(subacute), depending on bleeding, edema extension, and necrotic evolution. It may result in severe neurological, ophthalmological, and endocrinological consequences and may require prompt surgical decompression.

Case: Our patient is a 17 year old AA tall male (Ht=86%) that was initially seen by neurosurgery with a history of progressive headaches over a period of 7-8 months, fatigue and abnormal brain MRI findings. He also reported sudden episodes of "blacking out" prompted by loud sounds. He did not have any visual complaints. He was referred to endocrinology and his work up showed hypopituitarism with a low baseline cortisol 5.1 ug/dL (6.2-19.4) with inappropriately normal ACTH 23 pg/ml (7.2-63.3), low free T4 0.72 ng/dL (0.93-1.6) with an inappropriately normal TSH 1.7 uIU/ml, low testosterone 18.4 ng/dL (350-970) without an elevated LH 1.1 mIU/ml or FSH 1.9 mIU/ml; low IGF1 74 ng/ml (153-542), normal IGFBP3 3316 ug/L (2657-6319); slightly elevated PRL 36.6 ng/ml (4-15.2). The initial brain MRI showed a pituitary mass, measuring 13x14x17 mm, which was homogenous with minimal upward lifting of the optic chiasm with concern for possible hemorrhage in the adenoma. He was started on maintenance and prn stress doses of hydrocortisone and subsequently thyroid hormone and testosterone gel with improvement of symptoms. A repeat MRI approx. 3 months after showed no interval change. Decision was made to proceed with endoscopic transphenoidal hypophysectomy. There were no complications, (i.e., DI). The pathology report described "organizing hematoma and fragments of sinus mucosa." He had labs repeated nearly 4 weeks post op with an improvement in his IGF 1 211ng/ml (151-521), and PRL 12 ng/ml (3-18). His FT4 after thyroid hormone implementation was 1.64 ng/dl (0.93-1.6). His testosterone was slightly lower than initial one (testosterone 161.8 ng/dl) but he was less compliant with testosterone therapy.

Conclusion: Pituitary apoplexy is rare in the pediatric or adolescent population and is restricted to case reports. It remains a diagnostic and therapeutic challenge and specific guidelines are lacking. The outcome is highly variable and the optimal time of surgery is still a matter of debate. For our patient serial imaging will show if there is recurrence of a lesion and repeated pituitary function will allow us to determine need for hormone replacement over time since resolution of existing deficiencies or development of new ones have been reported.

Thyroid

THYROID NEOPLASIA AND CANCER

Detection of RAS Mutations and RET/PTC Fusions in Thyroid Cancer Using Microfluidic Digital PCR

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MON-514

Detection of RAS mutations and RET/PTC fusions in thyroid cancer using microfluidic digital PCR

Background:

The identification of somatic mutations and gene fusions is crucial for guiding therapeutic decisions in patients with thyroid cancer. Microfluidic digital PCR is currently considered as a technique of choice for highly sensitive detection of gene mutations/fusion. We recently demonstrated that dPCR is a useful tool for detection of BRAFV600E and TERT promoter mutations in thyroid tumors.

Objectives:

This study aimed to determine the analytic and clinical validity of dPCR for detection of RAS mutations and RET/PTC fusions in thyroid cancer tissue.

Material and Methods:

Thyroid tissues from 75 patients with PTCs (58 classical PTC (CPTC) and 17 follicular variant (FVPTC) were used for DNA and RNA extraction. The rare mutation SNP genotyping assays which were multiplexed for detection of mutant and wild type NRASQ61; as well as RET/PTC1 and RET/PTC3 were synthesized by Thermo Fisher Scientific. Digital PCR was performed using a QuantStudio 3D Digital PCR platform. QuantStudio Software was used for relative and quantitative data analysis.

Results:

NRASQ61 was detected in 0/58 CPTC and in 6/17 (35%) FVPTC. The ratios of mutant/total varying from 11.7% to 61.5%. Among patients with FVPTC there were no significant associations between the presence of NRASQ61 and patient's age, sex, multifocal growth, extra-thyroidal invasion and lymph node metastases. The ratios mutant/total correlated with tumor size in patients harboring NRASQ61. In 23 cases, RET/PTC1 and RET/PTC3 transcripts were examined. RET/PTC1 and RET/PTC3 transcripts were detected in 3 and 1 case, respectively. RET/PTCs were detected in CPTC, but not in FVPTC. RET/PTC positive tumors were characterized by multi-focal patterns of growth, presence of extra-thyroidal invasion, and presence of lymph node metastases (4 of 4 cases with RET/PTC). There were not RET/PTCs positive tumors harboring simultaneously anomalies in RAS oncogene.

Conclusions:

Microfluidic digital PCR allows specific, sensitive and rapid detection of RAS mutations and RET/PTC fusions in thyroid tissue samples. Implementation of dPCR-based assays may facilitate analysis of thyroid tumors and support research in patients with thyroid cancer.

Tumor Biology

ENDOCRINE NEOPLASIA CASE REPORTS I

Dual Ectopic Gastrin and ACTH Secretion Leading to Combined Zollinger-Ellison Syndrome and Cushing's Syndrome in a Patient with Metastatic Neuroendocrine Pancreatic Tumor

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SUN-907

Background:

Zollinger-Ellison Syndrome (ZES) is caused by ectopic secretion of gastrin from a gastrinoma. The annual