MASSACHUSETTS GENERAL HOSPITAL

Neuroendocrine & Pituitary Tumor Clinical Center (NEPTCC) Bulletin

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Cushing's disease in pregnancy

MELANIE SCHORR, MD

Case presentation

A 28-year-old woman was referred to the Neuroendocrine and Pituitary Tumor Clinical Center for management of Cushing's disease (CD). Four years prior to referral, she had a full-term uncomplicated pregnancy and delivered a baby girl. Post-partum, she experienced depression, fatigue, hirsutism, and easy bruising, as well as inability to lose weight and secondary amenorrhea. Three years prior to referral, an outside endocrinologist suspected Cushing's syndrome (CS), and checked 24-hour urine free cortisols (UFC), which were 10x the upper limit of normal (ULN), and late night salivary cortisols (LNSC), which were also elevated. Serum adrenocorticotropic hormone (ACTH) was elevated, suggesting ACTH-dependent CS. A pituitary MRI revealed a 12 x 9 x 6 mm right sellar lesion. The patient then underwent transsphenoidal surgery at an outside hospital, and pathology was consistent with an ACTH-producing pituitary adenoma. Post-operatively, UFC normalized, but she did not become adrenally insufficient, suggesting she was not in remission and was at a high risk of recurrent hypercortisolemia. Post-operative MRI did not show clear residual tumor.

Symptomatically, the patient felt much improved; her mood, fatigue, hirsutism and easy bruising improved, she was able to lose 20 lbs and had resumption of her menstrual periods. However, two years later, symptoms recurred and she gained 100 lbs. She was referred back to her outside endocrinologist, who suspected persistent CD, and checked UFC, which were 1.5x ULN, and LNSC, which were also elevated. Pituitary MRI demonstrated a small area of residual/recurrent disease in the medial wall of the right cavernous sinus. The patient was then referred to the Neuroendocrine and Pituitary Tumor Clinical Center for management of persistent CD. On exam, she did not appear particularly Cushingoid, as she had normal fat distribution, no extremity thinning and no posterior cervical or supraclavicular fat pads. She did have light violaceous striae over her abdomen. It was recommended that more UFC and LNSC be collected to determine the degree of cortisol excess. Plan was also made for referral to an expert pituitary surgeon, but in the interim, the patient called to report that she was pregnant. Cortisol testing, which had been performed when she was at 8 weeks gestation prior to knowledge of the pregnancy, revealed UFC that were approximately 4x ULN and elevated LNSC. This case raised a number of questions, namely (1) How do you interpret UFC and LNSC in pregnancy? (2) Does CS during pregnancy adversely affect maternal and fetal outcomes? (3) Does treatment of CS during pregnancy improve maternal and fetal outcomes? and (4) What are the treatment options for CS during pregnancy?

How do you interpret UFC and LNSC during pregnancy?

Pregnancy is associated with a number of physiologic changes, including activation of the maternal hypothalamic-pituitary-adrenal axis[1]. Placental estradiol increases serum cortisol binding globulin (CBG) levels, which raises total serum cortisol levels, as most cortisol is protein-bound in the circulation. Placental corticotropin-releasing hormone (CRH) stimulates placental and pituitary ACTH, which increases cortisol production from the maternal adrenal glands. Placental progesterone also induces a relative glucocorticoid resistance, which may further increase maternal serum cortisol levels. The fetus is partially protected from this maternal hypercortisolemia because placental 11β-hydroxysteroid dehydrogenase 2 converts 85% of maternal cortisol to biologically inactive cortisone prior to it entering the fetal circulation.

Recent longitudinal studies that have followed healthy women from early pregnancy to post-partum have demonstrated that 24hr UFC levels were elevated 1.7-, 2.4-, and 3.1- fold during the 1st, 2nd, and 3rd trimesters compared to healthy controls[2]. Studies documenting LNSC levels across gestation have reported variable results. One cross-sectional study reported that pregnant women in the 2nd and 3rd trimester had significantly higher LNSC compared to healthy controls[3], although another longitudinal study reported no significant differences in LNSC across gestation compared to healthy controls[4]. Studies more consistently demonstrate that within 2-3 months post-partum, UFC and LNSC are no longer elevated compared to healthy controls[2,4].

Consistent with these data, the 2008 Endocrine Society Clinical Practice Guideline on the diagnosis of CS recommended that "UFC values in the 2nd or 3rd trimester >3x ULN can be taken to indicate CS," but that "diagnostic thresholds for LNSC in pregnant patients are not known"[5]. Given that this patient's UFC was >5x ULN, we were confident that this was consistent with CD, and not physiologic hypercortisolemia of pregnancy.

Does CS during pregnancy adversely affect maternal and fetal outcomes?

The first description of CS during pregnancy was a case series of 7 patients published in 1953 by Hunt and McConahey[6]. The fetal or newborn mortality rate in this case series was 43%. More recent reports have confirmed that maternal and fetal complications are common in CS during pregnancy. In a review of 136 cases of treated or untreated CS during pregnancy, the maternal mortality rate was 2% and maternal morbidity included hypertension (68%), diabetes mellitus or impaired glucose tolerance (25%) and preeclampsia (14%)[7]. In a review of 128 cases of untreated CS during pregnancy, overall fetal loss was 31%, prematurity was 66%, and low birth weight was 68%[8].

Does treatment of CS during pregnancy improve maternal and fetal outcomes?

Studies have demonstrated that the treatment of CS during pregnancy reduces fetal mortality. In a review of 213 cases of CS during pregnancy, treatment was associated with a significant reduction in overall fetal loss (from 60.1% with no treatment to 11.3% with medical treatment, 23.9% with surgical treatment and 4.7% with medical and surgical treatment) (Table 1)[8]. There was no significant difference in the rate of preterm birth or low birth weight with treatment, although the heterogeneity of the groups in terms of the etiology, diagnosis and treatment of CS may explain the lack of significant difference. In addition to improving fetal mortality, treatment also improves maternal morbidity. In a review of 23 cases of adrenalectomy for adrenocortical adenoma causing CS during pregnancy, there was resolution of symptoms related to CS and/or improved control of diabetes mellitus and/or hypertension in 88% of patients[7]. However, the authors reported that the incidence of preterm delivery and intrauterine growth restriction were not significantly different from those of untreated CS during pregnancy.

Table 1 - Fetal outcome by treatment in women with active Cushing's syndrome during pregnancy

	No treatment $N=128$	Medical treatment $N=24$	Surgical treatment $N=49$	Medical and surgical treatment $N=10$	p-value
Overall fetal loss (%)	30.6	20.8	12.5	0	0.021
Preterm birth (%)	66.3	76.2	56.1	80	0.304
Low birth weight (%)	68.3	68.8	73.3	80	0.883

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What are the treatment options for CD during pregnancy?

Transsphenoidal surgery for CD during pregnancy is preferred over medical therapy if it is deemed safe for the mother and fetus. Surgery during the 2nd trimester is associated with a lower rate of maternal and fetal complications compared to surgery during the 1st or 3rd trimester[9]. However, in a review of cases of CS during pregnancy, only 20% of women with CD underwent transsphenoidal surgery during pregnancy, perhaps due to a lack of access to expert pituitary surgery teams who feel comfortable operating on pregnant women[7]. Currently, there are no FDA-approved medications to treat CD in pregnancy. For those patients who are not surgical candidates, decline surgery, or need adjunctive therapy, the medication most commonly used off-label for the treatment of CD in pregnancy is metyrapone (pregnancy category C)[10]. Metyrapone is an 11β-hydroxylase inhibitor, thereby blocking the conversion of 11-deoxycortisol to cortisol. However, 11β-hydroxylase inhibition also results in the build up of mineralcorticoid precursors, which can cause side effects such as hypertension, hypokalemia, and edema, and therefore blood pressure, serum potassium, and edema must be frequently monitored.

After a discussion of the benefits and risks of treatment with the patient, pituitary surgeon, and high-risk obstetrics, the decision was made to pursue transsphenoidal surgery in the 2nd trimester. Pathology from this 2nd transsphenoidal surgery was consistent with an ACTH-producing pituitary adenoma. However, the resection was not complete, presumably due to dural and/or cavernous sinus invasion, as post-operative 24hr UFC remained 3-4x ULN, so metyrapone was titrated to achieve a goal UFC 2-3x ULN for the 2nd trimester. Blood pressure and serum potassium were monitored weekly and remained normal. When the patient went into labor, metyrapone was stopped and stress dose glucocorticoids were started for labor and delivery of a full-term, healthy baby boy. Post-partum, glucocorticoids were rapidly tapered off, and UFC and LNSC were repeated 1 month post-partum revealing UFC 1.5-2x ULN and elevated LNSC. Cabergoline was then started to achieve short-term control of hypercortisolemia because dosing was more convenient compared to metyrapone and the patient had chosen to stop breast-feeding. Proton beam radiosurgery was then performed with the goal of achieving long-term control of hypercortisolemia. The patient remains on cabergoline with normalized UFC and LNSC.

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Effects of Growth Hormone on Thyroid Function in Patients with Growth Hormone Deficiency – A Potential Effect of GH on Type 2 Iodothyronine Deiodinase

JANET LO, MD

The growth hormone (GH)-insulin-like growth factor-1 (IGF-I) axis has notable effects on thyroid function and thyroid hormone metabolism. In the literature, concern had been raised that GH replacement may unmask undiagnosed central hypothyroidism[1, 2]. GH treatment was shown in several studies to lower levels of thyroxine (T4) and free thyroxine (free T4) in adults and children with growth hormone deficiency (GHD) [1-6]. Some studies have shown that GH and IGF-I can increase levels of triiodothyronine (T3)[7, 8], while others have shown no change in free T3 levels with GH replacement[3]. In healthy men, Grunfeld et al. demonstrated that recombinant human GH (rhGH) at a dose of 0.125mg/day subcutaneously for four days acutely reduced serum total T4 and free T4, increased total T3 and markedly decreased serum TSH, considered likely to be compensatory[9].

These prior observations have led endocrinologists to hypothesize that GH likely stimulates the peripheral conversion of T4 to T3. Conversely, effects of an acute decline in GH levels on thyroid function have also been assessed in patients with acromegaly, demonstrating that T3 levels transiently decreased after transsphenoidal surgery (TSS)[10]. Jorgensen et al. demonstrated that GH increased total and free T3 and reduced rT3 in a dose-dependent manner, and therefore, likely induced peripheral T4 to T3 conversion[8]. The exact mechanism whereby GH increases the conversion of T4 to T3 was previously uncertain although a GH-induced alteration in deiodinase activity was hypothesized. In light of these prior observations, recently Dr. Yamauchi and colleagues performed new mechanistic studies to further elucidate the physiologic mechanisms mediating the effects of growth hormone on thyroid function[11].

Yamauchi et al. first performed two retrospective observational studies in 1) adults with severe GHD before and after receiving GH therapy and 2) in adults with acromegaly before and after trans-sphenoidal surgery (TSS)[11]. In both studies, serum levels of free T3, free thyroxine (free T4) and free T3 and free T4 ratio were assessed before and after intervention. In the first study, consecutive adult patients with GHD (confirmed by peak serum GH <9ng/mL after GH-releasing peptide-2 test according to Japan Endocrine Society guidelines) identified using medical records in Kyoto University Hospital, Japan, who started GH replacement therapy between August 2008 and July 2015 were selected. After exclusion of patients with missing data on thyroid function, 20 patients were included in the analysis. Serum free T3, free T4 and TSH levels were obtained before and 3 months after initiation of GH replacement. The median dose of GH was 0.2mg/day

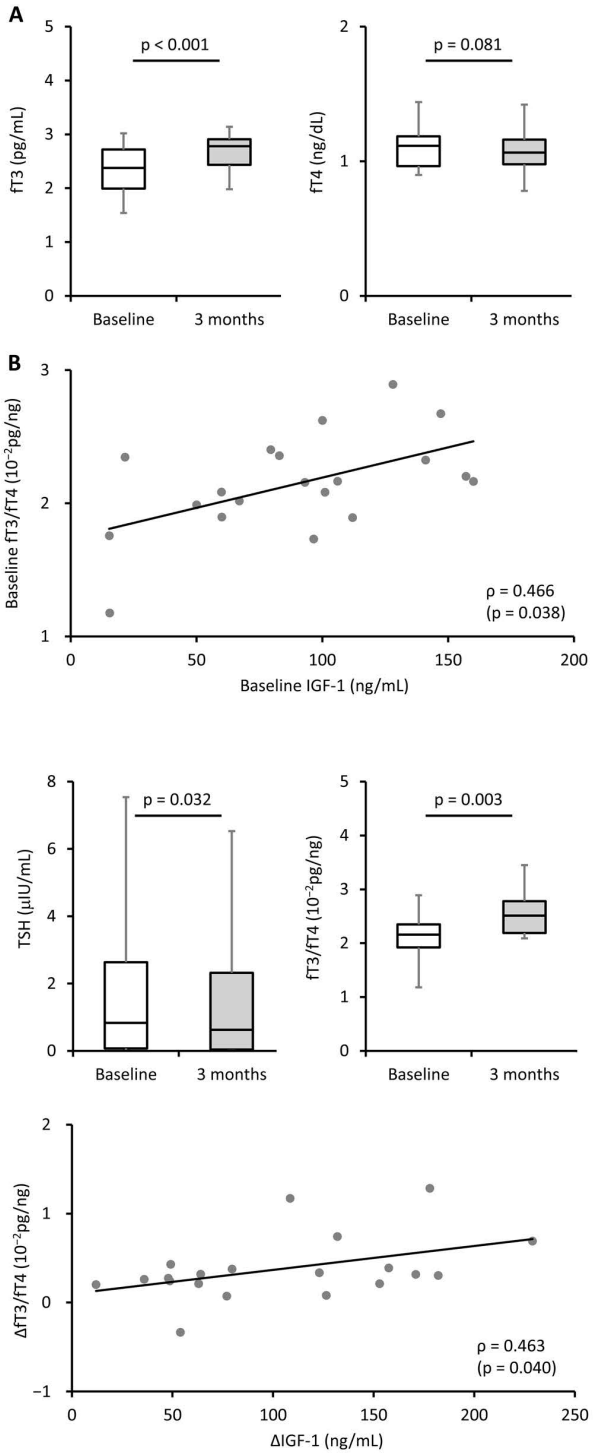


Figure 1. a Box-plot of thyroid function at baseline and 3 months after recombinant human growth hormone (rhGH) replacement therapy in 20 patients with adult GH deficiency. Each box represents the interquartile range. The horizontal line in each box represents the median. The ends of the vertical lines represent the minimum and maximum values. b Linear regression of insulin-like growth factor 1 (IGF-1) and free triiodothyronine /free thyroxine (fT3/fT4) ratio at baseline, and that of changes in IGF-1 (ΔIGF-1) and fT3/fT4 ratio (ΔfT3/fT4) between baseline and 3 months after rhGH therapy. ρ, Spearman's rank correlation coefficient. Reprinted by permission from: Endocrine/Springer Nature, Yamauchi I, Sakane Y, Yamashita T, Hirota K, Ueda Y, Kanai Y, Yamashita Y, Kondo E, Fujii T, Taura D, Sone M, Yasoda A, Inagaki N. Effects of growth hormone on thyroid function are mediated by type 2 iodothyronine deiodinase in humans. (2018)

at 3 months and median IGF-I level rose from 95ng/mL to 179 ng/mL. Median free T3 significantly rose (Figure 1). Free T4 tended to decrease, but did not meet statistical significance (Figure 1). The ratio of free T3/free T4 increased and TSH decreased (Figure 1). Changes in IGF-I positively correlated with changes in free T3 and free T4 ratio (Figure 1).

In the second study, the authors analyzed medical records of 27 consecutive patients with acromegaly, but had to exclude one patient because of missing data on thyroid function and one patient due to hyperthyroidism, so data from 25 patients were analyzed. Thyroid function tests were measured at post-operative day 16. Median serum free T3 level decreased and free T4 increased sixteen days after TSS. Free T3/free T4 ratio decreased. Changes in IGF-I also correlated positively with changes in free T3/free T4 ratio.

The authors then performed in vitro studies to assess the effects of GH administration on the expression of iodothyronine deiodinases types I, II, and III (D1, D2 and D3) in human cell lines HepG2m (derived from human hepatoblastoma), TSA201 (derived from human embryonic kidney cells), MCF7 (derived from human breast carcinoma), and HTC/C3 (derived from human thyroid undifferentiated carcinoma). Changes in mRNA levels of D1, D2 and D3 iodothyronine deiodinases (DIOs) in these cells were assessed in response to rhGH. The main finding was that mRNA levels of DIO2 increased significantly in HTC/C3 cells in response to GH. Western blot experiments confirmed that protein levels of D2 were significantly increased by GH. DIO1 mRNA expression in HTC/C3 and HepG2 cells was unchanged by GH. D3 protein levels were unchanged and DIO3 mRNA expression showed no change in TSA201, MCF7 and HTC/C3 cells in response to GH. These new studies by Dr. Yamauchi and colleagues shed additional light on the potential mechanism of GH's impact on thyroid function, suggesting an important effect of GH on DIO2 mRNA expression and increased protein levels of D2.

Serum T3 levels are normally maintained at constant levels in the body, and D1, D2 and D3 in peripheral tissues are the key determinants of serum T3 level12. D2 activity is thought to be an important source of extrathyroidal T3 production in humans. What are the clinical consequences of increased peripheral T4 to T3 conversion for patients with GHD as they initiate GH replacement therapy? It appears that in patients with idiopathic isolated GHD, the effects of GH on the thyroid function is likely to be transient as the normal hypothalamic-pituitary-thyroid (HPT) axis will regulate thyroid function via an intact HPT feedback system. However, in patients with hypopituitarism and those who are at risk for developing central hypothyroidism, close monitoring of thyroid function is warranted before and after initiating GH replacement therapy, as it may unmask abnormal thyrotroph function. Another unanswered question is why teleologically does this effect of GH on thyroid function occur? The stimulatory effects of GH on peripheral conversion of T4 to T3 have

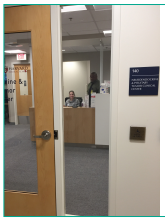
Research Studies Available

Patients may qualify for research studies in the Neuroendocrine and Pituitary Tumor Clinical Center. We are currently accepting the following categories of patients for screening to determine study eligibility. Depending on the study, subjects may receive free testing, medication and/or stipends.

SUBJECTS	STUDIES	CONTACT
Adults with active or treated acromegaly	Quality of life	Lisa Nachtigall, MD
	Cross-sectional bone density study	Karen Pulaski Liebert, RN
Men with hypopituitarism	Study evaluating sustained control with oral octreotide	Elizabeth Lawson, MD
	Characterization of oxytocin deficiency	

The Neuroendocrine and Pituitary Tumor Clinical Center is involved in many different research studies.

Types of studies and enrollment status changes frequently, so please call our office (617-726-3870) or check our webpage (massgeneral.org/neuroendocrine) for more information about potential studies which may not be listed here.



We moved!

The Neuroendocrine and Pituitary Tumor Clinical Center is now located on the MGH main campus directly across the street from our previous location.

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been conserved and have been observed in other species including rainbow trout[13], chickens[14], and lambs[15] although possibly via different deiodinases and mechanisms than in humans. The teleological rationale is still unknown.

In summary, the published data support that GH stimulates peripheral T4 to T3 conversion and now Yamauchi et al. have demonstrated that this effect may be mediated by GH-stimulated increase in D2. The long-term clinical implications are still unclear. The Endocrine Society Clinical Practice Guidelines for the Evaluation and Treatment of Adult Growth Hormone Deficiency recommend that "freeT4 levels should be monitored during GH treatment, and doses of T4 should be adjusted as necessary" [16]. In conclusion, data and guidelines suggest that it would be prudent for endocrinologists to routinely monitor thyroid function tests in all patients before and after initiating GH, especially in individuals with other pituitary hormonal deficits or pathology that may predispose them to central hypothyroidism.

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SPECIAL LECTURE

19th Annual Nicholas T. Zervas, M.D. Lectureship

Massachusetts General Hospital

Historic Ether Dome

Tuesday, May 15, 2018 at 12pm

"Melanocortin Regulation of Energy Balance and Pituitary Function: Human Translational Studies"

Sharon L. Wardlaw, M.D.
Robert C and Veronica Atkins Professor of Medicine
Department of Medicine-Endocrinology
Columbia University Medical Center
New York, NY

For further information call Philip at 617-726-3870

Pamela S. Jones, MD MS MPH

Pamela Jones, MD MS MPH, is a neurosurgeon who cares for people with all types of brain tumors, including glioblastoma, low and high-grade gliomas, metastatic brain tumors, and skull base tumors such as meningiomas, acoustic neuromas, and pituitary tumors. Her expertise includes performing minimally-invasive endoscopic neurosurgery for treating tumors in the skull base and pituitary.

Dr. Jones completed residency training in neurosurgery at the Massachusetts General Hospital. Her fellowship training in pituitary and endoscopic skull base was also performed at the Massachusetts General Hospital and the Massachusetts Eye and Ear Infirmary under Dr. Brooke Swearingen and Dr. William Curry. Dr. Jones earned her medical degree from Tulane University School of Medicine, where she graduated with Alpha Omega Alpha honors. She attended Stanford University for her undergraduate degree in biology. She also holds a master's degree in public health from Harvard School of Public Health and a master's degree in biomedical journalism from New York University. Dr. Jones' research focuses on neurosurgical outcomes and clinical trials, particularly within brain tumor and pituitary tumor, as well as quality and safety improvement initiatives.

Dr. Jones is thrilled to return to the MGH after being an assistant professor at UCSD. In her free time, Dr. Jones has a passion for ballet, both as a dancer and as a patron, and enjoys running, writing, traveling with friends and family, and watching Patriots and Stanford football.

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100 Blossom Street, Cox 140
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SERVICES AVAILABLE

FACILITIES

The Neuroendocrine and Pituitary Tumor Clinical Center is located on the 1st floor (Suite 140) of the Cox Building at the Massachusetts General Hospital. A test center is available for complete outpatient diagnostic testing, including ACTH (Cortrosyn) stimulation; insulin tolerance; CRH stimulation; oral glucose tolerance and growth hormone stimulation testing. Testing for Cushing's syndrome can also be arranged, including bilateral inferior petrosal sinus ACTH sampling for patients with ACTH-dependent Cushing's syndrome.

NEUROENDOCRINE AND PITUITARY TUMOR CLINICAL CONFERENCE

A weekly interdisciplinary conference is held to discuss all new patients referred to the Center and to review patient management issues. It is a multidisciplinary conference, attended by members of the Neuroendocrine, Neurology, Neurosurgery, Psychiatry and Radiation Oncology services. Physicians are welcome to attend and present cases.

PHYSICIANS' PITUITARY INFORMATION SERVICE (PPIS)

Physicians with questions about pituitary disorders may contact the PPIS at (617) 726-3965 within the Boston area or toll free at (888) 429-6863, or e-mail to pituitary.info@partners.org.

SCHEDULING

Outpatient clinical consultations can be arranged by calling the Neuroendocrine and Pituitary Tumor Clinical Center Office at (617) 726-7948.

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Dr. Beverly MK Biller, Editor of the MGH Neuroendocrine and Pituitary Tumor Clinical Center Bulletin, is the primary investigator on research grants to the Neuroendocrine Unit from NovoNordisk and Novartis and occasionally consults for Ferring, Merck-Serono, Novartis, NovoNordisk, Pfizer and Sandoz..

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