Chapter 11
Anxiety and Depression

ABSTRACT

It is an established entity that thyroid hormones play an important role in mood, behavior, and cognition. Autoimmune thyroiditis, including Hashimoto’s thyroiditis (HT), can lead to depression and anxiety disorders. It is thus important to enhance awareness among physicians about this connection to accelerate the diagnostic process. In patients with depression and anxiety disorders, a test for autoimmune thyroiditis should be performed, and in patients with autoimmune thyroiditis, a screening for psychiatric symptoms is necessary. Thus, patients presenting with anxiety or depression disorders should be treated and monitored by both an endocrinologist and a psychiatrist in liaison with each other so as to optimize their management. Moreover, an early recognition of an endocrine condition will help minimize psychiatric morbidity and hence improve health. This chapter explores anxiety, depression, and Hashimoto’s disease.

INTRODUCTION

Autoimmune thyroiditis (AIT) is considered a major health problem with implications for both patients and physicians as it causes somatic and psychiatric disorders the extent of which is not fully known so far.

Hashimoto’s thyroiditis denotes an autoimmune thyroid condition in which the thyroid gland is attacked by the immune system resulting in impaired thyroid function (hypothyroidism). During destruction of thyroid gland cells, it is possible to experience increase in hormone levels temporarily resulting

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in transient hyperthyroidism (Hashitoxicosis or thyrotoxicosis). When the high level of hormones is managed by the body, the thyroid function starts to decline resulting in hypothyroidism, which causes depression and anxiety.

Carta et al. (2004) suggested that people with thyroid autoimmunity are at increased risk of anxiety and mood related disorders. It indicated that both conditions are caused by the same aberrancy present in the immuno-endocrinal center, which is difficult to correct.

**Effect of Thyroid Hormone on the Central Nervous System**

Thyroid hormone affects and governs the central nervous system both during the development phase and also during the entire life. It regulates gene expression in myelination, differentiation of neuronal and glial cells, and neuronal viability and function. The effect of thyroid hormones on serotonin (5-hydroxytryptamine [5-HT]) is explained by desensitization of 5-HT1A auto-receptor at the site of raphe nuclei, which probably results in an increase in the release of serotonin from the raphe neurons (Bauer, 2002; Smith, 2002; Bernal, 1995).

The mechanism of thyroid hormone activity in the brain is unclear due to the complexity of neuro-transmission interaction with the thyroid. One hypothesis stated that thyroid hormone modulates postsynaptic beta-adrenergic receptors in the cerebral cortex and cerebellum, while others stated that the modulation of 5-HT and its receptors is responsible, which happens due to inhibition caused by the TH at the raphe, causing reduction in 5-HT levels (Atlerwillw, 2008; Belmaker, 2008).

Both the hyper- and hypo-thyroid states have co-morbidity with psychiatric conditions. Similarly, patients of mood disorders also show derangement in TSH and thyrotropin releasing hormone whereas T3 and T4 may remain normal. (Bauer, 2002; Smith, 2002; Linkowski, 1981; Loosen, 1985).

**Prevalence**

Autoimmune thyroiditis is a common disease with a prevalence of approximately 4% to 13% in the United States (Forman-Hoffman, 2006; Hollowell, 2002). It generally affects more women than men and its frequency increases with age, reaching up to 20% among elderly women (Canaris, 2000; Sarks, 2004; Chaudhary, 2014).
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