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Is the myocardium source or target of non-thyroidal illness syndrome? Observations on local thyroid hormone metabolism in chronic heart failure and controls.

Johannes W Dietrich[1], Axel Kloppe[2], Leif I. Bösche[2], Apostolos Chatzitomaris[1], Harald H. Klein[1], Andreas Mügge[2], Assem Aweimer[2]

[1]Department of Endocrinology and Diabetes, Medical Hospital I, Bergmannsheil University Hospitals, Ruhr University of Bochum, D-44789 Bochum, NRW, Germany
[2]Department of Cardiology and Angiology, Medical Hospital II,Bergmannsheil University Hospitals, Ruhr University of Bochum, D-44789 Bochum, NRW, Germany

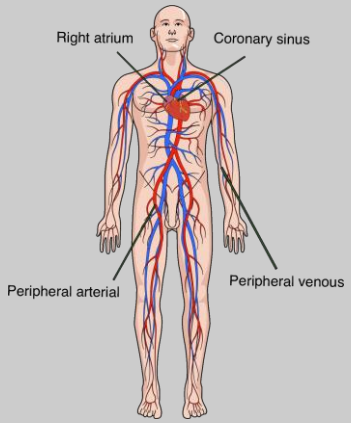
Background

The relation between thyroid homeostasis and myocardial function is governed by complex interactions. Recently published data suggest a U-shaped relationship between serum levels of FT3 and the risk of atrial fibrillation. In addition, heart failure (HF) may result from both hypo- and hyperthyroidism. In this study, we examined the intracardiac thyroid hormone metabolism in patients with and without systolic HF.

Methods

This study covered 11 patients with HF (LV-EF<35% and NYHA II/III) undergoing implantation of a CRT (cardiac resynchronization therapy) device, and a control group of six age- and sex-matched subjects without HF (LVEF> 55%) planned for electrophysiological testing. Exclusion criteria were thyroid disease or exposition to iodinated radiocontrast agents within three months before investigation.

In all patients, blood samples were obtained simultaneously from various compartments (peripheral-venous, peripheral-arterial, right atrium, coronary sinus). Concentrations of TSH, FT3, FT4 and BNP were determined in all samples. Total deiodination capacity (SPINA-GD) was calculated from measured values for FT3 and FT4. Distributions of hormone concentrations were compared with unpaired two-sided t test and confirmed with a linear mixed effects model.



Results

While TSH levels didn't differ among all groups and compartments, FT3 concentrations and SPINA-GD were significantly reduced and concentrations of FT4 and BNP were increased in peripheral venous samples of patients with HF. Similarly, FT3 was reduced in right atrial samples, and SPINA-GD was decreased in the coronary sinus of patients suffering from HF. BNP concentrations were increased in all compartments in patients with HF, but highest in samples from coronary sinus.

	Heart failure (Mean ± SD) [n = 11]	Control [n = 6]	p		Heart failure (Mean ± SD) [n = 11]	Control [n = 6]	p
Peripheral venous				**Right atrium**			
TSH mIU/l	1.46 ± 0.89	1.32 ± 0.48	0.7028	TSH mIU/l	1.61 ± 0.85	1.24 ± 0.44	0.2540
FT3 pmol/l	4.31 ± 0.72	5.30 ± 0.67	**0.0163***	FT3 pmol/l	4.30 ± 0.80	5.37 ± 0.52	**0.0052****
FT4 pmol/l	13.36 ± 2.16	10.66 ± 1.75	**0.0190***	FT4 pmol/l	12.92 ± 1.93	10.46 ± 2.34	0.0566
SPINA-GD nmol/sec	30.43 ± 5.44	47.29 ± 11.09	**0.0117***	SPINA-GD nmol/sec	33.11 ± 9.91	50.87 ± 19.53	0.1139
BNP pmol/l	148.55 ± 13.067	6.55 ± 3.83	**0.0048****	BNP pmol/l	154.64 ± 143.76	10.35 ± 4.98	**0.0076****
Peripheral arterial				**Coronary sinus**			
TSH mIU/l	1.53 ± 0.96	1.22 ± 0.38	0.3556	TSH mIU/l	1.66 ± 0.98	1.22 ± 0.42	0.2125
FT3 pmol/l	4.32 ± 0.96	5.02 ± 0.45	0.0609	FT3 pmol/l	4.18 ± 1.10	4.95 ± 0.55	0.0686
FT4 pmol/l	12.92 ± 2.78	11.94 ± 1.55	0.3697	FT4 pmol/l	12.33 ± 1.92	10.46 ± 1.70	0.0605
SPINA-GD nmol/sec	32.30 ± 10.08	39.64 ± 8.04	0.1254	SPINA-GD nmol/sec	31.89 ± 9.86	44.55 ± 6.86	**0.0080****
BNP pmol/l	156.23 ± 166.28	6.89 ± 4.72	**0.0194***	BNP pmol/l	437.36 ± 442.26	22.16 ± 12.75	**0.0110***

Conclusions

In the HF group samples from peripheral veins reflected a slight, but classical pattern of non-thyroidal illness syndrome (NTIS or TACITUS). A similar constellation was observable in samples from right atrium and coronary sinus. Probes from peripheral arteries didn't show, however, any pattern of TACITUS. The results suggest that local deiodinases are downregulated in myocardial tissue of patients with HF. Therefore, peripheral organs and the heart, but not the lung, may contribute to the constellation of NTIS. This spatial diversity in the pathogenesis of NTIS may be promotive for cardiac complications, including atrial fibrillation, in critical illness.