Opinion

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Acute Central Retinal Artery Occlusion Associated with Raised Homocysteine Levels

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Description

In patients with cardiovascular diseases, homocysteine is occasionally requested as a prognostic risk factor. As an intermediate product of the biosynthesis of methionine and cysteine and as a product of the demethylation of dietary methionine, homocysteine is an amino acid that contains sulfhydryls. By inducing oxidative stress and decreasing the amount of available nitric oxide, elevated plasma HCY may also increase the risk of thrombosis and cause direct endothelial cell injury. By acting as an agonist for glutamate receptors, metabotropic receptors, and ionotropic receptors, hyperhomocysteinaemia causes neurotoxic effects on neuronal cells, increasing levels of cytoplasmic calcium, free radicals, and caspases when these receptors are overstimulated. Additionally, it may disrupt the blood-brain barrier and cause damage to glial cells. An elevated plasma level of HCY was found to be a risk factor for ischaemic and recurrent strokes, but not for hemorrhagic stroke, according to a recent systematic review with meta-analysis of prospective observational studies. On the other hand, a different recent systematic review with a meta-analysis of case-control studies that included completely different articles from the previous systematic review has shown that haemorrhagic stroke patients have higher HCY levels than healthy controls, but there is no significant difference between ischaemic and haemorrhagic strokes in HCY levels. Additionally, homocysteine damages cardiovascular endothelium and smooth muscle cells as an independent risk factor for cardiovascular disease.

Anthraquinone Derivative

Treatment with vitamin B is thought to lower HCY levels, thereby lowering the risk of cardiovascular disease and stroke. However, observational studies and interventional trials have found opposite results, with the former group supporting the benefits of HCY-lowering vitamin B treatment for cardiovascular disease and stroke prevention while the latter group does not find any significant benefits. It has been demonstrated that the total homocysteine immunoassay performs well. Ion exchange chromatography and the immunoassay are correlated, but there are biases. If either method is used interchangeably, caution is required. Phosphate reabsorption in the kidney as a hormone synthesized by bone cells and the production and degradation of biologically active vitamin D are both controlled by fibroblast growth factor 23. In addition, there are additional paracrine effects in other organs. The FGF23 plasma concentration rises in renal and cardiovascular diseases and is linked to outcome as a biomarker. The regulation of FGF23 is influenced by a number of factors, including oxidative stress, and is poorly understood. L-homocysteine is an amino acid produced during methionine metabolism that can be transformed into additional metabolites if vitamin B is available. Hyperhomocysteinemia may increase the risk of cardiovascular disease. Our research sought to determine whether homocysteine has an effect on FGF23 synthesis. Consumption of a high-protein diet during pregnancy raises homocysteine levels in the adolescent rats' cerebral cortex. Consumption of a high-protein diet during pregnancy increases neuroinflammation in the adolescent rats' cerebral cortex. Consumption of a high-protein diet during pregnancy lowers serum histidine levels in adolescent rats.

Adolescent rats' memory and maturity are impaired when they consume a high-protein diet during pregnancy. Adolescent rats exhibit anxiety-like behaviors when fed a diet high in protein during pregnancy. In prepubertal and adult rats, administration of homocysteine to neonates resulted in hyperactivity and cognitive impairments. Homocysteine-administered prepubertal rats reverted to hyperactivity after receiving amphetamine. Administration of homocysteine to newborns: only prepubertal rats showed a decrease in social interaction after it was induced. In limbic regions that are associated with behavior and cognition, homocysteine decreased mushroom dendritic spines in newborns. The severe cardiac dysfunction brought on by elevated plasma homocysteine levels is closely linked to oxidative stress. It has been demonstrated that the naturally occurring anthraquinone derivative emodin has antioxidant and anti-apoptotic properties. However, it is unknown whether emodin could guard against Hcy-induced cardiac dysfunction. The purpose of this study was to investigate the molecular mechanisms of how emodin affected Hcy-induced cardiac dysfunction.

Cardiac Dysfunction

In order to construct the animal model of hyperhomocysteinemia rats were fed a diet high in methionine. In order to create a cell model of Hcy-injured cardiomyocytes, H9C2 cells were incubated with Hcy. In this study, ELISA, HE staining, cannulation of the carotid artery and left ventricle, MTT, fluorescence staining, flow cytometry, and western blotting were utilized. Emodin significantly reduced HHcy rats' cardiac dysfunction and structural myocardial damage. *In vitro*, Emodin inhibited apoptosis and MMP collapse in Hcy-treated H9C2 cells. The flow of blood through the uterus is a crucial aspect of embryonic development. Vascular damage may be associated with increased blood flow resistance in the uterine artery. Homocysteine can damage endothelial cells in a variety of ways. As a result, we look into the connection between uterine artery blood flow in the non-pregnant state and serum HCY levels in women who have experienced pregnancy loss.

A significant negative life event, pregnancy loss is defined as the spontaneous death of a pregnancy before the fetus reaches viability. A couple may experience varying degrees of anxiety and depression as a result of PL. Additionally; women with PL were more likely than women without PL to develop cardiovascular disease over time. Women and their families are negatively impacted by all of this. There are many different causes of pregnancy loss, but the etiology and pathogenesis of about 60% of PL remain a mystery. The only source of homocysteine is the byproduct of methionine demethylation, homocysteine. Homocysteine levels in the blood are typically low. HCY, which can cause a variety of blood vessel damage, is a significant biomarker for overall health status.