TIROIDE E SCOMPENSO CARDIACO

QUESTIONS & ANSWERS

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HYPOTHYROIDISM
HYPERTHYROIDISM
T3 availability controlled by deiodinases
T3 fundamental for cardiac morphology and performance
Non-genomic effects
Genomic effects: TR (α1, α2, β1, β2)
TRβ required for angiogenesis in cardiac development
“Foetal gene program” (lowT3): ↑ cells proliferation, ↓ differentiation
Restored in CHF, hypothyroidism, lowT3
20-30% of HF lowT3 in NYHA grado elevato

Low T3 syndrome leads to changes in cardiac function and gene expression similar to hypothyroidism

Low T3 is an adaptative response in HF.

It should be treated? Why? How? New drugs?
HYPERTHYROIDISM

↑ Cardiac output, ↑ Fc, ↑ preload, ↓ resistance, hyperdinamic circulation

↓ Renal perfusion, ↑ renin/aldo

↑ Oxygen demand, ↑ energy consumption, ↓ impossible adequate output during exercise

Prolonged Hyper, hypertrophy, ↓ ventricular performance, ↑ HF
HYPERTHYROIDISM

Both, overt and subclinical hyperthyroidism, cause HF

Why? Differences?

Increased cardiac mortality in treated hyperthyroid patients

First therapy?
Hypothyroidism

AIT type 1, hyper, pre-existing disease, low I intake, ↑ flow

AIT type 2, thyreotox, not pre-existing disease, IL6, no flow

AIT & impaired left ventricular ejection: high risk major CV events

Treatment?
Resistance to thyroid hormone (RTH): TR β mutation

TR β gene, chromosome 3, T3-binding isoforms (β1, β2, β3), hypothalamus-pituitary

TR α gene, chromosome 17, isoforms (α1, α2, α3), heart

Due to α predominance vs β in myocardium, possible relative sparing of the heart in RTH

Symptoms patients RTH (↑ cardiac contractility, ↑ Fc) similar to hyperthyroidism

Others hypothyroidism (Pulcrano JCEM 2009).

Why? Cardiac condition in RTH?
Thanks for your answers…