

between each E and the NR. Factors that likely contributed to difference in measurement are heterogeneity of the PA, MRI quality, selection bias in choosing “most appropriate” site to measure intensity of adenoma, gray and white matter. Es could be trained to interpret the T2 intensity, although reliability with NR is only moderate. Interestingly, in this sample majority of T2 PA were hyperintense, but densely granulated, suggesting that preoperative identification of densely granulated tumors, which are also predictive of favorable SRL response, might be limited. More studies are needed to assess T2 correlation with pathology. **Conclusion** As T2 intensity (hyper-, hypo- or iso-) on MRI might be predictive of biochemical response to medical therapy in some patients with PA, we recommend T2 intensity to be part of neuroradiology reporting protocol. Our pilot study showed that endocrinologists could read MRIs after adequate training, but there is only moderate correlation with neuroradiologists.

Neuroendocrinology and Pituitary PITUITARY TUMORS II

Pituitary Magnetic Resonance Imaging in the Postoperative Follow-Up of Patients with Acromegaly, Less Is More!

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Background Patients (pts) with acromegaly (A) require long term follow up, as up to 15% will develop recurrence. Current guidelines for MRI surveillance recommend 12 week post-operative (postop) imaging for all pts and yearly if on pegvisomant (PEG). Many pts with residual tumor postop undergo repetitive imaging even when controlled with pituitary (PIT) directed therapies. However, gadolinium retention and healthcare costs are of increased concern. **Aim** Assess tumor growth postop and necessity of serial MRI in medically treated A pts. **Methods** Retrospective, IRB-approved, data analysis of pathology-proven A pts. Included were pts with at least 1 MRI at ≥ 1 year postop. Initial tumor size, invasion status, pathology, postop remission, MRIs, radiation and medical therapy data were collected. Biochemical (biochem) remission = normal IGF-1 and GH < 1 at 3 mo postop. For pts with radiation, data was only collected up to radiation. Stats: t-test, chi-square. **Results** 83 pts were included; mean age 46 ± 16 years, 45% female, mean follow up 7.9 ± 5.3 years. 55 pts were on PIT-directed therapy (50 on somatostatin receptor ligands (SRL) alone, 1 on cabergoline (Cab) alone, 4 on SRL/Cab), 12 on PEG > 1 year (9 on PEG alone.) 11/83 (13.25%) had tumor growth at median 3.5 years (range 1-11). Tumors that grew were larger at diagnosis (25.21 ± 10.93 mm vs 17.45 ± 8.37 mm, $p=0.004$), had larger residuals postop (23.83 ± 5.0 mm vs 11.86 ± 7.47 mm, $p=0.0003$), and tended to be invasive (77.78% (7/9) vs 53.03% (35/66), $p=NS$). 7/11 were sparsely granulated and 4 mixed GH-PRL. Of 11 that grew, 8 had postop residual tumor, 3 in remission, 4 with discrepant IGF-1/GH, 2 uncontrolled and 2 with no data at 3 months

postop. At the time of growth, 9/11 pts were untreated (6 had active A, 1 with discrepant IGF-1/GH and 2 with no IGF-1/GH data), 1 was controlled on pasireotide and one in biochem remission. Only 1/50 (2%) pts on pasireotide had growth and no pts on PEG > 1 year. **Discussion** 86.75% of pts with A did not have tumor growth after surgery. Only one pt on PIT-targeted medications and none on PEG experienced tumor growth. Almost all pts who had growth had large invasive adenomas, majority were sparsely granulated, residual tumor postop, were biochemically uncontrolled and not on medication at the time of growth. A previous metanalysis of SRLs in A showed that tumor increase occurs in 1.4% (follow up 3-36 months). In our study pt follow up was longer and 1.82% (1/55) of pts who were on SRL/Cab had growth. **Conclusion** We recommend less frequent MRI monitoring for pts treated with PIT-targeted medications. Conversely, pts with residual adenoma not on medical therapy should be closely monitored biochemically and by serial MRIs. Further studies are needed to identify appropriate imaging interval for pts on medications and based on characteristics of aggression (such as sparsely granulated, large residual tumors, lack of biochemical control despite medications).

Thyroid

THYROID DISORDERS CASE REPORTS III

The Broken Heart That Hid Behind the Goiter

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Most goiters grow slowly over many years and are often asymptomatic. Although substernal goiters are estimated to represent between 5-24% of all mediastinal masses [1], a majority are benign. Symptoms may include neck fullness, nocturnal or positional dyspnea, or dysphagia due to tracheal and/or esophageal compression [2-3]. We present a case of a patient with new onset dyspnea that was initially attributed to a large intrathoracic goiter, but ultimately was found to be due to severe heart failure.

A 72-year-old man with a history of HTN, type 2 diabetes, and moderate aortic regurgitation presented to his primary care physician with exertional dyspnea, dry cough, and bilateral leg edema for 2 months. He was referred to pulmonology and a chest CT showed a large intrathoracic goiter, measuring 8.5 x 4.6 x 5.3 cm, extending from the left limb of the thyroid into the mediastinum with rightward tracheal and leftward aortic arch displacement. The patient had no prior history of thyroid disease, cancer, or neck radiation. He denied neck fullness, dysphagia, positional or nocturnal dyspnea, though his exertional dyspnea was progressive. Labs revealed that the patient was biochemically euthyroid. Due to concern for malignancy, the patient underwent a biopsy via EBUS/bronchoscopy, which was non-diagnostic. The case was ultimately discussed at cardiothoracic tumor board, and it was determined that since the mass had likely been present for several years and with the surgical risks being high, to continue monitoring with serial imaging.