composition were associated with islet function but not insulin independence after TPIAT surgery.

Thyroid

THYROID DISORDERS CASE REPORTS I

Thyroid Storm Followed by ATD Induced Agranulocytosis in Late Pregnancy: A Management Dilemma

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Background:
Management of Graves' disease (GD) in pregnancy presents challenges. Thionamide Antithyroid drugs are the treatment for GD hyperthyroidism with goal of achieving mild but persistent hyperthyroidism and avoiding over-treatment in pregnancy. ATD Induced Agranulocytosis is a rare but serous side effect and presents management dilemmas.
Clinical Case:
A 37-year-old woman with history of Graves' disease was admitted to our hospital at gestational age of 34 weeks with fever, palpitations and diarrhea. Burch-Wartofsky Point Scale was 35 concerning for impending thyroid storm. She had been diagnosed with hyperthyroidism 6 weeks into her pregnancy, initially treated with PTU which was then changed to methimazole in 2nd trimester. A work up for infection and PE was negative. Non-compliance was suspected, methimazole was resumed, and hydrocortisone and propranolol were added. After 2 days, her vital signs and free hormone levels normalized. Her methimazole dose was decreased and she was discharged home in a stable condition.
Five days after her discharge, she presented with sore throat, fever and chills. She had an absolute neutrophil count (ANC) of 0 and a positive rapid strep test. ATD Induced Agranulocytosis was suspected. Her labs showed elevated fT3 of 4.5(nl 1.7–3.7), normal fT4 and suppressed TSH with <0.01(nl 0.3–4.9). A CT scan of the neck showed no evidence of retropharyngeal or thyroid abscess. Methimazole was stopped and she was started on glucocorticoids (initially betamethasone for fetal lung maturity, then switched to prednisone) and cholestyramine. She was also started on Cefepime and G-CSF for her neutropenia. A thyroid ultrasound showed enlarged and hypervascular gland. TSI was 157%(nl. <122%), and thyroglobulin 155 ng/ml (nl. <33 ng/ml). After 4 days, her ANC started to recover. Simultaneously, she started to show worsening thyrotoxicosis but remined hemodynamically stable. A decision to induce labor was then made and was successfully done on the 6th day of her admission. Post-delivery, PTU was started at low dose along with SSKI to prepare her for total thyroidectomy which was done on day 3 post-delivery. Post-thyroidectomy, she had an uncomplicated course and was discharged on levothyroxine. Her child did well with no evidence of thyroid disease.
Conclusion:
We present a unique case of thyrotoxicosis in late pregnancy complicated by ATD Induced Agranulocytosis. Given the high risk of thyroid surgery during pregnancy, our multi-disciplinary team approach opted for labor induction, followed by preparation for thyroidectomy and subsequent surgery. Individualization of management approach using a multi-disciplinary team with emphasis on maternal and fetal well-being is of paramount importance with such challenging presentations.

Neuroendocrinology and Pituitary

CASE REPORTS IN SECRETORY PITUITARY PATHOLOGIES, THEIR TREATMENTS AND OUTCOMES

A Fierce Presentation of Cushing Disease

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Introduction: Cushing disease refers to the endogenous overproduction of glucocorticoid due to an ACTH-producing pituitary adenoma. It is important to recognize and treat due to the adverse health outcomes associated with it. We describe an unusual case of Cushing disease which presented very rapidly and progressively with extremely high cortisol levels mimicking those seen in ectopic production of ACTH.**Case Presentation:** A 43 year old Caucasian man, with no past medical history, presented with hypertensive crisis. He was discharged home with anti-hypertensive medications. Over the next 4 months, he gained 20 pounds, mainly around his abdomen, developed fatigue, and blood pressure continued to be high despite six anti-hypertensive medications, developed diabetes and hypokalemia, requiring 120 meq/day of potassium chloride. On exam, he had plethora, central obesity and wide, purple striae over his abdomen. Work-up for secondary causes of hypertension showed normal renal Doppler US, normal aldosterone and renin activity, normal plasma metanephrines, however, his 24 hour urinary free cortisol was dramatically elevated at 4022ug/day with a urine volume of 4 L, 1 mg dexamethasone suppression test showed unsuppressed serum cortisol of 55ug/dl. Morning ACTH of 125 pg/ml with concurrent serum cortisol level of 53.8 mcg/dl, indicated ACTH-dependent hypercortisolism. Inferior petrosal sinus sampling indicated a pituitary source of ACTH. Sellar MRI initially did not show a pituitary adenoma, however, repeat MRI with a 3-Tesla magnet showed a 4 mm pituitary adenoma. He was treated with ketoconazole and was started on atovaquone for PCP prophylaxis while awaiting trans-sphenoidal resection, which he had a month later. Pathology showed a 4 mm adenoma which stained strongly for ACTH. On postoperative day 1, serum cortisol dropped to 2.1 from 52.3 mcg/dl, and patient was discharged on hydrocortisone replacement. Three weeks later, he had lost 12 pounds, hyperglycemia improved with discontinuation of insulin, hypokalemia resolved and hypertension was well controlled on two anti-hypertensives. **Discussion:** ACTH-dependent Cushing syndrome is either caused by Cushing disease, or from ectopic ACTH production from a tumor.