Journal of Thyroid Disorders & Therapy
ISSN: 2167-7948

McAdams et al., Thyroid Disorders Ther 2015, 4:3
DOI: 10.4172/2167-7948.1000185

Case report Open Access

How does Alcohol Use affect Thyroid Function? Illustrative Case and Literature Review

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Rec date: May 27, 2015; **Acc date:** June 17, 2015; **Pub date:** June 23, 2015

Keywords Thyroid; Alcohol; Euthyroid sick syndrome

Introduction

Excessive alcohol use, in the form of either chronic dependence or binge drinking, is a large economic burden on the U.S. health care system, being responsible for over $2.9 billion yearly in health-related costs [1] and an estimated 1.8 million annual hospital admissions [2]. The classic symptoms of acute alcohol withdrawal include tremors, anxiety, nausea, emesis, palpitations, and irritability; these symptoms could easily be confused with those found in thyrotoxicosis states. Further, thyroid function studies in patients who consume alcohol are often abnormal and sometimes lead to unnecessary work up on the part of clinicians. Previous reports have demonstrated a normalization of thyroid function parameters with alcohol cessation [3]. The following case study describes changes in thyroid levels in a patient with alcohol use and highlights the need for their careful interpretation.

Case Report

A 61 year old female presented to the emergency department (ED) with complaints of dyspnea on exertion, palpitations, occasional tremor, and fatigue. She denied any past medical history; however, she had not seen a physician in more than twenty years. She denied a family history of thyroid dysfunction. She admitted to consuming two to three alcoholic mixed drinks every evening, with a greater intake on the weekends. Physical examination revealed an overweight disheveled female. She weighed 102 kg, her height was 162 cm, and her body mass index was 38.9 kg/m^2. Vitals included a blood pressure of 111/75 mmHg, and she had tachycardia with a heart rate of 106 beats per minute. Her thyroid was not enlarged and there were no palpable nodules. No evidence of thyroid bruit or cervical lymphadenopathy. She did not demonstrate exophthalmoses or lid lag. There was a fine tremor of the hands noted bilaterally. She was found to be anemic, possibly from bleeding from an upper gastrointestinal source, and noted to have cirrhotic liver changes on imaging.

Thyroid function tests revealed a subnormal thyroid stimulating hormone (TSH) of 0.24 uIU/ml (reference range 0.35-4.500 uIU/ml), free thyroxine (free T4) 1.85 ng/dl (reference range 0.76-1.46 ng/dl), and free triiodothyronine (free T3) 1.63 pg/ml (reference range 2.18-3.98 pg/ml). Thyroid peroxidase antibodies and thyroid stimulating immunoglobulin tests were negative. Thyroid ultrasound was unremarkable (Figure 1), and a radioactive iodine uptake (RAIU) test could not be performed as patient had computed tomography angiogram earlier in the hospitalization to evaluate for pulmonary embolism.

A clear etiology of the patient's abnormal thyroid hormone levels was uncertain; however, it was felt that alcoholism may be a contributing factor. No active treatment was started and observation was deemed appropriate. Three days later repeat laboratory testing revealed a near-normalization of free T4 (1.48 ng/dl) and free T3 (2.11 pg/ml), with an increase in TSH toward normal (0.39 uIU/ml).

Discussion

Studies describing the relationship between the hypothalamic-pituitary-thyroid (HPT) axis and alcoholism are found largely in the psychiatric literature, since an association between the severity of withdrawal and thyroid dysfunction has been noted for a long time [4]. Alcoholism has been known to cause both central HPT axis and peripheral thyroid hormone dysfunction [4,5]. The mechanism involved in the alterations found in thyroid parameters of alcoholic patients during withdrawal have been studied in an animal model [5]. They include a reduction of type II 5'-deiodinase activity, the enzyme that converts T4 to T3, which could explain the increase in T4 and decrease in T3. Most studies have demonstrated a blunting of TSH release in response to thyrotropin-releasing hormone (TRH), even after several weeks of sobriety, as well as decreased total and free triiodothyronine (T3) [5-7]. However, total and free thyroxine (T4) concentrations have been found to be normal, decreased, or elevated at various times during detoxification. Given the observational alterations of the HPT axis throughout the stages of alcohol withdrawal and abstinence, interpretation of thyroid dysfunction in alcoholism can be difficult as various patterns can occur.

Although there have been several small studies over the last sixty years assessing the relationship between alcohol and thyroid function, our literature search was limited to larger studies of greater than 30 patients over the last twenty years, using the Medline/PubMed key words 'thyroid hormone,' 'alcohol,' 'hypothalamic-pituitary-thyroid axis,' and 'alcoholism.' Peripheral thyroid hormone levels were assessed in 5 studies that included a total of 1719 patients (Table 1).

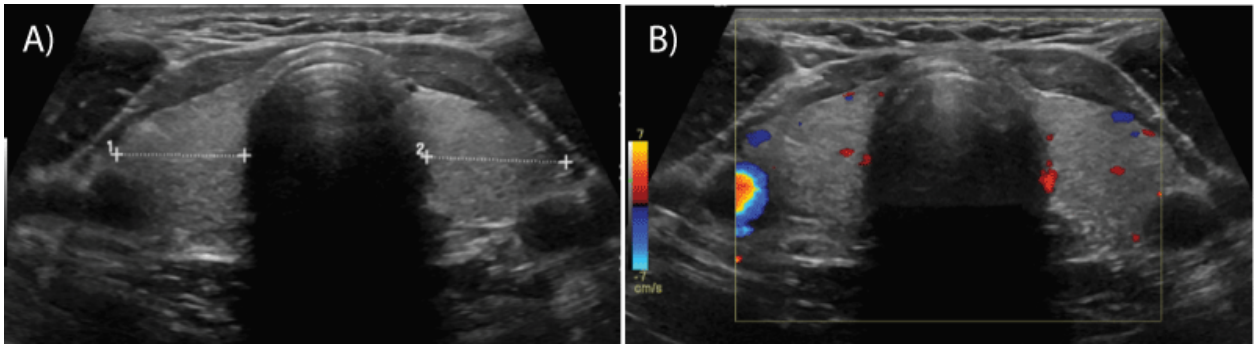

Figure 1: The transverse ultrasound (A) and color Doppler images (B) of the thyroid gland demonstrate both lobes of the thyroid being normal in size, contour, and echogenicity, without focal abnormalities. The isthmus was normal measuring 2 mm. There was no surrounded adenopathy or other abnormalities.

Study	Number of patients	TSH	Free T4	Total T4	Free T3	Total T3	Comment
Valeix [9]	1493	Normal	Decreased	-	-	-	Decrease in free T4 notable in men only and was associated with high alcohol consumption.
Liappas [10]	100 (followed on day 1 and week 4 of cessation)	Decreased Decreased	-	Decreased Normal		Decreased Elevated	30% of patients had normal T4, and elevated T3. Normalization was noted in majority of patients by week 4 of sobriety.
Ozsoy [11]	39 (Followed day 1 – 28)	Normal Decreased	Normal Decreased	-	Normal Decreased	-	Patients with normal values at day 1, reassessed at day 28
Leggio [12]	42	Decreased	Decreased	-	Decreased	-	Values returned to normal after abstinence but recurred with alcohol relapse.
Heinz [13]	45 (Day 1 -8)	Normal Normal	Increased	Decreased Increased	Increased	Normal	Study continued to follow patients at 3 and 6 months; those who relapsed into alcohol use had decrease in T4 compared with abstinent patients.

Table 1: Studies evaluating thyroid levels and alcohol intake.

Besides a search of large studies dating back over the last twenty years, appraisal of older systematic reviews demonstrated conflicting results. In one such review of 33 empirical studies assessing thyroid function in alcoholism from 1980-2001, the most consistent abnormality was reduction in T4, T3, and free T3 during early abstinence, with one-third of all alcoholics having a blunted TSH response to thyrotropin-releasing hormone test [8].

The variability of thyroid function tests in the above studies suggest that the mechanism by which alcohol abuse disrupts the HPT axis is multifactorial. In most situations alcoholism tends to cause a reduction of peripheral thyroid hormones, including total and free T3/T4, reminiscent of the euthyroid sick syndrome (ESS) [9]; though other studies demonstrate elevated levels. However, despite the appearance of a ESS type picture, alcoholism related thyroid dysfunction does not appear to share the same mechanism, as reverse triiodothyronine (rT3) has been found to be normal or reduced in several studies [5-7]. Perhaps concomitant depression and other psychiatric disorders common to this population are responsible for these inconsistencies [10,11]. Further, confounding the picture is both the physiological/psychological stress of detoxification, and the possibility of malnutrition with subsequent iodine deficiency in chronic abusers [7]. Still another consideration needs to be given to the direct cellular toxicity on thyroid cells by alcohol. Finally, regarding impairment at the hypothalamus and pituitary, it has been hypothesized that chronic alcohol abuse may cause an increase in TRH, thus down regulating pituitary TRH receptors and subsequently causing a reduced TSH response to TRH testing [12,13].

With alcohol being abused at an alarming rate, and ranking among the top five risk factors for disease, disability and death according to the World Health Organization [1], it is to be expected that physicians will encounter abnormal thyroid function tests in the setting of alcohol use, abuse, withdrawal, and detoxification. With several studies suggesting a complex and sometimes contradictory interrelation of alcohol and thyroid function, laboratory data needs to be interpreted

with caution. As was the scenario with our case report, most often a trend towards normalization of thyroid tests with continued sobriety is to be expected [11]. The literature also supports that close observation and avoidance of unnecessary testing rather than premature diagnostic decisions or hurried therapeutic intervention may be the correct course of action in these complex patients.

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