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If the thyroid has decreased reserve for any reason during pregnancy, the mother may develop mild/subclinical hypothyroidism during the pregnancy. In some, but not all, of these women, thyroid function returns to normal after delivery. This study examined the changes in thyroid function after pregnancy in women who developed subclinical hypothyroidism during pregnancy.

Li N et al. Postpartum follow-up of patients with subclinical hypothyroidism during pregnancy. *Thyroid*. 2020. ePub: June 5, 2020. DOI: 10.1089/thy.2019.0714. PMID 32375594

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Development of thyroid problems with immunotherapy drugs for certain cancers is associated with favorable survival

New chemotherapy drugs for the treatment of cancer have been activating the immune system to target and kill cancer cells. Prior studies have suggested that the cancer patients who develop thyroid problems while taking immunotherapy drugs have improved survival. The goal of this study is to evaluate the association between thyroid function test results, anti-thyroid antibody concentrations and survival rates in a large group of cancer patients treated with anti-PD-1 drugs.

Basak EA et al. 2020 Overt thyroid dysfunction and anti-thyroid antibodies predict response to anti-PD-1 immunotherapy in cancer patients. *Thyroid*. Epub 2020 Mar 10. PMID: 32151195.

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Obese women with hypothyroidism treated with levothyroxine have a reduced energy expenditure

Hypothyroidism decreases metabolism. Once thyroid hormone levels return to normal on levothyroxine, metabolism returns to normal. A previous small study has suggested that the resting energy expenditure is lower in patients with hypothyroidism even if they are treated with levothyroxine and their thyroid hormone levels are in the normal range. This study was done to determine the resting energy expenditure in a group of obese women with hypothyroidism attending a bariatric clinic

Muraca E et al 2020 Resting Energy Expenditure in Obese Women with Primary Hypothyroidism and Appropriate Levothyroxine Replacement Therapy. *J Clin Endocrinol Metab*. Apr 1;105.

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Being overweight is linked to a genetic mutation that causes increased risk of thyroid cancer

It is clear that obesity is a risk factor for different types of cancers, possibly may be associated with thyroid cancer. Part of this association is that the increase in thyroid cancer has mirrored the increase in obesity in the United States. To determine if there is a genetic link between these 2 disorders, this review was done to determine if *BRAF^{V600E}*-positive thyroid cancer was associated with obesity.

Rahman S et al 2020 Obesity is associated with *BRAF^{V600E}*-mutated thyroid cancer. *Thyroid*. Epub 2020 Mar 31. PMID: 32228152.

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Aggressive types of thyroid cancer are becoming more common and differ from one another in how dangerous they may be

Classical papillary thyroid cancer is the fastest rising type of thyroid cancer and is generally slow growing with an excellent prognosis. There are several more aggressive thyroid cancer subtypes tend to grow faster and spread. To date, it is unclear if these subtypes are also rising in frequency and if there are really more dangerous. The study described here tries to answer these two questions by evaluating the medical records of large groups of people previously treated for thyroid cancer.

Ho AS et al 2020 Incidence and mortality risk spectrum across aggressive variants of papillary thyroid carcinoma. *JAMA Oncol*. Epub 2020 Mar 5. PMID: 32134428.

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Detecting thyroid cancer recurrence following lobectomy

More frequently, patients diagnosed with thyroid cancer who are low risk and with the cancer confined to one lobe are offered to be treated a lobectomy. However, in patients with a normal lobe after a lobectomy, following the thyroglobulin levels are much less reliable as a cancer marker. In this study, the authors report on their experience from a single institution on measuring thyroglobulin levels after a lobectomy for thyroid cancer.

Ritter A et al 2020 Detecting recurrence following lobectomy for thyroid cancer: Role of thyroglobulin and thyroglobulin antibodies. *J Clin Endocrinol Metab* 105:1–7. PMID: 32219303.

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Clinical Thyroidology for the Public

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EDITOR'S COMMENTS

Welcome to another issue of *Clinical Thyroidology for the Public*. In this journal, we will bring to you the most up-to-date, cutting edge thyroid research. We also provide even faster updates of late-breaking thyroid news through [Twitter](#) at [@thyroidfriends](#) and on [Facebook](#). Our goal is to provide patients with the tools to be the most informed thyroid patient in the waiting room. Also check out our friends in the [Alliance for Thyroid Patient Education](#). The [Alliance](#) member groups consist of: the *American Thyroid Association*, *Bite Me Cancer*, *the Graves' Disease and Thyroid Foundation*, *the Light of Life Foundation*, *MCT8 – AHDS Foundation*, *ThyCa: Thyroid Cancer Survivors' Association*, *Thyroid Cancer Canada*, *Thyroid Cancer Alliance* and *Thyroid Federation International*.

We invite all of you to join our [Friends of the ATA](#) community. It is for you that the American Thyroid Association (ATA) is dedicated to carrying out our mission of providing reliable thyroid information and resources, clinical practice guidelines for thyroid detection and treatments, resources for connecting you with other patients affected by thyroid conditions, and cutting edge thyroid research as we search for better diagnoses and treatment outcomes for thyroid disease and thyroid cancer. We thank all of the *Friends of the ATA* who support our mission and work throughout the year to support us. We invite you to help keep the ATA mission strong by choosing to make a donation that suits you — it takes just one moment to give online at: www.thyroid.org/donate and all donations are put to good work. The ATA is a 501(c)3 nonprofit organization and your gift is tax deductible.

The Covid-19 pandemic has caused an unprecedented upheaval in our daily lives and presented extremely difficult challenges to our healthcare system. There is a lot of information circulating around. We at the American Thyroid Association would like to make sure that you all have access to most accurate, reliable, fact-based and updated information. (<https://www.thyroid.org/covid-19/>)

August is [Thyroid and Pregnancy Awareness Month](#).

In this issue, the studies ask the following questions:

- Do women with subclinical hypothyroidism during pregnancy continue to have hypothyroidism after delivery?
- Do thyroid problems during cancer immunotherapy treatment affect prognosis?
- Does levothyroxine return metabolism to normal in obese hypothyroid women?
- Is obesity a risk factor for thyroid cancer?
- Are aggressive papillary thyroid cancer subtypes increasing?
- Can thyroglobulin levels in thyroid cancer patient be followed after a lobectomy?

We welcome your feedback and suggestions. Let us know what you want to see in this publication. I hope you find these summaries interesting and informative.

— Alan P. Farwell, MD, FACE



THYROID AND PREGNANCY

Postpartum thyroid function in women with subclinical hypothyroidism during pregnancy

BACKGROUND

Thyroid hormone is essential for normal development of the baby during pregnancy. Key to the baby getting enough thyroid hormone is the level of thyroid hormones in the mother. Thus, the thyroid status of the mother during pregnancy is very important and continues to be the subject of numerous research studies. It is very important to maintain normal thyroid hormone levels in the mother during pregnancy for the best pregnancy outcomes. During pregnancy, the thyroid needs to increase the production of thyroid hormone due to hormonal changes in the thyroid hormone binding proteins. If the thyroid has decreased reserve for any reason, the mother may develop mild/subclinical hypothyroidism during the pregnancy. In some, but not all, of these women, thyroid function returns to normal after delivery. This study examined the changes in thyroid function after pregnancy in women who developed subclinical hypothyroidism during pregnancy. They sought to determine the risk of long term hypothyroidism after pregnancy in these women.

THE FULL ARTICLE TITLE

Li N et al. Postpartum follow-up of patients with subclinical hypothyroidism during pregnancy. *Thyroid*. 2020. ePub: June 5, 2020. DOI: 10.1089/thy.2019.0714. PMID 32375594

SUMMARY OF THE STUDY

A total of 393 women who developed subclinical hypothyroidism during pregnancy, defined as TSH > 4 mIU/mL and normal free T₄, were recruited to participate the study. They were all treated with thyroid hormone during pregnancy, but all stopped taking the medication after delivery. Thyroid function and thyroid peroxidase

(TPO) antibody levels were measured 6 weeks after delivery and then periodically thereafter. At the 6 week postpartum visit, 248 women (63.1%) had normal thyroid function, 134 (34.1%) were hypothyroid (all by 1 had subclinical hypothyroidism) and 11 (2.8%) were hyperthyroid. A total of 216 women were followed for more than 6 months after delivery. Of the 131 women who had normal thyroid function at the 6 week postpartum visit and had more than 6 months follow up, 37 women (28.2%) went on to develop hypothyroidism. At the last follow up evaluation (average follow up 11 months after delivery), 132 women (61.1%) had normal thyroid function, 84 (38.9%) were hypothyroid and none were hyperthyroid. Women diagnosed with subclinical hypothyroidism earlier in their pregnancy and those with positive TPO antibodies were more likely to develop persistent hypothyroidism after delivery.

WHAT ARE THE IMPLICATIONS OF THE STUDY?

Women with subclinical hypothyroidism during pregnancy are at increased risk of developing persistent long-term hypothyroidism. They are more likely to develop long term hypothyroidism if they also have positive TPO antibodies or developed thyroid problems early during pregnancy. Consequently, it is recommended that women with subclinical hypothyroidism be monitored postpartum for the development of persistent hypothyroidism. Although duration or timing of monitoring for hypothyroidism postpartum remains unclear, women should at least be educated about potential signs and symptoms of thyroid problems that could signify the need for repeat evaluation.

— Whitney W. Woodmansee MD





THYROID AND PREGNANCY, continued

ATA THYROID BROCHURE LINKS

Thyroid Disease in Pregnancy: <https://www.thyroid.org/thyroid-disease-pregnancy/>

Hypothyroidism (Underactive): <https://www.thyroid.org/hypothyroidism/>

Postpartum Thyroiditis: <https://www.thyroid.org/postpartum-thyroiditis/>

Thyroid Function Tests: <https://www.thyroid.org/thyroid-function-tests/>

ABBREVIATIONS & DEFINITIONS

Hypothyroidism: a condition where the thyroid gland is underactive and doesn't produce enough thyroid hormone. Treatment requires taking thyroid hormone pills.

Subclinical Hypothyroidism: a mild form of hypothyroidism where the only abnormal hormone level is an increased TSH. There is controversy as to whether this should be treated or not.

TPO antibodies: these are antibodies that attack the thyroid instead of bacteria and viruses, they are a marker for autoimmune thyroid disease, which is the main underlying cause for hypothyroidism and hyperthyroidism in the United States.

TSH: thyroid stimulating hormone — produced by the pituitary gland that regulates thyroid function; also the best screening test to determine if the thyroid is functioning normally.

AUGUST
Thyroid & Pregnancy
Awareness Month

AMERICAN THYROID ASSOCIATION®
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THYROID AND CANCER

Development of thyroid problems with immunotherapy drugs for certain cancers is associated with favorable survival

BACKGROUND

New chemotherapy drugs for the treatment of cancer have been activating the immune system to target and kill cancer cells. One major new category of immunotherapy drugs is known as immune checkpoint inhibitors that activate the immune system. These immunotherapy drugs have significantly improved survival for various types of cancers. However, these treatments may be associated with many immune-related side effects. In particular, many patients treated with the immune checkpoint inhibitor anti-PD-1 develop thyroid problems. Most often, this results in an inflammation of the thyroid (thyroiditis) that causes a short period of hyperthyroidism followed by hypothyroidism. Compared to common forms of thyroiditis (subacute thyroiditis, post-partum thyroiditis), there is usually no associated thyroid pain and the time from hyperthyroidism to hypothyroidism is shorter (3 months vs 6-9 months). This is often associated with an increase in levels of anti-thyroid antibodies (TPO and thyroglobulin antibodies).

Prior studies have suggested that the cancer patients who develop thyroid problems while taking immunotherapy drugs have improved survival. The goal of this study is to evaluate the association between thyroid function test results, anti-thyroid antibody concentrations and survival rates in a large group of cancer patients treated with anti-PD-1 drugs.

THE FULL ARTICLE TITLE

Basak EA et al. 2020 Overt thyroid dysfunction and anti-thyroid antibodies predict response to anti-PD-1 immunotherapy in cancer patients. *Thyroid*. Epub 2020 Mar 10. PMID: 32151195.

SUMMARY OF THE STUDY

This is a study of 168 patients with non-small-cell lung cancer, renal-cell cancer and melanoma followed at Erasmus Medical Center in the Netherlands. The patients started anti-PD-1 treatment with nivolumab (every 2 weeks) or pembrolizumab (every 3 weeks). Thyroid function tests, including thyroid-stimulating hormone

(TSH) and free thyroxine (FT₄) levels were measured every 2 or 3 weeks before each anti-PD-1 infusion. Antibody levels to thyroid peroxidase (anti-TPO) and thyroglobulin (anti-Tg) were measured at baseline and after 2 months of treatment. The patients were followed for an average time of 15 months. Overall survival (OS) was defined as the period between the start of therapy until death, while progression-free survival (PFS) was calculated until tumor progression, based on standard Response Evaluation Criteria In Solid Tumors (RECIST) criteria or death.

A total of 27 patients (16%) had pre-existing thyroid problems, 9 patients being hyperthyroid and 18 patients being hypothyroid before starting treatment. Among these patients, 22 patients had mild thyroid disease. During the study period, 34 patients (20%) developed subclinical thyroid dysfunction and 20 patients (12%) developed overt thyroid dysfunction on treatment. The average time to develop thyroid dysfunction was 2.8 months.

Patients who developed overt thyroid dysfunction during anti-PD-1 treatment had significantly higher survival rates than patients without thyroid dysfunction at 1 year (OS, 94% vs. 64%; PFS, 64% vs. 33% patients). During treatment, patients with higher anti-thyroid antibody levels had higher survival rates than patients with lower anti-thyroid antibody level, with 1-year OS rates of 83% and 49% and PFS rates of 54% and 20%, respectively. For the majority of patients (84%), the antibody status did not change from baseline until the end of treatment. Most patients with higher levels of antibodies during the treatment were also observed to have a higher antibody count before treatment.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

Development of overt thyroid problems and higher antithyroid antibody levels during anti-PD-1 treatment were associated with a significant improvement in both overall and progression-free survival rates. These data suggest that this could be used as a predictive marker



THYROID AND CANCER, continued

for response to treatment. Additionally, higher baseline antithyroid antibody levels usually remain at the same level during the treatment and predict better survival

rates among cancer patients treated with anti-PD-1 immunotherapy.

— Alina Gavrilă, MD, MMSC

ATA THYROID BROCHURE LINKS

Hypothyroidism (Underactive): <https://www.thyroid.org/hypothyroidism/>

Hyperthyroidism (Overactive): <https://www.thyroid.org/hyperthyroidism/>

Thyroiditis: <https://www.thyroid.org/thyroiditis/>

ABBREVIATIONS & DEFINITIONS

Immunotherapy: a type of treatment that helps a person’s immune system fight diseases, such as cancer. A class of immunotherapy drugs is known as immune checkpoint inhibitors.

Anti-programmed cell death 1 (anti-PD-1) immunotherapy: treatment that targets the programmed cell death protein 1 (PD-1) to activate the immune system to attack cancer. PD-1 is an immune checkpoint protein found on the surface of human cells that decreases the response of the immune system to its own cells. PD-1 prevents the development of autoimmune diseases, however, it also prevents the immune system from killing cancer cells.

Autoimmune thyroid disease: a group of disorders that are caused by antibodies that get confused and attack the thyroid. These antibodies can either turn on the thyroid (Graves’ disease, hyperthyroidism) or turn it off (Hashimoto’s thyroiditis, hypothyroidism).

Thyroiditis: inflammation of the thyroid, most commonly cause by antibodies that attack the thyroid as seen in Hashimoto’s thyroiditis. It can also occur in response to a viral infection.

Hypothyroidism: a condition where the thyroid gland is underactive and doesn’t produce enough thyroid hormone. Subclinical hypothyroidism is a mild form where the only abnormal hormone level is an increased TSH. Overt hypothyroidism is clear hypothyroidism with

an increased TSH and a decreased T₄ level. All patients with overt hypothyroidism are usually treated with thyroid hormone pills.

Hyperthyroidism: a condition where the thyroid gland is either overactive or inflamed and produces too much thyroid hormone. Subclinical hyperthyroidism is a mild form where the only abnormal hormone level is a decreased TSH. Overt hyperthyroidism is clear hyperthyroidism with a decreased TSH and an increased T₄ level.

TSH: thyroid stimulating hormone — produced by the pituitary gland that regulates thyroid function; the best screening test to determine if the thyroid is functioning normally.

Thyroxine (T₄): the major hormone produced by the thyroid gland. T₄ gets converted to the active hormone T₃ in various tissues in the body.

TPO antibodies and Thyroglobulin antibodies: these are antibodies that attack the thyroid instead of bacteria and viruses, they are a marker for autoimmune thyroid disease, which is the main underlying cause for hypothyroidism and hyperthyroidism in the United States.

RECIST: Response Evaluation Criteria in Solid Tumors — this is a set of published rules that define when cancer patients improve (“respond”), stay the same (“stable”) or worsen (“progression”) during treatments.





HYPOTHYROIDISM

Obese women with hypothyroidism treated with levothyroxine have a reduced energy expenditure

BACKGROUND

It is well known that thyroid hormone regulates some aspects of our metabolism. When thyroid levels are high (hyperthyroidism), our metabolism increases, we burn more calories and tend to lose weight. When thyroid levels are low (hypothyroidism), our metabolism slows down, we burn less calories and tend to gain weight. Once thyroid hormone levels return to normal after treating these 2 conditions, metabolism returns to normal.

One of the ways to measure metabolism is to look at the energy our body spends at rest. This is called resting energy expenditure. A previous small study has suggested that the resting energy expenditure is lower in patients with hypothyroidism even if they are treated with levothyroxine and their thyroid hormone levels are in the normal range. This study was done to determine the resting energy expenditure in a group of obese women with hypothyroidism attending a bariatric clinic

THE FULL ARTICLE TITLE

Muraca E et al 2020 Resting Energy Expenditure in Obese Women with Primary Hypothyroidism and Appropriate Levothyroxine Replacement Therapy. *J Clin Endocrinol Metab.* Apr 1;105.

SUMMARY OF THE STUDY

This study enrolled 649 obese women (body mass index (BMI) >30 kg/m²) attending a bariatric surgery clinic in Monza, Italy. Of these women, 564 (average age 44)

had no history of thyroid problems, while 85 had a past history of hypothyroidism and were currently treated with levothyroxine (average age 49). Both groups had serum TSH levels within the laboratory reference range (0.4-4.0 mU/L). Resting energy expenditure was measured and eating behavior and physical activity were assessed by questionnaires during a single visit to the clinic.

The hypothyroid group on levothyroxine had slightly higher levels of physical activity and lower levels of insulin resistance. The resting energy expenditure was 6% lower in this group compared to the group without hypothyroidism. There was no correlation between resting energy expenditure and TSH levels in either group. There were no differences between the two groups in body mass index (BMI).

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

Obese hypothyroid women with normal TSH levels have slightly but significantly lower REE than obese women who were not hypothyroid. Even though the energy expenditure was lower in the group that had hypothyroidism and were treated with levothyroxine, their BMI was unaffected as compared to the group without hypothyroidism. While interesting, more studies are needed before changing current recommendations for weight management in patients with hypothyroidism.

—Vibhavasu Sharma, MD, FACE

ATA THYROID BROCHURE LINKS

Hypothyroidism (Underactive): <https://www.thyroid.org/hypothyroidism/>

Thyroid Hormone Treatment: <https://www.thyroid.org/thyroid-hormone-treatment/>

Thyroid and Weight: <https://www.thyroid.org/thyroid-and-weight/>





HYPOTHYROIDISM, continued

ABBREVIATIONS & DEFINITIONS

Body-mass index (BMI): a standardized measure of obesity calculated by dividing the weight in kilograms by the square of the height. A normal BMI is 18.5-24.9, overweight is 25-30 and obese is >30.

Levothyroxine (T4): the major hormone produced by the thyroid gland and available in pill form as Synthroid™, Levoxyl™, Tyrosint™ and generic preparations.

Thyroid hormone therapy: patients with hypothyroidism are most often treated with Levothyroxine in order

to return their thyroid hormone levels to normal. *Replacement therapy* means the goal is a TSH in the normal range and is the usual therapy. *Suppressive therapy* means that the goal is a TSH below the normal range and is used in thyroid cancer patients to prevent growth of any remaining cancer cells.

Hypothyroidism: a condition where the thyroid gland is underactive and doesn't produce enough thyroid hormone. Treatment requires taking thyroid hormone pills.





THYROID CANCER

Being overweight is linked to a genetic mutation that causes increased risk of thyroid cancer

BACKGROUND

The medical community has recognized that excess weight (obesity) is a risk factor for different types of cancers, including breast, colon and pancreatic cancers. Some studies have suggested that obesity is also associated with thyroid cancer. Part of this association is that the increase in thyroid cancer has mirrored the increase in obesity in the United States. Since there has been a rise in the cases of thyroid cancer at the same time that there are more people who are obese, it is important to find the cause of this relationship. Some studies suggest that obese patients seek more medical care and are more likely to be diagnosed with thyroid cancer. Others point to chemicals in the environment that increase both obesity and thyroid cancer. Still others have looked at cancer gene mutations as a cause.

One such cancer gene mutation that is known to promote thyroid cancer is called *BRAF^{V600E}*. This review was done to determine if *BRAF^{V600E}*-positive thyroid cancer was associated with obesity.

THE FULL ARTICLE TITLE

Rahman S et al 2020 Obesity is associated with *BRAF^{V600E}*-mutated thyroid cancer. *Thyroid*. Epub 2020 Mar 31. PMID: 32228152.

SUMMARY OF THE STUDY

This study looked at the Body Mass Index (BMI) and the presence of BRAF mutation in participants ages 18 to 79 between the years 2013 and 2016. The study included

1013 thyroid cancer patients and 1057 individuals without thyroid cancer. The average age of participants was 52 years, 73% were women, and 88% had papillary thyroid carcinoma, of which approximately 59% were *BRAF^{V600E}*-positive. Age and sex distributions were similar between patients with and those without the *BRAF^{V600E}* mutation.

Overweight and obesity were significantly associated with an increased risk of thyroid cancer (as high as double the risk compared to normal weight subjects). This was noticed in thyroid cancers with and without the *BRAF^{V600E}* mutation. In regards to having more *BRAF^{V600E}*-positive thyroid cancers - this was only seen in obese/overweight women (not in men). There was no association between the participants' weight and how aggressive the cancer was.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

In this study, overweight/obesity was associated with general increased risk of thyroid cancer with the *BRAF^{V600E}* mutation in women. This is important because it suggests a genetic link to both obesity and thyroid cancer. At this point, it is unclear if treatment of the obesity or the thyroid cancer would have any effect on the other disorder. Importantly, most obese patients do not have thyroid cancer. However, it is important that doctors and patients are aware of this risk.

— Maria Brito, MD

ATA THYROID BROCHURE LINKS

Thyroid Cancer (Papillary and Follicular): <https://www.thyroid.org/thyroid-cancer/>

Thyroid and Weight: <https://www.thyroid.org/thyroid-and-weight/>





THYROID CANCER, continued

ABBREVIATIONS & DEFINITIONS

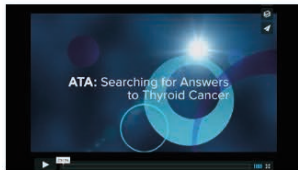
Body-mass index (BMI): a standardized measure of obesity calculated by dividing the weight in kilograms by the square of the height. A normal BMI is 18.5-24.9, overweight is 25-30 and obese is >30.

Cancer-associated genes: these are genes that are normally expressed in cells. Cancer cells frequently have mutations in these genes. It is unclear whether mutations in these genes cause the cancer or are just associated with the cancer cells. The cancer-associated genes important in thyroid cancer are BRAF, RET/PTC and RAS.

BRAF gene: this is gene that codes for a protein that is involved in a signaling pathway and is important for cell growth. Mutations in the BRAF gene in adults appear to cause cancer.

Papillary thyroid cancer: the most common type of thyroid cancer. There are 4 variants of papillary thyroid cancer: classic, follicular, tall-cell and noninvasive follicular thyroid neoplasm with papillary-like nuclear features (NIFTP).

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THYROID CANCER

Aggressive types of thyroid cancer are becoming more common and differ from one another in how dangerous they may be

BACKGROUND

The number of people diagnosed with thyroid cancer in the United States has been increasing steadily over the last several decades. Fortunately, most cases of this disease will be a non-aggressive subtype, called classical papillary thyroid cancer, which, when treated correctly, is very unlikely to be dangerous. This being said, there are other, less common forms of thyroid cancer that are more dangerous than papillary thyroid cancer. These more aggressive thyroid cancer subtypes tend to grow faster and spread (metastasize) to other parts of the body sooner. Such aggressive cancer subtypes include diffuse sclerosing variant, tall cell variant, poorly differentiated thyroid cancer and insular variant.

There are a number of important questions about these uncommon, more aggressive thyroid cancer subtypes that remain to be answered. Unlike classical papillary thyroid cancer, we do not know for sure if the number of people diagnosed with these thyroid cancer types is increasing over time. We also do not know exactly how dangerous these cancer types are, at least compared to classical papillary thyroid cancer. The study described here tries to answer these two questions by evaluating the medical records of large groups of people previously treated for thyroid cancer. By answering these questions, the study authors hope to better understand aggressive types of thyroid cancer and, in particular, to get a better idea of how to treat them.

FULL ARTICLE TITLE

Ho AS et al 2020 Incidence and mortality risk spectrum across aggressive variants of papillary thyroid carcinoma. JAMA Oncol. Epub 2020 Mar 5. PMID: 32134428.

SUMMARY OF THE STUDY

The study authors reviewed the medical records of people who were treated for any kind of thyroid cancer in the United States during the 16 year period between 2000 and

2016. These medical records were identified by examining two large data bases that store information about people being treated for cancer, including the U.S. National Cancer Data Base and the U.S. Surveillance, Epidemiology, and End Results (SEER) data base. In the end, the authors found 5,447 cases of aggressive subtype thyroid cancer, compared to 35,812 cases of classical papillary thyroid cancer. They also found that these aggressive cancer types became increasingly more common during the 16 year study period and that the number of aggressive type thyroid cancer cases grew faster than the number of classical papillary thyroid cancer cases diagnosed in the same timeframe. Similarly, the authors learned that the aggressive subtype thyroid cancers identified were, on average, bigger than the classical papillary thyroid cancer type and were more likely to spread into both neighboring tissues and to other parts of the body. In addition, when the aggressive thyroid cancer types were evaluated individually, the study team found that the four thyroid cancer types evaluated were not equally dangerous. The insular variant type was found to be the most dangerous, with the lowest survival rate, followed by poorly differentiated thyroid cancer, tall cell variant and, least dangerous, diffuse sclerosing variant. In fact, the overall rate of survival for people diagnosed with diffuse sclerosing variant was similar to those diagnosed with classical papillary thyroid cancer, suggesting that the diffuse sclerosing variant subtype may not be particularly dangerous.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

The study authors concluded that aggressive thyroid cancer types are becoming increasingly common and confirmed that 3 of the 4 aggressive cancers are more dangerous than classical papillary thyroid cancer. The diffuse sclerosing variant was found to be similar to the classical papillary thyroid cancer. Moreover, these investigators found that the rate at which these cancer types





THYROID CANCER, continued

are being diagnosed is growing faster than that of other classical papillary thyroid cancer. The findings of this study are important because they alert us that relatively aggressive forms of thyroid cancer appear to be increasing-

ly common and need to be treated more aggressively when diagnosed. Additionally, more studies are needed into how and why these thyroid cancer subtypes develop.

— Jason D. Prescott, MD PhD

ATA THYROID BROCHURE LINKS

Thyroid Cancer (Papillary and Follicular): <https://www.thyroid.org/thyroid-cancer/>

ABBREVIATIONS & DEFINITIONS

Papillary thyroid cancer: the most common type of thyroid cancer. There are several variants of papillary thyroid cancer: classic and follicular are generally non-aggressive while tall-cell, diffuse sclerosing and insular variants are more aggressive. Recently, the noninvasive follicular thyroid neoplasm with papillary-like nuclear features (NIFTP) variant has been described that may be a pre-cancer.

Poorly differentiated thyroid cancer: a less common form of thyroid cancer that can't be clearly identified as papillary or follicular. It appears to be a more aggressive subtype of thyroid cancer.

Cancer metastasis: spread of the cancer from the initial organ where it developed to other organs, such as the lungs and bone.

SEER: Surveillance, Epidemiology and End Results program, a nation-wide anonymous cancer registry generated by the National Cancer Institute that contains information on 26% of the United States population. Website: <http://seer.cancer.gov/>





THYROID CANCER

Detecting thyroid cancer recurrence following lobectomy

BACKGROUND

The initial treatment for thyroid cancer is surgery. Many patients are treated with a total thyroidectomy and these patients are followed for cancer recurrence by measuring levels of the thyroid protein thyroglobulin as a cancer marker. More recently, patients who are low risk and with the cancer confined to one lobe are offered to be treated a lobectomy. Indeed, the number of lobectomies done for thyroid cancer has been increasing recently. However, in patients with a normal lobe after a lobectomy, the thyroglobulin level is much less reliable as a cancer marker.

In this study, the authors report on their experience from a single institution on measuring thyroglobulin levels after a lobectomy for thyroid cancer.

THE FULL ARTICLE TITLE

Ritter A et al 2020 Detecting recurrence following lobectomy for thyroid cancer: Role of thyroglobulin and thyroglobulin antibodies. *J Clin Endocrinol Metab* 105:1–7. PMID: 32219303.

SUMMARY OF THE STUDY

The authors looked at all their adult patients over a 15 period that had a thyroid lobectomy for thyroid cancer

and were monitored by serial serum thyroglobulin and thyroglobulin antibody for cancer recurrence. Most of the 167 patients were female and all were disease stage I (low risk for cancer recurrence). The average cancer size was 9.5 mm and only a small percentage had some aggressive features. Overall, the blood thyroglobulin levels did not correlate with cancer size or more aggressive cancer features and there was fairly equal distribution in patients whose thyroglobulin levels declined, increased, or remained stable over time. Of the group, 10% of patients had a completion thyroidectomy during the study period, and 12 of them had cancer and 6 had benign nodules. The trend in serum thyroglobulin was the same for those patients with cancer in the other thyroid lobe compared to those that had benign disease.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

The authors conclude that serum thyroglobulin and thyroglobulin antibody levels after thyroid lobectomy are not sensitive to detect cancer recurrence. They therefore recommend ultrasound surveillance as well as more research to determine if there is a certain thyroglobulin level that would prompt suspicion for recurrence.

— Melanie Goldfarb, MD

ATA THYROID BROCHURE LINKS

Thyroid Cancer (Papillary and Follicular): <https://www.thyroid.org/thyroid-cancer/>

Thyroid Surgery: <https://www.thyroid.org/thyroid-surgery/>

ABBREVIATIONS & DEFINITIONS

Thyroglobulin antibodies: these are antibodies that attack the thyroid instead of bacteria and viruses, they are a marker for autoimmune thyroid disease, which is the main underlying cause for hypothyroidism and hyperthyroidism in the United States.

Thyroglobulin: a protein made only by thyroid cells, both normal and cancerous. When all normal thyroid tissue is destroyed after radioactive iodine therapy in patients with thyroid cancer, thyroglobulin can be used as a thyroid cancer marker in patients that do not have thyroglobulin antibodies.





THYROID CANCER, continued

Thyroidectomy: surgery to remove the entire thyroid gland. When the entire thyroid is removed it is termed a *total thyroidectomy*. When less is removed, such as in removal of a lobe, it is termed a *partial thyroidectomy*.

Lobectomy: surgery to remove one lobe of the thyroid

Papillary thyroid cancer: the most common type of thyroid cancer. There are 4 variants of papillary thyroid cancer: classic, follicular, tall-cell and noninvasive follicular thyroid neoplasm with papillary-like nuclear features (NIFTP).

Thyroid Ultrasound: a common imaging test used to evaluate the structure of the thyroid gland. Ultrasound uses soundwaves to create a picture of the structure of the thyroid gland and accurately identify and characterize nodules within the thyroid. Ultrasound is also frequently used to guide the needle into a nodule during a thyroid nodule biopsy.

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ATA Alliance for Thyroid Patient Education

GOAL The goal of our organizations is to provide accurate and reliable information for patients about the diagnosis, evaluation and treatment of thyroid diseases. We look forward to future collaborations and continuing to work together toward the improvement of thyroid education and resources for patients.



ThyCa: Thyroid Cancer Survivors' Association, Inc.SM
www.thyca.org



MCT8 - AHDS Foundation



American Thyroid Association

www.thyroid.org

ATA Patient Resources:

www.thyroid.org/thyroid-information/

Find a Thyroid Specialist: www.thyroid.org

(Toll-free): 1-800-THYROID

thyroid@thyroid.org

Bite Me Cancer

www.bitemecancer.org

info@bitemecancer.org

Graves' Disease and Thyroid Foundation

www.gdatf.org

(Toll-free): 877-643-3123

info@ngdf.org

Light of Life Foundation

www.checkyourneck.com

info@checkyourneck.com

MCT8 – AHDS Foundation

mct8.info

Contact@mct8.info

Thyca: Thyroid Cancer Survivors' Association, Inc.

www.thyca.org

(Toll-free): 877-588-7904

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Thyroid Cancer Alliance

www.thyroidcanceralliance.org

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PLEASE JOIN OUR JOURNEY TO ADVANCED DISCOVERIES AND TREATMENT FOR THYROID DISEASE AND THYROID CANCER

As patients with thyroid disease navigate the challenges to their quality of life and researchers and physicians look for more effective directions, we at the ATA have our own destination—**funding for critical thyroid research, prevention, and treatment.** For 94 years, the ATA has led the way in thyroidology. It's a daily obstacle course to find new drugs, better treatments, advanced surgical methods, and more rapid diagnoses for the 20 million Americans who have some form of thyroid disease.



“The ATA was a valuable resource for our family when my dad was diagnosed with Anaplastic Thyroid Cancer. When you're faced with a detrimental diagnosis where even a few days can make the difference in life or death, understanding your options quickly is critical. The ATA website offers a one-stop shop for patients and caregivers to find specialists, current clinical trials, general thyroid cancer information, and links to other patient support groups and information.”

Mary Catherine Petermann

- Father who was diagnosed with Anaplastic Thyroid Cancer in 2006
- He was treated at Mayo Clinic
- He has clean scans as of October 2016

The ATA has paved the way with management guidelines for clinicians who diagnose and treat thyroid disease. For physicians treating pregnant women diagnosed with thyroid disease, our recent publication presents 97 evidence-based recommendations making sure that best practices are implemented with the latest, most effective treatment.



Through your generous support and donations, research takes the lead and hope is on the horizon. **Will you join us** in our campaign to raise **\$1.5 million** for thyroid research, prevention, and treatment? Your compassionate, tax-deductible gift will provide funds for:

- Research grants that pave the way for 1,700 ATA physicians and scientists who have devoted their careers to understanding the biology of and caring for patients affected by thyroid disease.
- Patient education for individuals and families looking for life-changing clinical trials, the best thyroid specialists, and cutting edge treatment and drugs.
- Professional education that offers a wealth of knowledge and leading-edge research for trainees and practitioners.
- A website that is the go-to resource for thyroid information for patients and practitioners alike. In 2016 alone, there were more than 3,700,000 website views of ATA's library of online thyroid information patient brochures.

Donations **of all sizes** will change the future for thyroid patients. You will make a direct impact on patients like Mary Catherine's father as he deals with Anaplastic Thyroid Cancer. You will help scientists like ATA Associate Member Julia Rodiger, Ph.D., a scientist at the National Institutes of Health, as she analyzes thyroid hormones for intestinal stem cell development.

Hyperthyroidism in Pregnancy

WHAT IS THE THYROID GLAND?

The thyroid gland is a butterfly-shaped endocrine gland that is normally located in the lower front of the neck. The thyroid's job is to make thyroid hormones, which are secreted into the blood and then carried to every tissue in the body. Thyroid hormone helps the body use energy, stay warm and keep the brain, heart, muscles, and other organs working as they should.

WHAT ARE THE NORMAL CHANGES IN THYROID FUNCTION ASSOCIATED WITH PREGNANCY?

HORMONE CHANGES. A normal pregnancy results in a number of important physiological and hormonal changes that alter thyroid function. These changes mean that laboratory tests of thyroid function must be interpreted with caution during pregnancy. Thyroid function tests change during pregnancy due to the influence of two main hormones: human chorionic gonadotropin (hCG), the hormone that is measured in the pregnancy test and estrogen, the main female hormone. HCG can weakly turn on the thyroid and the high circulating hCG levels in the first trimester may result in a slightly low TSH. When this occurs, the TSH will be slightly decreased in the first trimester and then return to normal throughout the duration of pregnancy. Estrogen increases the amount of thyroid hormone binding proteins in the serum which increases the total thyroid hormone levels in the blood since >99% of the thyroid hormones in the blood are bound to these proteins. However, measurements of "Free" hormone (that are not bound to protein, representing the active form of the hormone) usually remain normal. The thyroid is functioning normally if the TSH and Free T4 remain in the trimester-specific normal ranges throughout pregnancy.

SIZE CHANGES. The thyroid gland can increase in size during pregnancy (enlarged thyroid = goiter). However, pregnancy-associated goiters occur much more frequently in iodine-deficient areas of the world. It is relatively uncommon in the United States. If very sensitive imaging techniques (ultrasound) are used, it is possible to detect an increase in thyroid volume in some women. This is usually only a 10-15% increase in size and is not typically apparent on physical examination by the physician. However, sometimes a significant goiter may develop and prompt the doctor to measure tests of thyroid function.

WHAT IS THE INTERACTION BETWEEN THE THYROID FUNCTION OF THE MOTHER AND THE BABY?

For the first 18-20 weeks of pregnancy, the baby is completely dependent on the mother for the production of thyroid hormone. By mid-pregnancy, the baby's thyroid begins to produce thyroid hormone on its own. The baby, however, remains dependent on the mother for ingestion of adequate amounts of iodine, which is essential to make the thyroid hormones. The World Health Organization recommends iodine intake of 250 micrograms/day during pregnancy to maintain adequate thyroid hormone production. Because iodine intakes in pregnancy are currently low in the United States, the ATA recommends that US women who are planning pregnancy, pregnant, or breastfeeding should take a daily supplement containing 150 mcg of iodine.

HYPERTHYROIDISM & PREGNANCY

WHAT ARE THE MOST COMMON CAUSES OF HYPERTHYROIDISM DURING PREGNANCY?

Overall, the most common cause of hyperthyroidism in women of childbearing age is Graves' disease (see [Graves' Disease brochure](#)), which occurs in 0.2% of pregnant patients. In addition to other usual causes of hyperthyroidism (see [Hyperthyroidism brochure](#)), very high levels of hCG, seen in severe forms of morning sickness (hyperemesis gravidarum), may cause transient hyperthyroidism in early pregnancy. The correct diagnosis is based on a careful review of history, physical exam and laboratory testing.

WHAT ARE THE RISKS OF GRAVES' DISEASE/ HYPERTHYROIDISM TO THE MOTHER?

Graves' disease may present initially during the first trimester or may be exacerbated during this time in a woman known to have the disorder. In addition to the classic symptoms associated with hyperthyroidism, inadequately treated maternal hyperthyroidism can result in early labor and a serious complication known as pre-eclampsia. Additionally, women with active Graves' disease during pregnancy are at higher risk of developing very severe hyperthyroidism known as thyroid storm. Graves' disease often improves during the third trimester of pregnancy and may worsen during the post partum period.

Hyperthyroidism in Pregnancy

WHAT ARE THE RISKS OF GRAVES' DISEASE/HYPERTHYROIDISM TO THE BABY?

The risks to the baby from Graves' disease are due to one of three possible mechanisms:

1) UNCONTROLLED MATERNAL HYPERTHYROIDISM:

Uncontrolled maternal hyperthyroidism has been associated with fetal tachycardia (fast heart rate), small for gestational age babies, prematurity, stillbirths and congenital malformations (birth defects). This is another reason why it is important to treat hyperthyroidism in the mother.

2) EXTREMELY HIGH LEVELS OF THYROID STIMULATING IMMUNOGLOBULINS (TSI): Graves' disease is an autoimmune disorder caused by the production of antibodies that stimulate the thyroid gland referred to as thyroid stimulating immunoglobulins (TSI). These antibodies do cross the placenta and can interact with the baby's thyroid. High levels of maternal TSI's have been known to cause fetal or neonatal hyperthyroidism, but this is uncommon (only 1-5% of women with Graves' disease during pregnancy). Fortunately, this typically only occurs when the mother's TSI levels are very high (many times above normal). Measuring TSI in the mother with Graves' disease is recommended in early pregnancy and, if initially elevated, again around weeks 18-22.

When a mother with Graves' disease requires antithyroid drug therapy during pregnancy, fetal hyperthyroidism is rare because antithyroid drugs also cross the placenta and can prevent the fetal thyroid from becoming overactive. Of potentially more concern to the baby is when the mother has been treated for Graves' disease (for example radioactive iodine or surgery) and no longer requires antithyroid drugs. It is very important to tell your doctor if you have been treated for Graves' Disease in the past so proper monitoring can be done to ensure the baby remains healthy during the pregnancy.

3) ANTI-THYROID DRUG THERAPY (ATD). Methimazole (Tapazole) or propylthiouracil (PTU) are the ATDs available in the United States for the treatment of hyperthyroidism (see [Hyperthyroidism brochure](#)). Both of these drugs cross the placenta and can potentially impair the baby's thyroid function and cause fetal goiter. Use of either drug in the first trimester of pregnancy has been associated with birth defects, although the defects associated with PTU are less frequent and less severe. Definitive therapy (thyroid surgery or radioactive iodine treatment) may be considered prior to pregnancy in order to avoid the need to use PTU or methimazole in pregnancy. When ATDs are required, PTU is preferred until week 16 of pregnancy. It is recommended that the lowest possible dose of ATD be used to control maternal hyperthyroidism in order to minimize the development of hypothyroidism in the baby. Overall, the benefits to the baby of treating a mother with hyperthyroidism during pregnancy outweigh the risks if therapy is carefully monitored.

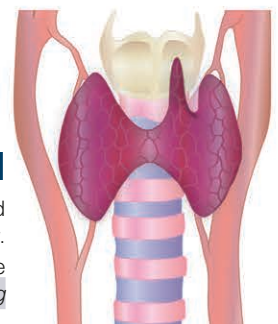
WHAT ARE THE TREATMENT OPTIONS FOR A PREGNANT WOMAN WITH GRAVES' DISEASE/HYPERTHYROIDISM?

Mild hyperthyroidism (slightly elevated thyroid hormone levels, minimal symptoms) often is monitored closely without therapy as long as both the mother and the baby are doing well. When hyperthyroidism is severe enough to require therapy, anti-thyroid medications are the treatment of choice, with PTU being preferred in the first trimester. The goal of therapy is to keep the mother's free T4 in the high-normal to mildly elevated range on the lowest dose of antithyroid medication. Addition of levothyroxine to ATDs ("block-and-replace") is not recommended. Targeting this range of free hormone levels will minimize the risk to the baby of developing [hypothyroidism](#) or [goiter](#). Maternal hypothyroidism should be avoided. Therapy should be closely monitored during pregnancy. This is typically done by following thyroid function tests (TSH and thyroid hormone levels) monthly.

FURTHER INFORMATION

Further details on this and other thyroid-related topics are available in the patient thyroid information section on the American Thyroid Association® website at www.thyroid.org.

For information on thyroid patient support organizations, please visit the [Patient Support Links](#) section on the ATA website at www.thyroid.org



Hyperthyroidism in Pregnancy

In patients who cannot be adequately treated with anti-thyroid medications (i.e. those who develop an allergic reaction to the drugs), surgery is an acceptable alternative. Surgical removal of the thyroid gland is safest in the second trimester.

Radioiodine is contraindicated to treat hyperthyroidism during pregnancy since it readily crosses the placenta and is taken up by the baby's thyroid gland. This can cause destruction of the gland and result in permanent hypothyroidism.

Beta-blockers can be used during pregnancy to help treat significant palpitations and tremor due to hyperthyroidism. They should be used sparingly due to reports of impaired fetal growth associated with long-term use of these medications. Typically, these drugs are only required until the hyperthyroidism is controlled with anti-thyroid medications.

WHAT IS THE NATURAL HISTORY OF GRAVES' DISEASE AFTER DELIVERY?

Graves' disease typically worsens in the postpartum period or may occur then for the first time. When new hyperthyroidism occurs in the first months after delivery, the cause may be either Graves' disease or postpartum thyroiditis and testing with careful follow-up is needed to distinguish between the two. Higher doses of anti-thyroid medications may be required during this time. As usual, close monitoring of thyroid function tests is necessary.

CAN THE MOTHER WITH GRAVES' DISEASE, WHO IS BEING TREATED WITH ANTI-THYROID DRUGS, BREASTFEED HER INFANT?

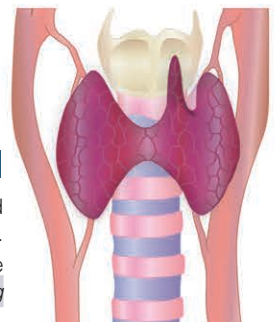
Yes. Although very small quantities of both PTU and methimazole are transferred into breast milk, total daily doses of up to 20mg methimazole or 450mg PTU are considered safe and monitoring of the breastfed infants' thyroid status is not required.



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Hypothyroidism in Pregnancy

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HORMONE CHANGES. Thyroid function tests change during normal pregnancy due to the influence of two main hormones: human chorionic gonadotropin (hCG) and estrogen. Because hCG can weakly stimulate the thyroid, the high circulating hCG levels in the first trimester may result in a low TSH that returns to normal throughout the duration of pregnancy. Estrogen increases the amount of thyroid hormone binding proteins, and this increases the total thyroid hormone levels but the "Free" hormone (the amount that is not bound and can be active for use) usually remains normal. The thyroid is functioning normally if the TSH and Free T4 remain in the trimester-specific normal ranges throughout pregnancy.

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however, remains dependent on the mother for ingestion of adequate amounts of iodine, which is essential to make the thyroid hormones. The World Health Organization recommends iodine intake of 250 micrograms/day during pregnancy to maintain adequate thyroid hormone production. Because iodine intakes in pregnancy are currently low in the United States, the ATA recommends that US women who are planning to become pregnant, who are pregnant, or breastfeeding, should take a daily supplement containing 150 mcg of iodine.

HYPOTHYROIDISM & PREGNANCY

WHAT ARE THE MOST COMMON CAUSES OF HYPOTHYROIDISM DURING PREGNANCY?

Overall, the most common cause of hypothyroidism is the autoimmune disorder known as Hashimoto's thyroiditis (see [Hypothyroidism brochure](#)). Hypothyroidism can occur during pregnancy due to the initial presentation of Hashimoto's thyroiditis, inadequate treatment of a woman already known to have hypothyroidism from a variety of causes, or over-treatment of a hyperthyroid woman with anti-thyroid medications. Approximately, 2.5% of women will have a TSH of greater than 6 mIU/L (slightly elevated) and 0.4% will have a TSH greater than 10 mIU/L during pregnancy.

WHAT ARE THE RISKS OF HYPOTHYROIDISM TO THE MOTHER?

Untreated, or inadequately treated, hypothyroidism has increased risk of miscarriage, and has been associated with maternal anemia, myopathy (muscle pain, weakness), congestive heart failure, pre-eclampsia, placental abnormalities, and postpartum hemorrhage (bleeding). These complications are more likely to occur in women with severe hypothyroidism. Some risks also appear to be higher in women with antibodies against thyroid peroxidase (TPO). Women with mild hypothyroidism may have no symptoms or attribute symptoms they have to the pregnancy.



Hypothyroidism in Pregnancy

WHAT ARE THE RISKS OF MATERNAL HYPOTHYROIDISM TO THE BABY?

Thyroid hormone is critical for brain development in the baby. Children born with congenital hypothyroidism (no thyroid function at birth) can have severe cognitive, neurological and developmental abnormalities if the condition is not recognized and treated promptly. With early treatment, these developmental abnormalities largely can be prevented. Consequently, all newborn babies in the United States are screened for congenital hypothyroidism so they can be treated with thyroid hormone replacement therapy as soon as possible.

Untreated severe hypothyroidism in the mother can lead to impaired brain development in the baby. Recent studies have suggested that mild developmental brain abnormalities also may be present in children born to women who had mild untreated hypothyroidism during pregnancy. At this time, there is no general consensus of opinion regarding screening all women for hypothyroidism during pregnancy. However, the ATA recommends checking a woman's TSH as soon as pregnancy is confirmed in women at high risk for thyroid disease, such as those with prior treatment for hyper- or hypothyroidism, a family history of thyroid disease, a personal history of autoimmune disease, and those with a goiter.

Women with established hypothyroidism should have a TSH test as soon as pregnancy is confirmed. They also should immediately increase their levothyroxine dose, because thyroid hormone requirements increase during pregnancy. (See below for specific dosing recommendations.) If new onset hypothyroidism has been detected, the woman should be treated with levothyroxine to normalize her TSH values (see *Hypothyroidism brochure*).

WHO SHOULD BE TREATED FOR HYPOTHYROIDISM DURING PREGNANCY?

Women found to have a TSH level greater than 10 mIU/L in the first trimester of pregnancy should be treated for hypothyroidism. Conversely, women with a TSH of 2.5 or less, do not need levothyroxine treatment. For women with TSH measured between these (2.5-10), ATA recommendations for treatment vary and may depend on whether or not the mother has TPO antibodies. When TPO antibodies are positive, treatment is recommended when the TSH is above 4 and should be considered when the TSH is between 2.5-4.0. However, when

there are no TPO antibodies (i.e. negative), current ATA recommendations are less strong and suggest that treatment 'may be considered' when TSH is between 2.5-10.0 mIU/L. These recommendations are based on the degree of evidence that exists that treatment with levothyroxine would be beneficial.

HOW SHOULD A WOMAN WITH HYPOTHYROIDISM BE TREATED DURING PREGNANCY?

The goal of treating hypothyroidism in a pregnant woman is adequate replacement of thyroid hormone. Ideally, hypothyroid women should have their levothyroxine dose optimized prior to becoming pregnant. Levothyroxine requirements frequently increase during pregnancy, usually by 25 to 50 percent. Hypothyroid women taking levothyroxine should independently increase their dose by 20%–30% as soon as pregnancy is diagnosed and should notify their doctor for prompt testing and further evaluation. One means of accomplishing the dose increase is to take two additional tablets weekly of their usual daily levothyroxine dosage. Thyroid function tests should be checked approximately every 4 weeks during the first half of pregnancy to ensure that the woman has normal thyroid function throughout pregnancy. As soon as delivery of the child occurs, the woman may go back to her usual pre-pregnancy dose of levothyroxine. It is also important to recognize that prenatal vitamins contain iron and calcium that can impair the absorption of thyroid hormone from the gastrointestinal tract. Consequently, levothyroxine and prenatal vitamins should not be taken at the same time and should be separated by at least 4 hours.

SPECIAL CONSIDERATIONS FOR WOMEN WITH A HISTORY OF GRAVES' DISEASE

In addition to the dosing and testing considerations explained in this brochure, women with a history of *Graves' disease* who were treated with radioiodine (RAI) or surgical thyroidectomy should also have Graves' antibodies (TRAb) tested early in pregnancy to assess the risk of passing antibodies on to the fetus. If antibodies are elevated, follow-up testing is recommended at weeks 18-22, and if antibodies are still elevated, additional follow-up is recommended at weeks 30-34 to evaluate the need for fetal and neonatal monitoring.



FURTHER INFORMATION

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