Minerva Med. 1998 Nov-Dec;89(11-12):397-403.

# Are failured cardiomyopathies a zinc-deficit related disease? A study on Zn and Cu in patients with chronic failured dilated and hypertrophic cardiomyopathies.

Ripa S[1], Ripa R, Giustiniani S.

Author information

## Abstract

**BACKGROUND:** Muscle zinc deficit may be important in cardiac failure pathogenesis. We studied whether Zn deficit is present in dilated (DCM) and hypertrophic (HCM) cardiomyopathies with chronic cardiac failure. The aim was to find out whether it might be a diagnostic and prognostic marker.

**METHODS:** Zn and Cu values were measured in plasma, urine and red blood cells by flame atomic absorption spectrophotometer in normal subjects, in 15 patients with DCM and in 11 patients with HCM in chronic cardiac failure.

**RESULTS:** There is a statistically important increase of zincuria as well as low plasmatic and erythrocytic zinc in DCM; low plasmatic and erythrocytic zinc was found in HCM.

**CONCLUSIONS:** It is likely that chronic cardiac failure, by atrial natriuretic peptide activation, increases zincuria and involves a secondary loss of plasmatic and erythrocytic zinc. Primitive or secondary zinc deficit causes marked structural and functional myocardial cell impairment. Hypozinchemia from increased zincuria seems to represent an important diagnostic and prognostic marker in chronic failured cardiomyopathies. Dilated cardiomyopathy, particularly in elder subjects, may be considered as a primitive or a secondary Zn-deficit myocardial disease. In the essay, the possible pathogenetic mechanisms underlying hypozinchemia and determining functional and structural myocardial cell changes are discussed. The main consequence of present results is pharmacological Zn administration as a new pathogenetic therapy in chronic failured

cardiomyopathies.

PMID: 10212663

[Indexed for MEDLINE]

## MeSH terms, Substances

## LinkOut - more resources