

CONCLUSION

Early course of AMTT can rarely present as AST, both being progressive and fatal diseases. Our case had a typical presentation of AST, causing delay in diagnosis of his very aggressive form of thyroid cancer. Gross pathology showed predominantly hemorrhagic areas in the tumor with central necrosis, which was likely the cause of acute mass with systemic signs suspicious for infection. One should consider an alternative diagnosis in suspected cases of AST in the absence of positive cultures or lack of improvement with parenteral antibiotics. It is imperative to make a diagnosis without any delay as prognosis for either condition depends on prompt recognition and treatment.

Thyroid

THYROID CANCER CASE REPORTS I

An Unusual Case of Poorly Differentiated Thyroid Carcinoma with an Excellent Prognosis

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Background: Poorly differentiated thyroid carcinoma (PDTC) is a rare and aggressive subtype with morphological/behavioral features between differentiated thyroid carcinoma (DTC) and anaplastic thyroid carcinoma (ATC).

Clinical case: A 43-year-old female presented with 3 cm right thyroid mass noted on US neck. FNA biopsy showed undifferentiated carcinoma, large cell type. Additional immune-stains were suggestive of ATC. Pre-surgery non-stimulated thyroglobulin (NSTG) was 311 (RR 0–55 ng/dl). Pathology post-total thyroidectomy with bilateral level VI lymph node dissection showed a 3.2 x 2.5 x 2.5 cm carcinoma with vascular and capsular invasion. Most of the mass consisted of very atypical pleomorphic cells, mitosis was difficult to find. The tumor did not show the widely invasive-destructive pattern commonly seen in ATC. An adjacent differentiated component showed predominantly follicular pattern and was described as dedifferentiated follicular carcinoma. All lymph nodes were negative for metastatic disease. Post-surgery NSTG was <0.2 (RR <0.1ng/ml as athyreotic), stimulated TG was 2.22 with negative TG antibodies. Four months later, she received 193.5 mCi radioactive iodine (RAI) therapy. The post-therapy scan showed no Iodine-131 avid uptake in neck or distant metastasis. Neck imaging and TG levels done periodically showed no structural or biochemical evidence of recurrence. Currently the patient is cancer-free for 14 years since diagnosis with no need for additional therapies.

Discussion: PDTC accounts for 1–15% of all thyroid cancers. Although PDTC is rare, it is a clinically significant histological diagnosis as it represents the main cause of death from non-anaplastic follicular cell-derived thyroid carcinoma. The Turin proposal published in 2007 suggested three criteria for the diagnosis of PDTC which included the pattern of growth and high-grade features. PDTC presents more frequently with locally invasive extra-thyroidal disease, metastasis to regional lymph nodes and distant organs compared to DTC. Despite the capacity to have RAI

uptake, there has been no evidence of significant improvement in survival due to tumor heterogeneity in differentiation. Recent data suggest that age more than 45 years, tumor size more than 4cm, extra-thyroidal extension, higher pathological T stage, positive margins, and distant metastasis predict worse prognosis.

Conclusion: Our patient showed an excellent response to therapy in spite of having PDTC with positive margins. We hypothesize that this could be likely due to young age at the time of diagnosis, early detection of tumor while it was localized in the thyroid without distant metastasis as well as heterogeneity in the tumor with differentiated cells that are responsive to RAI. We conclude that with early detection, timely surgery, and adjuvant therapy, excellent prognosis can be achieved in patients with PDTC.

Adrenal

ADRENAL CASE REPORTS II

Pseudo-Cushing Syndrome Secondary to Malnutrition and Gluco-Toxicity Mimicking Type 1 Diabetes Mellitus

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Background: Pseudo-Cushing Syndrome (PCS) is an under-recognized clinical entity that is a reversible consequence of alterations in cortisol production. We present a case of a patient with presumed type 1 Diabetes Mellitus (DM) who was found to have PCS secondary to malnutrition. Once the nutritional status normalized, the patient's glycemic control remarkably improved and became well-controlled on metformin alone.

Clinical case: A 54-year-old female with poorly controlled insulin-dependent DM for 10 years was referred for concern for adrenal insufficiency after an ACTH came back elevated in the setting of intractable nausea, vomiting and considerable weight loss over 1 year. Prior HbA1c was 16.2% (RR 4.4–6.7). On exam her vitals were normal, body mass index (BMI) was 15 kg/m². Workup confirmed an elevated ACTH of 100 pg/ml (RR 6–50 pg/ml), however, random PM cortisol was unexpectedly elevated at 26.58 ug/dL (RR 4.46 – 22.7). 8 AM labs for ACTH and cortisol were similarly elevated at 91 pg/ml and 28.33 ug/dl, respectively. She had no evidence of classic Cushingoid features. Subsequent low dose dexamethasone suppression test and 24-hour urine free cortisol were negative. Over 18 months, with optimization of her insulin therapy, BMI improved to 19 kg/m², ACTH and cortisol started to downtrend spontaneously. After 30 months, her BMI improved to 20 kg/m². Repeat blood work showed A1C 6.5%, ACTH and cortisol completely normalized to 42 pg/dl and 8 ug/dL, respectively. After being adherent to insulin for a few years, her gluco-toxicity state resolved. A month prior to following up, she self-discontinued insulin due to hypoglycemia but continued on metformin. Currently she continues to remain off insulin.

Discussion: PCS is a challenging diagnosis to recognize and differentiate from Cushing Disease (CD) especially due to overlap in biochemical profile. It is important to be aware of