death. [5] The definition of stroke is clinical, and laboratory studies including brain imaging are used to support the diagnosis [5]. The diagnosis of stroke (versus not stroke) is still a matter of clinical skill often without the help of many, or any, confirmatory investigations. However, this does have the advantage that the diagnosis is independent of the availability and quality of rapidly changing technology (such as CT scanners), which is often not available at all in developing countries, or even universally available in developed countries. [4,5]

Once the diagnosis of stroke is made, a brain imaging study is necessary to determine if the cause of stroke is ischemia or hemorrhage. The clinical manifestation of stroke is highly variable because of the complex anatomy of the brain and its vasculature.

There are several common causes of sudden onset neurologic symptoms that may mimic stroke, including seizure, intracranial tumor, migraine and metabolic encephalopathy.[6] A fall in cerebral 6blood flow to zero causes death of brain tissue within 4-10 minutes; values <16-18 ml/l00 gm tissue per minute cause infarction within an hour; and values <20 ml/100 gm tissue per minute cause ischemia without an infarction unless prolonged for several hours or days.[5] Neurologic symptoms are manifest within seconds because neurons lack glycogen, so energy failure is rapid. If the cessation of blood flow lasts more thana few minutes, infarction or death of brain tissue results. [5,6]

Stroke mortality rises rapidly with age. The burden of stroke arises largely from the elderly population. However, there remains a small but significant subset of younger patients with ischemic stroke, in whom conventional vascular risk factors play a smaller role. About 85% of all first ever stroke is ischemic, 10% are due to primary intracerebral hemorrhage and about 05% are due to subarachnoid hemorrhage.[4,5,6] The ischemic stroke, 25% are caused by large artery disease, 25% by small vessel disease, 20% by cardiac embolism, 05% by other rare causes. .[4,5,6] The standard definition of Transient Ischemic Attack (TIA) requires that all neurologic signs and symptoms resolve within 24 hours regardless of whether there is imaging evidence of new permanent injury; stroke has occurred if the neurologic signs and symptoms lasts for >24 hours. [4,5,6]

There are many risk factors for stroke including age, sex, family history of stroke, hypertension, smoking, diabetes, obesity, hyperlipidemia and atrial fibrillation. Many studies indicate a plethora of conventional risk factors for stroke. Nevertheless, cerebrovascular events do occur sometimes in the individuals without any of the previously mentioned risk factors. As a consequence, it is very likely that other risk factors exist. Identification of modifiable risk factors for stroke may lead to more effective prevention of first and recurrent episodes of cerebrovascular disease.

Hyperhomocysteinemia, defined as an elevated plasma total homocysteine concentration (>10 μM), is one such factor.[10] Some studies have shown that elevated serum homocysteine is an independent risk factor for stroke. (Ischemic) [7,9] Hyperhomocysteinemia is common and is the main prothrombotic factor associated with cerebrovascular accident. Hyperhomocysteinemia is an independent risk factor for arterial dysfunction in healthy middle-aged adults. Hyperhomocysteinemia has also been associated with myocardial infarction, Alzheirner's disease and vascular dementia. Hyperhomocysteinemia causes increased arterial blood pressure thereby increasing the risk of cerebrovascular accidents. Elevated plasma homocysteine has also been shown to induce oxidative injury to vascular endothelial cells and cause impairment of the endothelial production of nitric oxide, a strong vascular relaxing factor. Other proposed mechanisms include enhancement of platelet adhesion to endothelial cells, promotion of the growth of vascular smooth muscle cells and association of increased homocysteine with higher levels of prothrombotic factors such as thromboglobulin, tissue plasminogen activator and factor VIIc[10].

Homocysteine is an amino acid in the blood. It is not obtained from the diet and is biosynthesized from methionine via multi step process. Plasma homocysteine

levels are strongly influenced by diet, as well as by genetic factors. The dietary components with the greatest effects are folic acid and vitamins B6 and B_{12}. Folic acid and other B vitamins help break down homocysteine in the body. Several studies have found that higher blood levels of B vitamins are related, at least partly, to lower concentrations of homocysteine. Other recent evidence shows that low blood levels of folic acid are linked with a higher risk of fatal coronary heart disease and stroke.[11] Several clinical trials are under way to test whether lowering homocysteine will reduce coronary heart disease risk. Recent data show that the institution of folate fortification of foods has reduced the average level of homocysteine in the United States population.

Recent findings suggest that laboratory testing for plasma homocysteine levels can improve the assessment of risk. It may be particularly useful in patients with a personal or family history of cardiovascular disease, but in whom the well- established risk factors (smoking, high blood cholesterol, high blood pressure) do not exist. Although evidence for the benefit of lowering homocysteine levels is lacking, patients at high risk should be strongly advised to be sure to get enough folic acid and vitamins B_6 and B_{12} in their diet. Foods high in folic acid include green leafy vegetables and grain products fortified with folic acid. But this is just one of the risk factor. A physician taking any type of nutritional approach to reducing risk should consider a person's overall risk factor profile and adjust the diet accordingly. The reason for the decline in the incidence of major stroke in recent years are unclear, may be due to the treatment of risk factors such as hypertension and elevated cholesterol. It has been estimated that full implementation of currently available preventive strategy could reduce stroke incidence by as much as 50 - 80 %.[12]

Aims and Objective

It is an observational study. It includes 73 cases and 40 controls. After hospitalization, a detailed history was taken from all cases and controls including history of lifestyle and dietary habit and a thorough physical examination was performed so as to fulfill the inclusion and exclusion criteria laid down in the study protocol.

Experimental Methods

Table-1 Comparison of mean SR.homocystene with diet

Diet	No.of pts		%		Sr.homocysteine Level Mean+_sd		P-value Significance
	Cases	Cont	Cases	Cont	Cases	Cont	
Vegeterian	4	4	10	10	21.76±17.38	10± 2.4	P-<.0002
Mixed diet	38	38	90	90	17.5± 14.38	10.2± 2	Highly
Total	42	42	100	100	19.64±15.88	10.1± 2.2	significant

Mean serum homocysteine levels were higher (21.76 ± 17.38) in patients on vegetarian diet than in patients with mixed diet where it was 17.5 ± 14.35 but in control however it is approximately same in both groups.

Table 2: Comparison of Mean SR. Homocystene With Blood Pressure

Blood pressure	No. Of pts		%		Sr. Homocysteine LevelMean ± SD		P-Value Significance
	Case	Control	Case	Control	Case	Control	
Hypertensive	27	0	37	0	18.06 ± 5.20	0	P-<.0001 Extremely significant
Normotensive	46	40	63	100	19.4 ± 15.53	8.5±2.7	
Total	42	42	100	100	18.7± 12.96	4.3±1.35	

Mean serum homocysteine levels were higher (19.4±15.53) in normotensive patients than in hypertensives where it was 18±10.4.

As a whole the mean serum homocysteine levels were much higher (18.7±12.96)in cases than in controls where it was 4.3±1.35 and the statistical difference was extremely significant (p<.0001).

Table -3 Comparison of Mean SR. Homocystene With onset stoke

Newely onset stoke	No.of pts		%		Sr. Homocysteine Level Mean+SD		P-value Significance
	Case	Control	Case	Control	Case	Control	
Newly onset	36	_	86	_	21.9±14.9	_	P=. 6300 (>.05) Non-significant
Re-stroke	6	_	14	_	18.9±4.19	_	
Total	42	_	100	_	20.4 ±9.54		

Mean and standard deviation of serum homocysteine levels were 21.9+_14.9 in patients with newly onset stroke and 18.9+_4.19 in re-stroke. The difference was statistically not significant (p=.6300).

Table-4: Comparison of Mean Serum Homocystene With Life Style

Lifestyle	No.Of Pts		%		Sr. Homocysteine Level Mean+_SD		P-Value Significance
	Case	Control	Case	Control	Case	Control	
Sedentary	36	36	86	86	19.78 ±12.38	10.2 ±2.8	P-<.0044 Very Significant
Active	6	6	14	14	11.01±10.42	10 ±3.1	
Total	42	42	100	100	15.44±11.40	10.1±3	

Mean serum homocysteine levels were higher (19.78 ± 12.38) in patients with sedentary lifestyle than in patients with active lifestyle where it was 11.01 ± 10.42).

But in controls mean serum homocysteine levels were almost same in both the groups.As a whole mean serum homocysteine levels were much more (15.44 ± 11.40) in cases than in controls where it was 10.13. The difference was statistically very significant (p<.0044).

Table-5: Comparision Of Mean Sr. Homocystene With Diet

Diet	No.of pts		%		SR.homocysteine Level Mean+_SD		P-value Significance
	Case	Control	Case	Control	Case	Control	
Vegeterian	4	4	10	10	21.76± 17.38	10 ± 2.4	P-<.0002 Highly significant
Mixed diet	38	38	90	90	17.5 ± 14.38	10.2 ± 2	
Total	42	42	100	100	19.64± 15.88	10.1± 2.2	

Result and Discussion

Mean serum homocysteine levels were higher (21.76 ± 17.38) in patients on vegetarian diet than in patients with mixed diet where it was 17.5 ± 14.35 but in control however it is approximately same in both groups. In total serum homocysteine levels were 19.64 micro mole/l in cases than 10.1 in control However the difference was statistically highly significant (p<.0002).

Conclusions

1) As a whole the mean serum homocysteine levels were much higher (18.7±12.96) in cases than in controls where it was 4.3±1.35 and thestatistical difference was extremely significant (p<.0001).
2) As a whole the mean serum homocysteine levels were much higher (18.7±12.96) in cases than in controls where it was 4.3±1.35 and thestatistical difference was extremely significant (p<.0001).
3) Mean and standard deviation of serum homocysteine levels were 21.9+_14.9 in patients with newly onset stroke and 18.9+_4.19 in re-stroke. The difference was statistically not significant (p=.6300).
4) Mean serum homocysteine levels were higher (19.78 ± 12.38) in patients with sedentary lifestyle than in patients with active lifestyle where it was 11.01 ± 10.42).
5) In total serum homocysteine levels were 19.64 micro mole/l in cases than 10.1 in control However the difference was statistically highly significant (p<.0002).

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