

A Treatable Hormone Imbalance May Dramatically Accelerate Progression to Heart Failure

A Martin Gerdes*

Department of Biomedical Sciences, New York College of Osteopathic Medicine, New York Institute of Technology, USA

Introduction

Heart failure is currently the number one killer and most expensive single diagnosis in developed countries. Despite improved treatment over the past few decades, more people are living with heart failure, leading to an increase in the overall incidence. Clearly, more effective treatments are needed. What if we learned that a treatable, cardiac hormone deficiency that is known to actually cause heart failure may be present in most cardiac patients but largely going undetected and/or ignored? What if correction of this hormone deficiency could be done safely, improve coronary blood flow and contractile function, reverse neurohormonal activation, and also reverse most of the abnormally functioning cardiac genes associated with heart failure? Certainly, any newly developed, patentable drug accomplishing this feat would receive considerable attention. But alas, I am referring to generic thyroid hormones, something that would save, rather than incur billions in global health costs if proven effective. Yes, thyroid hormone treatment of heart disease has been suggested before and the medical community is flatly opposed, believing the issue was settled long ago. But, if one takes a fair and objective look at the older literature that largely shaped the current opinion, the conclusion should be that “toxic” doses of thyroid hormones can lead to increased arrhythmias and mortality. The issue of long-term use of “therapeutic” doses of thyroid hormones in chronic heart failure has never been adequately studied, despite positive results from many short-term studies. More recent information also provides important mechanistic insight and begs re-examination of the topic [1]

In the past, it appears that thyroid hormones were considered a possible treatment largely for their potential to stimulate a weakly performing heart. It is understandable that initiating treatment with this mindset could easily lead to overdosing. New evidence from animal studies suggests that heart diseases, possibly of all etiologies, promote cardiac tissue hypothyroidism by activating the D3 deiodinase. D3 activation leads to increased conversion of both T4 (to rT3) and T3 (to T2) to inactive forms. Cumulative data from humans suggests the same situation likely occurs, although no cardiac tissue thyroid hormone data are available at this time. It is clear that chronic, low cardiac tissue T3 levels leads to impaired coronary blood flow, impaired contractility, impaired relaxation, maladaptive remodeling of cardiac myocytes and small arterioles, and eventual heart failure. Rather than thinking of thyroid hormones as another positive inotrope, the target should be restoration of normal thyroid hormone function at the cardiac tissue level.

How extensive is low cardiac tissue T3 in human heart disease? A study by Iervasi et al. [2] indicated that 20% of cardiac patients at their institution had overt hypothyroidism, another 30% had low T3 syndrome, and most of the remaining patients were in the lower half of the normal reference range. Based on this information, it is not unreasonable to suspect that most cardiac patients are likely to have low cardiac T3 levels. Importantly, our lab and others have observed evidence of low cardiac T3 in virtually every animal model of heart disease examined to this point (e.g. hypertension, myocardial infarction, and idiopathic dilated cardiomyopathy), despite normal serum thyroid hormone levels in some cases.

Can thyroid hormones be safely administered in humans with heart disease? Many short-term studies have shown that thyroid hormones can be safely used in heart patients. Importantly, thyroid hormone diagnostics have improved considerably since many of the previous studies were conducted. Which form should be used, T3 or T4? Since it appears that the heart is actively destroying T4 in individuals with heart disease, T3 will likely prove to be a more direct, better alternative. Nonetheless, comparative studies are needed. Since it is impractical to obtain cardiac tissue T3 levels from heart patients, how can the most effective dose be determined? This is an important question that is largely unresolved by the current literature. Fortunately, two just completed studies from our lab may provide an answer. Oral T3 was administered in drinking water to two different rat models of heart disease. Untreated rats from both models had low cardiac tissue T3 with serum thyroid hormone profiles showing hypothyroidism in one case and euthyroidism in the other. T3 dose was increased until serum TSH and T4 showed suppression but remained within the normal reference range. Serum T3 also remained within normal limits in both treated models. Using this approach, cardiac tissue T3 levels were restored in both heart disease models along with significant improvements in cardiac function. This approach could easily and safely be translated to patients. Positive responders would likely be obvious within a few days. What about the possibility of inducing arrhythmias with treatment? Clearly, hyperthyroidism can lead to increased incidence of arrhythmias. Interestingly, hypothyroidism and subclinical hypothyroidism may also lead to increased incidence of arrhythmias. Since most cardiac patients likely have tissue hypothyroidism, it is likely that withholding thyroid hormones, the current approach, may actually result in more arrhythmias.

Conclusion

Cumulative evidence, particularly within the past few years, suggests that low cardiac tissue thyroid hormones likely play a major adverse role in progression of heart diseases to heart failure. It is time to move past long-standing prejudices based largely on weak and inadequate science, recognize the cumulative impact of new information, and move forward with clinical trials. Rarely does medicine have an opportunity to improve health care while also reducing costs.

*Corresponding author: A Martin Gerdes, Department of Biomedical Sciences, New York College of Osteopathic Medicine, New York Institute of Technology, Northern Blvd, Old Westbury, New York, USA, Tel: 516-686-4039; Fax 516-686-3832; E-mail: agerdes@nyit.edu

Received November 27, 2012; Accepted November 27, 2012; Published November 29, 2012

Citation: Gerdes AM (2013) A Treatable Hormone Imbalance May Dramatically Accelerate Progression to Heart Failure. *Thyroid Disorders Ther* 2: e111. doi:10.4172/2167-7948.1000e111

Copyright: © 2013 Gerdes AM. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

References

1. Gerdes AM, Iervasi G (2010) Thyroid replacement therapy and heart failure. *Circulation* 122: 385-393.
2. Iervasi G, Pingitore A, Landi P, Raciti M, Ripoli A, et al. (2003) Low-T3 syndrome: a strong prognostic predictor of death in patients with heart disease. *Circulation* 107: 708-713.