Journal of Thyroid Disorders & Therapy
ISSN: 2167-7948

Gerdes et al, Thyroid Disorders Ther 2015, 4:3
DOI: 10.4172/2167-7948.1000e121

Editorial Open Access

When to Treat Borderline Hypothyroidism? Time to Listen to Your Patient!

A Martin Gerdes[1] **and Mervat Mourad**[2]

[1]*Department of Biomedical Sciences, New York Institute of Technology, College of Osteopathic Medicine, Old Westbury, NY 11568-8000, USA*

[2]*Department of Medicine, New York Institute of Technology, College of Osteopathic Medicine, Old Westbury, NY 11568-8000, USA*

[*]**Corresponding author**: Department of Biomedical Sciences, New York Institute of Technology, College of Osteopathic Medicine, Old Westbury, NY 11568-8000, USA, Tel: 516-686-4039; E-mail: agerdes@nyit.edu

Rec date: May 26, 2015; **Acc date:** May 27, 2015; **Pub date:** June 5, 2015

Editorial

Subclinical hypothyroidism is a common problem that affects about 3-8% of the population [1]. Subclinical hypothyroidism is defined as mildly elevated TSH levels with normal free thyroxine levels [2,3]. Subclinical hypothyroidism carries a risk of progression to overt hypothyroidism, a condition effecting 2-5% of the population annually [1]. Patients with overt hypothyroidism may present a wide array of symptoms and signs that include fatigue, slow movement, slow speech, eye problems, joint aches, cold intolerance, constipation, weight gain, menstrual disturbances, infertility, bradycardia, depression, neurological problems, encephalopathy and even coma. This long list of symptoms and signs can be confused with other medical conditions and can obfuscate the true and primary issue [4]. Some of the symptoms can be misinterpreted as depression, chronic fatigue syndrome or anxiety disorder. To this backdrop, a physician is presented with a crucial decision that many must make when they encounter a patient with subclinical hypothyroidism: whether or not to treat the patient with thyroid replacement therapy. In the context of our busy clinical settings, there is heavy reliance on lab values. Often times, the physician's action may be over-reliant on the lab values without paying particularly close attention to the patient's symptoms in these borderline cases.

While almost all experts recommend treatment of patients with serum thyroid-stimulating hormone (TSH) concentrations >10 mU/L, the routine treatment of asymptomatic patients with TSH values between 4.5 and 10 mU/L is still controversial [3, 5-9]. Recent studies have shown strong correlation between subclinical hypothyroidism and the risk of cardiovascular diseases such as coronary artery disease and heart failure [10,11]. This risk is even higher in younger populations [12,13].

Thyroid hormones are deemed to be key regulators that control different chemical and physiological processes in the cardiovascular system. Recent studies have shown increased mortality in heart failure patients with low thyroid function [14-16]. Therefore, low thyroid hormone levels are related to and involved in cardiovascular death, which is the number one cause of death in the United States [17]. Despite the fact that cardiovascular disease and stroke are closely related, the data demonstrating a correlation between subclinical hypothyroidism and stroke are still inadequate and inconsistent [18]. Recent animal studies may provide some insight (reviewed in [17]). It appears that heart diseases, in general, trigger low cardiac T3 levels, a change that is believed to result from increased local expression of the D3 deiodinase. Importantly, it appears that a reduction in cardiac tissue T3 can lead to pronounced cardiac dysfunction even though serum hormone levels may be normal [19]. While a discussion of this topic is well beyond the current editorial, this new information suggests that maladaptive effects on the heart may extend beyond individuals with diagnosable borderline thyroid dysfunction. It does not help matters that cardiac manifestation of heart failure and hypothyroidism are largely indistinguishable. Clearly, a serum biomarker tracking with low cardiac tissue T3 would be extremely helpful.

Many patients with insufficient thyroid hormone levels likely go underdiagnosed and untreated. In contrast, they are given non-specific diagnoses like “chronic fatigue syndrome,” “fibromyalgia,” “depression,” and “anxiety disorder.” These diagnoses, in reality, are just a constellation of their symptoms, since doctors don’t realize the exact cause. These symptoms provide an opportunity for excessive use of anti-depressants, anti-psychotics, muscle-relaxants, stimulants, anti-epileptics, pain relievers and other products. These drugs do not fix the problem and can often make it worse. They intervene with normal brain function producing changes in feelings, thought and mood that may be misinterpreted as an improvement. Medical ethics demands clinicians search for the cause and fix the primary problem if feasible.

Furthermore, treatment of subclinical hypothyroidism will prevent transformation to overt hypothyroidism, especially in those with serum TSH concentrations greater than 10 to 15 mU/L. Treatment in patients with smaller elevations in serum TSH concentrations may possibly ameliorate nonspecific symptoms of hypothyroidism, such as fatigue, constipation or depression. Treatment may also increase cardiac muscle contraction and improve serum lipid profile in some patients and consequently reduce the risk of coronary heart disease. Therefore, when a diagnosis of symptomatic subclinical hypothyroidism is made, physicians should adequately treat to ensure mitigation of the hormonal imbalances given the potential benefits. Importantly, we rarely know the normal thyroid hormone status of a given patient when they were well. A given patient who has moved from one end of the normal hormone reference curve to other may be symptomatic despite testing within the normal range. Paying particularly close attention to those patients at the extremes of the normal reference range is certainly a good idea.

Conclusion

In conclusion, clinicians should remain vigilant when it comes to treating a patient with solely elevated TSH or subclinical hypothyroidism. Clinicians should always look into the clinical context and take their patients’ complaints and symptoms into consideration. Given the potential to develop overt hypothyroidism and the possible adverse effects of unattended hormonal imbalances, clinicians have to periodically follow their asymptomatic patients with subclinical hypothyroidism. Physicians should treat their patients, not the lab values.

References

1. Khandelwal D, Tandon N (2012) Overt and subclinical hypothyroidism: who to treat and how. Drugs 72: 17-33.
2. Bell RJ, Rivera-Woll L, Davison SL, Topliss DJ, Donath S, et al. (2007) Well-being, health-related quality of life and cardiovascular disease risk profile in women with subclinical thyroid disease - a community-based study. Clin Endocrinol (Oxf) 66:548-556.
3. Biondi B, Cooper DS (2008) The clinical significance of subclinical thyroid dysfunction. Endocr Rev 29: 76-131.
4. McDermott MT (2009) In the clinic. Hypothyroidism. Ann Intern Med 151: ITC61.
5. Biondi B, Cooper DS (2008) The clinical significance of subclinical thyroid dysfunction. Endocr Rev 29: 76-131.
6. Cooper DS (2001) Clinical practice. Subclinical hypothyroidism. N Engl J Med 345: 260-265.
7. Garber JR, Cobin RH, Gharib H, Hennessey JV, Klein I, et al. (2012) Clinical practice guidelines for hypothyroidism in adults: cosponsored by the American Association of Clinical Endocrinologists and the American Thyroid Association. Thyroid 22: 1200-1235
8. Gharib H, Tuttle RM, Baskin HJ, Fish LH, Singer PA, et al (2005) Subclinical thyroid dysfunction: a joint statement on management from the American Association of Clinical Endocrinologists, the American Thyroid Association, and the Endocrine Society. The Journal of clinical endocrinology and metabolism 90: 581-585.
9. McDermott MT, Ridgway EC (2001) Subclinical hypothyroidism is mild thyroid failure and should be treated. J Clin Endocrinol Metab 86: 4585-4590.
10. Gencer B, Collet TH, Virgini V, Bauer DC, Gussekloo J, et al. (2012) Subclinical thyroid dysfunction and the risk of heart failure events: an individual participant data analysis from 6 prospective cohorts. Circulation 126: 1040-1049
11. Rodondi N, den Elzen WP, Bauer DC, Cappola AR, Razvi S, et al. (2010) Subclinical hypothyroidism and the risk of coronary heart disease and mortality. JAMA 304: 1365-1374.
12. Gussekloo J, van Exel E, de Craen AJ, Meinders AE, Frölich M, et al. (2004) Thyroid status, disability and cognitive function, and survival in old age. JAMA 292: 2591-2599.
13. Razvi S, Shakoor A, Vanderpump M, Weaver JU, Pearce SH (2008) The influence of age on the relationship between subclinical hypothyroidism and ischemic heart disease: a metaanalysis. J Clin Endocrinol Metab 93: 2998-3007.
14. Chuang CP, Jong YS, Wu CY, Lo HM (2014) Impact of triiodothyronine and N-terminal pro-B-type natriuretic peptide on the long-term survival of critically ill patients with acute heart failure. Am J Cardiol 113: 845-850.
15. Mitchell JE, Hellkamp AS, Mark DB, Anderson J, Johnson GW, et al. (2013) Thyroid function in heart failure and impact on mortality. JACC Heart Fail 1: 48-55.
16. Tseng FY, Lin WY, Lin CC, Lee LT, Li TC, et al. (2012) Subclinical hypothyroidism is associated with increased risk for all-cause and cardiovascular mortality in adults. J Am Coll Cardiol 60: 730-737.
17. Gerdes AM (2015) Restoration of thyroid hormone balance: a game changer in the treatment of heart failure? Am J Physiol Heart Circ Physiol 308: H1-10.
18. Chaker L, Baumgartner C, Ikram MA, Dehghan A, Medici M, et al. (2014) Subclinical thyroid dysfunction and the risk of stroke: a systematic review and meta-analysis. Eur J Epidemiol 29: 791-800.
19. Weltman NY, Ojamaa K, Schlenker EH, Chen YF, Zucchi R, et al. (2014) Low-dose T replacement restores depressed cardiac T levels, preserves coronary microvasculature, and attenuates cardiac dysfunction in experimental diabetes mellitus. Mol Med 20:302-312.