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Homocysteine, brain natriuretic peptide and chronic heart failure: a critical review

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Abstract

Chronic heart failure (CHF) is a major public health problem causing considerable morbidity and mortality. Recently, plasma homocysteine (HCY) has been suggested to be significantly increased in CHF patients. This article reviews the relation between hyperhomocysteinemia (HHCY) and CHF. Clinical data indicate that HHCY is associated with an increased incidence, as well as severity, of CHF. In addition, HCY correlates with brain natriuretic peptide (BNP), a modern biochemical marker of CHF, which is used for diagnosis, treatment guidance and risk assessment. Animal studies showed that experimental HHCY induces systolic and diastolic dysfunction, as well as an increased BNP expression. Moreover, hyperhomocysteinemic animals exhibit an adverse cardiac remodeling characterized by accumulation of interstitial and perivascular collagen. In vitro superfusion experiments with increasing concentrations of HCY in the superfusion medium stimulated myocardial BNP release independent from myocardial wall stress. Thus, clinical and experimental data underline a correlation between HHCY and BNP supporting the role of HHCY as a causal factor for CHF. The mechanisms leading from an elevated HCY level to reduced pump function and adverse cardiac remodeling are a matter of speculation Existing data indicate that direct effects of HCY on the myocardium, as well as nitric oxide independent vascular effects, are involved. Preliminary data from small intervention trials have initiated the speculation that HCY lowering therapy by micronutrients may improve clinical as well as laboratory markers of CHF. In conclusion, HHCY might be a potential etiological factor in CHF. Future studies need to explore the pathomechanisms of HHCY in CHF. Moreover, larger intervention trials are needed to clarify whether modification of plasma HCY by B-vitamin supplementation improves the clinical outcome in CHF patients.

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