Evaluation of thyroid dysfunction and postpartum depression in the postpartum female

Catherine Gail Lindback

The University of Toledo
Evaluation of Thyroid Dysfunction and Postpartum Depression in the Postpartum Female

Catherine Gail Lindback

The University of Toledo

2012
Acknowledgments

Thank you to my major advisor, Vivian Moynihan MD, MPH, for her constant support, guidance, and encouragement throughout this process. Also, thank you to her for her heartfelt dedication to physician assistant education.
# Table of Contents

I: Introduction ..................................................................................................................................1

II: Methods .......................................................................................................................................3

III: General Overview of Thyroid Function .....................................................................................4

IV: Hormonal Changes in Pregnancy and Postpartum Period .........................................................5

V: Endocrine Dysfunction in the Postpartum Female .......................................................................8

VI: Postpartum Thyroid Dysfunction and Association with Postpartum Depression ...............10

VII: Current Screening Mechanisms for Postpartum Depression ..............................................13

VIII: Discussion .............................................................................................................................14

Conclusion .....................................................................................................................................17

References ......................................................................................................................................19

Abstract .......................................................................................................................................22
Introduction

The postnatal period is a time of great adjustment for new mothers. There is much joy and excitement associated with the long-awaited arrival of a new infant. Along with such joy and excitement comes a broad spectrum of alternate sensations including worry, fatigue, stress, and anxiety. Forty to eighty percent of postpartum women develop changes in mood, most often within two to three days of delivery, and experience symptoms ranging from great happiness to irritability, tearfulness, insomnia, and sadness (Pearlstein, Howard, Salisbury, & Zlotnick, 2009). These symptoms typically resolve within two weeks, but for some new mothers symptoms persist and worsen.

Dramatic hormone changes during and after pregnancy play a significant role in the initiation of the above symptoms and associated disease states. Altered endocrine gland function with varying levels of estrogen and progesterone contribute to such symptoms. Postpartum depression arises in greater than 1 in 10 women up to a year after childbirth and requires appropriate medical attention in order to alleviate debilitating symptoms such as extreme fatigue, irritability, anxiety, depressed mood, and impaired concentration (Pearlstein et al., 2009). Postpartum thyroid dysfunction and postpartum thyroiditis are two associated medical conditions that share such symptoms.

The prevalence of postpartum thyroid dysfunction is currently a topic of much debate and uncertainty in the medical community. Reported figures of the occurrence of this disease state vary significantly and universal screening standards do not exist at this time. Postpartum thyroid dysfunction, particularly postpartum thyroiditis, has been shown to be an overlooked condition with significant patient morbidity. Recurrent postpartum thyroiditis is observed in two thirds of all pregnancies with evidence of 20 to 60% of women developing permanent hypothyroidism.
within 10 years of the initial event (Azizi, 2005). As a result, there is a need for heightened awareness of the symptoms associated with this disease both for afflicted patients and health care providers. In reviewing current research and clinical literature, there is a need to bring such evidence to the forefront and to devise an appropriate screening strategy with which to detect this underlying medical condition.
Methods

Research was conducted using the following databases: CINAHL, Dissertation Abstracts, JAMA - Journal of American Medical Association, NIH - National Institutes of Health, ProQuest Nursing & Allied Health Source, PsycINFO, PsycLIT, and PubMed. Several search terms were used including: Endocrine disorders in the postpartum period, Endocrine dysfunction in the postpartum period, Evaluation of postpartum thyroid dysfunction, Postnatal period, Postpartum blues, Postpartum depression, Postpartum endocrine dysfunction, Postpartum hypothyroidism, Postpartum period, Postpartum thyroid dysfunction, Postpartum thyroid dysfunction and depression, Postpartum thyroid dysfunction and postpartum depression, Postpartum thyroid function screening, Postpartum thyroiditis, Postpartum thyroiditis and depression, Postpartum thyroiditis and postpartum depression, and Thyroid function screening in the postpartum period.

All articles reviewed came from peer-reviewed journals and fell within a 12-year time frame. The research population lied within the United States and Europe and all articles were written in or translated into English.

First tier studies consisted of original research articles evaluating large sample sizes of postpartum females. Second tier studies resembled first tier studies but consisted of smaller sample sizes from the population. Third tier studies consisted of general medical textbooks along with literature reviews of general articles containing relevant background information.

Exclusion criteria included articles that were not associated with peer-reviewed journals, had serious identifiable methodological errors, and evidence of incorrect statistical analysis. I was wary of articles funded by particular corporations and associations and took the time to assess that the evidence presented was accurate and unbiased.
General Overview of Thyroid Function

The thyroid gland is an endocrine gland consisting of clustered spheroid follicles lying in front and to the sides of the upper part of the trachea. The adult thyroid gland contains two lobes that wrap along the right and left aspects of the trachea. Each lobe is separated into three poles - upper, middle, and lower. Right and left thyroid lobes connect to form the thyroid isthmus at the anterior portion of the trachea just below the cricoid cartilage (Goldman & Ausiello, 2008). The clustered spheroid follicles contained in thyroid tissue contain thyrocytes that surround colloid. The main component of this colloid is thyroglobulin which is a thyrocyte-specific protein. Thyrocytes play an active role in the synthesis and secretion of thyroid hormone. “Thyroid hormone increases oxygen consumption, thermogenesis, and expression of the low-density lipoprotein (LDL) receptor, resulting in accelerated LDL cholesterol degradation. Other physiologic effects of thyroid hormone include increased mental alertness, ventilatory drive, gastrointestinal motility, and bone turnover” (Goldman & Ausiello, 2008). Overall, thyroid hormone helps regulate growth and development while controlling metabolism and body temperature and is therefore an essential factor in human life.

Dietary iodine is an essential factor in thyroid hormone synthesis and secretion. Dietary iodine, mainly in the form of iodide (I⁻), is absorbed from the gastrointestinal tract and distributed in extracellular fluid. From here, it is actively transported into thyrocytes where it is oxidized and later involved in the generation of thyroxine (T₄) and triiodothyronine (T₃), the thyroid hormones primarily responsible for regulation of metabolism (Goldman & Ausiello, 2008). As a result, dietary intake of iodine is of great significance in regulation of metabolism. The current recommended daily intake of iodine in the United States is 150 µg for optimal physiologic function and health (Zimmermann, 2009).
Hormonal Changes in Pregnancy and Postpartum Period

Postpartum refers to the time period following delivery and extends to approximately six weeks after childbirth (Hacker, Gambone, & Hobel, 2010). Significant hormonal changes occur throughout pregnancy and their effects continue into the postpartum period. Placenta-derived human chorionic gonadotropin (hCG) has a thyroid-stimulating hormone (TSH) effect on the thyroid gland which can result in TSH suppression. Elevated estrogen levels lead to increased hepatic synthesis of thyroxine-binding globulin (TBG) and corticosteroid-binding globulin (CBG). TBG is the primary carrier protein for circulating thyroid hormone in the form of thyroxine (T₄). As a result, the body raises total circulating bound thyroid hormone. Adrenocorticotropic hormone (ACTH) and plasma cortisol levels increase late in the first trimester of pregnancy and remain elevated to the time of delivery with circulating cortisol primarily bound to CBG (Hacker et al., 2010).

The pituitary gland is one of the most affected organs during this time, undergoing great anatomic and physiologic change. The pituitary gland is a pea-sized endocrine gland located at the base of the brain which helps control the release of hormones from other endocrine glands throughout the body and also releases hormones that directly affect body tissues. Pituitary gland volume is found to increase during pregnancy with the highest volume being observed during the first three postpartum days (Karaca, Tanriverdi, Unluhizarci, & Kelestimur, 2010). Pregnancy is associated with increased maternal hypothalamic-pituitary-adrenal (HPA) axis activity in which ACTH release from the anterior pituitary gland is increased in response to decreased free cortisol levels (Karaca et al., 2010).

In addition, the thyroid-releasing hormone-thyroid-stimulating hormone (TRH-TSH) axis is altered throughout pregnancy due to the TSH effect of placental hCG on the thyroid gland. As
a result, maternal TSH levels decrease. TRH (thyroid-releasing hormone) is released from the hypothalamus and acts on the anterior lobe of the pituitary gland. TSH (thyrotropin or thyroid-stimulating hormone) is a glycoprotein hormone produced by the anterior lobe of the pituitary gland that stimulates the growth and function of the thyroid gland. The changes in maternal thyroid function that occur throughout pregnancy usually plateau and resolve, but failure to adapt to these physiological changes can result in thyroid dysfunction (Mannisto et al., 2010).

Significant changes in iodine metabolism occur during this time as well. After dietary consumption, iodine is absorbed by the stomach and duodenum and used in the production of thyroid hormone. Pregnancy increases demand on the maternal thyroid gland, with T4 production increasing approximately 50% (Yarrington & Pearce, 2011). Increased iodine loss through excretion in urine also occurs and can potentially cause an increase in thyroid volume due to the resulting elevated levels of TSH (Lazarus, 2005).

As a result of hormonal changes during pregnancy and throughout the postpartum period, changes in mood often occur. Dramatic changes in estrogen and progesterone levels occur during this time and are linked to such altered emotions (Steiner, Dunn, & Born, 2003). Postpartum blues refers to a period of great emotional change following childbirth. During this time, emotions of great joy and happiness may be present in the initial days after delivery followed by anxiety, irritability, confusion, and frequent crying episodes. Anywhere from 15 to 85% of women may experience symptoms of postpartum blues. Associated symptoms typically arise throughout the first five days after delivery. Symptoms typically resolve entirely after a short period and therefore do not require treatment. However in approximately 20% of cases, symptoms persist for more than three weeks and lead to a more serious disorder (Born, Zinga, & Steiner, 2004).
Postpartum depression is the new onset of depression during the postpartum period in which women experience disabling and persistent symptoms of depression, most frequently manifesting in the first 6 to 12 weeks after delivery, but onset of symptoms can occur even later following a period of initial stability. Symptoms include depressed mood, sleep disturbance, loss of appetite, lack of energy, anxiety, and suicidal thoughts. Overwhelming feelings of guilt or inadequacy in regards to the ability to care for the infant often arise. The new mother may also have an extreme preoccupation with the infant’s safety and well-being. The prevalence of postpartum depression ranges between 10 to 15% in new mothers with reports of approximately 10% of women still experiencing symptoms one year after childbirth (Born et al., 2004).
Endocrine Dysfunction in the Postpartum Female

Postpartum thyroid dysfunction is an immunological disorder occurring more frequently in women with particular human leukocyte antigen haplotypes. It is usually characterized by the presence of circulating thyroid peroxidase (TPO) antibodies in the bloodstream and is most often caused by postpartum thyroiditis (Lazarus, 2011). Postpartum thyroiditis is an autoimmune occurrence, in the postpartum period, of transient hyperthyroidism and/or transient hypothyroidism, with most women returning to the euthyroid state by one year postpartum. It is considered an exacerbation of an underlying autoimmune thyroiditis. Prevalence rates in the general population range from 0.9% to 11.7% (Nicholson, Robinson, Smallridge, Ladenson, & Powe, 2006). Autoimmune thyroiditis is often suppressed during pregnancy due to the physiologic suppression of immune activity during this time. It is subsequently potentiated in the postpartum period due to immunological rebound (Stagnaro-Green, 2002).

Postpartum thyroiditis presents most commonly as hypothyroidism without preceding hyperthyroidism. In a study of 371 episodes of postpartum thyroiditis, hypothyroidism occurred in 43% of the cases without preceding hyperthyroidism. Hyperthyroidism alone occurred in 32% of the cases and hyperthyroidism followed by hypothyroidism occurred in 25% of reported cases (Stagnaro-Green, 2002). The hyperthyroid phase associated with postpartum thyroiditis occurs in the postpartum period between 2 and 10 months, most commonly presenting at three months. Symptoms include palpitations, nervousness, fatigue, and heat intolerance (Stagnaro-Green, 2002).

The hypothyroid phase of postpartum thyroiditis occurs between 2 and 12 months into the postpartum period and is typically diagnosed around six months. Symptoms include decreased energy, depressed mood, impaired concentration, weight gain, dry skin, and cold
intolerance (Stagnaro-Green, 2002). Most women with postpartum thyroiditis are euthyroid at
the completion of the first postpartum year, but permanent hypothyroidism is possible.
“Prospective studies have shown a prevalence rate of hypothyroidism of 23% and 29% at 3.5 to
8.7 years postpartum. Progression to permanent hypothyroidism was more common in women
who presented with higher TSH levels and higher titers of thyroid peroxidase antibodies in the
hypothyroid phase of postpartum thyroiditis” (Stagnaro-Green, 2002).
Postpartum Thyroid Dysfunction and Association with Postpartum Depression

Although a distinct relationship between postpartum thyroid dysfunction and postpartum depression is yet to be proven, an overlap of symptoms is evident. Common symptoms of postpartum depression include depressed mood, weight gain/loss, sleep disturbance, loss of appetite, lack of energy, and anxiety. These symptoms are also associated with postpartum thyroid dysfunction. There is a direct correlation between thyroid dysfunction and depression/anxiety in the generalized population. Abnormalities in thyroid function are associated with an increased frequency of psychiatric symptoms. Hyperthyroidism has been associated with anxiety, depression, and cognitive deficit while hypothyroidism has been associated with an increased incidence of depression (Lucas, Pizarro, Granada, Salinas, & Sanmarti 2001). The postpartum period is considered a time of heightened risk for developing psychiatric disorders. “Women are particularly at risk of developing depression, having a 1 year prevalence, in general, of 8-12%, which rises to 15% during the postpartum period” (Kuijpen, Vader, Drexhage, Wiersinga, van Son, & Pop, 2001). As a result, the possibility of postpartum thyroid dysfunction should be further investigated and screening considered.

In a study conducted in June of 1997, 641 healthy Caucasian women were selected between their 36th week of pregnancy and fourth day postpartum for biological and clinical evaluation. Both clinical and laboratory follow-up continued throughout the postpartum period at one ($n = 605$), three ($n = 552$), six ($n = 574$), nine ($n = 431$), and twelve ($n = 444$) months (Lucas et al., 2001). Blood samples to determine free $T_4$ and TSH concentrations were drawn at each visit and subsequent thyroid antibodies were determined in patients with abnormal thyroid hormone concentrations. Reference ranges were 0.69 to 2.3 ng/dl for free $T_4$ and 0.4 to 4 mIU/l for TSH. Reference ranges for thyroperoxidase and thyroglobulin antibodies were less than 66
IU/ml and less than 275 IU/ml. The Beck Depression Inventory (BDI) was administered at baseline and with each follow-up as a means of depression screening with total BDI scores of 21 and above being indicative of depression. Postpartum thyroid dysfunction was diagnosed in patients with the initial presentation of thyroid dysfunction in the postpartum period.

“Postpartum thyroiditis was considered to be present in women with overt or subclinical (only abnormalities in TSH concentrations, levels less than 0.1 mIU/l) transient hyperthyroidism between 1 and 3 months postpartum and/or overt or subclinical hypothyroidism (only abnormalities in TSH concentrations, levels above 4 mIU/l) between 3 and 6 months postpartum” (Lucas et al., 2001).

Throughout the study, 56 women developed postpartum thyroid dysfunction, corresponding to an incidence rate of 11%, with 45 women developing postpartum thyroiditis (incidence rate of 7.8%). Fifty women (incidence rate of 7.8%) scored greater than 21 on the BDI, but the diagnosis of postpartum depression was not confirmed. Out of these 50 women, five had a concurrent diagnosis of postpartum thyroid dysfunction. Eleven of the evaluated women (incidence rate of 1.8%) were diagnosed as having postpartum depression with a BDI score over 21 and required psychiatric treatment. Out of these 11 women, none had a concurrent diagnosis of postpartum thyroid dysfunction (Lucas et al., 2001).

In a 2001 prospective observational study, 310 women were evaluated at 12 and 32 weeks gestation along with at 4, 12, 20, 28, and 36 weeks postpartum. TSH, free T4, and TPO antibody testing was performed at each visit. Clinical thyroid dysfunction was defined as both abnormal TSH and free T4 while subclinical thyroid dysfunction was defined as abnormal TSH in combination with normal free T4. The TSH reference interval was defined as 0.15 to 2.0 mU/l while free T4 was 8.7 to 19.6 pmol/l. In regards to TPO antibody testing, a concentration of
greater than 50 U/ml was considered positive. Depression was defined according to the Research Diagnostic Criteria in which one investigator established a syndromal diagnosis of depression during a semi-structured interview with the patient (Kuijpens et al., 2001).

Results of this study showed that during pregnancy and in the early postpartum period, women with depression presented more often with elevated TPO antibody concentrations. In addition, both clinical and subclinical thyroid dysfunction in combination with depression were significantly more prevalent in TPO antibody positive women (Kuijpens et al., 2001). Overall, 41 women (14.1%) were found to be TPO antibody positive at some point in the postpartum period. Two hundred and thirty-two women (80%) remained euthyroid while seven women (2.4%) showed clinical thyroid dysfunction. One hundred and fifty-eight women (54.3%) presented with at least one episode of depression and 117 women (40.1%) had depression at one or more time(s) in the postpartum period. There is still no explanation for the relationship between particular forms of thyroid dysfunction and depression, but this study does suggest that TPO antibodies can be regarded as important markers for the future occurrence of depression in at-risk groups of patients (Kuijpens et al., 2001).
Current Screening Mechanisms for Postpartum Depression

Postpartum depression, along with depression in general, is a major health problem that often goes undiagnosed. “It is estimated that more than half of subjects…do not seek professional care. Moreover, in subjects who visit their general practitioner, the diagnosis is missed in up to 50% of cases” (Kuijpens et al., 2001). As a result, significant social, psychological, and financial burdens occur. Detection of postpartum depression at an early stage is crucial in alleviating such burdens and evaluation of potential causes is necessary. The Edinburgh Postnatal Depression Scale (EPDS) is currently the most common instrument utilized to screen for postpartum depression, having a sensitivity of 86% and specificity of 78% (Pedersen et al., 2007). The EPDS is a 10-item self-report questionnaire designed to identify symptoms of emotional distress in mothers during the postnatal period. A score above 10 requires repeat screening within 2 weeks and two scores above 12 require further assessment and treatment (Hanusa, Scholle, Haskett, Spadaro, & Wisner, 2008).

Proper screening for postpartum depression is important because it is often the first step in the pathway to treatment. The EPDS should be utilized regularly at all postpartum examinations throughout the first postpartum year. Careful evaluation of results should be carried out and follow-up screenings administered as necessary. The EPDS was specifically developed for assessing postpartum depression and reduces the focus on specific somatic symptoms that are common to both women in the postpartum period and to women with depression (Hanusa et al., 2008). Routine use of the EPDS has consistently been shown to increase the detection of postpartum depression when compared to usual care and usage of other depression screening inventories (Sharp & Lipsky, 2002).
Discussion

The incidence of postpartum thyroid dysfunction and its association with postpartum depression is an ongoing topic of much debate and uncertainty in the medical community. In reviewing current research and clinical literature, it is difficult to determine a direct correlation between postpartum thyroid dysfunction and postpartum depression. Specific studies exploring such a correlation are limited in number and provide inconclusive results.

In the study of 641 healthy Caucasian women, postpartum thyroid dysfunction was diagnosed in only 56 women. In addition, subjects were evaluated between their 36th week of pregnancy and fourth day postpartum. This is a very limited time frame for diagnosis and evaluation of postpartum thyroid dysfunction and postpartum depression. A diagnosis of postpartum depression was derived using the Beck Depression Inventory, instead of the preferred Edinburgh Postnatal Depression Scale, with 50 women in the study scoring greater than 21 (incidence rate 7.8%; confidence interval 95%). Only five of these women had a simultaneous diagnosis of postpartum thyroid dysfunction. Eleven of the evaluated women were diagnosed as having postpartum depression and required psychiatric treatment (incidence rate 1.7%; confidence interval 95%). None of these women had a simultaneous diagnosis of postpartum thyroid dysfunction (Lucas et al., 2001).

Despite the overlap of symptoms between postpartum thyroid dysfunction and postpartum depression, results of this study fail to show a correlation between the two. Instead, analysis of this study places focus on proper screening mechanisms for postpartum depression and the appropriate time frame in which to screen. Numerous studies have shown that the Edinburgh Postnatal Depression Scale accurately identifies women who are depressed or at risk of developing depression in the first six months postpartum and it is therefore the instrument of
choice in screening (Hanusa et al., 2008). The formal definition of the postpartum period extends to approximately six weeks after childbirth, but assessment of postpartum depression and potential postpartum thyroid dysfunction should not end here (Hacker et al., 2010). There are several reports of symptom onset after the six-week postnatal checkup. As a result, a longer period of clinical surveillance is warranted (Stowe, Hostetter, & Newport, 2004).

In the 2001 prospective observational study, a much broader time frame existed in which to adequately diagnose and evaluate postpartum thyroid dysfunction and its potential correlation with postpartum depression. Both clinical and subclinical thyroid dysfunction were defined and appropriately measured in terms of TSH, free T4, and TPO antibody testing at each visit. However, depression was defined according to the Research Diagnostic Criteria. As a result, a single investigator conducted a semi-structured interview with each subject in order to establish a diagnosis of depression. Such a method creates uncertainty regarding whether a standardized and accurate diagnosis of postpartum depression was made. Diagnoses of both clinical and subclinical thyroid dysfunction along with the diagnosis of depression were significantly more prevalent in TPO antibody positive subjects. As a result, a correlation between positive TPO antibody testing and postpartum depression is identified and further investigation is warranted (Kuijpers et al., 2001).

Analysis of this study places emphasis on appropriate laboratory testing for diagnosis of postpartum thyroid dysfunction. Symptoms of postpartum thyroid dysfunction may be subtle and confused with postpartum depression, therefore complete testing is necessary (Swelam, Bakr, & Mansour, 2011). Laboratory testing includes thyroid function tests (serum TSH, T3, and free T4) along with TPO antibodies. Thyroid peroxidase (TPO) is an enzyme found on the surface of thyroid follicular cells and presence of serum TPO antibodies suggests an autoimmune
etiology of thyroid dysfunction. It is important to know that afflicted patients may become hypothyroid, hyperthyroid, or develop hyperthyroidism followed by hypothyroidism. As a result, complete laboratory testing is required for diagnosis and management (Swelam et al., 2011).
Conclusion

At this time, a direct correlation between postpartum thyroid dysfunction and postpartum depression is yet to be proven, but a distinct overlap of symptoms between the two certainly exists. Such symptoms include depressed mood, weight gain/loss, sleep disturbance, loss of appetite, lack of energy, and anxiety. It is the presence of such symptoms in the postpartum female that warrants further investigation, evaluation, and treatment. Clinicians must be aware of such symptoms in their patients and utilize the appropriate screening instruments and testing mechanisms with which to arrive at the correct diagnosis. Clinicians must also obtain a greater understanding of postpartum thyroid dysfunction for it is a diagnosis that often goes unevaluated. A greater societal awareness of the potential onset of postpartum depression must exist as well, especially among pregnant and postpartum females. Postpartum depression is a common affliction in such a population that often goes unreported, undiagnosed, and ultimately untreated. Adverse and potentially severe consequences for mother, child, and immediate family result and persist without appropriate medical care.

Clinicians must first and foremost listen to their patients with great attention and always take a detailed account of past medical history, family history, and social history. These are seemingly simple concepts, but can often be hastened or overlooked throughout the course of a busy day. In regards to pregnant and postpartum females, special focus must be placed on prior episodes of major depression, depression arising during previous pregnancies, personal or family history of thyroid dysfunction or autoimmune disorders, social support, and dietary intake. Although rare in the United States, iodine deficiency directly contributes to thyroid dysfunction. The current recommended daily intake of iodine in the United States must increase to 250 µg during pregnancy and postpartum due to maternal demands for increased T₄ production, iodine
transfer to the fetus, increase in renal iodine clearance, and excretion of iodine in breast milk. A urine iodine concentration of 100 µg/liter corresponds to an average daily intake of 150 µg (Zimmermann, 2009). Clinicians must know their practice demographics and be aware of areas throughout the United States where iodine deficiency is potentially prevalent, namely in the Midwest and Great Lakes regions of the country (Zimmermann, 2009).

The Edinburgh Postnatal Depression Scale should be utilized at each postpartum visit throughout the first postpartum year. It is designed specifically for screening and early detection of postpartum depression. A score above 10 requires repeat screening within two weeks and two scores above 12 require further assessment and possible treatment for postpartum depression (Hanusa et al., 2008). A score of 10 or above should also create suspicion of underlying postpartum thyroid dysfunction. It is important to consider this diagnosis as a possible cause of postpartum depression symptoms and therefore order TSH, T₃, free T₄, and TPO antibody testing. A past medical history or family history positive for thyroid dysfunction or autoimmune disorders should initiate TSH, T₃, free T₄, and TPO antibody testing as well.

Evaluation of thyroid dysfunction and depression in the postpartum female comprises an area of medicine that is still evolving. An overlap of symptoms between the two is evident, but a direct correlation remains unproven. The role of the clinician in proper diagnosis and subsequent treatment is essential. As a result, the clinician must keep these two related diagnoses in mind when caring for patients throughout the postpartum period.
References


doi:10.1530/eje.1.01976


doi:10.1530/eje.0.1450579


Abstract

Objective: To explore the potential correlation between postpartum thyroid dysfunction and postpartum depression and devise an appropriate strategy with which to detect and distinguish these conditions.

Methods: Research was conducted using the following databases: CINAHL, Dissertation Abstracts, JAMA, NIH, ProQuest Nursing & Allied Health Source, PsycINFO, PsycLIT, and PubMed. Reviewed articles came from peer-reviewed journals and fell within a 12-year time frame. The research population lied within the United States and Europe and articles were written in or translated into English.

Results: A direct correlation between postpartum thyroid dysfunction and postpartum depression is yet to be proven, but a distinct overlap of symptoms exists.

Conclusion: The role of the clinician in proper diagnosis is essential. The clinician must keep these related diagnoses in mind when caring for patients throughout the postpartum period and utilize the Edinburgh Postnatal Depression Scale with TSH, T3, free T4, and TPO antibody testing when necessary.