Thyrotoxicosis

An Uncommon Presentation of Thyrotoxicosis

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SAT-462

Coronary Vasospasm-Induced Myocardial Infarction: an uncommon presentation of Thyrotoxicosis

Background: Coronary artery vasospasm-induced myocardial infarction is a rare cardiac complication of untreated thyrotoxicosis. Diagnosis is difficult due to the transient and unpredictable occurrence of coronary spasm [1].

Clinical Case: A 47-year-old Hispanic female smoker presented with a one-week history of severe, intermittent substernal chest pain radiating to the left arm. The pain was associated with palpitations and shortness of breath. She was afibrile with a heart rate of 100, a blood pressure of 119/59, a fine tremor, and brisk reflexes. No lid lag or proptosis was appreciated. The thyroid was enlarged, non-tender, without palpable nodules. ECG showed T-wave inversions in leads V1-V2 and ST depressions in V4-V5.

Discussion: Rarely, hyperthyroidism can present with transient myocardial ischemia secondary to coronary artery vasospasm. The etiopathogenesis is unclear and may relate to a direct metabolic effect of excess thyroid hormone on the myocardium. In a Korean study evaluating chest pain in patients who underwent coronary angiography, the incidence of coronary vasospasm was 5%, occurring most frequently in women under 50 years of age with thyrotoxicosis [2].

Conclusion: Patients who present with angina and are thyrotoxic should be evaluated for vasospasm. Females under 50 years old with Graves’ disease are at highest risk. Treatment includes antithyroid medications along with nitrroglycerin, and we can consider calcium channel blockers including diltiazem. Treatment of thyrotoxicosis eliminates recurrence of vasospasm [3].

References
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3. Marah N, Bryant K, Haq S, Khan M: Graves’ disease-induced coronary vasospasm. JACC: Cardiovascular Interventions. 2016, 9(23):2452-2453.
Case report: 18 years old female with type one diabetes on insulin pump present with epigastric abdominal pain for three days associated with nausea and vomiting of three days duration. On examination; conscious alert oriented young female looks in pain, vital sign were stable temperature 37°C, heart rate 89 beat per minute, blood pressure 103/72 mm Hg, respiratory rate 20 per minute, oxygen saturation 100% and random blood sugar (RBS) 179 mg/dl. Abdominal examination revealed soft and lax abdomen with tenderness in the epigastric area and right renal angle. No sign of rigidity or rebound tenderness. Bowel sound was present. No sign of ascites, splenomegaly or hepatomegaly. Investigations showed; WBC: 10.2, neutrophil 65%, urine analysis WBCs 30-50 per high field microscopy, RBC 5-10, PH 7, negative nitrate and culture did not show any growth. ESR was 48 and CRP was 4.2. Thrombophilic screen was done and all within normal. Computed tomography (CT) revealed reduced enhancement of right kidney likely indicating acute pyelonephritis and portal vein edema with complete occlusion of left branch of portal vein. Local factors and prothrombotic disorders were ruled out. The patient was managed with ciprofloxacin, enoxaparin and warfarin. The patient was symptomatic free and discharge home with a therapeutic range INR.

Conclusion: Portal vein thrombosis is uncommon condition in absence of liver diseases. Few case report liking been reported. This case report is a rare presentation of accelerated osteoporosis in a young diabetic patient.

Neuroendocrinology and Pituitary
CASE REPORTS IN SECRETORY PITUITARY PATHOLOGIES, THEIR TREATMENTS AND OUTCOMES

Accelerated Osteoporosis - a Rare Presentation of Cushing’s Disease
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SAT-266
Background: Osteoporosis in post-menopausal women is usually due to bone loss from estrogen deficiency and/or age. Secondary osteoporosis (SO) is less common. Up to 30% of postmenopausal women and 50% of men with osteoporosis may have an underlying cause. Cushing’s disease (CD) is one cause of SO but rarely the presenting symptoms. The prevalence of osteoporosis (69.6% vs 37.8%) is significantly higher in patients with adrenal rather than pituitary CD. Diagnosing CD remains a challenge to physicians in spite of advances in diagnostic techniques. We report a case of CD in a post-menopausal woman presenting as accelerated osteoporosis.

Clinical Case
A 63-year-old Caucasian female with a history of hypertension and hysterectomy in her 50s on transdermal estrogen was referred to our Endocrine clinic for evaluation of osteoporosis and incidental finding of bilateral adrenal hyperplasia on CT spine. The patient rapidly developed kyphoscoliosis within the past 2 years. She was debilitated by pain and decreased mobility from compression fractures and spinal stenosis, and underwent thoracic and lumbar fusion surgery.

On physical examination, her heart rate was 64 beats per minute, blood pressure 130/92 mm Hg, weight 188 lbs. and Height 5.1 ft; a year ago it was 5.5 ft. Her face appeared round but not red. Buffalo hump and supraclavicular pad were noted. No striae or bruises noted. Healing surgical scars over the thoracic and lumbar spine were violaceous.

The patient’s urine free cortisol levels, tested a month apart, were 190 mcg and 132 mcg (n 3.5-45 mcg/24h). Midnight salivary cortisol levels taken consecutive nights were 160 ng/dl and 513 ng/dl, (n < 100 ng/dl). Morning serum Cortisol and ACTH were 20.2 ug/dl and 14 pg/mL (n Cortisol 6.0-27.0 ug/dl and ACTH 7.2-63 pg/mL). Following low dose (1mg) dexamethasone suppression testing, her serum cortisol were 12.6 ug/dl, ACTH levels were 32 pg/mL and dexamethasone were 187 ng/dl (n < 30 ng/dl).

25-Hydroxy D total, TSH, Free T4, PTH intact, calcium, renin, aldosterone and SLEP levels were normal. Renal and liver functions were normal except alkaline phosphatase was 142 U/L (n 34-104 U/L). Pituitary MRI with contrast showed pituitary microadenoma. The patient was referred to a neurosurgeon and is planned for an inferior petrosal sinus sampling prior to transsphenoidal resection.

Conclusion
Cushing’s disease as a cause of osteoporosis is rare. Diagnosis of Cushing’s can be challenging in patients without obvious signs, as in our patient who was referred to an endocrinologist due to incidental finding of bilateral adrenal hyperplasia. A high degree of clinical suspicion is needed when investigating CD, as initial test results can be indecisive. As in our patient, initial ACTH and cortisol levels were normal, the low dose dexamethasone suppression test helped us direct our diagnosis towards CD.

Thyroid
THYROID CANCER CASE REPORTS II

Prophylactic Thyroidectomy in a Patient with Codon 891 Mutation of the RET Proto-Oncogene
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MON-450
Background: Medullary thyroid cancer (MTC) is a neuroendocrine tumor of the parafollicular or C cells of the thyroid gland, accounting for 1-2% of thyroid cancers in the United States. About 25% MTCs are familial as part of the MEN2 syndrome or familial MTC (FMTC). Germline mutations in codon 891 are predominantly associated with MTC. Case: 65-year-old Caucasian male was referred to the Endocrinology clinic after bilateral thyroid nodules were found on thyroid US. The patient had requested an ultrasound of his thyroid after his brother was diagnosed with MTC following fine-needle aspiration for an incidental thyroid nodule prompting total thyroidectomy and genetic testing. The patient’s brother was found to be heterozygous for RET mutation (c.2617T>G;pSer891Ala). This resulted in screening of the other siblings including this patient, also found to be heterozygous for this mutation. Both parents were deceased, and their clinical history is not known. Only one of the remaining two siblings had genetic testing; one brother refused testing for the mutation and one sister was positive for the mutation but had no thyroid