Dealing with the Problems of Elevated Homocysteine

HELEN KIMBER BSC (HONS) PGCE

Heart disease is the nation's number one killer and we are all aware that high cholesterol is indeed a risk factor. However, many heart attack victims have cholesterol levels that would qualify as normal. So what else may be contributing to this frightening epidemic?

Smoking, obesity and high blood pressure all pose well known hazards. However, this does not explain why many people who suffer heart attacks have no known risk factors.

Fortunately, thanks to the pioneering work of Dr Kilmer McCully M.D. the amino acid homocysteine has been identified to play a critical role in destroying our arteries. Perhaps to the same extent as smoking!

Homocysteine is an amino acid. It is derived from methionine, an amino acid found in animal proteins. During normal metabolism any excess homocysteine is quickly eliminated with the help of certain nutrient co-factos, B6, B12 and Folic Acid. When the process is disrupted, levels of homocysteine build up and the consequences can be very troublesome.

Homocysteine metabolism also influences the biosynthesis of numerous compounds including glucosamine sulphate, coenzyme Q10, melatonin, carnitine, taurine and methionine.

Elevated plasma homocysteine levels (hyper-homocysteinaemia) appear to be related to dysfunction in the remethylation of homocysteine to methionine.

Homocysteine levels should be low in healthy individuals. Hyper-homocysteinaemia is now associated with cardiovascular and cerebrovascular diseases, [1-4] congenital defects of the heart and neural tube, [5,6] Type II diabetes mellitus, [7,8] rheumatoid arthritis, [9] psychogeriatric dementia [10] and schizophrenia.

Several nutrients are needed for optimal homocysteine metabolism. Moreover, nutritional intervention is the only therapy that has proven effective in reducing hyper-homocysteinaemia.

Vitamin B-6 is required for the conversion of homocysteine to cysteine. Dietary and plasma vitamin B-6 levels were found to be lower in people with hyper-homocysteinaemia, and were inversely correlated with the risk of myocardinal infarction. [12,13]

Pyridoxal-5-Phosphate (P5P) is the biologically active form of vitamin B-6, and is required for the trans-sulfuration of homocysteine. Low levels of P5P correlate with increased homocysteine, and confer an independent risk for coronary artery disease. [14]

Folic Acid may be the most significant vitamin in correcting homocysteine imbalances. It is crucial for the remethylation of homocysteine to methionine. A 14-year prospective cohort study published in JAMA found that higher intakes of folate alone, or in combination with vitamin B-6, substantially lowered rates of coronary heart disease. [15]

Vitamin B-12 deficiency is significantly correlated with elevated plasma homocysteine.[16] As with folate, the remethylation of homocysteine is dependent on adequate levels of Vitamin B-12.

Betaine (trimethylglycine) acts as a methyl donor in the metabolism of homocysteine to methionine. Research has demonstrated that long-term betaine supplementation substantially lowers plasma homocysteine levels, and that these levels can be maintained as long as betain is taken. [17]

One way of testing for elevated levels of homocysteine is to carry out a comprehensive cardiovascular risk profile from Great Smokies Diagnostic Laboratory.

As well as homocysteine levels this test also identifies an array of critical independent markers for cardiovascular disease. These are:-

- Lipoprotein (a)
- Apo A-1
- Apo B
- Fibrinogen
- Total Cholesterol
- C-reactive protein
- Triglycendes
- HDL Cholesterol
- LDL Cholesterol

By carrying out this blood profile a persons overall cardiac risk may be assessed. This can enable the practitioner to design a regime, particularly suited to the patients needs.

If the cardiovascular risk profile indicates high levels of homocysteine, the practitioner can take appropriate action by incorporating Vitamin B6, Vitamin B12, Folic Acid and Betaine into the nutritional support programme.

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