Pediatric Endocrine Tumors

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Endocrine tumors represent a minority of all neoplasms observed in the pediatric population and are generally clinically benign or low-grade cancers, although a small percentage of these tumors are high-grade malignancies requiring multimodality therapies. Although rare, pediatric endocrine tumors are important clinical entities that can have unique clinical presentations and genetic causes. As with any rare childhood disease, treatment is best provided at referral centers with multidisciplinary expertise in the management of such tumors. The Pediatric Endocrine Tumor Program at the Children’s Cancer Hospital of the University of Texas M. D. Anderson Cancer Center is a multidisciplinary program that provides primary evaluation and treatment to children and young adults suspected to have or who are already diagnosed with thyroid neoplasms, adrenal tumors, pituitary adenomas, and/or hereditary tumor syndromes. Our team currently consists of two board-certified adult and pediatric endocrinologists, Steven Waguespack, M.D. and Anita Ying, M.D., and Sarah Bottomley, RN, MSN, CPNP, a pediatric nurse practitioner who works closely with both Drs. Waguespack and Ying.

This issue of EndoPerspectives is focused on pediatric endocrine tumors. We start with a general overview of the most common endocrine neoplasms encountered in the pediatric population. Dr. Ying and Ms. Bottomley review late effects of endocrine tumors and their treatment. Thereasa Rich, M.S., C.G.C., reviews the genetic syndromes most commonly associated with pediatric endocrine tumors and emphasizes the role of genetic counseling when taking care of these children.

Pediatric Endocrine Tumors, An Overview
Pituitary Tumors

Pituitary adenomas (PA) represent less than 3% of all supratentorial tumors diagnosed during childhood. Over 75% of PAs occur in children older than 12 years, and females are more commonly affected than males. Pituitary tumors are usually diagnosed secondary to symptoms of hormone excess or mass effect, such as visual disturbance (classically a bitemporal hemianopsia), headache, or ophthalmoplegia. Patients may also present with delayed growth and pubertal development due to pituitary hormone hypersecretion, as seen with hyperprolactinemia, or hyposecretion, as seen with large PAs that compress the normal adenohypophyseseal tissue. The initial workup of a PA is similar to adults and includes imaging of the sella turcica, a comprehensive hormonal evaluation to assess for hyper- and hypopituitarism, and visual field testing for children with large tumors that approach the optic chiasm. Prolactinomas are the most common PA in childhood, representing about 50% of cases, followed by ACTH-secreting adenomas (Cushing disease), somatotroph adenomas (gigantism/acromegaly), and lastly, clinically nonfunctioning PAs. In contradistinction to adults, (continued on Page 2)
nonfunctioning PAs represent only <5% of pediatric pituitary tumors. Although the clinical presentation of PAs during childhood is often different compared with adults, the approach to diagnosis and treatment of these neoplasms is similar.

8-year-old with Cushing disease and a cystic pituitary macroadenoma (arrows).

Thyroid Tumors
Benign Thyroid Tumors
Benign thyroid tumors represent up to 80% of all thyroid nodules presenting in the pediatric population. The younger a child is at the time of presentation, the more likely it is for a solitary nodule to be malignant. The work up of a thyroid nodule in a child includes the laboratory assessment of thyroid function, ultrasound to assess the nodule and regional lymph node characteristics, and fine needle aspiration (usually under ultrasound guidance) for definitive diagnosis. Solid lesions with increased blood flow and the presence of microcalcifications are more likely to be malignant whereas pure cystic lesions are almost always benign. Nuclear scintigraphy with 123I or Tc-99m pertechnetate is generally not useful in the diagnostic evaluation, except in the event of a suppressed TSH.

Malignant Thyroid Tumors
Malignant neoplasms of the thyroid are rare in the pediatric population, with an incidence of ≤1 case/million/year in children under ten years of age to 15.4 cases/million/year in adolescents ages 15-19, which is the most commonly affected pediatric age group. Over 90% of thyroid cancers occurring in childhood are papillary thyroid cancer (PTC). Medullary thyroid carcinoma (MTC) is a very uncommon disease in childhood that almost always occurs in the context of one of two hereditary endocrine tumor syndromes that arise secondary to activating mutations of the RET (REarranged during Transfection) proto-oncogene: multiple endocrine neoplasia type 2a (MEN2A) and type 2b (MEN2B). In addition to the almost complete penetrance of MTC, 50% of patients with MEN2A and MEN2B develop pheochromocytomas and up to 30% of MEN2A patients have parathyroid adenomas, although these tumors are rarely diagnosed during childhood. Patients with MEN2B have a distinct phenotype with a characteristic facial appearance, Marfanoid body habitus, and a generalized ganglioneuromatosis, manifested most obviously by the presence of oral mucosa neuromas.

Children with thyroid cancer usually present with an asymptomatic thyroid mass and/or cervical lymphadenopathy. Lymph node metastases are present in the majority of PTC cases and lung metastases are identified in up to 20% of cases; metastases to other sites occur but are rare.

CT scan of a 6-year-old with papillary thyroid cancer (PTC) and bulky bilateral lymph node (LN) metastases.

Despite a more advanced presentation, children with thyroid cancer usually have an excellent prognosis with anticipated survival over decades. Part of this clinical phenomenon may be due to mutational differences between children and adults, in addition to the fact that pediatric PTC is usually very iodine avid, typically leading to successful treatment of distantly metastatic disease after one or more courses of radioactive iodine (RAI) therapy.

Total thyroidectomy and a compartment-oriented lymph node dissection, as indicated, are best accomplished by surgeons with extensive experience in the management of thyroid malignancies. In DTC, radioiodine (using 131I) is often administered a few weeks after surgery to ablate any residual normal thyroid tissue and treat any residual thyroid cancer that is iodine-avid; children with MTC do not require radioiodine ablation. The thyroid stimulating hormone (TSH) level is suppressed by giving
supraphysiologic levothyroxine doses in DTC but the TSH is kept normal in MTC. Long-term follow up involves monitoring of tumor markers (thyroglobulin in DTC and calcitonin/carcinogenic embryonic antigen in MTC) as well as routine imaging based upon the initial extent of disease presentation. Neck US is generally the most useful imaging modality. Traditional chemotherapy has generally not been shown to be effective in thyroid cancer, but newer anti-cancer agents, the oral tyrosine kinase inhibitors, are showing promise in the treatment of children with disease not amenable or responsive to standard therapeutic approaches.

In MEN2, there is a correlation between genotype and phenotype, and the biological aggressiveness of MTC depends on the hereditary setting in which it develops. With the advent of genetic testing for RET mutations, MTC has become one of the few malignancies that can be prevented or cured via prophylactic thyroidectomy before it becomes clinically relevant. Recent guidelines have updated recommendations regarding the age of prophylactic thyroidectomy in children who are carriers of a RET mutation.

Adrenal Tumors
Adrenocortical tumors (ACT) arise from the outer adrenal cortex whereas pheochromocytomas (PHEO) derive from the catecholamine-producing chromaffin cells of the adrenal medulla. The pathologic categorization of ACT in children as benign or malignant does not always correlate to the clinical behavior of these tumors, making it difficult to differentiate clinically significant adrenocortical carcinomas from those tumors that retain a good prognosis. ACT are very rare and tend to present at an age <5 years. They have a female predominance and are functional tumors (producing androgens and/or glucocorticoids, typically) in >90% of cases. ACT may also present as an abdominal mass or pain. In children, ACT are associated with the Li-Fraumeni syndrome (germline inactivating mutations in the p53 tumor suppressor gene), the Beckwith-Wiedemann syndrome (BWS), hemihypertrophy other than that seen as part of BWS, and rarely congenital adrenal hyperplasia. Other rare causes of nodular adrenocortical disease, which usually present with Cushing syndrome, include Carney’s complex and macronodular adrenal hyperplasia. PHEO are more likely to be bilateral, extra-adrenal, malignant, and secondary to a heritable tumor syndrome in children. Most familial PHEO presenting in children are associated with Von-Hippel Lindau syndrome, although MEN2, the familial paraganglioma syndromes due to mutations in the succinate dehydrogenase gene, and neurofibromatosis type 1 are also in the differential diagnosis. Compared with adults, hypertension is usually sustained in children and they may lack the typical triad of headache, palpitations, and diaphoresis. The initial test recommended for diagnosis is measurement of plasma and/or urine metanephrine levels.

Most children with ACT or PHEO are treated with surgical resection, but systemic approaches to therapy are typically required in the case of malignant disease that is present outside of the adrenal gland. Children with PHEO who proceed to surgery are pretreated with alpha (and possibly beta) blockade.

(Waguespack, continued from Page 1)
Notes from the Endocrine Faculty Team

Publications:

- Schlumberger M, Sherman SI. Clinical trials for progressive differentiated thyroid cancer: patient selection, study design, and recent advances. Thyroid. 2009 Dec;19(12):1393-400.

Wish to refer a patient to M. D. Anderson?

Online Referrals:
M. D. Anderson has created an online referral process, myMDAnderson, to help you get your patient into M. D. Anderson as quickly as possible. You can use myMDAnderson to follow the treatment your patients receive by viewing transcribed reports and accessing your patients’ 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 to the Dept. of Endocrine Neoplasia and H.D., please call 713-792-2841.
To speak to 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

We want to hear from you!

We are always looking for suggestions to improve this newsletter and your input is valuable to us. If you have any suggestions or articles to share with us, please contact Charles Stava, Program Manager, Department of Endocrine Neoplasia and Hormonal Disorders, The University of Texas M. D. Anderson Cancer Center at: cstava@mdanderson.org, or write to him at: 1515 Holcombe Blvd, Unit 1461, Houston, Texas, 77030.

Multiple Endocrine Neoplasias Patient Education Conference

The Clinical Cancer Genetics (CCG) Program is pleased to announce an upcoming patient-directed conference on Multiple Endocrine Neoplasia Types 1 and 2 (MEN1 & MEN2) to be held on June 26, 2010. The conference will be a wonderful opportunity for patients and caregivers to learn about the latest developments in clinical care and research focusing on these hereditary syndromes.

The agenda will mirror the mission of the CCG program to provide research-driven information about genetic syndromes, genetic risk assessment, testing, and screening for people and their family members who are at increased risk for cancer. Registration is currently on-going for this conference and is free of charge. For more information please see visit our website at www.mdanderson.org/departments/ccg or to register please call 713-745-7391 or email ccg@mdanderson.org. Please see the enclosed flyer for more information about the conference.
The late effects of endocrine tumors are dependent on the organ involved, extent of disease, age at diagnosis, and treatment modality, which may include surgery, radiation, chemotherapy or other therapies. Additionally, compliance with prescribed hormone replacements may impact overall outcomes and quality of life. Lifelong surveillance by providers familiar with endocrine tumors and potential sequelae will be required to optimize a survivor’s physical and psychosocial health, ensure early diagnosis of sequelae and provide appropriate intervention.

**Pituitary Tumors**

Long-term effects from pituitary tumors can result from treatment. For those tumors that require surgical resection or more rarely radiation therapy, varying degrees of hypopituitarism can occur that require lifelong hormone replacement. Treatment of central adrenal insufficiency, diabetes insipidus, and hypothyroidism are similar to adult patients with adjustment for patient weight and age. Depending on the age of onset, graduated dosing of pubertal hormones to assist with induction or maintenance of puberty and fertility assistance may be needed. Growth hormone replacement prior to fusion of growth plates is generally needed, but continued treatment in adulthood is not always necessary. Studies have shown no increased incidence of tumor recurrence in adult patients with prior nonfunctioning pituitary tumors who receive growth hormone.

Cushings syndrome, as a result of a pituitary or adrenal tumor, can affect growth, puberty, and accrual of bone mass. After hypercortisolism is controlled, these issues can resolve with catch-up growth and restoration of bone mass.

Prolactinomas are generally treated with medical therapy of dopamine agonists. There has been an association between valvular heart disease and dopamine agonist use in patients treated for Parkinson’s disease. The applicability to patients treated for hyperprolactinemia is unclear. Parkinson’s patients with significant valvular disease have been exposed to much higher doses (25 mg/week) than those usually used in hyperprolactinemic patients (0.5-3mg/week). And advanced age is strongly associated with increasing prevalence of valvulopathy. However, given the longevity with which some patients with prolactinomas must be treated, this data has concerned endocrinologists. There is insufficient evidence to provide consensus guidelines to endocrinologists, but patients should be educated about potential risks, treatment should be given at the lowest possible dose for the shortest duration possible, and baseline echocardiograms can be considered in adulthood in those patients who will likely be on long-term cabergoline therapy.

**Thyroid Cancer**

The child treated for differentiated thyroid cancer (DTC) can usually anticipate excellent outcomes and long-term survival, and treatment is generally well tolerated with limited side effects. However, with the advent of increasingly aggressive therapies and the prospect of decades of survivorship, it is important to maintain an awareness of the potential early and late adverse events that may impact the patient’s quality of life.

One of the unique aspects of DTC is the use of radioactive iodine (RAI) in the evaluation and treatment of patients with this disease. Therapy with RAI is generally well tolerated and safe. Systematic reviews of RAI effects on the gonadal system of men and women with DTC provide reassuring results. In men, there are lab abnormalities in the first 6 months after RAI, with normalization usually by 18 months, suggesting transient gonadal dysfunction. However, there is no evidence of increased rates of infertility. There are recommendations to wait at least four months before attempting to have children.

In women, FSH values normalize by 12 months and there is no significantly increased risk of infertility, miscarriage, stillbirths, congenital defects, or cancers identified in offspring. Current recommendations state that pregnancy should be avoided for 6-12 months after receiving therapeutic RAI doses so that thyroid function can stabilize and remission of cancer can be verified. RAI should not be given to women who are breast feeding and should be deferred for at least six to eight weeks after breast feeding cessation to decrease radiation exposure to breast tissue.

There is growing understanding of the possible late effects of RAI, which can include: permanent damage to the salivary glands resulting in chronic xerostomia or salivary duct stones, excessive dental caries, reduced taste, pulmonary fibrosis (in those with diffuse pulmonary metastases), fertility issues, and the possibility of the development of other cancers (stomach, bladder, colon, salivary gland, breast, and leukemia) after very high cumulative doses of 131I. Therefore, caution should be exercised when giving multiple high doses of RAI to children, particularly in those patients whose disease is more indolent and may not require such aggressive therapy.

In the patient treated for DTC or medullary thyroid cancer (MTC), surgical resection can be associated with potential late effects, including voice difficulties and hypoparathyroidism. If the recurrent laryngeal nerves or the external branches of the superior laryngeal nerve are damaged, patients may be hoarse following surgery and require additional surgical procedures to improve phonation. Continued on page 6
Patients who develop permanent hypoparathyroidism will require lifelong vitamin D and oral calcium preparations to maintain eucalcemia.

For the majority of patients, lifelong thyroid hormone suppressive therapy is needed, compliance of which can be more challenging during the adolescent/young adult years. There has also been concern about the adverse cardiac effects of long-term thyroid hormone suppressive therapy for DTC. A few studies have shown increased left ventricular mass index (LVMI) in treated patients compared to controls, with resolution of the abnormalities with reduction in suppressive doses of LT4. In other populations, increased LVMI has been associated with left ventricular hypertrophy, which is an important risk factor for cardiovascular disease. The importance of this finding for demonstrable cardiac disease is unclear and other adverse cardiac effects have not been consistently confirmed.

Many survivors of thyroid cancer express ongoing difficulty with problems that may impact quality of life. When compared to survivors of other cancers, thyroid cancer survivors are more likely to report memory problems, psychological issues, and migraine headaches. In another series, over 50% of patients reported fatigue as a chronic complaint. Fatigue is frequently reported, even in those with normal or mildly suppressed TSH levels, and it is unclear how to intervene effectively. Studies combining L-thyroxine with triiodothyronine therapy have not shown clear benefit, but this may be considered in individual cases. Other reported problems include anxiety, insomnia, temperature sensitivity, skin changes, dryness, and pruritus. At University of Texas M. D. Anderson, we have developed a multidisciplinary survivorship clinic to focus on late effects from thyroid cancer.

**Thyroid Cancer as a Second Malignancy**

Survivors of childhood cancer who have had radiation therapy to the neck or total-body irradiation as part of a stem cell transplant are at a higher risk for the development of thyroid cancer later in life. The risk is increased for those patients receiving up to 30 Gy to the neck, five years after irradiation. Therefore, it is important to be monitored with thyroid exam annually by healthcare providers who are familiar with this potential late-effect of therapy. Ultrasound with FNA is recommended for palpable nodules, but not as regular screening.

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**Genetic Counseling and Genetic Aspects of Pediatric Endocrine Tumors**

Thereasa A. Rich, MS, CGC, Certified Genetic Counselor, Department of Surgical Oncology and Endocrine Center

Patient autonomy and informed consent are central guiding ethical principles surrounding genetic testing. These concepts are of special consideration in genetic testing for children, since individuals under 18 years of age legally cannot give informed consent. The genetic counseling issues outlined by the American Society of Human Genetics (ASHG) 1995 policy on genetic testing in minors will be reviewed and examined in the context of hereditary endocrine tumor syndromes, including multiple endocrine neoplasia types 1 and 2 (MEN1 and MEN2), von Hippel-Lindau syndrome (VHL), Li-Fraumeni syndrome (LFS) and the familial paraganglioma syndromes (FPGL).

Genetic testing is considered appropriate in childhood when the information gained will result in a clear medical benefit for the child and inappropriate when the information will not result in medical benefit.

MEN2 has served as a prototypical example of the usefulness of genetic testing in childhood. Patients with MEN2 have over a 90% lifetime risk to develop medullary thyroid cancer (MTC), which can be prevented or completely treated in those diagnosed at an early stage. Children with high risk RET mutations are recommended to undergo prophylactic thyroidectomy in early childhood to prevent the development of MTC. Other select cases involving RET mutations associated with later onset and less aggressive MTC may be able to undergo expectant monitoring to delay thyroidectomy until later in life.

Similarly, patients with VHL benefit from an early diagnosis. While there are no preventive options, surveillance can significantly impact outcomes. Children with VHL are primarily at risk for hemangioblastomas of the central nervous system, retinal angiomas, and pheochromocytomas. Early detection and treatment of such tumors can prevent serious complications from tumor growth, such as vision loss in the case of retinal angiomas.

Mutations in SDHB, SDHD, and SDHC cause FPGL, which is associated with a high lifetime risk to develop paragangliomas (PGLs). The typical site that PGLs develop, the risk for a PGL to become malignant, and the inheritance pattern varies between each gene. SDHD mutations are associated with PGLs that tend to develop predominately from the parasympathetic chain, have a low risk of malignancy, and mutations are transmitted in an autosomal dominant pattern with parent-of-origin effects—children who inherit a SDHD mutation from their mother are not expected to have an increased risk for PGLs, but those that inherit a mutation from their father do have a high risk. Mutations in SDHC are associated with predominately benign parasympathetic PGLs and are inherited in a straightforward autosomal dominant pattern. SDHB mutations are associated with predominately sympathetic PGLs, a relatively high risk for malignancy, and autosomal dominant transmission of mutations. In all three syndromes, childhood onset of paragangliomas is possible. Most published management guidelines, which are based on expert opinion, recommend initiating predictive genetic testing in childhood and screening of mutation carriers. It is thought that screening and early tumor detection should, theoretically, benefit a child medically given the slow-growing nature of the tumors and the potential for catecholamine hypersecretion. The exception is testing children at risk to inherit a SDHD mutation from their mother; in such cases, predictive genetic testing should be delayed until adulthood since the information would not benefit the child medically.

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The utility of genetic testing for other hereditary endocrine tumor syndromes is less clear. For example, patients with MEN1 may occasionally present with childhood onset endocrine tumors, mainly primary hyperparathyroidism and pituitary adenomas, however in those that do, the disease is typically not life-threatening. It is not clear whether outcomes would be improved if these diseases are detected and treated in childhood versus waiting until early adulthood. In situations such as this, where the medical benefit is uncertain, the ASHG recommends careful counseling about the benefits, risks, and limitations of genetic testing, and allowing the parents to make the decision.

Li-Fraumeni Syndrome is a difficult situation in that patients do have a high risk to develop life-threatening cancers in childhood, but there are no screening measures that are likely to improve outcomes. LFS is associated with risk for many different cancer types, but primarily brain tumors, soft tissue sarcomas, osteosarcomas, adrenocortical carcinomas, and breast cancer; and all but the latter may be childhood onset. Since individuals with LFS are particularly prone to radiation-induced malignancies, diagnostic TP53 testing in children who are already affected with a LFS cancer could impact the decision-making when radiation could be a treatment option. In addition, identification of LFS in an affected child would alert to the possibility that the parents could be at risk for LFS-associated cancers. Predictive genetic testing in childhood is generally discouraged because there is little medical benefit to the child and the potential for psychological harm becomes the predominant consideration.

Genetic testing in childhood may be associated with positive or negative psychological effects and may impact family dynamics and family planning.

Predictive genetic testing in childhood is discouraged in cases where there is no medical benefit to the child because there is a real potential for psychosocial harm. While there is still very little research addressing this topic, multiple concerns have been raised by experienced geneticists and child psychologists. For the child, a genetic diagnosis could negatively impact self-esteem, self-image, and expectations out of life. Parents of a child with a genetic diagnosis may perceive their child to be sick or vulnerable resulting in overprotective behaviors, over-reaction to common childhood symptoms, and differential treatment of the child. Even in cases of children who did not inherit the genetic condition in the family, there is the potential for survivor guilt (“why NOT me?”) and resentment of the extra attention often paid to the affected sibling.

While much of the literature on psychosocial implications of genetic testing focuses on potential harms, there is also the potential that genetic testing could have a psychological benefit. For instance, identifying children who are NOT at risk for the hereditary condition in the family can create a tremendous amount of relief and the child and family would be spared from the burden of expensive and time-consuming screening exams. For other patients, confirmation of a diagnosis reduces uncertainty and allows the family to plan ahead for the medical care their child is expected to need. It is also possible that growing up with the knowledge of a genetic condition from an early age could be less impactful on self-image as an older child, adolescent, or young adult learning they have a genetic disease for the first time.

The diagnosis of a genetic syndrome may also result in changes in life planning in terms of the child’s educational goals, occupational choices, living arrangements (for example, feeling forced to live close to home, caregivers, and their doctors), and financial planning, including retirement planning and obtaining various forms of insurance. The impact of the genetic condition on planning may be appropriate and helpful (for instance, positioning oneself in a situation of financial stability and insurability by taking advantage of anti-discrimination laws), or inappropriate and detrimental (for instance, not pursuing educational or career opportunities or financial planning that otherwise would have been pursued).

Finally, the diagnosis of a genetic condition in a child reveals information about future reproductive risks for the child and their parents. Children growing up with the knowledge of their reproductive risk could experience significant anxiety, particularly as it relates to relationships, dating, marriage, and their perceived desirability as a reproductive partner. However it could also be argued that having the information from a young age could help the child develop a mature view of family planning. Parents have the option to consider reproductive options, such as preimplantation genetic diagnosis and prenatal genetic testing, which reduce the risk of having another child with a genetic condition. However this raises special concerns in terms of how the parents’ reproductive choices may impact the psychology of the affected child (“my parents avoided having another child like me”).

Childhood genetic testing often involves consent from a family unit rather than a single individual. As children get older; they have increasing emotional and intellectual capacity to understand the implications of genetic testing, and should be increasingly involved in decisions about their own medical care.

Legally, a child is unable to provide informed consent until 18 years of age; however most people can draw from personal experience that emotional and intellectual maturity cannot always be predicted from age alone. There is a growing trend of involving children in their own medical care since a child’s assent may foster stronger and more trusting relationships with the healthcare team and potentially empower the child such that their long-term health outcomes might be improved.

In most cases, parental authority is ultimately honored given the lack of the legal ability of the child to provide consent and the presumption that parents are promoting the best interests of the child. While uncommon, complex ethical and legal issues can arise if parents and clinicians disagree about whether a genetic test is appropriate in a child.

Geneticists and genetic counselors are available to help families and clinicians tackle issues related to childhood genetic testing, and in the rare instances of ethical dilemmas, a medical ethics committee can be employed.
Do you need a Resource for a Suspicious Thyroid Nodule?

Thyroid nodules are fairly common, representing the most common endocrine neoplasia problem in the United States, but effective evaluation is extremely important to rule out thyroid cancer.

Dr. Naifa Busaidy, Director of the Thyroid Nodule Clinic now open at M. D. Anderson Cancer Center says, “The clinic serves as a resource for our physicians and all patients with thyroid nodules. We want to be a part of your team in providing an exceptional experience for the community physician and their adult and pediatric patients.

Getting a rapid and accurate diagnosis in one place at one time for a patient anxious about whether or not they might have cancer, improves the experience for all those involved. The experienced multidisciplinary team of endocrinologists, surgeons, mid-levels, cytopathologists, radiologists and ultrasonographers at M. D. Anderson are here to help you. We also have two pediatric endocrinologists who can evaluate pediatric patients of all ages.

All patients receive within one day:
- Consultation with a thyroid specialist
- Thyroid ultrasound
- Thyroid biopsy, if needed
- Multidisciplinary conference to discuss treatment options, if needed.

The Thyroid Nodule Clinic is located inside the Endocrine Center at M. D. Anderson Cancer Center at 1515 Holcombe in Houston, Texas.

For more information or to refer a patient for an appointment:
New Patient Referral Coordinators: 713-563-4400, and 713-792-5410 for patients under 18 years of age.
Physician to Physician Referrals: 713-792-2841
Online Referrals: https://my.mdanderson.org/