REVIEW ARTICLES

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The role of homocysteine in endothelial dysfunction

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Abstract

Background: Homocysteine is a sulfur-containing intermediate product in the normal metabolism of methionine, an essential amino acid. Hyperhomocysteinemia defines the state in which concentrations of homocysteine exceeds normal level. Homocysteine is located at a metabolic branch point and can either be irreversibly degraded to cysteine via the transsulfuration pathway, or conserved by remethylation back to methionine. Folic acid, vitamin B_{12}, and vitamin B_6 deficiencies and reduced enzyme activities inhibit the breakdown of homocysteine, thus increasing the concentration of intracellular homocysteine. Being cytotoxic, homocysteine is increasingly exported from the cell to become detectable in plasma. In recent years the amino acid homocysteine has achieved the status of an important factor in vascular disease, diseases of aging, and other fundamental processes in biology and medicine. Hyperhomocysteinemia may alter vascular morphology, stimulate inflammation, activate the endothelium and the blood clotting cascade, and inhibit fibrinolysis. As a result, hyperhomocysteinemia is associated with loss of endothelial antithrombotic function and induction of a procoagulant environment. The role of homocysteine in endothelial dysfunction is thought to be mediated by mechanisms including oxidative stress. Vascular injury could be caused by an imbalance between nitric oxide production from dysfunctional endothelial cells and homocysteine concentrations.

Conclusions: Hyperhomocysteinemia is associated with alterations in vascular morphology, loss of endothelial antithrombotic function, and induction of a procoagulant environment.

Key words: homocysteine, endothelial dysfunction, hyperhomocysteinemia, endothelium, oxidative stress.