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Effects of Elevated Thyroid Stimulating Hormone on Lactation  
in the Breastfeeding Woman  
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## PERMISSION

Title: Effects of Elevated Thyroid Stimulating Hormone on Lactation in the Breastfeeding Woman

Department: Nursing

Degree: Master of Science

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### Abstract

Pregnancy introduces a vast array of hormonal fluctuations which progress into the postpartum period. The thyroid gland responds to pregnancy by increasing thyroid hormone production in response to the growing fetus. In the postpartum period, thyroid hormone dysfunction is the thyroid transitioning from the suppressed state during pregnancy in an attempt to revert to a euthyroid state. This transition in the woman is evidenced by thyroid stimulating hormone (TSH) levels  $>10\text{mU/L}$ . Levels of TSH in the postpartum period impact regulatory hormones of lactation which may lead to difficulties with milk supply. It is estimated that Postpartum thyroiditis (PPT) occurs in 7-10% of postpartum women, with variations regarding genetic factors influencing thyroid disease (Ablovich, Amino, Barbour, Cobin, Degroot & Glinoyer, 2007). It is recommended by the Center for Disease Control and Prevention (CDC) (2017) that infants be exclusively breastfed for the first six months after birth to achieve optimal growth, development, and health. The postpartum period compiles a multifactorial initiation of physiologic adaptations in the female body, most notable being lactation and the practice of breastfeeding. The postpartum female who has TSH levels  $>10\text{mU/L}$  is indicative of a hypothyroid state and requires pharmacological management and further monitoring. Adequate treatment with Levothyroxine to a euthyroid state restores hormones of lactation and positively impacts milk supply. It is important for the health care provider to be aware of the clinical evaluation and management of hypothyroidism in the postpartum period and how this may affect one's ability to successfully lactate.

## Background

Effective management of thyroid disease in the postpartum period is important for optimal maternal and fetal health. A hypothyroid state in the postpartum female alters the functioning of prolactin and oxytocin involved in milk production and supply by negatively influencing their release and feedback from the hypothalamic-pituitary axis (HPA). Lactation and breastfeeding are supported as providing emotional and physical benefits to the mother and infant. Breastfeeding has long been reviewed as the best source of nutrition and a reliable indicator of growth prediction for the newborn. Infants who are breastfed are less likely to be diagnosed with asthma, obesity, type 2 diabetes, ear and respiratory infections, sudden infant death syndrome, and gastrointestinal infections (CDC, 2019). Not only are there benefits to the newborn, but the exclusively breastfeeding mother may also benefit from breastfeeding. The Center for Disease Prevention and Control (2019) suggests breastfeeding may help lower a women's risk of high blood pressure, type 2 diabetes, ovarian cancer, and breast cancer.

Disruptions in one's ability to adequately lactate may be related to endocrine changes in the postpartum female. Specifically, fluctuations in excess levels of TSH during the postpartum period have a negative impact on breastfeeding when desired. Understanding of the hormonal feedback mechanisms is pertinent to further discussing alterations in the neuroendocrine changes observed in the postpartum female. According to Stuebe et al. (2015) disorders of thyroid hormone have been associated with low milk supply in the lactating woman. Hormonal regulation is pertinent to assess in the presenting female experiencing challenges with breastfeeding. The purpose of this report is to address the management of lactation disturbances in the breastfeeding female by assessing thyroid function, explicitly addressing elevated levels of TSH in the postpartum female.

### Case Report

The case being presented is a 32-year-old female (name CT) who is otherwise healthy and comes to the primary care provider with symptoms of fatigue, dry skin, difficulty breastfeeding, and intermittent constipation. CT reports she is three months postpartum of delivering a healthy baby boy and has felt physical symptoms of fatigue more than usual. CT has been battling constant dry skin, is having trouble breastfeeding, and has been experiencing constipation at times. Her newborn is at a healthy weight for age and length. CT includes that she was having difficulty breastfeeding with her newborn upon delivery and at home and has been formula feeding for the last three weeks. CT states her fatigue is “more pronounced than ever, even with my other 2 children I wasn’t this tired all the time postpartum.” CT endorses using lotions for her dry skin to no avail. She also states the constipation doesn’t bother her much, and her Culturelle Probiotic helps keep her bowel movements regular. She notes the only medications she takes consist of a Women’s Prenatal Multivitamin and Culturelle Probiotic daily. CT reports the fatigue and inability to breastfeed are interfering with her ability to attend to her family. CT also states, “I wonder if it’s my thyroid, my mom and sister are on medication for their thyroid, but my thyroid labs have always been normal.”

Upon completing a review of systems, a physical exam includes the following pertinent positives: Bilateral forearm epidermis is dry. Notable for thin, brittle hair to her scalp. Delayed +1 deep tendon reflexes bilaterally to biceps, brachioradialis, patellar and Achilles tendons, mood tired and appears extensively fatigued. Labs completed with complete blood count and basic metabolic panel within normal limits. TSH notably high at 18.61mIU/L (normal level 0.4-4.0 mU/L) and Free T4 1.27 ng/dL (normal level 0.7-1.9 ng/dl) indicating a normal level. In reviewing previous lab results prior to her pregnancy, TSH levels were within normal ranges for

the last 8 years with no data recorded in the electronic record system prior to that time.

Given CT's presentation and supportive lab work, we discussed the diagnosis of Postpartum Thyroiditis (PPT), indicating a hypothyroid state. Today we will initiate Levothyroxine 50mcg PO daily 30 minutes before breakfast. She was reassured this is a safe medication to take if she attempts to breastfeed at home. CT has agreed to come back to the clinic in 6 weeks for a TSH recheck to assess need for dosing adjustments and evaluate improvement in symptoms. She was also encouraged to try Fish Oil 500mg tablet PO daily and introduce ground flaxseeds into her diet for management of dry skin. Difficulties with lactation were also discussed, and CT reports with her other two children she was able to breastfeed until 18 months of age without difficulty. With her most recent pregnancy she states she was able to breastfeed after delivery for the first 2 months of being at home with her baby. Denies issues with latching onto the breast but states "I don't feel like I have enough milk." CT reports she met with a lactation nurse in the hospital after delivery and was successful with breastfeeding and utilizing the breast pump. In review of the literature, current research supports elevated TSH ( $>10\text{mU/L}$ ) being related to complications with lactation in the postpartum period.

CT's physical presentation and current complaints of low milk supply, suspicion related to her thyroid function should be considered. Discussion with CT included the hormonal feedback mechanisms which occur with milk production and release, and how her elevated TSH levels may be playing a role in difficulties with lactation. CT is willing to attempt breastfeeding while starting on Levothyroxine with hopes of improvement in milk supply and release for her newborn's nutrition while correcting her TSH levels and promoting a euthyroid state.

### Literature Review

Pregnancy elicits many physiological changes in the female body from the time of conception through the postpartum period. Changes in metabolic functioning of hormones occur because of increased metabolic demands during both pregnancy and lactation (Arfuso, Fazio, Levanti, Rizzo, Di Pietro, Giudice, 2016). Gestation produces numerous metabolic alterations and suppressive states, especially with respect to the thyroid gland. The neuroendocrine adaptations such as elevated levels of TSH occurring in the postpartum female impacts mammary gland function by negatively interacting with the hormones of lactation (Campo Verde Arbocco, Sasso, Nasif, Hapon, & Jahn, 2015) as well as milk supply (Stuebe, Meltzer-Brody, Pearson, Pederson, & Grewen, 2015). Such hormonal interplay affects how the hormones of lactation appropriately supply and release milk for breastfeeding. Physical alterations which may accompany pregnancy and the postpartum period include but are not limited to: increased fatigue, nausea, vomiting, and healthy weight gain in relation to fetal number, size and the woman's weight prior to pregnancy. Physical feelings experienced because of pregnancy and the postpartum period may also disguise an underlying disease process such as thyroid disease. Symptoms of fatigue, weight gain, irritability, and depression may resemble a mental health need, thyroid disease, or the postpartum female simply adapting to caring for an infant. It is important for the provider to understand the expected alterations which occur in the female throughout pregnancy and in the postpartum period. The following literature review will focus on the postpartum female who presents with a subjective report, physical exam, and lab work consistent with postpartum thyroiditis in the hypothyroid state.

Post-Partum Thyroiditis (PPT) is the occurrence of thyroid dysfunction in the absence of a toxic thyroid nodule or thyrotoxin receptor antibodies in the first postpartum year in women

who were euthyroid prior to pregnancy (Muller, Drexhage, & Berghout, 2001). Post-Partum Thyroiditis has an autoimmune component which reflects the rebound of the thyroid gland after being in a suppressive state during pregnancy leading to inflammation of the thyroid gland and release of thyroid hormones into circulation (Yalamanchi & Cooper, 2015). Increases in thyroid hormone production in pregnancy result from fetal and maternal increases in iodine need and supplementation. PPT may occur in the postpartum individual who was otherwise euthyroid prior to and throughout her pregnancy (Alexander, Pearce, Brent, Brown, Chen, Dosiou, Grobman, Laurberg, Lazarus, Mandel, Peeters, & Sullivan, 2017).

Post-Partum Thyroiditis most commonly presents in the postpartum female as an initial hyperthyroid state followed by hypothyroidism with a return to a euthyroid state by the end of the postpartum year (Qaseem, Snow, Owens, & Shekelle, 2010). Approximately 20-40% of women with PPT develop permanent primary hypothyroidism within 3-12 years (Stuckey, Kent, & Ward, 2010). Some individuals may experience symptoms consistent with isolated hypothyroidism in the postpartum period which may be hard to detect from normal variations in the postpartum woman given in the pronounced fatigue, impaired memory, and weight gain consistent with pregnancy. According to Alexander et al., (2017) PPT reflects the rebounding mechanism within the thyroid gland in the postpartum period following the relative immune suppression occurring in pregnancy.

In the early stages of a healthy pregnancy, placental human chorionic gonadotropin (hCG) stimulates thyroid hormone secretion, which naturally decreases maternal TSH concentrations (Alexander et al., 2017). This mechanism reflects the natural rebounding of the immunosuppressed state of the thyroid gland throughout pregnancy. Post-Partum Thyroiditis reflects the adaptations in the thyroid gland from a gestational state to the postpartum period.

It has been demonstrated that elevated TSH concentrations ( $>10\text{mU/L}$ ) in the postpartum female exhibit changes with milk production and let down, thereby affecting one's ability to lactate. Maternal hormone fluctuation, alterations in breast anatomy, difficulties with infant latch and feeding patterns influence the woman's ability to lactate (Steube et al., 2015). Low milk supply is cited by many women as the primary reason for discontinuing to breastfeed.

The interplay of hormones involved in lactation and milk management affect milk initiation and ongoing milk production, also known as galactopoesis (Sriraman, 2017). Lactation is the process of continued secretion of milk and requires removal of milk and stimulation of the female's nipple. Reduced milk production may be affected by many internal and external factors including hormonal imbalances, interactions of certain medications the individual may be taking, improper development of the breast during pregnancy, or previous breast surgery or radiation treatment (Spencer, Abrams, & Hoppin, 2017).

There are significant neuroendocrine adjustments which promote and maintain lactation in the postpartum period. These include alterations in the primary hormones involved in lactation including prolactin and oxytocin which are regulated through a negative feedback mechanism in the HPA. Disruptions to the HPA such as elevations in TSH  $>10\text{mU/L}$  indicating a hypothyroid state negatively alters feedback mechanisms in milk supply and hinders the ability to breastfeed. Interestingly, animal research studies have shown a relationship is found in relation to TSH values and positive milk yield which suggests the role of the thyroid gland on the maintenance of lactogenesis (Fiore, Arfuso, Giancesella, Vecchio, Morgante, Mazzotta, Badon, Rossi, Bedin, & Piccione, 2018). Research proving such a relationship in humans has yet to be conducted.

Further assessment in thyroid dysfunction also include discussing the alterations in lactation experienced by the female. This provides opportunity to discuss her goals of feeding the infant and restoring her milk supply if able to return with treatment to a euthyroid state (Kellams, Harrel, Omage, Gregory, Rosen-Carol, 2017). As mentioned, it may be difficult to determine PPT in the form of hypothyroidism in the postpartum female as the clinical symptoms can mimic the typical fatigue following delivery and caring for a newborn. Careful assessment and knowledge of the postpartum female's adaptations in endocrine functioning is critical to diagnosis and management of PPT. Women in the postpartum period who present to the primary care provider with decreased milk supply should heighten the providers awareness to the possibility of PPT and appropriately include TSH screening in her evaluation.

There are differences of opinion in how to screen for thyroid disease during pregnancy and in the postpartum period. Guidelines have been established by the American Thyroid Association (ATA) for recommendations of monitoring thyroid function during pregnancy and in the postpartum period. Revisions to the ATA guidelines for thyroid monitoring in pregnancy and the postpartum period has not been updated and should be noted for further research needs. Indications for thyroid testing before and during pregnancy include presence of a goiter, family history of autoimmune thyroid disease, on current thyroid therapy, or history of high-dose neck radiation, PPT, previous delivery of an infant with thyroid disease, type I diabetes mellitus, or an autoimmune disorder (De Groot, Abalovich, & Alexander, 2012). Those who may be at higher risk for developing PPT include those with a family history of thyroid disease, smokers, personal history or autoimmune thyroid disease, those with positive TPO antibodies during early pregnancy, and those needing Levothyroxine replacement during pregnancy (Nochev, Argatska, Pehlivanov, & Orbetzova, 2017).

Assessing one's risk factors for thyroid disease in pregnancy or the postpartum period requires knowledge of one's family history and current health status. The ACOG recommends testing high-risk women who are symptomatic or have a personal history of an autoimmune disease (American College of Obstetrics and Gynecology, 2007), and no current revisions have been made to their recommended screening guidelines. In contrast, the Endocrine Society supports testing for thyroid dysfunction in women 30 years of age or older, with a family history of thyroid disease, presenting with signs and symptoms of thyroid disease, have a personal history of thyroid disease or thyroid surgery, have had prior neck irradiation, a positive thyroid peroxidase (TPO) antibody, history of autoimmune disease, infertility, history of miscarriage or preterm delivery, or residence in an iodine-deficient area (Degroot et al., 2012). The ATA recommendations are similar to screening recommendations to that of the Endocrine Society, but also includes screening women with body mass index (BMI)  $>40\text{kg/m}^2$  and women who have used amiodarone, lithium, or received recent iodinated contrast (Stagnaro-Green, 2011). With differences in screening recommendations during the state of pregnancy, opinions of how to assess thyroid dysfunction on the postpartum state have been well established. An individual without significant risk factors for PPT but who present to the providers office in the postpartum period with significant symptoms consistent with hypothyroidism should have their TSH and thyroxine (T4) level drawn to assess the need for thyroid hormone supplementation.

Diagnosis of hypothyroidism in the postpartum period is evidence by elevated TSH values  $>10\text{mU/L}$  in women who are also reporting significant symptoms and troubles with lactation (Stagnaro-Green, 2012) and it is recommended that they should be treated with Levothyroxine. Duration of therapy is not distinguished given that postpartum hypothyroidism may be transient or lead to overt hypothyroidism. Recommendations have been made to wean

the individual off their Levothyroxine after 6-12 months of treatment unless the woman is pregnant, attempting pregnancy, or continuing to breastfeed (Stagnaro-Green, 2012). The goal of postpartum hypothyroidism treatment is lowering TSH to a euthyroid state (TSH of 0.4-4.0mU/L), which typically occurs within 6-12 months after the initial diagnosis. Research suggests up to 30% of women never recover from the initial hypothyroid phase as evidenced by an increasing TSH level while the individual is taken off their Levothyroxine. This persistent rise in TSH indicates permanent hypothyroidism (Aziz, 2005). Such individuals will require long term T4 replacement. Some research suggests being able to detect the severity of the initial hypothyroid postpartum episode and the predicted development of permanent hypothyroidism using antithyroid peroxidase antibodies for assessment. For example, Aziz (2010) reports women with very elevated initial TSH values (50-100mU/L) and presence of high titers of antithyroid peroxidase antibodies suggest the possibility of permanent hypothyroidism. After initiation of Levothyroxine, thyroid function should be rechecked in six weeks. For women who have recovered from PPT and been weaned from medication therapy, serum TSH levels should be measured annually for the following 5-10 years after initial diagnosis (Degroot, et al., 2012). Individuals who have been diagnosed with PPT are at an increased risk of overt hypothyroidism indefinitely (Abalovich et al., 2007). Proper monitoring of one's TSH after the occurrence of PPT provides measurement of adequate thyroid functioning and surveillance of isolated hypothyroidism.

In summary, the performance of the thyroid gland fluctuates in the postpartum period due to hormonal alterations occurring after delivery of the infant and onset of lactation. The shift in thyroid hormone, specifically elevated TSH levels play an important role in negatively impacting hormones of lactation, namely prolactin and oxytocin. Such adjustments in TSH and hormones

of lactation reduce milk supply and may inhibit the woman's ability to breastfeed. Being aware of the neuroendocrine relationship between TSH and hormones of lactation allows health care providers to assess for PPT when the female presents with lactation concerns and symptoms of hypothyroidism. Proper diagnosis and treatment of PPT should restore the postpartum female to a euthyroid state, allowing for return of proper lactation.

#### Learning Points

- The importance of understanding the relationship between hormones of lactation and thyroid function allowed for adequate assessment of TSH in the postpartum female presenting with the inability to breastfeed.
- Pregnancy induces a suppressive state which can carry over into the postpartum period which alters hormonal feedback mechanisms required for lactation and decreasing milk supply.
- The diagnosis of PPT is often challenging given its broad range of presenting symptoms. Providers should include hypothyroidism in their differential when working with this population.
- Following recommended guidelines in screening women who plan to get pregnant and during pregnancy helps to distinguish those who are at risk for PPT. In the postpartum period, diagnostic levels of TSH  $>10\text{mU/L}$  in the woman presenting with symptoms of hypothyroidism who also report a decrease in milk supply are requirements for diagnosis and treatment of PPT.

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