Department of Endocrine Neoplasia and Hormonal Disorders NEWSLETTER Volume 5, Issue 2, 2012

Metastatic Differentiated Thyroid Cancer: When is Systemic Therapy Beyond RAI Warranted?



Department of Endocrine Neoplasia and Hormonal Disorders

Making Cancer History*



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Introduction

Patients with widely metastatic differentiated thyroid cancer (DTC; includes papillary, follicular, and Hurthle cell carcinoma) can pose a challenge to physicians, especially those who see few cases of this relatively rare presenta-

tion. While the 10 year median survival after the discovery of metastatic disease is 42%, the prognosis can be vastly different among patients and depends on the age of patient, histology, location and size of the distant disease, as well as whether the disease takes up and responds to radioactive iodine (RAI)¹. For example, younger patients (<40 years) with micronodular (<1cm) lung disease have an excellent overall survival of 95% at 10 years, while older patients with macronodular lung metastases or multiple bone metastases have a 14% overall survival at 10 years¹.

Metastatic DTC tends to be indolent in the majority of patients whereas a minority of others have metastatic disease that grows rapidly from the outset. Also, patients who initially had indolent disease may later demonstrate a more aggressive course. An understanding of the natural history and appropriate management of this disease is important so that patients are not under or over treated. Because metastatic DTC is often slow growing and asymptomatic, a more restrained approach, compared with other solid tumors, is needed. In some cases, patient with rapidly progressive and/or symptomatic disease or disease in areas that are lifethreatening or have the potential to become life-threatening require a more aggressive approach.

Management of metastatic DTC

Patients who present with newly diagnosed DTC with distant metastases should be treated with first line, standard therapy, which consists of surgery and subsequent RAI. The thyroid and any regional disease are removed prior to RAI in order to remove a source of large iodine uptake. Once the thyroid and involved cervical lymph nodes are removed, RAI can be highly effective to target distant metastatic disease. However, if the distant disease is RAI-refractory (defined below),

consideration of local therapy or systemic treatment with the newer tyrosine kinase inhibitors or cytotoxic chemotherapy is appropriate once significant progression is documented. Figure 1 shows the algorithm for treatment of patients who present with metastatic disease and those who later develop metastatic disease after first line therapy.

Definition of RAI-refractory

A patient is considered RAI-refractory when radioiodine is no longer effective either because the disease does not take up iodine (usually determined on a pre- or post-treatment, diagnostic whole body scan) at known sites of metastatic disease or because the disease continues to grow despite previous treatment. Radioiodine can continue to have efficacy over 9-12 months (sometimes longer) and therefore the patient is considered RAI-refractory only when the distant disease grows over a 1 year period after RAI. Because a cumulative dose of RAI beyond 600 mCi has been shown to have little efficacy¹, patients who have received this amount of RAI can also be considered RAI-refractory, especially for the purpose of clinical trial entry criteria. In addition, some patients should not be considered for additional RAI if they have had adverse events associated with high doses of RAI. One example is bone marrow damage resulting in cytopenias.

When are local therapies considered?

Searching for bone metastases in patients with musculoskeletal pain and elevated (Continued on Page 2)

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(Cabanillas, continued)

thyroglobulin should be considered. Local therapy with external beam radiation is often considered in two scenarios: to palliate painful metastases and to provide local control of disease when RAI is no longer effective or rapid disease control is imperative such as treating bony metastatic disease in the vertebral spine. Embolization of a site of bony metastatic disease is useful in some cases and can lead to tumor shrinkage and palliation of pain. Metastatectomy is sometimes performed if there is only one site of disease. Zolendronic acid administered intravenously can also be used for pain and may be helpful for disease control in patients with bony metastastic disease, although little data exists to support the latter. It is not uncommon for brain metastases to be asymptomatic. Therefore, imaging of the brain should be considered prior to proceeding with any systemic

therapy. Treatment of unifocal brain metastases includes surgery or stereotactic radiation. Whole brain irradiation is considered in patients with extensive multifocal metastatic disease.

When to 'watch and wait' in patients who are RAI refractory

Dormancy of the metastatic disease is common in DTC. The events which lead to progression likely are due to genetic and epigenetic events in the tumor which cause the disease to become more aggressive². The rationale for the 'watch and wait' approach is based mainly on the fact that most patients with widely metastatic, RAI-refractory DTC enjoy a long indolent phase where the tumor is stable or slowly progressive and asymptomatic. These patients can enjoy a good quality of life for many years before requiring systemic therapy. Because systemic therapies have many side effects, some of which can be fatal, delaying therapy until the disease accelerates is prudent. TKIs are considered chronic therapies, as stopping these drugs usually results in progression of disease. Furthermore, the TKIs have not cured any patient thus far and it is not clear whether they will prolong overall survival.

Patients should have full staging exams to determine extent of disease and pace of progression. Distant disease limited to the mediastinum and lung that is asymptomatic and does not threaten vital structures can usually be followed with serial imaging. Metastatic, RAI-refractory disease to the brain, liver, and bone may indicate that the disease is more aggressive and warrant local treatment or very close follow up for progression and subsequent initiation of local and/or systemic therapy.

Monitoring for progressive disease

Serial cross-sectional imaging with the same modality is the most accurate way of monitoring for progression of disease. While RECIST guidelines (Response Evaluation Criteria In Solid Tumors)^{3,4} have many problems, this is a standardized method of determining progression in patients with solid tumors, including thyroid cancer. In general, a 20% increase in target lesion tumor measurements is considered progressive disease, 30% decrease is considered a partial response, and anything in between (-29 to +19%) is stable disease.

When to begin systemic treatment

RAI-refractory patients with progression within 6 months, particularly those with symptomatic or lifethreatening disease, should be considered for systemic therapy. Most clinical trials in thyroid cancer require progression within 1 year in order to qualify for enrollment in a trial⁵ and therefore this is the standard that we use for initiating systemic therapy in asymptomatic patients. Symptomatic patients or patients with bulky disease that compromises organ function and who cannot be managed with local therapies should initiate systemic therapy. High FDG uptake on PET scan has been considered as a criteria for initiating systemic therapy because these patients exhibit a more aggressive course and are usually RAIrefractory^{6,7}. However, it is important to prove RAI-refractoriness prior to recommending systemic therapy. Systemic therapies:

Choices for systemic therapy include the newer targeted agents (tyrosine kinase inhibitors, TKIs) or cytotoxic chemotherapy. TKIs should be considered first because these have been shown to be efficacious in DTC whereas cytotoxic chemotherapy has limited efficacy. While there are no TKIs approved by the FDA for DTC, the ATA⁸ and NCCN⁹ guidelines recommend "off-label" use of TKIs (such as sorafenib, sunitinib, and pazopanib) or treatment on a clinical trial (clinicaltrials.gov). Table 1 shows the list of drugs that have been studied in DTC and their targets. Entry into phase 2 and 3 clinical trials is usually limited to patients with RAI-refractory, progressive disease. Phase 1 trials can be considered for patients who do not meet entry criteria. For example, a patient with very rapidly progressive disease who has an unresectable primary tumor in the thyroid would likely not benefit from RAI. Rather than administer a treatment known to be ineffective, this patient could be offered a phase 1 trial.

Sorafenib, sunitinib, and pazopanib are oral antiangiogenics approved for other indications. These drugs have been used in phase 2 trials and are promising agents for patients with progressive, RAI-refractory disease. Common side effects include hypertension, skin toxicity (rash and hand-foot syndrome), diarrhea, weight loss and fatigue. Less common but potentially fatal adverse events include congestive heart failure, slow wound healing or wound dehiscence, bleeding, and tracheoesophageal (TE) fistula formation. The antiagniogenic agents should be used with caution or avoided if possible in patients at high risk for bleeding or TE fistula. Use of concomitant medications metabolized via the CYP4A pathway such as coumadin and verapamil can cause changes in drug levels and/or QT prolongation and therefore require careful monitoring or discontinuation of the CP4A medication. TKIs should be prescribed only by endocrinologists and oncologists who are well versed in their use and have multidisciplinary care available to manage potential side effects.

Vemurafenib is a TKI which selectively targets BRAF mutations. This drug is FDA approved for use in BRAFmutated metastatic melanoma but is currently being studied in a phase 2 trial in patients with BRAF-mutated papillary thyroid cancer. Off-label use of vemurafenib is not recommended at this time until more information regarding efficacy in PTC is reported.

(Continued on Page 3)

Upcoming Events

American Society of Clinical Oncologists (ASCO) 2011 Annual Meeting June 1-5, 2012. Chicago, IL. (http://chicago2012.asco.org)

Amerian Diabetes Association (ADA) 72nd Scientific Sessions. June 8-12, 2012, Philadelphia, PA. (www.scientificsessions.diabetes.org)

ENDO: The Endocrine Society Annual Meeting 2012. June 23-26, 2012. Houston, Texas. (www.endo-society.org)

13th International Workshop on Multiple Endocrine Neoplasia. Sept 5-8, 2012. Liege, Belgium. (www.iwmen. org/event/Liege2012/)

American Thyroid Association (ATA) 82nd Annual Meeting. Sept 19-23, 2012. Quebec City, PQ, Canada. (www.thyroid.org)

American Society for Bone and Mineral Research (ASBMR) 2012 Annual Meeting. Oct 12-15, 2012. Minneapolis, MN. (www.asbmr.org)

ThyCa Conference 2012. Oct 19-21, 2012. Chicago, Ill. (www.thyca.org)

12th International Conference on Cancer Induced Bone Disease. Nov 15-17, 2012. Lyon, France (www.ibms.org)

The Second World Congress on Thyroid Cancer. July 10-14, 2013. Toronto, CA. (www.thyroidworldcongress.com)



(Cabanillas, continued)

Summary

The majority of patients with distant metastatic DTC can be effectively managed with surgery, RAI, TSH suppression and local therapies. Few patients require systemic therapies with cytotoxic chemotherapy or TKIs; however it is important to be able to identify these patients so that they may initiate treatment when appropriate. In general, only patients with progressive and/or symptomatic, RAI-refractory disease should be started on a TKI since these treatments are not curative and can have serious side effects.

Figure 1: Algorithm for management of DTC patients presenting with metastatic disease and DTC patients who develop metastatic disease

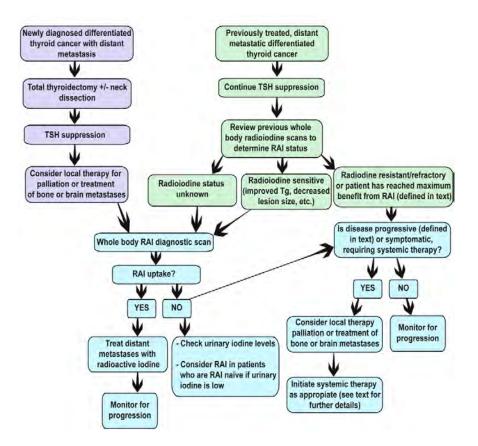


Table 1: Tyrosine kinase inhibitors studied in DTC (adapted from Busaidy et al¹⁰)

Drug	VEGFR1	VEGFR2	VEGFR3	RET	BRAF	Other	Response; PFS	Citation
Motesanib	Х	Х	Х	Х			14% PR; 9 mos	Sherman <i>et al</i> ¹¹
Axitinib	x	х	х				31% PR; 18 mos (MTC included)	Cohen <i>et al</i> ¹²
Sorafenib		х	х	х	х		15-23% PR; PFS 10-21 mos	Kloos <i>et al</i> ¹³ ; Gupta-Abramson <i>et al</i> ¹⁴ ; Cabanillas <i>et al</i> ¹⁵
Sunitinib	х	х	х	х			28% CR+PR; TTP 13 mos	Carr <i>et al</i> ¹⁶
Pazopanib	х	х	х	х			49% PR; PFS 12 mos	Bible <i>et al</i> ¹⁷
Lenvatanib (E7080)	х	х	х	х		FGFR	50% PR; PFS 13 mos	Sherman <i>et al</i> ¹⁸
Cabozantinib (XL184)	Х	х		х		c-MET	53% PR; PFS n/a	Cabanillas <i>et al</i> ¹⁹

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Case Files From The Department: ACTH-Independent Macronodular Adrenal Hyperplasia (AIMAH)

Mouhammed Amir Habra, MD, F.A.C.P., F.A.C.E., Assistant Professor, Department of Endocrine Neoplasia and Hormonal Disorders

A 40 year-old man presented with an unexplained 15 Kg weight gain over one year despite attempts to control weight through diet and exercise.

He reported intermittent lower extremity edema and new onset of purple striae. His past medical history is remarkable only for a history of nephrolithiasis 5 years earlier and newly diagnosed hypertension and type 2 diabetes mellitus.

His medications included metformin for blood glucose control and 4 antihypertensive medications (spironolactone, lisinopril, carvedilol, and amlodipine). He denied any family history of endocrine disorders. On examination he had a BMI of 36 Kg/m2 and blood pressure of 150/90 mmHg. His examination was remarkable for facial plethora, dorsocervical fat pad, and central obesity with purple striae on his abdomen (Figure 1 on page 5).

Laboratory work-up showed ACTH<5 pg/ml, 24-hour urine free cortisol of 536 micrograms/ 24 hour (reference range 3.5-45), and DHEA-S of 22.7 micrograms/dL (reference range 48- 244).

CT of the abdomen revealed bilateral non-obstructing renal stones, diffuse fatty infiltration of liver, and innumerable large solid nodules in both adrenal glands with attenuation of 8.2 Hounsfield units (HU) before contrast with washout of about 60% (Figure 2 on page 5). The presence of ACTH independent Cushing syndrome with multiple adrenal macronodules on CT scan was compatible with ACTH-independent macronodular adrenal hyperplasia.

The patient started metyrapone 1500 mg daily in preparation for surgery and then underwent bilateral adrenalectomy. On pathology, the right adrenal gland weighed 124 grams and the left adrenal gland weighed 240 grams (Figure 3 on page 5). Normalization of blood pressure and blood glucose occurred within a few days of adrenal gland resection, and the patient was maintained on replacement steroids.

Discussion

ACTH-Independent Macronodular Adrenal Hyperplasia (AIMAH) is among the least common causes of Cushing's syndrome (CS). While most cases are sporadic, recently familial AIMAH cases have been described. It involves both adrenal glands that contain multiple macronodules (>0.5 cm each). Plasma ACTH is often suppressed or near the lower end of the normal range depending on the degree of autonomous cortisol production. Subclinical Cushing's syndrome is the more common than overt CS in these patients.

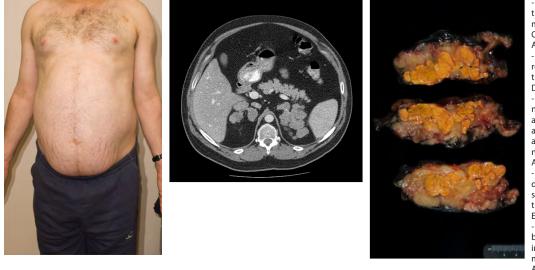
The exact cause of AIMAH is unknown but some cases are associated with aberrant hormone receptors (gastric inhibitory peptide, vasopressin, leutenizing hormone, beta adrenergic, angiotensin, or serotonin receptors). Screening for aberrant hormonal receptors has been recommended by some groups with the aim to offer medical options to control CS. The long term implications of testing are unclear in most cases as only few patients with aberrant hormonal receptors could achieve reasonable long term cortisol control with medical therapy and most patients will eventually require surgical resection. Our patient was not tested for aberrant receptor expression considering the clinical picture of overt Cushing's syndrome

Bilateral adrenalectomy is often done in AIMAH patients with severe CS while unilateral adrenalectomy is often sufficient in cases with subclinical or mild features of CS. (Habra, continued)



Figure 2

Figure 3



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The Emergence of Oncologic Endocrinology as a Clinical and Research Field

Steven I Sherman, MD, Professor and Chair, Department of Endocrine Neoplasia and Hormonal Disorders

In 1999, I was asked to speak on the topic of "Multicenter Clinical Trials in Thyroid Cancer," to summarize the state of the field and identify both the challenges to and opportunities for advancement of improved clinical care and outcomes for patients with this particular endocrine malignancy. Surveying the medical literature led to certain unsatisfying conclusions about the state of affairs:

• No data existed from randomized controlled trials testing any of the primary treatments commonly used for thyroid carcinoma.

• The few prospective clinical trials that had been performed for metastatic disease were generally unsuccessful in demonstrating treatment benefits, but these studies suffered from insufficient numbers of patients.

• Pharmaceutical companies, with rare exception, had not developed or supported clinical trials to address new therapies for thyroid carcinoma.

• Endocrinologists, as the providers of primary oncologic management for thyroid cancer patients, were not trained to perform clinical trials or manage the complexities of patients with morbidly advanced malignancies.

Today, major advances in oncologic endocrinology have led to important steps to overcome each of these earlier deficiencies, and provide us with exciting opportunities for the future.

The formal clinical trial to test therapies for cancer emerged in the mid 1950s with successful experiments to treat acute leukemias. Oncologists were emboldened by these early gains, and attempted to re-create this experience with many of the common solid tumors. With recognition of the need for large numbers of patients to participate in pivotal trials, regional cooperative oncology groups were established throughout the United States, merging oncologists from multiple institutions with expertise in clinical trials design, biostatistics, drug development, and patient care. During the next two decades, these groups developed and implemented multiple studies that demonstrated benefit from many of the now-standard chemotherapeutic agents for many malignancies.

From this background, cancer researchers hypothesized that some of these new cytotoxic drugs being tested in various malignancies might also benefit patients with advanced endocrine cancers, particularly thyroid. Beginning in the 1970s, trials were performed testing drugs like doxorubicin and cisplatin. However, it was soon realized that poor accrual was leading to disappointingly inadequate evaluations of treatment efficacy. Attempts to reproduce the success of the cooperative groups with other malignancies were similarly unproductive for endocrine cancers. Of 15 clinical trials focused specifically on thyroid carcinoma that were initiated between 1975 and 1999, only five led to published results and none reached full, planned enrollment. This poor experience, both in terms of patient's response rates and lack of completion of clinical trials, resulted in the conclusion that "conventional chemotherapy has prove[n] to be ineffective for most patients with progressive metastases from thyroid cancer."

Multiple factors can probably be identified that led to these unsatisfying outcomes. Most tested therapies were compounds that attacked dividing cells, and thyroid tumors are typically slowly growing relative to many other solid tumors. A relatively narrow therapeutic index for some cytotoxic drugs led to marked side effects at doses necessary for even marginal effectiveness. Inadequate recruitment of patients may therefore have been secondary to the emerging reality that these chemotherapies were insufficiently active to justify the toxicities. But perhaps of equal importance, negligible participation in these trials by endocrinologists may have restricted awareness among potential referring physicians and patients, thus leading to the common but mistaken impression that such trials were in fact not available.

One major scientific initiative that transformed chemotherapy was the creation of a novel oral therapy for chronic myelogenous leukemia, an inhibitor of the protein tyrosine kinase pathophysiologically linked with the leukemia. The demonstration that targeting a specific abnormally expressed or activated molecular pathway could lead to successful therapy ushered a new era in medical oncology, further enhanced by approval of other targeted tyrosine kinase inhibitors for solid tumors such as renal cell carcinoma, gastrointestinal stromal tumors, and melanoma. Often not associated with the myelosuppressive and common life-threatening toxicities seen with earlier chemotherapies, such "targeted therapies" opened multiple opportunities for researchers and patients alike.

Endocrine investigators soon recognized that these same pathways of disordered intracellular signaling are associated with development, maintenance, and progression of the malignant phenotype in thyroid carcinomas. As novel, anti-angiogenic targeted therapies were introduced in early phase testing, the endocrinologists at M.D. Anderson identified that these drugs could have potential benefit to treat patients with progressive, metastatic endocrine cancers. (Continued on Page 8)

(Sherman, continued)

Our endocrinologists established critical collaborations with medical oncologists to create pathways for patients to enter these phase I clinical trials. Multidisciplinary clinical research teams emerged, combining sophisticated endocrinologic care with oncologic specialists, and our endocrinologists acquired new skill sets, focusing on use of targeted therapies such as tyrosine kinase inhibitors, toxicity management, and supportive care for symptomatic metastases. Working with collaborators at the National Cancer Institute, pharmaceutical industry and other academic centers, our oncologic endocrinologists participated in the design of phase II and III trials, many of which are now leading to drugs in consideration for approval to treat thyroid cancer. Outside of clinical research, oncologic endocrinologists are treating selected thyroid cancer patients with targeted therapies as part of routine care, expertly identifying those patients most likely to benefit from such advanced treatment modalities. Moving beyond thyroid, these specialists are now applying their new knowledge to treatment of other endocrine malignancies, such as adrenal cortical carcinoma and pheochromocytoma. And, coming full circle, the principles that oncologic endocrinologists have learned in the design of trials for patients with advanced disease are now being applied to multicenter studies of primary treatments, such as adjuvant radioiodine.

This model of multidisciplinary care, with major components provided by oncologic endocrinologists, has been replicated at other institutions. Recognizing exciting career opportunities for young endocrinologists, new training programs are emerging as well that will contribute to shaping this new, focused subspecialty (see article by Dr. Mimi Hu in this issue). New collaborative research networks are being created and led by oncologic endocrinologists, such as the International Thyroid Oncology Group, that will bring multidisciplinary research programs and clinical trials to endocrine cancer patients worldwide.

New Oncologic Endocrinology Fellowship Training Program

Mimi I. Hu, MD, Assistant Professor, Department of Endocrine Neoplasia and Hormonal Disorders

The incidence and mortality of endocrine malignancies, especially thyroid carcinoma, has been increasing more rapidly in the U.S. than most other malignancies. Among the elderly, mortality from thyroid cancer has been rising 1-2% per year. Additionally, within the last decade, the field of endocrine neoplasia has advanced remarkably in areas of diagnostic testing, medical management and surgical techniques.

With the development of specialized therapies for endocrine malignancies and expanded treatment options with proven efficacy, such as antiangiogenic agents and other small molecule tyrosine kinase inhibitors (TKIs), there is a need for specialized training of physicians beyond what is currently provided by standard endocrine or oncology fellowship training programs. With the FDA approval of vandetanib last April 2011 for advanced medullary thyroid cancer (MTC) and the availability other promising agents in the pipeline for MTC and differentiated thyroid cancer, there is a urgent need for well-trained physicians experienced with the management of these cancers and the toxicities associated with systemic treatment.

Additionally, newer agents developed for thyroid cancer or other malignancies are associated with specific adverse endocrine complications (e.g. hypothyroidism with TKIs¹, hyperglycemia and dyslipidemia with mTOR inhibitors², hypophysitis with anti-CTLA-4 therapy for melanoma³, central hypothyroidism with bexarotene⁴), which warrant greater investigation into causative mechanisms and correlations with oncologic therapeutic and survivorship outcomes. Management of established endocrine-related complications of cancer therapy (e.g., steroid-induced diabetes, osteoporosis, hypogonadism) has been shown to decrease the morbidity and improve quality of life for cancer survivors.

I am extremely pleased to announce the recent approval of the Oncologic Endocrine Fellowship Training Program at MD Anderson Cancer Center. This non-accredited one-year advanced fellowship is the first program of its kind in the nation providing training focused on the evaluation and treatment of endocrine neoplasias in the outpatient and inpatient setting. A physician, who has completed an accredited fellowship in either endocrinology or medical oncology, will acquire the skills and knowledge base for evaluating and treating patients with these complex tumors.

The Department of Endocrine Neoplasia and Hormonal Disorders at MDACC is the optimal setting for a program of this type. The Department is comprised of 11 clinical and 4 basic science faculty members who evaluate,

manage, and investigate a wide spectrum of endocrine tumors/malignancies of the thyroid, adrenals, parathyroid, pituitary, pancreas, and hereditary endocrine syndromes. Additionally, we have a unique advantage of having two faculty members with dual board certification in adult and pediatric endocrinology. The Endocrine Center at the institution is a high-referral, multidisciplinary center for advanced endocrine neoplasias and conducts a robust clinical trials program. The Center has well-established collaborative relationships with head and neck surgery, surgical endocrinology, medical oncology, genetics, radiology, nuclear medicine, urology, and pathology.

The curriculum will include outpatient and inpatient management of patients with complicated endocrine neoplasias and hormonal disorders, including patients enrolled into Phase I-III clinical trials, and rotations with non-endocrine specialties, such as medical oncology, surgery, pathology and radiology. Research activities will be encouraged with opportunities to present at local, national or international meetings. Academic opportunities will include multiple-routine conferences and the education of medical students, residents, or fellows.

The program is accepting applications for the 2013-2014 academic year. Eligible candidates require the following:

- Completion of a US or Canadian-accredited fellowship in Endocrinology, Diabetes and Metabolism or Medical Oncology

- Valid certificate from the Educational Commission for Foreign Medical Graduates (ECFMG) for graduates of medical schools and specialty training programs in acceptable fellowships outside of the US or Canada

- An active license from the Texas Medical Board or eligibility to obtain a license or training permit from the Texas Medical Board

Please contact Mimi I. Hu, MD, program director, for inquiries: mhu@ mdanderson.org, 713-792-2841.

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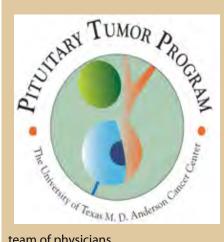
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Come visit us at the ENDO 2012 Expo! Booth #1011 and 1013

The Department of Endocrine Neoplasia will be hosting an exhibit booth at the Endocrine Society's ENDO 2012 Expo at the George Brown Convention Center, here in downtown Houston, TX. Please drop by to visit with our faculty members, nurse practitioners and learn about our department and the services we offer.

The Pituitary Tumor Program



The Pituitary Tumor Program is a group of physicians dedicated to caring for patients with all types of pituitary disorders. We provide high quality care for patients with newly diagnosed and recurrent pituitary tumors, and long-term patient follow-up related to their pituitary disorder. Our endocrinologists work with colleagues in neurosurgery, neuroradiology, neuro-ophthalmology, neuropathology and radiation oncology to provide expert management of pituitary disease.

The Pituitary Tumor team provides coordinated outpatient evaluation and care, including:

- Dynamic hormonal testing
- A wide range of pituitary function tests
- Testing and diagnosis of Cushing's Syndrome
- Pituitary-dedicated MRI imaging
- Neuro-ophthalmologic exams
- Stereotactic radiosurgery and conventional radiation therapy

New patients are routinely presented at the monthly pituitary conference so that an individualized treatment plan can be formulated and carried out by a multidisciplinary

team of physicians.

To refer a patient to the Pituitary Tumor Program, please call our new patient referral coordinators at: 713-563-4400 (Dept. of Endocrine Neoplasia and Hormonal Disorders) or 713-792-7728 (Dept. of Neurosurgery). For patients less than 18 years of age, please call the Children's Cancer Hospital at 713-792-5410.

The Diabetes Program

The faculty and staff of the Diabetes Program at The University of Texas MD Anderson Cancer Center strive to improve care for cancer patients with pre-existing diabetes mellitus and treatment-related hyperglycemia. The goals of the program include:

- Management of pre-existing diabetes mellitus for patients undergoing treatment for cancer, including insulin programs for patients on high-dose glucocorticoids, and management of insulin pumps during chemotherapy and surgery.
- Improvement of the long-term health of MDACC patients with previously untreated diabetes by arranging for follow-up diabetes management consistent with current standards of care.
- Development and validation of algorithms for management of hyperglycemia related to cancer treatments such as high-dose glucocorticoids, inhibitors of insulin and IGF-I signaling pathways, tube feedings and parenteral nutrition.
- Research that can help define new standards of care for patients with diabetes
- Promotion of prevention by assessment of diabetes mellitus, obesity, and insulin resistance as potential risk factors for cancer.
- Assessment of the roles of diabetes mellitus, obesity, and insulin resistance as factors that can impair response to the treatment of cancer.

For more information, please visit us at the Diabetes Program page of the Department of Endocrine Neoplasia and Hormonal Disorders webpage at: http://www.mdanderson.org/education-and-research/departments-programs-and-labs/programs-centers-institutes/diabetes-program/index.html. For guestions about the program, please call the department at 713-792-2841

The Pediatric Endocrine Tumor Program



The Pediatric Endocrine Tumor Program is a component of the Children's Cancer Hospital and one of the few pediatric endocrinology practices in the nation that specifically focuses on the diagnosis, evaluation, and treatment of children with thyroid nodules and thyroid cancer, adrenal tumors, pituitary tumors, and hereditary tumors such as the multiple endocrine neoplasias and von Hippel-Landau disease. All of our patients are treated in a multidisciplinary fashion, and our colleagues in nuclear medicine, oncology, pathology, radiology, and surgery are equally poised to provide excellent clinical care to this population.

The pediatric endocrinology team also evaluates and treats all endocrine disorders that occur in the pediatric cancer population.

If your child has been diagnosed with or is suspected to have an endocrine tumor, we're here to help. Call the Children's Cancer Hospital Patient Access Center at 713-792-5410 or toll-free at 888-543-2435. To learn more about the Children's Cancer Hospital, please visit www.mdanderson.org/children



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Wish to make a donation to Endocrine Research?

You can make a huge difference in the lives of those with endocrine tumors and hormonal disorders by donating to our endocrine research fund. New discoveries pertaining to endocrine malignancies can also combat other types of cancers. We now have a webpage for your convenience: http://www.mdanderson.org/education-and-research/departments-programs-and-labs/departments-and-divisions/endocrine-neoplasia-and-hormonal-disorders/endocrine-research.html

How to refer a patient to MD Anderson

Online Referrals:



MD Anderson has created an online referral process, myMDAnderson, to help you get your patient into MD Anderson as quickly as possible. You can use myMDAnderson to follow your patient's treatment regimen by viewing transcribed reports and accessing your patient's schedules. To qualify for this free service, you must be a licensed, practicing physician. To start a referral through myMDAnderson, please access this portal: https://my.mdanderson.org/public/physicians/user/

Telephone Referrals:

- Physician to Physician referrals, please call 713-792-2841.
- To speak with a New Patient Referral Coordinator, please call 713-563-4400.
- For Pediatric Referrals (patients less than 18 years of age), please call 713-792-5410.

Department of Endocrine Neoplasia and Hormonal Disorders Faculty

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