Clinical THYROIDOLOGY FOR PATIENTS

AMERICAN THYROID ASSOCIATION
FOUNDED 1923

VOLUME 4 • ISSUE 5 • MAY 2011

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EDITOR'S COMMENTS

Welcome to Clinical Thyroidology for Patients. This publication is a collection of summaries of the top articles from the recent medical literature that cover the broad spectrum of thyroid disorders. *Clinical Thyroidology for Patients* is published on a monthly basis and includes summaries of research studies that were discussed in the previous month's issue of *Clinical Thyroidology*, a publication of the American Thyroid Association for physicians. This means that you, the patients, are getting the latest information on thyroid research and treatment almost as soon as your physicians. The Calendar of Events highlights educational forums and support groups that are organized around the country by members of the Alliance for Thyroid Patient Education. The *Alliance* member groups consist of: the *American Thyroid Association*, the *Graves' Disease Foundation*, the *Light of Life Foundation* and *ThyCa: Thyroid Cancer Survivors Association*.

In this issue, the studies ask the following questions:

- Can large nodular goiters be treated with radioactive iodine?
- Does obesity case thyroid cancer?
- Can iodide help patients with Graves' hyperthyroidism respond to treatment quicker?
- When is the best time to take thyroid hormone?
- Do you need to worry about cancer in nodules in patients with autoimmune thyroid disease?
- Can we predict who will develop thyroid disease?
- Do most children with Prader-Willi Syndrome have hypothyroidism?

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

HOW TO NAVIGATE THIS DOCUMENT: The Table of Contents and the Bookmarks are linked to the articles. To navigate, move your cursor over the article title you wish to see (either in the Contents or in the Bookmarks panel) and the hand will show a pointing finger, indicating a link. Left-click the title and the article will instantly appear. To return to the Contents, move the cursor to the bottom of the page and left-click Back to Table of Contents.

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GOITER

Modified-release recombinant human TSH and its effect on large goiters treated with radioactive iodine

BACKGROUND

A thyroid gland that is enlarged for any reason is called a goiter. A goiter can be seen when the thyroid is overactive, underactive or functioning normally. If there are nodules in the goiter it is called a multinodular goiter. Sometimes multinodular goiters get large enough to cause symptoms such as difficult swallowing or choking. When this happens, surgery is often the main option. However, several studies have shown that treatment with radioactive iodine may produce enough shrinking in size of the goiter to improve symptoms. This is especially helpful if the patient cannot undergo surgery or refuses this option. The limiting factor in treating multinodular goiters with radioactive iodine is getting enough radioactive iodine into the gland to be effective. This study was done to determine if stimulating the goiter with a small dose of recombinant human TSH (modified-release recombinant human TSH, MRrhTSH) would allow a higher dose of radioactive iodine to get into the thyroid and cause more shrinking in the size of the goiter, resulting in a more effective treatment. This would provide patients with an alternative treatment to surgery for this condition.

THE FULL ARTICLE TITLE:

Graf H et al. Modified-release recombinant human TSH (MRrhTSH) augments the effect of 131I therapy in benign multinodular goiter: results from a multicenter international, randomized, placebo-controlled study. J Clin Endocrinol Metab. February 23, 2011.

SUMMARY OF THE STUDY

A total of 95 patients with multinodular goiters that were working normally participated in this study. A total of 30 patients did not get the MRrhTSH and were compared to 65 patients who were treated with MRrhTSH before receiving the radioactive iodine. The age of patients ranged from 35-80 years. The results showed that treating with MRrhTSH caused a greater shrinkage in the goiter volume after radioactive iodine as compared to just treating with radioactive iodine alone. In the MRrhTSH group, one patient developed hyperthyroidism and one patient developed an abnormal heart rhythm. There were no complications in the patients that did not get the MRrhTSH.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study shows that radioactive iodine can be effective in treating large symptomatic goiters and that pre-treating with MRrhTSH can make radioactive iodine more effective. MRrhTSH was generally safe. Thus, patients with large multinodular goiters could potentially have an additional treatment option with radioactive iodine and MRrhTSH instead of surgery.

— Heather Hofflich, MD

ATA THYROID BROCHURE LINKS

Radioactive Iodine: http://thyroid.org/patients/patient_brochures/radioactive_iodine.html

Thyroid Surgery: http://thyroid.org/patients/patient_brochures/surgery.html

Goiter: http://thyroid.org/patients/patient_brochures/goiter.html

ABBREVIATIONS & DEFINITIONS

Goiter: a thyroid gland that is enlarged for any reason is called a goiter. A goiter can be seen when the thyroid is overactive, underactive or functioning normally. If there are nodules in the goiter it is called a nodular goiter; if there is more than one nodule it is called a multinodular goiter.

Recombinant human TSH (rhTSH): human TSH that is produced in the laboratory and used to produce high levels of TSH in patients after an intramuscular injection. This is mainly used in thyroid cancer patients before treating with radioactive iodine or performing a whole body scan. The brand name for rhTSH is Thyrogen™.

Modified-release recombinant human TSH (MRrhTSH): a lower dose of rhTSH that may be used in patients with intact thyroid grands.

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.

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THYROID CANCER

Is obesity directly involved in causing thyroid cancer?

BACKGROUND

Thyroid cancer is the fastest rising cancer in women. Obesity is increasing in the general population in both women and men. Obesity is considered to be a risk factor in several cancers, including breast cancer. There are several studies that have examined the risk of obesity on several different types of cancer. This study looked at these studies to examine the risk of obesity on thyroid cancer.

THE FULL ARTICLE TITLE:

Kitahara CM et al Obesity and thyroid cancer risk among U.S. men and women: a pooled analysis of five prospective studies. Cancer Epidemiol Biomarkers Prev 2011;20:464-72. Epub January 25, 2011.

SUMMARY OF THE STUDY

This study looked at the connection between BMI and thyroid cancer using the data from five prospective U.S. National Cancer Institute studies that were performed over various periods between 1979 and 2009 (mean follow-up, 10 years). Baseline questionnaires from approximately 414,000 women and 435,000 men provided information on demographics, lifestyle and medical history. Cancer information was obtained from self-reports (three studies), cancer registry linkage (four), death certificates (three) and/or the National Death Index (four). The type of thyroid cancer was established from clinical and pathology record, and cancer registries. The type of thyroid cancer was known in 1024 patient: 80% were papillary, 15% follicular, 3% medullary and 2% anaplastic. The mean age at study entry was 58 years and 20% of participants were obese.

This study suggested that thyroid cancer was related to BMI. Overweight patients (BMI 25.0 to 29.9) had a 20% increased risk for developing thyroid cancer while obese patients (BMI>30) had a >50% risk of developing cancer as compared to individuals with a normal BMI. There was no significant difference between types of thyroid cancer. This increased risk appeared to be similar in men and women.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This interesting study suggests that obesity is a significant independent risk factor for thyroid cancer in both men and women. At this point there is no evidence that screening obese patients for thyroid cancer is indicated. However, this study should prompt physicians to make a careful thyroid exam a priority on their overweight and obese patients.

— Alan P. Farwell, MD

ATA THYROID BROCHURE LINKS

Thyroid cancer: http://thyroid.org/patients/patient_brochures/cancer_of_thyroid.html

Thyroid and Weight: http://thyroid.org/patients/patient_brochures/weight.html

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.

Papillary thyroid cancer: the most common type of thyroid cancer.

Follicular thyroid cancer: the second most common type of thyroid cancer.

Anaplastic thyroid cancer: a very rare but very aggressive type of thyroid cancer. In contrast to all other types of thyroid cancer, most patients with anaplastic thyroid cancer die of their cancer and do so within a few years.

Medullary thyroid cancer: a relatively rare type of thyroid cancer that often runs in families. Medullary cancer arises from the C-cells in the thyroid.

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HYPERTHYROIDISM

Rapid control of hyperthyroidism from Graves' Disease with iodide

BACKGROUND

Graves' disease is the most common cause of hyperthyroidism. Antithyroid drugs such as methimazole or propylthiouracil are widely used to block the production of thyroid hormones in individuals with Graves' disease, slowing down the overactive thyroid and returning thyroid function to normal. These drugs take 5-10 days to start to improve symptoms and 4-6 weeks or longer to return thyroid hormones to the normal range. Remaining on antithyroid drugs for 6 -18 months sometimes results in the Graves' disease going into remission and allows for medications to be stopped. Potassium iodide taken in large doses acutely blocks the release of the thyroid hormone from the thyroid and can decrease thyroid hormone levels rapidly. However, after a few weeks patients usually escape from the effects of potassium iodide and may become more resistant to the effects of the antithyroid drugs. This study was designed to determine if adding a smaller dose of potassium iodine to treatment with methimazole would improve the hyperthyroidism faster than methimazole alone.

THE FULL ARTICLE TITLE:

Takata K et al Benefit of short-term iodide supplementation to antithyroid drug treatment of thyrotoxicosis due to Graves' disease. Clin Endocrinol (Oxf) 2010;72:845-50.

SUMMARY OF THE STUDY

A total of 134 Japanese patients with Graves' disease completed the study. They were divided into four groups: (1) 30 mg of methimazole alone; (2) 30 mg of methimazole with 50 mg potassium iodide; (3) 15 mg of methimazole alone; and (4) 15 mg of methimazole with 50 mg potassium iodide. They measured levels of thyroxine (T_4), triiodothyronine (T_3) and TSH. When the T_4 entered the normal range, the potassium iodide was stopped, while the methimazole was continued with the dose adjusted to keep the T_4 and TSH in the normal range. Methimazole was discontinued after the blood results were normal for 6 months.

The patients who received the potassium iodide in addition to either dose of methimazole achieved a more rapid normalization of their T_4 than did those patients who received only methimazole. The results were significantly different at 2 weeks of therapy but all groups were similar at 4 weeks of therapy. At one year, there was no significant difference between any of the groups in the percentage of patients who were in remission of the Graves' disease. The addition of potassium iodide to the methimazole did not cause worsening of the hyperthyroidism.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

The authors concluded that the combined treatment of methimazole and potassium iodide improved the short-term control of hyperthyroidism. However, there was no difference between the groups after one month of combined therapy and there was a lack of a difference in the remission rates of hyperthyroidism at one year. At the present time, this should not become standard therapy for the majority of patients with Graves' disease who can be adequately managed with antithyroid drug therapy alone.

— Glenn Braunstein, MD

ATA THYROID BROCHURE LINKS

Graves disease: http://thyroid.org/patients/patient_brochures/graves.html

Hyperthyroidism: http://thyroid.org/patients/patient_brochures/hyperthyroidism.html

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HYPERTHYROIDISM, continued

ABBREVIATIONS & DEFINITIONS

Graves' disease: the most common cause of hyperthyroidism in the United States. It is caused by antibodies that attack the thyroid and turn it on.

Hyperthyroidism: a condition where the thyroid gland is overactive and produces too much thyroid hormone. Hyperthyroidism may be treated with antithyroid meds (Methimazole, Propylthiouracil), radioactive iodine or surgery.

Methimazole: an antithyroid medication that blocks the thyroid from making thyroid hormone. Methimazole is used to treat hyperthyroidism, especially when it is caused by Graves' disease.

Propylthiouracil (PTU): an antithyroid medication that blocks the thyroid from making thyroid hormone. Propylthiouracil is used to treat hyperthyroidism, especially in women during pregnancy.

Thyroxine (T_4): the major hormone secreted by the thyroid gland. Thyroxine is broken down to produce Triiodothyronine which causes most of the effects of the thyroid hormones.

Triiodothyronine (T_3): the active thyroid hormone, usually produced from thyroxine.

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.

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HYPOTHYROIDISM

When is the best time to take thyroid hormone?

BACKGROUND

Hypothyroidism is treated by replacement of thyroid hormone in pill form, specifically levothyroxine. It is well documented that food and a number of medications can decrease the absorption of levothyroxine. This is especially true with calcium and iron pills. Consequently, many patients are instructed to take their levothyroxine on an empty stomach before breakfast and to wait up until an hour before eating. Some patients find this timing inconvenient. A prior study suggested that taking levothyroxine at bedtime was equally as effective in providing stable thyroid hormone levels. The goal of this study was to compare the effect of taking levothyroxine at bedtime as opposed to taking it before breakfast.

THE FULL ARTICLE TITLE:

Bolk N et al. Effects of evening vs. morning levothyroxine intake: A randomized double-blind crossover trial. Arch Intern Med. 2010;170(22):1996-2003.

SUMMARY OF THE STUDY

All patients had hypothyroidism and had been on stable doses of levothyroxine for 6 months when they were assigned to either take levothyroxine on an empty stomach 30 minutes before breakfast or at bedtime. After three months, they switched the timing of levothyroxine to the alternate time (either pre-breakfast or bedtime) for another 3 months, such that all patients experienced both schedules of levothyroxine ingestion. Thyroid hormone, TSH and cholesterol levels were measured every 6 weeks and quality of life questionnaires were assessed every 12 weeks for the 6 month duration of the study. Bedtime levothyroxine ingestion resulted in significantly lower TSH values and higher thyroid hormone levels, indicating improved absorption of levothyroxine when taken at bedtime. There were no significant differences in cholesterol levels or quality of life measures.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study suggests that taking levothyroxine at bedtime results in better absorption than taking it before breakfast. This confirms that taking levothyroxine at different times can result in different levels of thyroid hormones in the blood, emphasizing the need to take it at the same time every day. However, this also confirms that taking levothyroxine at bedtime is an effective alternative to taking it before breakfast. Further, bedtime may be better in patients who appear to have problems absorbing levothyroxine.

—Whitney Woodmansee, MD

ATA THYROID BROCHURE LINKS

Hypothyroidism: http://thyroid.org/patients/patient_brochures/hypothyroidism.html

Thyroid Hormone Treatment: http://thyroid.org/patients/patient_brochures/hormonetreatment.html

ABBREVIATIONS & DEFINITIONS

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.

Levothyroxine: the major hormone produced by the thyroid gland and available in pill form as Levoxyl™, Synthroid™, Levothroid™ 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.

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THYROID CANCER

Thyroid nodules in patients with autoimmune thyroid disease should be evaluated for thyroid cancer

BACKGROUND

Autoimmune thyroid disease is caused by your body making antibodies that attack the thyroid and cause it to be hypothyroid (Hashimoto's thyroiditis) or hyperthyroid (Graves' disease). Autoimmune thyroid disease occurs in 10-12% of the population, although over half of those affected have normal thyroid function. Thyroid cancer is the fastest rising cancer in women. Prior studies have shown that autoimmune thyroid disease and thyroid cancer may occur at the same time. Indeed, patients with autoimmune thyroid disease frequently have enlarged thyroids with nodules found on ultrasound. Scientists have found common genes activated in both conditions. Some studies suggest that inflammation caused by the antibodies in autoimmune thyroid disease may increase the likelihood of thyroid cancer. Another explanation is that the antibodies may stimulate thyroid cancer growth. Other studies suggest these antibodies may actually prevent the development of thyroid cancer. The purpose of this study was to determine the risk of thyroid cancer in patients with autoimmune thyroid diseases.

THE FULL ARTICLE TITLE:

Mukasa K et al Prevalence of malignant tumors and adenomatous lesions detected by ultrasonographic screening in patients with autoimmune thyroid diseases. Thyroid 2011;21:37-41. Epub October 9, 2010.

SUMMARY OF THE STUDY

The patients studied included 1652 patients with Graves' disease and 2036 patients with Hashimoto's thyroiditis. A thyroid ultrasound was done on all patients. Those found to have nodules greater than 1 cm in size or appearance suggestive of thyroid cancer underwent fine needle biopsy. In patients with Hashimoto's thyroiditis, 1.8% were found to have papillary thyroid cancer. In patients with Graves' disease, ~1% were found to have papillary thyroid cancer. Among patients who underwent thyroid biopsies, 5.7% were diagnosed with thyroid cancer, which is similar to the frequency in patients without autoimmune thyroid disease.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

Patient with autoimmune thyroid disease frequently have enlarged thyroids with nodules found on ultrasound. This study suggests that these patients have the same risk of developing cancerous thyroid nodules as patients without autoimmune thyroid disease. This study is an important reminder that patients with autoimmune thyroid disease may develop cancerous thyroid nodules.

— Ruth Belin, MD

ATA THYROID BROCHURE LINKS

Thyroid Nodules: http://thyroid.org/patients/patient_brochures/nodules.html

Hypothyroidism: http://thyroid.org/patients/patient_brochures/hypothyroidism.html

Graves' disease: http://thyroid.org/patients/patient_brochures/graves.html

ABBREVIATIONS & DEFINITIONS

Autoimmune thyroid disease: a diverse group of disorders that are caused by antibodies that get confused and attack the body's own tissues. The disorder depends on what tissue the antibodies attack. Graves' disease and Hashimoto's thyroiditis are examples of autoimmune thyroid disease.

Graves' disease: the most common cause of hyperthyroidism in the United States. It is caused by antibodies that attack the thyroid and turn it on.

Hashimoto's thyroiditis: the most common cause of hypothyroidism in the United States. It is caused by antibodies that attack the thyroid and destroy the gland.

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THYROID CANCER, continued

Thyroid nodule: an abnormal growth of thyroid cells that forms a lump within the thyroid. While most thyroid nodules are non-cancerous (Benign), ~5% are cancerous.

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.

Thyroid fine needle aspiration biopsy (FNAB): a simple procedure that is done in the doctor's office to determine if a thyroid nodule is benign (non-cancerous) or cancer. The doctor uses a very thin needle to withdraw cells from the thyroid nodule. Patients usually return home or to work after the biopsy without any ill effects.

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AUTOIMMUNE THYROID DISEASES

Can we predict who will develop thyroid dysfunction, and when?

BACKGROUND:

Hashimoto's thyroiditis and Graves' disease are autoimmune disorders where the immune system damages or alters the function of the thyroid gland. In Hashimoto's thyroiditis, antibodies attack the thyroid and destroy the gland, producing hypothyroidism. In Graves' disease, antibodies attack the thyroid and turn it on, producing hyperthyroidism. These disorders often run in families. Thyroid patients often wonder if their children are at risk for developing thyroid problems and, if so, when they are likely to occur. This study was done to follow a group of people at risk for the development of thyroid disease to answer these questions.

FULL ARTICLE TITLE:

Effraimidis et al. Natural history of the transition from euthyroidism to overt autoimmune hypo- or hyperthyroidism: a prospective study Eur J Endocrinol 2011;164:107-113. Epub October 18, 2010.

SUMMARY OF THE STUDY:

Close to 800 healthy women in Amsterdam ages 18-65 who had at least one relative with documented autoimmune thyroid disease were followed annually for 5 years. Approximately 5 percent became hypothyroid while 1.6 % became hyperthyroid. While everyone had normal thyroid levels at the beginning the study, the people who became hypothyroid had higher titers of TPO antibodies, higher TSH levels and lower levels of T_4 as compared with those patients that did not develop hypothyroidism. It took an average of three years for hypothyroidism to develop.

In the people who became hyperthyroid, TPO antibodies were more common but there was no difference in thyroid hormone levels, even at the last annual study before development of hyperthyroidism.

IMPLICATIONS OF THE STUDY:

As has been shown in different ways in previous studies, the development of Hashimoto's thyroiditis is a relatively slow process over many years. On the other hand, Graves' disease seems to develop rapidly in susceptible people and may be triggered by as yet unknown stresses.

This study raises the question whether some patients who may develop Hashimoto's thyroiditis should be treated with thyroid hormone replacement even before they develop hypothyroidism. However, precisely who could benefit from this remains quite unclear since, in this study, only a small fraction of people became hypothyroid.

— Henry Fein, MD

ATA THYROID BROCHURE LINKS

Hypothyroidism: http://thyroid.org/patients/patient_brochures/hypothyroidism.html

Graves disease: http://thyroid.org/patients/patient_brochures/graves.html

Hyperthyroidism: http://thyroid.org/patients/patient_brochures/hyperthyroidism.html

ABBREVIATIONS & DEFINITIONS

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

Hashimoto's thyroiditis: the most common cause of hypothyroidism in the United States. It is caused by antibodies that attack the thyroid and destroy the gland.

Hyperthyroidism: a condition where the thyroid gland is overactive and produces too much thyroid hormone. Hyperthyroidism may be treated with antithyroid meds (Methimazole, Propylthiouracil), radioactive iodine or surgery.

Graves' disease: the most common cause of hyperthyroidism in the United States. It is caused by antibodies that attack the thyroid and turn it on.

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AUTOIMMUNE THYROID DISEASES, continued

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.

Thyroxine (T_4): the major hormone secreted by the thyroid gland. Thyroxine is broken down to produce Triiodothyronine which causes most of the effects of the thyroid hormones.

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.

Antibodies: proteins that are produced by the body's immune cells that attack and destroy bacteria and viruses that cause infections. Occasionally the antibodies get confused and attack the body's own tissues, causing autoimmune disease.

Autoimmune disorders: a diverse group of disorders that are caused by antibodies that get confused and attack the body's own tissues. The disorder depends on what tissue the antibodies attack. Graves' disease and Hashimoto's thyroiditis are examples of autoimmune thyroid disease. Other Autoimmune disorders include: type 1 diabetes mellitus, Addison's disease (adrenal insufficiency), vitiligo (loss of pigment of some areas of the skin), systemic lupus erythematosus, pernicious anemia (B12 deficiency), celiac disease, inflammatory bowel disease, myasthenia gravis, multiple sclerosis and rheumatoid arthritis.

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HYPOTHYROIDISM

Children with Prader–Willi Syndrome frequently have central hypothyroidism

BACKGROUND

Prader–Willi syndrome is a rare genetic disease with an incidence of 1 in 10,000 to 1 in 16,000 of newborns. Patients with this disorder have a variety of problems including moderate to severe mental retardation, short stature and severe obesity that is a result of a marked increase in appetite. There are multiple endocrine abnormalities that affect pituitary function. The pituitary produces TSH that regulates thyroid function. The vast majority of hypothyroidism is cause by thyroid gland failure and is associated with increased TSH levels. Hypothyroidism due to pituitary problems (central hypothyroidism) is associated with normal or low TSH levels in the setting of low thyroid hormone levels. Central hypothyroidism has been reported in 20% to 30% of patients. This study examined thyroid function in all infants diagnosed at the Hospital de Pediatria Garraham in Buenos Aires over a period of 5 years.

THE FULL ARTICLE TITLE:

Vaiani E et al. Thyroid axis dysfunction in patients with Prader–Willi syndrome during the first 2 years of life. Clin Endocrinol 2010;73:546-50.

SUMMARY OF THE STUDY

Eighteen patients (11 boys and 7 girls) up to 2 years of age were included in this study. The diagnosis was documented by genetic testing. The diagnosis of central hypothyroidism was considered if serum T_4 was less than the 2.5th percentile of the normal range and if there was absence of the expected increase in serum TSH.
In 61% of patients (11 of 18), T_4 values were below the 2.5 percentile and in 2 additional cases the value was just borderline. With the exception of one case, all patients had normal serum T_3 levels. Interestingly, body length was significantly shorter in the hypothyroid patients than in the small group of patients with normal T_4 levels.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This small study suggests that central hypothyroidism is common in Prader–Willi syndrome and is seen at a very early stage of life. At present, it is not clear whether this hypothyroidism is short-lived or permanent. While these findings do need to be confirmed, this study suggests that pediatricians treating patients with Prader–Willi syndrome should have a low threshold for diagnosing and treating central hypothyroidism.

— Alan P. Farwell, MD

ATA THYROID BROCHURE LINKS

Hypothyroidism: http://thyroid.org/patients/patient_brochures/hypothyroidism.html

ABBREVIATIONS & DEFINITIONS

Prader–Willi syndrome: a rare genetic disorder associated with moderate to severe mental retardation, short stature and obesity due to over-eating.

Pituitary gland: this endocrine sits at the base of the brain and secretes hormones that control thyroid and adrenal function, growth and reproduction. The pituitary gland secretes TSH to control thyroid function.

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

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.

Thyroxine (T_4): the major hormone secreted by the thyroid gland. Thyroxine is broken down to produce Triiodothyronine which causes most of the effects of the thyroid hormones.

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.

WHO WE ARE

AMERICAN THYROID ASSOCIATION

www.thyroid.org
ATA Patient Resources: http://www.thyroid.org/patients/
Find a Thyroid Specialist: www.thyroid.org
Phone (toll-free): 1-800-THYROID
e-mail: thyroid@thyroid.org

ATA Mission: The ATA leads in promoting thyroid health and understanding thyroid biology.
ATA Vision: The ATA is the leading organization focused on thyroid biology and the prevention and treatment of thyroid disorders through excellence and innovation in research, clinical care, education, and public health.
ATA Values: The ATA values scientific inquiry, clinical excellence, public service, education, collaboration, and collegiality.

To further our mission, vision and values the ATA sponsors "Friends of the ATA" online to advance the information provided to patients and the public such as this publication, *Clinical Thyroidology for Patients*. We welcome your support.

GRAVES' DISEASE FOUNDATION

www.ngdf.org
Phone (toll-free): 1-877-NGDF-123 or 643-3123
e-mail: Gravesdiseasefd@gmail.com

Founded in 1990, the Graves' Disease Foundation offers support and resources to Graves' disease patients, their families, and health care professionals. Their mission is to find the cause of and the cure for Graves' thyroid disease through research, to improve the quality of life for persons with Graves' disease and their caregivers and to educate persons with Graves' disease, their caregivers, healthcare professionals, and the general public about Graves' disease and its treatment. The web site features a monitored bulletin board.

LIGHT OF LIFE FOUNDATION

www.checkyourneck.com
email: info@checkyourneck.com

The Light of Life Foundation, founded in 1997, is a nonprofit organization that strives to improve the quality of life for thyroid cancer patients, educate the public and professionals about thyroid cancer, and promote research and development to improve thyroid cancer care.

THYCA: THYROID CANCER SURVIVORS' ASSOCIATION, INC.

www.thyca.org
Phone (toll-free): 877 588-7904
e-mail: thyca@thyca.org

ThyCa: Thyroid Cancer Survivors' Association, Inc., founded in 1995, is an international nonprofit organization, guided by a medical advisory council of renowned thyroid cancer specialists, offering support and information to thyroid cancer survivors, families, and health care professionals worldwide.

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Graves' Disease Foundation
Light of Life Foundation checkyourneck.com
ThyCa

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ATA Alliance for Thyroid Patient Education
CALENDAR OF EVENTS

Educational forums, patient support groups and other patient-oriented meetings

ATA Conferences www.thyroid.org

Saturday, October 29, 2011
1:00 pm – 3:00 pm — Indian Wells, CA
FREE Public Health Forum — Thyroid Disease and You

Graves' Disease Conferences www.ngdf.org

Fall, 2011 — Boston, MA
Annual Patient & Family Conference

Light of Life Foundation www.checkyourneck.com

Ongoing — www.checkyourneck.com
Thyroid Cancer Awareness campaign with Cindy Crawford and Brooke Shields

June 12, 2010 — a previous symposium available online at:
http://www.checkyourneck.com/About-Thyroid-Cancer/Thyroid-Cancer-Symposium-Presentations

Thyroid Cancer Symposium Presentations: What's New in Thyroid Cancer?
A Day for Patients and Their Families

Please visit the Light of Life Foundation website to view the Patient Educational Symposium which took place in NYC in 2010. As part of the Patient Educational Program these online presentations provide valuable information in hopes that patients everywhere can gain further information and support about their disease.

ThyCa Conferences www.thyca.org

October 14–16, 2011 — Los Angeles, California
14th International Thyroid Cancer Survivors' Conference
(at the Hilton Los Angeles Airport Hotel, 5711 West Century Boulevard, Los Angeles, California)

September, 2011 — Worldwide
Thyroid Cancer Awareness Month

FREE Public Health Forum

AMERICAN THYROID ASSOCIATION®
FOUNDED 1923
www.thyroid.org

Thyroid Experts from the American Thyroid Association and thyroid patients join together to inform the general public, other thyroid patients, and their friends and families about:

Thyroid Disease and You

Have you experienced a significant change in:

- Energy?
- Memory?
- Fatigue level after a good night's sleep?
- Depression?
- Rapid heart beat?
- Restlessness?
- Infertility?
- Weight?
- Hair?
- A lump on your neck?

Could it be your thyroid?

Public Forum will be held on Saturday, October 29, 2011

1:00 pm – 3:00 pm • Indian Wells, California

Renaissance Esmeralda Resort and Spa, 44-400 Indian Wells Lane, Indian Wells CA 92210-8708
Phone: 760-773-4444 or toll free at 800-446-9875

Physician experts will discuss thyroid disorders. This program is free and all are welcome, including walk-in-attendees. Reservations are encouraged to ensure we have enough seating. For more information and to register, please e-mail ThyCa at thyca@thyca.org.

Who should attend? Anyone who has had an overactive or underactive thyroid, thyroiditis, a thyroid nodule, thyroid cancer, or a family history of thyroid problems or related disorders, including rheumatoid arthritis, juvenile diabetes, pernicious anemia, or prematurely gray hair (starting before age 30) Please come if you have questions, symptoms, or concerns about a thyroid problem. Receive free educational materials.

Reservations requested. Walk-ins welcome. E-mail thyca@thyca.org to RSVP
(Please indicate in your message the thyroid condition you are most concerned about.)

Online educational information for patients is provided by all members of the ATA Alliance for Patient Education co-sponsoring this forum: ThyCA: Thyroid Cancer Survivors' Association, Light of Life Foundation, and Graves' Disease Foundation. Go online to www.thyroid.org and click on "Patients and Public" to access the resources you need.