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WOMEN'S HEALTH

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Editorial Note:

Dear Doctor,

It's our immense pleasure to inform you that we have published our newsletter, "Women's Health". In this issue we are focusing on Management of Depression in Pregnancy and Thyroid Disorders in Pregnancy: risk scoring in women.

Your comments and suggestions will encourage us for upcoming issues. Please participate in quiz competition and win prizes.

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WOMEN'S HEALTH

Management of Depression in Pregnancy

Depression is the leading cause of disease-related disability in women and depression rates are highest during reproductive years. Given that close to half of all pregnancies are unplanned, it is crucial to consider a potential pregnancy when treating depression in women of reproductive age. With the increase in utilization of antidepressants to treat depression in women of reproductive age, there has been a parallel rise in antidepressant use (>7%) during pregnancy. Historically, pregnancy has been considered a time of emotional wellbeing; however, recent studies suggest that 10-15% of pregnant women will suffer from mood symptoms. The risk of prenatal depression is highest in women with a history of depressive episodes; yet it is not uncommon for women to experience their first depressive episode during pregnancy. Findings from a prospective study of more than 1000 pregnant women show that 7.3% had their first depressive episode within the perinatal period. However, depression during pregnancy has received far less attention than postpartum depression, despite being the most significant predictor of postpartum wellbeing.

Routine screening for prenatal depression remains uncommon and, even when implemented, can miss signs of depression, owing to the high overlap of normative complaints during pregnancy. Even when depression is diagnosed, it typically goes untreated or undertreated. Many clinicians prefer not to prescribe medications to a pregnant woman for fear of teratogenicity. Typically, this fear is not evidence-based, but rather a cautionary response attributable to a lack of randomized controlled trials in pregnant women or difficulty in understanding the often complex epidemiologic observational studies. Historically, medications were prescribed more liberally during pregnancy out of the belief that most drugs did not cross the placental barrier. With the discovery of the teratogenicity of thalidomide, the pendulum swung in the opposite direction and currently, antidepressants, in particular, are not prescribed at all or if they are, with the utmost caution, often with inadequate dosing. However, since illnesses during pregnancy, such as diabetes and hypertension, require treatment, so too, does depression.

In a recent survey, perinatal psychiatrists perceived that there were multiple barriers surrounding pharmacologic treatment during pregnancy including: general stigma of mental illness; pregnant women's fear of using medications during pregnancy; conflicting evidence-based information on the safety of antidepressants in pregnancy; biased media reporting of risks of psychotropic drugs; difficulty with understanding complex disseminated scientific information; and other healthcare providers' misperception of the risk of drugs and other misunderstandings surrounding mental health.

Screening for depression during pregnancy: risk factors

In order to screen and identify women suffering from prenatal depression, it is important to first recognize common risk factors. By far, the single most predictive risk factor of depression during pregnancy is a history of depression or anxiety prior to pregnancy, with a family history of psychiatric illness also being an important predictor. Other psychosocial risk factors that have been identified include: unplanned pregnancy; lack of social support or an unsupportive relationship with a partner; single status; exposure to domestic violence; younger age at conception; lower socioeconomic status; and lack of education.

Treatment decisions for depression during pregnancy.

In patients seeking preconception counselling regarding treatment options for a potential pregnancy, it is imperative to explore the severity of previous episodes of depression and prior attempts to discontinue psychotropic medications. Women who have not had a serious impairment secondary to depressive episodes or who have been successful in discontinuing their antidepressant and remained euthymic, may want to consider weaning off medications prior to pregnancy, with close monitoring, as well as engaging in weekly or, if necessary, biweekly psychotherapy. However, if the woman presents with prior episodes of psychiatric hospitalizations, suicide attempts, antenatal/postpartum depression or relapse after attempts to discontinue medications, it is often advisable to continue the antidepressant that previously maintained euthymia during pregnancy. A general treatment algorithm for managing depression in pregnancy (Figure 1) is included in this article.

Untreated depression during pregnancy.

During pregnancy, the risks of untreated depression may be more serious than the risks of antidepressant use. Although women with mental illness are frequently encouraged to discontinue psychotropic medications, studies have demonstrated that discontinuation prior to conception is associated with higher rates of recurrence of depression compared with those who remain on antidepressants. The previous belief that pregnancy is protective against depression has also been dispelled. Untreated prenatal depression can have many risks to the mother, the pregnancy and the fetus. Symptoms of depression, such as poor sleep, anxiety, irritability, ruminations, impairment in functioning and increased risk of suicide, can cause a substantial burden to the mother, who is already experiencing other pregnancy-related physical and emotional symptoms, as well as to the rest of her immediate family, particularly the father of the baby. Pregnant women have been reported to manage depression in their own way, by using tobacco, alcohol and illicit drugs, which are not appropriate.

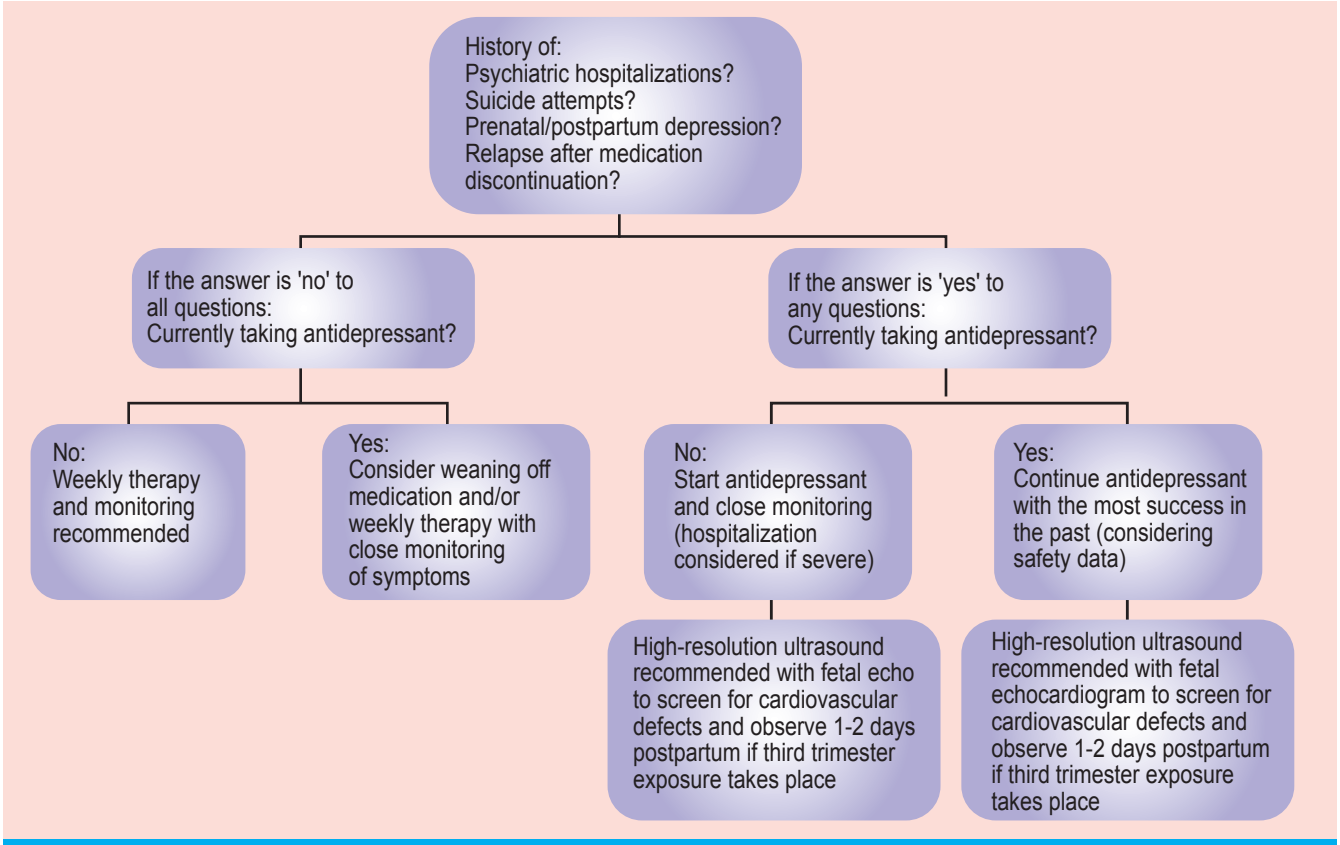

Figure 1. Treatment algorithm. Guidelines are general and each patient should be considered individually.

Additionally, a study by Newport et al. reports a decrease in prenatal vitamin compliance and increased use of over-the-counter medications targeting the physical symptoms associated with depression. Depression can also affect the mother's ability to think clearly about pregnancy-related decisions; for example, she may choose to terminate an wanted pregnancy, which can have long-term ramifications. When considering the risk-benefits of managing depression in pregnancy, both the direct impact of maternal symptoms and indirect consequences of maternal health behaviours -including the increased use of other medications and substances -must be addressed.

Risks to the fetus are also a major consideration in untreated depression. Maternal depression and anxiety are independently associated with greater neonatal irritability and poorer neonatal adaptation. Offspring of depressed mothers have been found to be less attentive, as well as having demonstrated impaired activity and behavioral problems. Furthermore, irregular ECGs and lower vagal tone is more common in offspring of depressed mothers. Although transient perinatal adverse events have been demonstrated, prospective follow-up of affected infants did not find an adverse impact on intelligence, internalizing behaviors, depression or anxiety at the age of 4-5 years. The question of whether the risk of autistic spectrum disorder is increased in children born to mothers taking SSRIs has been studied recently; however, owing to confounding variables and study limitations, no definitive association has been identified at this time. Unfortunately, to date, it has not been possible to separate whether the adverse outcomes on the infant are due to the underlying, untreated maternal illness, the antidepressant or maybe a combination of both. One group of researchers attempted to address potential perinatal adverse effects of SSRI use during pregnancy, while accounting for depression in the mother. The complex description of propensity scores lacked validity and, consequently, provided no solid evidence that they actually succeeded in doing so. Further research needs to be conducted, where it is possible to actually control for maternal depression, so there can be a better understanding of this complex medical condition.

Safety of antidepressant use during pregnancy: challenges in evaluating the current literature.

Owing to fear of teratogenicity, some healthcare providers recommend abrupt discontinuation of antidepressants following the discovery of a pregnancy. This is often without a thorough understanding of the evidence from studies demonstrating that the mother may be at serious risk of a depressive relapse. The current growing literature regarding the risks of antidepressants during pregnancy is often conflicting and changes constantly. Given that the current use of SSRIs during pregnancy is 3-7%, it is important to understand the current literature, including the limitations of

published studies, ascertain how to apply the cumulative evidence of risk and be able to make treatment decisions for each individual pregnant woman. Given obvious ethical limitations, there are no randomized controlled trials conducted with pregnant women so, currently, clinicians have to use the evidence from observational studies, which have many limitations, including potentially confounding factors, that are often not accounted for. These include maternal depression and anxiety, socioeconomic status, access to prenatal care, substance, tobacco and alcohol use, concurrent medications, and other illnesses.

Treating depression pharmacologically during pregnancy: overview of the safety data of antidepressants.

In this paper, the main focus is how to manage depression during pregnancy, and although the safety of antidepressants during pregnancy is of primary importance, this paper only summarizes outcomes from the most current evidence-based information derived from several comprehensive reviews in the peer-reviewed literature. These data suggest that if there are any risks associated with the use of antidepressants during pregnancy, they are, at most, marginal and should not preclude a pregnant woman diagnosed with depression from receiving pharmaceutical treatment.

Spontaneous abortion: There have been reports of an increased risk of spontaneous abortion in groups exposed to antidepressants in some studies but not in others. However, the higher rates are still within the normal range in the general population. It has also been reported in one study that women in the antidepressant group are more likely to undergo a therapeutic abortion, owing to fear of teratogenicity, compared with women in the non-exposed group.

Major malformations: There is no definitive evidence that antidepressant (as a group) exposure during pregnancy increases the risk for major malformations, including cardiac malformations, above the baseline rate.

Persistent pulmonary hypertension: Owing to conflicting evidence regarding the association of antidepressant exposure during pregnancy and risk for persistent pulmonary hypertension of the newborn (PPHN), the US FDA has concluded that "it is premature to reach any conclusion about a possible link between SSRI use in pregnancy and PPHN". It would seem that if there is an associated risk, the absolute risk increase is very small (from 1.2/1000 base rate to 3-6/1000) and would not justify discontinuing antidepressants prior to delivery, given the high risk for postpartum depression. There have been six published studies reporting on the possible association with an increased risk for PPHN associated with antidepressant use during late pregnancy. However, because of small sample sizes and quality issues in the studies, the absolute risk cannot be determined, although it is probably less than 1%.

Poor neonatal adaptation syndrome: A number of published studies suggest that newborns exposed to SSRIs and other classes of antidepressants may be at risk for developing adverse symptoms following delivery. These include a constellation of symptoms: jitteriness, sleep disturbance, poor feeding, poor muscle tone, weak cry, hypoglycemia and (rarely) seizure. This syndrome has been described to occur in approximately 10-25% of antidepressant-exposed infants. Symptoms are transient, lasting 2-3 days, usually with no need for medical intervention. Follow-up studies have found that antidepressant-exposed infants are indistinguishable from infants with no known exposure.

Birth weight & other related outcomes: There have been a number of studies linking exposure to antidepressants to low birth weight and SGA children, prematurity, and lower Apgar scores. However, the clinical importance appears to be negligible: as the preterm birth was less than 1 week early, there was 30-100 g lower birth weight, and less than half a point difference on Apgar score, compared with non-exposed group. Note that a child born weighing less than 2500 g is considered low birth weight, and a birth that occurred prior to 37 weeks gestation is considered a preterm birth, and both outcomes involve an increased risk of morbidity and mortality of the newborn, but do not represent different end points. A low birth weight baby can be born full term and a premature baby may not be low birth weight. A measure used to combine these aspects is intrauterine growth retardation, known as SGA, and is a baby whose birth weight is below the 10th percentile, based on birth weight reference curves, and stratified by infant gender and gestational age.

Long-term neurodevelopment: In a review of the literature, the authors could only find 12 studies that reported the neurodevelopmental outcomes of infants exposed prenatally to antidepressants and most had serious limitations, primarily owing to the methodology and inadequate sample sizes. In addition, there was no standardization for scales and measurements that were used and the ages of the children varied between 6 weeks and, only up to, 6 years old. Despite these limitations, the majority of studies found no difference between those exposed and the controls on any of the neurodevelopmental outcomes that were measured. One study reported that exposure to bupropion during pregnancy (OR: 3.63; $p = 0.02$), and notably during the second trimester (OR: 14.66; $p < 0.001$), was strongly associated with an increased risk of attention-deficit disorder, where, on the other hand, exposure to SSRIs was not (OR: 0.91; $p = 0.74$). However, the authors acknowledged that this could not be proven as a causal effect and, in addition, this study was derived from a claims benefit database, which are known to have many limitations. Larger long-term studies on neurodevelopmental follow-up are required; however, these studies are much more costly and time-consuming than the others and many institutions do not have the resources to conduct large studies such as these.

General management during pregnancy

During pregnancy, there should be careful monitoring of mood symptoms, particularly for women with a prior history of depression. If depression is identified, treatment should be instituted immediately, whether with psychotherapy for minor depression to prevent it worsening, or using the lowest effective dose of the appropriate antidepressant (whichever one is effective) if the depression is moderate-to-severe. Owing to physiological changes

during pregnancy, some women may need to increase the dosage of their medication to maintain euthymia.

A structured, high-resolution ultrasound can be offered for a detailed evaluation of fetal anatomy for a baby exposed to an antidepressant during pregnancy, mainly to reassure the woman. In addition, infants exposed to antidepressants during late pregnancy, should be observed for a longer period than the typical 1-2 days postpartum, so that neonatal symptoms of late trimester antidepressant exposure can be recognized and, if necessary, treated. Currently, affected infants are generally treated conservatively with observation in a special-care nursery with low-light environment and frequent small feedings. Mother-infant contact should be encouraged as this appears to improve temperature regulation, breathing regularity, behavioral state and overall infant health.

Conclusion & future perspective

Depression can be an overwhelming illness during pregnancy and can pose various risks to both the mother and fetus/infant. Therefore, when evaluating women of reproductive age for depression, clinicians should always be thinking of treatment strategies that would be appropriate to continue during a pregnancy. Women who previously required pharmacotherapy for depression and discontinue medications when planning for, or during, pregnancy are at a very high risk of relapse. Antidepressants remain the most studied medication during pregnancy and are the treatment of choice for childbearing women with moderate-to-severe depression. It is crucial for all physicians and treatment providers to be aware of both the risks of exposure to antidepressants, as well as the risks of untreated affective illness during the period of fetal development, so that the woman and her physician can make an informed joint decision regarding treatment options. The goal is to formulate a treatment plan that offers the least risk in the long term, and with the most benefits to both mother and child. Each patient needs to be evaluated on an individual basis and, ultimately, no decision is completely risk free.

Reference: Reminick, A., Cohen, S., & Einarson, A. (2013). Managing depression during pregnancy. *Women's Health*, 9(6), 527-535

Thyroid Disorders in Pregnancy

The increased metabolic demands during normal pregnancy result in alterations in maternal thyroid physiology and the effect is mediated by 3 main factors: (i) an increase in thyroid-binding globulin (TBG), (ii) stimulation of thyroid-stimulating hormone (TSH) receptors by human chorionic gonadotrophin (hCG) and (iii) the supply of iodine available to the thyroid.

TBG increases during pregnancy in response to estrogen, which stimulates hepatic production of TBG and reduces its peripheral degradation. TBG levels peak at approximately 20 weeks and then stabilize at approximately double prepregnancy levels. Thyroid hormone levels are also increased due to the effect of hCG. hCG stimulation of maternal TSH receptors protects the developing fetus from the potential effects of low TSH until fetal thyroid function is sufficient to maintain normal thyroid hormone levels in the fetus. Maternal renal iodine clearance is increased in pregnancy, and in addition some maternal iodine is lost to the fetus. Thus, iodine requirements are higher in pregnancy in order to maintain maternal euthyroidism. The current World Health Organization (WHO) recommendation is 250 mcg iodine daily for pregnant and lactating women.

In the normal thyroid gland, the combination of these physiological changes results in increased thyroid hormone secretion, and thus suppressed TSH levels. However, it should be noted that there is some variability in thyroid adjustments during normal pregnancy, with approximately one-third of women exhibiting relatively lower T4 secretion and higher TSH levels. Levels of TSH and T4 are often different in a pregnant woman as compared to a nonpregnant one, but the normal maternal state remains euthyroid.

Hypothyroidism disorders

Traditionally, hypothroidism is defined as elevation of TSH above 5 mIU/L. In the clinical context, it is classified as either subclinical hypothyroidism (elevated TSH with normal free T4 (FT4) levels) or overt hypothyroidism (elevated TSH with low FT4 levels). While women with subclinical hypothyroidism are asymptomatic, those with overt hypothyroidism may present with symptoms of tiredness, cold intolerance, constipation and muscle cramps. Clinical findings include dry and cold skin, bradycardia, delayed reflexes and goiter.

TSH and FT4 measurement is used to establish the diagnosis of hypothyroidism. Normal values vary over the course of pregnancy. The pregnancy-adjusted normal range for TSH is 0.08-2.99 mIU/L in first trimester, 0.2-3.0 mIU/L in the second trimester and 0.3-3.0 mIU/L in the third trimester. The degree of biochemical abnormality does not always correlate well with the severity of clinical symptoms. Direct measurement of FT4 in pregnancy is unreliable, due to the elevation in TBG level which skews the results. Total serum T4 measurement is more accurate during pregnancy. However, total T3 and T4 are about 1.5 times higher during pregnancy due to physiological changes.

Primary hypothyroidism in pregnancy is most commonly caused by Hashimoto's thyroiditis, an autoimmune disorder characterized by the presence of antithyroid peroxidase (anti-TPO) and/or antithyroglobulin (anti-Tg) antibodies which attack the thyroid gland. Thus, once the diagnosis of hypothyroidism is made, anti-TPO antibodies should be measured to confirm the etiology. Other causes include previous thyroid ablation therapy (surgical or medical) and in some areas, iodine deficiency is the most prevalent etiology.

Neonatal sequelae: In most cases, Hashimoto's thyroiditis does not result in fetal thyroid dysfunction. Only 2% of neonates born to women with hypothyroidism are affected by a transient congenital hypothyroid disorder. Maternal hypothyroidism due to iodine deficiency may have a significant impact on the fetus and neonate, and is known to be associated with fetal hypothyroidism and cretinism. Some research has also suggested a link between maternal subclinical hypothyroidism and fetal neuropsychological development, with findings of lower IQ scores in children born to mothers who were subclinically hypothyroid during pregnancy.

Screening and prevention: American College of Obstetricians and Gynecologists (ACOG) and the American Thyroid Association (ATA) recommends for targeted screening of high-risk women, rather than universal screening due to lack of evidence. Screening to be done in areas of iodine deficiency and those with a family or personal history of thyroid disease, miscarriage or preterm birth, thyroid peroxidase antibodies, head and neck radiation, type 1 diabetes and other autoimmune conditions, morbid obesity or infertility. To screen for hypothyroidism, TSH and anti-TPO antibodies should be measured early in pregnancy (1st trimester), or when contemplating pregnancy. Those with abnormal results should receive treatment to normalize thyroid function as soon as possible. In women in whom hypothyroidism is known or identified prior to pregnancy, treatment should bring TSH levels below 2.5 mIU/L before conception.

Treatment

L-thyroxine is the treatment of choice for hypothyroidism. The suggested initial dose is 0.150 mg daily, with a subsequent maintenance dose of between 0.125 mg and 0.250 mg daily (though requirements may be higher in certain cases such as following total thyroidectomy). Patients with pre-existing hypothyroidism require early pregnancy and regular TSH measurements throughout pregnancy, as their requirement for L-thyroxine may increase due to the greater demands on the thyroid. Immediately after delivery they should return to their previous dose of L-thyroxine.

Goiter

Goiter is a result of chronic thyroiditis, most commonly chronic immune thyroiditis and Hashimoto's thyroiditis. It is present in 80% of patients with chronic thyroiditis. Goiter during pregnancy is a common finding in countries where iodine deficiency is prevalent; however, in areas with adequate iodine intake, the finding of a goiter during pregnancy is pathological and requires further assessment in addition to L thyroxin therapy.

Thyrotoxicosis/hyperthyroidism disorders

Hyperthyroidism complicates about 0.1-0.4% of pregnancies and is defined by a markedly depressed (<0.1 mJ/L) or undetectable (<0.01 mU/L) serum TSH, accompanied by elevated serum free T4 and/or T3. Subclinical hyperthyroidism, defined as depressed TSH with normal free T4/T3, does not result in adverse pregnancy outcomes.

A certain degree of TSH depression occurs in normal pregnancy, particularly in the first trimester in association with rising hCG levels. Therefore, healthy women in the first trimester may have TSH levels between 0.03-0.1 mIU/L; most women with significant thyrotoxicosis in pregnancy have a first trimester TSH level of <0.01 mU/L. A TSH level of <0.1 mU/L warrants measurement of serum free T4. If free T4 is not elevated, free T3 should be measured, as elevated T3 may also, though rarely, cause hyperthyroidism.

TSH receptor antibodies (TSRAb) of the stimulating type (thyroid-stimulating immunoglobulins, TSIs) are found in 95% of patients with Graves' disease, and assays to determine the presence of these antibodies may be used if the cause of hyperthyroidism in pregnancy cannot be clinically determined. Other indications for measurement of TSIs are for the purpose of predicting the possibility of fetal or neonatal thyroid dysfunction, such as known active Graves' disease or past history of fetal or neonatal hyperthyroidism.

The pathology of transient hyperthyroidism of hyperemesis gravidarum (HEG thyroiditis) is related to the structural similarity between TSH and hCG. Most likely, the high levels of hCG in early pregnancy stimulate the TSH receptors on the thyroid gland, causing excess release of thyroid hormone and high serum FT4. Clinically, the presentation is dominated by symptoms of hyperemesis gravidarum, with severe nausea and vomiting requiring intravenous hydration, weight loss, ketonuria, hypokalaemia and elevated liver enzymes. The only symptoms of hypermetabolism may be mild palpitations and heat intolerance. Similarly, on physical examination exophthalmos and goiter are absent, and only mild tachycardia and tremor of the outstretched hands may be observed.

Further clues to the diagnosis are the close parallels of the symptoms and biochemical abnormalities with those of hyperemesis gravidarum. Patients give a clear history of onset of symptoms during pregnancy (rather than prior to conception), and in most cases the condition resolves spontaneously between 14-20 weeks gestation with the resolution of vomiting. It should be noted, however, that hyperthyroidism may persist past 20 weeks in 15-25% of cases, and TSH suppression may still be present a few weeks after normalization of FT4 levels. Finally, in HEG thyroiditis, the extent of the biochemical abnormality is directly related to the severity of hyperemesis symptoms. Thus, all women with hyperemesis gravidarum that is accompanied by substantial weight loss and dehydration should undergo an assessment of thyroid function. Other, less common, causes of hyperthyroidism in early pregnancy are gestational trophoblastic disease, molar pregnancies and choriocarcinoma, all of which are characterized by an excessively high hCG level.

Maternal and fetal complications

Untreated or poorly controlled overt maternal hyperthyroidism in pregnancy has been associated with higher rates of pre-eclampsia and, less commonly, maternal heart failure. Thyroid storm, an acute, life-threatening augmentation of thyrotoxicosis, is rare in pregnancy, however, may be precipitated by stress such as labor, caesarean section or infection.

Hyperthyroidism in pregnancy has also been shown to cause fetal complications including spontaneous abortion, preterm delivery, IUGR and stillbirth In contrast, subclinical hyperthyroidism has not been associated with adverse pregnancy outcomes. Early control of overt hyperthyroidism has been shown to reduce the rate of complications.

Treatment - acute and chronic

HEG thyroiditis does not require treatment, as it resolves spontaneously with resolution of hyperemesis gravidarum. Thyrotoxicosis induced by gestational trophoblastic tumor secreted hCG is also transient, resolving once the hCG level decreases. Rarely, symptomatic treatment is required. In contrast, treatment of maternal hyperthyroidism due to Graves' disease in pregnancy should be instituted as early as possible to minimize the potential for complications. Treatment is aimed at normalizing maternal thyroid function tests using the lowest possible dose of antithyroid medication; excessive amounts crossing the placenta can cause fetal hypothyroidism and goiter.

Thyroid nodules and neoplasia

There is a wide range in the reported prevalence of thyroid nodules and thyroid malignancies in pregnancy. Thyroid nodules occur in anywhere between 3-21% of pregnant women, and between 5-10% of these lesions are found to be malignant.

Assessment and management of thyroid nodules discovered during pregnancy is the same as in nonpregnant patients. TSH and FT3/FT4 are measured, and thyroid ultrasound performed to determine the size and characteristics (solid or cystic) of the nodule. Fine needle aspiration (FNA) biopsy is safe to perform during pregnancy, and the indications for this diagnostic procedure are similar to those in the nonpregnant state. In general, nodules greater than 1 cm in size should be evaluated using FNA. Radioiodine scanning is generally considered to be contraindicated in pregnancy, although with tracer doses there is minimal risk of fetal irradiation.

Evidence from a number of studies suggests that diagnosis of thyroid cancer during pregnancy does not have a significant impact on its prognosis. Differentiated thyroid carcinoma requires surgical management, however studies have shown no adverse impacts on morbidity or mortality when treatment is delayed until the postpartum period. If the decision is made to delay surgery, regular thyroid ultrasound scans should be performed to assess the growth of the lesion. If significant growth is noted, surgery should be performed during the second trimester. In cases where surgery is delayed, consideration should be given to suppression therapy with L-thyroxine to maintain low TSH levels and prevent excessive growth of the cancer. FT4 levels should also be kept within the normal range for pregnancy.

In women diagnosed with thyroid cancer prior to pregnancy, any persistent disease may progress during pregnancy. Thus, in patients with biochemical or ultrasound evidence of persistent disease prior to pregnancy, monitoring of T4 levels and thyroid ultrasound should be performed each trimester. In addition, suppression therapy with L-thyroxine should be continued in pregnancy. Higher doses may be required to maintain TSH in the desired range, thus TSH should be monitored regularly throughout pregnancy to ensure adequate suppression.

Summary and conclusions

Thyroid disorders are common and might have a significantly detrimental effect on the gravida, fetus and neonate. Though universal screening is not currently recommended, screening of high-risk patients should be performed. Such cases include women with personal or family history of thyroid dysfunction, positive thyroid autoantibodies, and autoimmune disorders especially polyglandular failure. It is also our belief that thyroid testing should be very liberally offered during or before pregnancy because of its simplicity, low cost and potential to prevent serious fetal and neonatal morbidity. The target range of TSH that applies for these patients, for both screening and treatment, differs from that of the general population. Prompt treatment and achievement of euthyroidism within the recommended TSH range is of greater importance to pregnant women than to the general population.

Reference: Parkes, I. L., Schenker, J. G., & Shufaro, Y. (2012). Thyroid disorders during pregnancy. *Gynecological Endocrinology*, 28(12), 993-998.

Congratulations !

The Winners of WOMEN'S HEALTH *Quiz Competition*

Volume : 7, No. :2, Sept - Dec 2014

1. **Dr. Hasina Akhtar**
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