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Hyperhomocysteinemia in Young Adult with Stroke: A Case Report.

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ABSTRACT

Stroke is the third leading cause of death in the world. Stroke in young individual is rare and poses a major problem. The mechanisms of ischemic stroke in young adults are poorly understood. There are several studies to suggest the role of genetic factors and hyperhomocysteinemia predisposing to hypercoagulability states. In this case, a 37 year old male diagnosed as ischemic cerebro vascular accident with markedly elevated fasting serum homocysteine levels is presented. Fasting serum sample taken by venepuncture. Serum homocysteine levels were measured by fully automated chemiluminescence immunoassay. In this case, a 37 year old male, a known smoker and alcoholic presented with weakness of left upper limb, CT scan of brain revealed acute ischemic infarct in right parietal and semi ovale region. He had a markedly elevated fasting serum homocysteine level of $63\mu\text{mol/L}$, which responded well with folate supplements. Hyperhomocysteinemia could serve as an important etiological factor in young stroke. Nutritional deficiency plays an important role in hyperhomocysteinemic condition in this young stroke patient.

Keywords: Hyperhomocysteinemia, Stroke, Nutritional deficiency.

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INTRODUCTION

Homocysteine, a sulphur containing amino acid is formed as an intermediary metabolite during the conversion of essential amino acid methionine to cysteine. Hyperhomocysteinemia originates from any alteration in methionine homocysteine metabolism including enzyme abnormalities, vitamin deficiencies and other factors. Hyperhomocysteinemia has been associated with increased risk for cerebrovascular disease. The advantages of treating hyperhomocysteinemia with folic acid and vitamin supplementations are that they are cost effective and safe. So, determination of serum homocysteine levels will be valuable for patients who present with ischemic cerebrovascular accident in young age.

Case history

A 37 year old male, admitted with complaints of weakness of left upper limb and deviation of angle of mouth to the right side with past history of hemianopia 2 years back which improved spontaneously. Patient is a chronic smoker and alcoholic for ten years duration. There is no history of epilepsy, vasculitis, headache, infections or coagulation disorders. There is no family history of cerebrovascular disease.

On examination, his blood pressure was 160/100 mmHg and there was left UMN type facial palsy with no cerebellar signs.

Routine biochemical and hematological investigations were done. Echocardiogram was normal. CT brain findings were suggestive of acute ischemic infarct in right parietal and semi ovale region.

Patient was put on salt restricted diet, physiotherapy and was started on oral aspirin 150mg OD. ANA, ENA profile and serum homocysteine levels were obtained as per rheumatologist advise. ANA, ENA profile were negative with elevated serum homocysteine levels of about 63 μ mol/L.

On further evaluation for vitamin deficiencies, the results revealed a low serum folic acid levels 1.2ng/ml (ref : 2-9 ng/ml) and serum vitamin B12 levels of about 160 pg/ml (ref : 197-866 pg/ml).

Patient was then started with folic acid and vitamin B12 supplements following which serum homocysteine levels returned to normal levels, following which he even recovered from limb weakness.

DISCUSSION

Hyperhomocysteinemia has been associated with increased risk for cerebrovascular disease. Various causes could lead to hyperhomocysteinemia. Hereditary abnormalities that leads to disturbances of enzymes in methionine-homocysteine metabolism, vitamin deficiencies and other lifestyle factors like smoking, alcoholism are some of them.

Welch et al, Kaul et al, Spence et al suggested that hyperhomocysteinemia is an independent risk factor for atherosclerosis of coronary, cerebral and peripheral blood vessels. There is a synergistic effect between serum homocysteine levels, systemic hypertension, smoking and alcohol intake as etiological risk factors in leading to ischemic cerebrovascular accident in young adults [1-4].

This patient had all these risk factors present. Hankey et al [5] showed that it is not fully known how homocysteine act to cause atherosclerosis but it causes endothelial cell

injury and thereby it initiates pre mature atherosclerosis. An increase of $5\mu\text{mol/L}$ of homocysteine in serum elevates the risk of cerebro vascular disease by as much as cholesterol increase by 20mg/dl . Homocysteine interacts with lysyl residues of collagen interfering with collagen cross linking. It forms homocysteine thiolactone, a highly reactive free radical which thiolates LDL particles. These particles tend to aggregate, are endocytosed by macrophages and increase the tendency for atherogenesis [6].

Homocysteine can have other effects, including lipid oxidation and platelet aggregation, which in turn leads to fibrosis and calcification of atherosclerotic plaque. Tetrahydrofolate and vitamin B12 are also required for the conversion of homocysteine to methionine. Vitamin B12 and folate deficiency is common in alcoholics. Alcohol causes pathological alterations of the gastrointestinal tract that directly interfere with absorption of certain nutrients. Alcohol interferes with folate absorption [7]. Vitamin B 12 dependent methionine synthesis is the only pathway by which N5 methyl tetra hydro folate can return to the tetra hydrofolate pool. Thus in vitamin B12 deficiency and essentially all folate becomes trapped. Thus in the absence of these vitamins homocysteine – methionine metabolism is altered leading to hyperhomocystenemia which had happened in our case.

CONCLUSION

It will be useful to check homocysteine level in patients presenting as cerebrovascular accident in young age who show no clue for coagulation disorders. Treating those patients with combined vitamin supplements and physiotherapy will help the patient to recover from his weakness.

REFERENCES

- [1] Welch NG, Loscalzo J. N Engl J Med 1998;338:1042-50.
- [2] Spence JD, Bang H, Chambless LE, Stampfer MJ. et al. Stroke 2005;36:2404-2409.
- [3] Spence JD. Lancet Neurol 2007;6:830-838.
- [4] Spence JD. Clin Chem Lab Med 2007;45:1582-1585.
- [5] Hankey GJ. Nat Clin Pract Neurol 2006;2:26-33.
- [6] Mccully KS. Ann Clin Lab Sci 1993;23:477.
- [7] Lieber CS. JAM College Nutr 1991;10:602.