Pallab Kumar Ganguly, J Diabetes Metab 2014, 5:10
http://dx.doi.org/10.4172/2155-6156.S1.026

OMICS Group Conferences
Accelerating Scientific Discovery

5th World Congress on
Diabetes & Metabolism
November 03-05, 2014 Embassy Suites Las Vegas, USA

Diabetes, hyperhomocysteinemia and stroke

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Until recently it has been unclear whether non-insulin dependent diabetic (NIDD) people with the methylenetetrahydrofolatereductase (MTHFR) mutation have an increased risk of cardiovascular disease; however a recent meta-analysis has shown a significantly higher risk of ischemic heart disease in people with the MTHFR mutation. Prospective studies confirm that hyperhomocysteinaemia and MTHFR mutation are independent risk factor for cardiovascular morbidity and mortality in end-stage renal disease. Therefore, hyperhomocysteinaemia in NIDD patients with both incipient and clinical nephropathy may partly contribute to the increased risk of vascular disease.Elevated plasma levels of homocysteine are associated with the development of stroke but the mechanistic association of stroke with hyperhomocysteinemia is unclear. Because hyperhomocysteinemia is related to the development of atherosclerosis, this may explain the association of homocysteinemia with a stroke. However, the relationship of homocysteine with atherosclerosis has been questioned recently. In addition, hyperhomocysteinemia has now been associated with a variety of brain disorders including dementia and Alzheimer's disease. Thus, the search for mechanisms to explain the neuronal effects of homocysteine is an important, valid avenue of study in many neurological disorders. It is possible that the additional effects of homocysteine may involve a more direct action on the brain itself that could augment neuronal damage in a stroke. In support of such a hypothesis, homocysteine can induce direct neurotoxicity by activating the N-methyl-D-aspartate (NMDA) subtype of the glutamate receptor. Our research is based on a hypothesis that increased level of homocysteine in blood can trigger the release of excitatory amino acids and catecholamines in specific areas of hypothalamus. The effect may be more predominant in hypertension superimposed with NIDD. Therefore, both catecholamines and excitatory amino acids are detrimental factors in hyperhomocysteinemia, the missing links for the development of stroke in NIDD.

Biography

Pallab Kumar Ganguly completed his medical degrees before joining a Cardiovascular Group at St. Boniface Hospital Research Centre, Winnipeg, Canada. He worked several years at the University of Manitoba as a Postdoctoral fellow and then as a faculty. He is presently the Chairman of the Department of Anatomy at Alfaisal University and is heavily engaged in research in medical education and understanding homocysteine-induced pathophysiology of heart disease. He has edited two books: Catecholamine and Heart Disease (CRC press) and Education in Anatomical Sciences (Nova Publisher). He has also over 125 original publications in peer reviewed international journals. He is a well-known scientist and has received several awards for his scholarly work.

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