radioactive iodine (RAI) or thyroideotomy. Thyroidectomy has been documented to have the lowest rate of recurrence amongst treatment options. Data regarding long-term recurrence rates is limited beyond 54 months.

**Clinical Case:** An asymptomatic 59-year-old female was found to have recurrent thyrotoxicosis on routine laboratory testing. The patient underwent thyroidectomy at age 19 years for Graves’ disease. Prior records unavailable to clarify initial surgical intervention. The patient had post-surgical hypothyroidism which was managed with levothyroxine 100mcg once daily for over 20 years. A biochemically euthyroid state was clearly documented on prior laboratory testing. Initial laboratory testing with TSH <0.01mIU/L (0.45-4.50), FT3 2.8ng/dL (0.8-1.7). Levothyroxine was discontinued with persistent thyrotoxicosis after 8 weeks: TSH <0.01, FT3 5.7, FT4 1.74. Radioactive Iodine Uptake and scan was obtained after administration of 6uCi of iodine-131 which demonstrated 50.8% uptake of radioactive iodine at 24 hours (Normal 10-30%). The left thyroid gland was noted to be in normal position and enlarged with diffuse increase intensity of radiotracer uptake. The right thyroid gland was surgically absent. The patient subsequently underwent completion thyroidectomy with endocrine surgery with resolution of hyperthyroid state. Surgical pathology was benign and consistent with Graves’ disease and multinodular goiter. The patient did become hypothyroid post-operatively and required levothyroxine replacement. She is clinically and biochemically euthyroid on levothyroxine 100mcg once daily 14 months post-operatively.

**Conclusion:** This is a case of recurrent hyperthyroidism approximately 40 years after definitive treatment with thyroidectomy. Although it is unclear whether patient underwent total thyroidectomy or subtotal thyroidectomy for initial intervention, the recurrence of thyrotoxicosis after such a long period of time has not previously been reported in the literature to the knowledge of this writer. This has important implications regarding the underlying pathophysiology of Graves’ disease and the ability of remnant thyroid tissue to regenerate over time. This also has important implications for long-term monitoring in patients with history of thyroidectomy for Graves’ disease.

**Reference:** 1. Sundaresh, V., Brito, J. P., Wang, Z., Prokop, L. J., Stan, M. N., Murad, M. H., & Bahn, R. S. (2013). Comparative effectiveness of therapies for Graves’ hyperthyroidism: a systematic review and network meta-analysis. The Journal of clinical endocrinology and metabolism, 98(9), 3671–3677.

**Thyroid**

**THYROID DISORDERS CASE REPORT**

**Improvement of Treatment Resistant Depression in a Patient With Primary Hypothyroidism and Thr92Ala5’ Type 2 Deiodinase Gene Polymorphism With Multiple Daily Doses of Triiodothyronine**

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**Introduction:** The augmenting pharmacologic therapy used in patients with treatment-resistant depression (TRD) includes drugs such as lithium, buspirone, triiodothyronine (LT3) and other drugs. We report a patient with TRD and primary hypothyroidism who responded to a combination of LT3, given in divided doses, and levothyroxine (LT4), rather than LT4 alone, even though the serum TSH levels were in the normal range with these treatments. Interestingly, the patient had a Thr92Ala5’ type 2 deiodinase polymorphism. **Case Report:** A 54-year-old male presented to our emergency room with suicidal ideation 8 years ago. The patient had severe depression and developed uncontrollable urges to surf the internet, generally prohibited sites, approximately 3 months prior to his visit to the emergency room. He had noted a 12-lbs weight gain, cold intolerance, dry skin, and excessive sleepiness for 3 months. Patient was admitted to the psychiatry ward and laboratory testing showed a serum TSH 180 µIU/mL, FT4 0.48 ng/dL, total T3 46 ng/mL, and TPO antibody 278 IU/mL. A thyroid ultrasound was consistent with Hashimoto’s thyroiditis. A diagnosis of major depressive disorder and primary hypothyroidism was made. He was started on citalopram (20 mg/day) and levothyroxine (175 mcg/day). The Beck Depression Scores (BDS) during the initial weeks was 37.5 ± 5.1 (Mean ± SD) (normal 0-9) with corresponding TSH 164 ± 133 µIU/mL, FT4 0.70 ± 0.25 ng/dL, and total T3 61 ± 7.9 ng/mL. Two weeks later the dose of citalopram was increased to 40 mg/day and then buspirone 10 mg/day was added. At the end of 11 months the BDS was 27.81 ± 2.1 with a corresponding TSH 1.5 ± 0.1 µIU/mL. After 4 months, 7.5 mg of aripiprazole was added. After 11 months of treatment, he was treated with a combination of LT4 + LT3 (5 mcg once daily) and TSH became 0.76 ± 0.1 µIU/mL with a corresponding BDS of 18.0 ± 1.9. Twelve months later, the patient was switched back to LT4 alone and during LT4 treatment the BDS score was 24.2 ± 22.2 with a TSH of 1.44 ± 0.11. Nine months later patient was changed to LT4 + LT3 (5 mcg three times daily) and his BDS score was 10.3 ± 1.2 with a TSH of 0.72 ± 0.09 µIU/mL. When he was on LT4 + LT3 TID he was able to discontinue all the antidepressant drugs and had no urge to surf on internet. His depression was controlled by over-the-counter antidepressant drugs (S-adenosylmethionine and rhodiola). A genetic test confirmed Thr92Ala5’ type 2 deiodinase polymorphism. **Discussion and Conclusion:** In our patient, there was a good correlation between the BDS improvement and the serum T3 levels (r: -0.7 p-value: 0.01). Thus, in patient with Thr92Ala5’ type 2 deiodinase polymorphism TID T3 dosing may significantly improve depression. Additional studies are needed.
Introduction: Intrathyroidal branchial cleft cysts are a rare entity and are usually associated with Hashimoto’s thyroiditis. Etiology is not very clear at this point, but they are thought to arise from the ultimobranchial body remnant during embryogenesis of the gland versus originating secondary to chronic inflammation as a form of squamous metaplasia. They are a common finding in the lateral neck but not with the thyroid. We present a case of a suspicious appearing bilateral thyroid nodules which were found to branchial cleft cysts on biopsy.

Clinical Case: A 56-year-old woman presented to us for evaluation of thyroid nodules. Her past medical history was pertinent for endometrial cancer status post surgery and radiation, obesity, sleep apnea, hyperlipidemia and hypothyroidism. She was diagnosed with hypothyroidism around 30 years ago and had been on Levothyroxine since then. She was recently noted to have a low TSH level on routine labs done by her primary care provider and therefore her Levothyroxine dose was adjusted. She also underwent a thyroid ultrasound due to exam findings of a palpable thyroid nodule. The ultrasound revealed a suspicious 2.2 cm hypoechoic solid nodule with irregular margins and microcalcifications in the left mid thyroid lobe. Additionally, she was also noted to have a 0.8 cm hypoechoic nodule with irregular margins in the right mid thyroid lobe. No concerning cervical lymphadenopathy was identified. She was hence referred to us for further evaluation. She denied having compressive symptoms and did not have history of head/neck radiation or family history of thyroid cancer. Both nodules were biopsied but were interpreted as non-diagnostic with insufficient follicular cells and colloid. She underwent repeat biopsy of both nodules which again was interpreted as non-diagnostic. After discussion with patient, given her suspicious ultrasound findings and inconclusive biopsy results she was referred to endocrine surgery for surgical evaluation. Shortly after she underwent total thyroidectomy with pathology consistent with bilateral benign branchial cleft like cysts associated with adjacent thyroid follicles undergoing squamous metaplasia admixed with chronic inflammation.

Conclusion: Pathogenesis of these intrathyroidal branchial cleft cysts is still unclear and not many similar cases have been reported in the literature so far. These commonly present as a painless mass but sometimes can also be an incidental finding. There are not a lot of specific details in the literature regarding imaging or pathology characteristics of these lesions therefore resulting in surgical intervention to reach a definitive diagnosis.

Thyroid

Thyroid DISORDERS CASE REPORT

Intrathyroidal Branchial Cleft Cysts Presenting as Bilateral Thyroid Nodules

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Background: Over the past few decades consumption of soy products has gained popularity in the U.S. in part due to reports of potential health and weight loss benefits. However, concerns have emerged regarding soy as a potential endocrine-disrupting chemical (EDC) leading to thyroid dysfunction amongst other health issues. Studies have shown a potential association of high soy intake with risk of hypothyroidism and simple goiter (1), but less is known regarding its impact on multinodular goiter.

Clinical Case: A 33-year-old female originally from Bolivia without significant medical history presented to our endocrine clinic with complaints of right-sided neck swelling. The swelling was insidious in onset, had gradually increased in size over the past 3-4 months, and become uncomfortable. She denied dysphagia, cough, or shortness of breath. Prior to the onset of symptoms, she had enrolled in a commercial weight loss program. The diet program consisted of limiting caloric intake to multiple meal replacement bars during the day followed by a light dinner of fish and non-starchy vegetables. Examination revealed a palpable right-sided thyroid mass and enlarged thyroid gland. Lab work showed normal TSH, fT4, and T3 levels, positive anti-thyroid peroxidase antibody (anti-TPO Ab) 588 (<=35.0 IU/ml), and negative thyroid-stimulating immunoglobulins (TSI). Ultrasound of the thyroid confirmed the presence of a 4 cm dominant right thyroid nodule and multinodular goiter. Subsequent fine-needle aspiration of thyroid nodule was consistent with benign nodular goiter. Upon further investigation, it was discovered that the meal replacement bars contained a significant amount of soy protein resulting in an excessive intake of 20-40 grams of soy protein daily. We discussed treatment options, and the patient declined thyroid surgery in favor of surveillance following discontinuation of the weight loss program. The patient was advised to consume a varied diet and given follow-up appointments for monitoring. Conclusion: Inadvertent excessive soy intake via meal replacement bars may have triggered the rapid growth of a multinodular goiter in our patient despite reported adequate dietary iodine intake. Soy products often contain isoﬂavones that may exert an adverse effect on the thyroid by inhibition of TPO, disruption of iodine metabolism, and/or estrogenic activity. We advise that caution be exercised with high soy protein consumption especially in patients with underlying risk factors for multinodular goiter. References: Messina, M. and Redmond, G., 2006. Effects of soy protein and soybean isoﬂavones on thyroid function in healthy adults. J Endocrine Soc, Volume 5, Issue Supplement_1, April-May 2021