department, he was tachycardic at 116 bpm, widened pulse blood pressure of 131/50 mmHg with normal respiratory rate 24 BR/min. He had diffused and significant muscle weakness on his all extremities including grip strength.
His potassium was 1.6 mmol/L (3.5 - 5.2) and magnesium 1.6 mmol/L (1.6-2.3). The rest of his chemistry panel was unremarkable. He had EKG changes consistent with hypokalemia with U waves, also revealing atrial rhythm with first degree AV block, intraventricular conduction delay, and QTc prolongation at 588 (<450). His chest x-ray was normal. Normal saline was
administered, and potassium replacement was given with 40 mEq of KCl followed by D5 NS with 40 meq/L KCl at maintenance. He continued taking atenolol and methimazole. He was also given an IV dose of magnesium. His muscle strength returned completely and potassium level returned to normal range at 4.6 mmol/L after 24 hours of treatment.
Conclusion: TPP is a rare cause of acute paralysis and can lead to cardiac arrhythmia and death without accurate diagnosis and prompt treatment. Our case should raise awareness of this disorder among pediatricians, emergency department physicians and endocrinologists. Acute paralysis with hypokalemia should also prompt the physician to consider evaluating thyroid function as a differential diagnosis in young Asian men.

Tumor Biology

NOVEL REGULATORS OF BREAST CANCER PROGRESSION

Lethal ERα-Dependent Hyperactivation of the Unfolded Protein Response Induces Complete Regression Without Recurrence of Primary and Metastatic Breast Cancer

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Metastatic estrogen receptor α (ERα) positive breast cancer is presently incurable and most patients die within 7 years. From a medicinal chemistry program, we identified a novel small molecule that acts through ERα to kill breast cancer cells and often induces complete regression without recurrence of large, therapy-resistant primary breast tumors and of lung, bone, and liver metastases. To target metastatic ERα positive breast cancer, we exploited our finding that estrogen-ERα activates an extranuclear tumor-protective, signaling pathway, the anticipatory unfolded protein response (UPR). We repurposed this tumor protective pathway by targeting it with the small molecule, ErSO. ErSO kills cancer cells by acting non-competitively through ERα to induce lethal hyperactivation of the anticipatory UPR, triggering rapid necrotic cell death. Using luciferase to image primary tumors and metastases containing lethal ERαD538G and ERαY537S mutations seen in metastatic breast cancer, oral and injected ErSO exhibited unprecedented antitumor activity. In mouse xenografts bearing large breast tumors, oral and injected ErSO induced complete regression (>115,000 fold mean regression) in about 45% of mice (18/39). Although durable response for 4-6 months without additional treatment was common, tumors that did recur remained fully sensitive to ErSO re-treatment. Consistent with the essential nature of the UPR pathway targeted by ErSO, in more than 100 tumor-bearing mice, we have never seen an ErSO-resistant tumor. In just 7 days, oral ErSO induced complete regression of most lung, bone, and liver metastases. ErSO is well-tolerated in mice and blood-brain-barrier penetrant. Injected ErSO induced profound regression of challenging brain tumors. On average, ErSO-treated tumors were >180-fold smaller than vehicle-treated tumors. These xenograft studies used human cancer cells in mice that lack a functional immune system and therefore did not exploit the known ability of inducers of necrotic cell death to activate immune cells and induce immunogenic cell death. Notably, medium from breast cancer cells killed by ErSO contained high levels of immune cell activators, robustly activated mouse and human macrophages and increased macrophage migration. Moreover, use of ErSO is not limited to breast cancer. ErSO rapidly kills ERα positive ovarian and endometrial cancer cells that do not require estrogen for growth. ErSO's potent activity against advanced primary and metastatic ERα-positive breast cancers represents a paradigm shift in leveraging ERα for anticancer efficacy.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS I

Severe Primary Hyperparathyroidism, Hypercalcemic Crisis, Acute Pancreatitis and Fatality: Complications of Atypical Parathyroid Adenoma, a Case Report

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Background: Atypical parathyroid adenomas (APAs) are a controversial and rare entity. These tumors show some features of carcinomas including adherence to adjacent structures, banding fibrosis, mitotic activity, and tumor cells trapped within capsule; however, they lack definitive capsular, vascular, or perineural invasion. Patients are often asymptomatic, or have vague symptoms such as fatigue. Studies and literature have reported that the elevation in parathyroid hormone (PTH) and calcium levels in APAs are intermediate between those of adenomas and carcinomas, and that the clinical course is generally benign. **Clinical Case:** A 41-year old woman with diabetes mellitus, Hashimoto's thyroiditis, and obesity was noted to have mild asymptomatic hypercalcemia during routine tests performed at an outside hospital several months prior to presentation. A few weeks prior to presentation, she started complaining of anorexia, nausea, vomiting and abdominal pain. At the time of presentation, she was critically-ill with