We report a case of a 57-year-old woman who developed severe musculoskeletal pain after taking one dose of Alendronate 70mg for treatment of osteoporosis. The pain was so debilitating that the patient was unable to sit up, stand, or move without assistance, and any kind of movement aggravated the pain.

Key words: bisphosphonate, severe muscle pain, case report, osteoporosis

Methods
A detailed case report is presented. A review of the current literature on the side effects of bisphosphonates was made for reference.

Results
Development of severe, disabling musculoskeletal pain was noted after taking one dose of Alendronate 70 mg tablet. The pain was severe enough to hinder the patient from sitting or standing up, and any form of movement seemed to worsen the pain. Discontinuation of the drug, pain relievers and complete bed rest afforded slight relief of symptoms. Complete resolution of pain only occurred after 72 hours. Review of literature showed that muscle pain with intake of bisphosphonates occurred in a few patients, and were mostly found in case reports or case series. Other commonly associated side effects with bisphosphonate therapy were also briefly discussed.

Conclusion
The case presented emphasizes that bisphosphonate therapy, although frequently used uneventfully, may still produce adverse effects in a few patients. A handful may present with severe debilitating muscle pain as seen in our patient. Literature review showed that another subset of patients may react differently and present with cardiac or ocular symptoms. What must be taken from this paper is the importance of patient education regarding these possible adverse events that although infrequent, may still happen. These unpleasant adverse effects may greatly affect compliance and ultimately have a bigger impact in the fight against osteoporosis.

Bone and Mineral Metabolism
BONE AND MINERAL CASE REPORTS I

A Rare Existence
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SAT-LB65
A Rare Existence: Rugger-Jersey Spine in Primary Hyperparathyroidism secondary to a Giant Parathyroid Adenoma with coexistent Thymoma
A GIANT PARATHYROID ADENOMA IN A 54-YEAR OLD FEMALE WITH CO-EXISTENT SILENT THYMOMA: RADIOLOGIC AND MANAGEMENT ISSUES
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Abstract
Primary hyperparathyroidism is a common endocrine disorder of metabolism usually due to a parathyroid adenoma. Although, the clinical presentation of primary hyperparathyroidism has changed from Albright’s description of a disease of bones and stones, the central target organs affected by this disorder continue to be the skeleton and kidneys. With the advent of routine biochemical screening, the typical diagnosis of primary hyperparathyroidism is no longer accompanied by overt skeletal and renal involvement. Majority of the cases of primary hyperparathyroidism are due to parathyroid adenomas. Giant glands were defined as greater than the 95th percentile, characterized as glands weighing > 3.5 grams.

This present case in a 54-year old female is a rare case of primary hyperparathyroidism secondary to a giant parathyroid adenoma measuring 10.7 x 8.0 x 40.0 cm and weighing 145 grams, the largest giant parathyroid adenoma reported to date, with co-existent silent thymoma, multinodular goiter and osteosclerosis of the vertebral spine, metaphorically known as the “rugger-jersey spine”. The association between thymoma and parathyroid adenoma is rare, and only 3 cases have been reported in the literature. We characterized the correlation of preoperative imaging, intraoperative location, and postoperative course, including significant postoperative hypoparathyroidism, as compared to other patients with PHPT to determine whether giant adenomas represent a clinical entity with distinct clinical characteristics.

Keywords: primary hyperparathyroidism, giant parathyroid adenoma, rugger-jersey spine, thymoma

Abbreviation
PHPT
Primary hyperparathyroidism

Diabetes Mellitus and Glucose Metabolism
DIABETES COMPLICATIONS I

POEMS Syndrome: Rare Presentation of New Onset Diabetes Mellitus
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SAT-LB117
Background: POEMS syndrome (Polyneuropathy, Organomegaly, Endocrinopathy, Monoclonal protein, Skin changes) is characterized by the presence of a monoclonal plasma cell disorder, peripheral neuropathy, and
one or more of the following features: osteosclerotic myeloma, Castleman disease, increased levels of serum vascular endothelial growth factor (VEGF), organomegaly, endocrinopathy, edema, typical skin changes, and papilledema. Clinical Case: 45 year old male with no chronic medical issues presented initially to the orthopedic clinic with right hip pain, pelvic MRI showed a right iliac crest lesion. CT Guided biopsy was done and showed plasmacytoma. SPEP showed elevated IgG lambda level in the gamma zone. In the meantime he was complaining of ascending numbness and weakness in hands and feet and he progressively became wheelchair bound. During his treatment and follow ups with Hematology/Oncology he was noted to have elevated blood sugars in the 500s. Hemoglobin A1C was elevated at 9.5 which confirmed the diagnosis of new onset diabetes. He was also noted to have splenomegaly which confirmed the diagnosis of POEMS syndrome. He was started on insulin and he managed to achieve good diabetes control with insulin and dietary changes. He is currently status post stem cell transplantation with a good response and the weakness and polyneuropathy improved with PT and OT. POEMS syndrome has major and minor criteria for diagnosis, mandatory major criteria includes polyneuropathy, monoclonal plasma cell proliferative disorder (almost always lambda). Additional major criteria are sclerotic bone disease, castleman disease, elevated VEGF. Minor criteria are organomegaly, extravascular volume load, endocrinopathy, and skin changes. In order to diagnose the syndrome, mandatory major criteria, and one major and one minor criteria need to be clinically present. Endocrinopathy includes the adrenal, pituitary, thyroid, gonadal, parathyroid, and pancreatic glands. Two-thirds of patients had at least one endocrine abnormality at presentation. Endocrine abnormalities can also develop later, during the course of the disease. Hypogonadism is the most common endocrine abnormality. Elevated levels of follicle stimulating hormone in the absence of primary hypogonadism levels have been reported, hence history and physical examination is crucial to detect the development of endocrinopathies in POEMS syndrome. There are no current guidelines or recommendations about the frequency of screening for endocrinopathies but it is suggested to obtain a baseline of thyroid function test, pituitary, gonadal, and adrenal axis. In addition to baseline parathyroid hormone level, close monitoring of calcium and blood glucose levels once the diagnosis is confirmed in patients with suggestive symptoms. Conclusion: POEMS syndrome is a rare condition that involves multiple endocrine organs, currently there are no guidelines or recommendations to obtain baseline endocrine labs once the diagnosis is confirmed, but it might be appropriate if there is a high clinical suspicion. References: 1. Castillo JJ (2016). “Plasma Cell Disorders”. Primary Care. 43 (4): 677-691. doi:10.1016/j.pop.2016.07.002. PMID 27386585. 2. Warsame R, Yenamandra U, Kapoor P (2017). “POEMS Syndrome: an Enigma”. Current Hematologic Malignancy Reports. 12 (2): 85-95. doi:10.1007/s11599-017-0367-0. PMID 28299525. 3. Dispensieri A (2017). “POEMS syndrome: 2017 Update on diagnosis, risk stratification, and management”. American Journal of Hematology. 92 (8): 814-829. doi:10.1002/ajh.24502. PMID 28699668. 4. Kaushik M, Pulido JS, Abreu R, Amselem L, Dispensieri A (2011). “Ocular findings in patients with polynuropathy, organomegaly, endocrinopathy, monoclonal gammopathy, and skin changes syndrome”. Ophthalmology. 118 (4): 778-82. doi:10.1016/j.ophtha.2010.08.013. PMID 21035860.

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

A Case of Cushing’s Syndrome in a Patient With Addison’s Disease

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SAT-LB52

This is a case of a 56-year-old female with history of Addison’s disease, T1DM, hypothyroidism and Asthma. She was diagnosed with Addison’s disease after multiple hospital visits consistent with adrenal crises (Fatigue, weakness, salt craving, abdominal pain, hypotension, hyperpigmentation and hyponatremia). Multiple abdominal images including CT abdomen did not reveal any pathology. A random cortisol and ACTH were found to be abnormal at 0.5 and 3362, respectively. She was started on Hydrocortisone 10mg in the morning and 5mg in the evening. Her physical exam revealed diffuse hyperpigmentation including palmar creases and oral mucosa. There was no facial plethora or striae. She had no history of proximal myopathy, easy bruising, hypertension or osteoporosis. Upon follow up, exam revealed progressive worsening of her diffuse hyperpigmentation. A random cortisol and ACTH, without holding her Hydrocortisone, revealed 64.3 and >2000, respectively. Concern arose for the possibility for a pituitary corticotrophin adenoma or ectopic secretion of ACTH driving her hyperpigmentation. MRI showed the pituitary gland had normal appearances. As there is a positive correlation between basal plasma ACTH values and the size of the pituitary adenoma in patients with Cushing’s disease the differential of a pituitary corticotrophin adenoma was originally thought to be dropped lower on the list of differentials. Further workup was pursued with an 8mg Dexamethasone suppression test to take advantage of the fact that ACTH secretion by the pituitary adenomas in Cushing’s disease are only relatively resistant to negative feedback regulation by glucocorticoids while most ectopic ACTH production from non-pituitary tumors are completely resistant to feedback inhibition. ACTH levels went from >2000 down to 29 post 8mg Dexamethasone, which led to decreasing suspicion of an ectopic source of ACTH and further concern for a pituitary corticotrophin adenoma in addition to Addison’s disease. ACTH measurements are not widely used in documenting the adequacy of treatment of primary adrenocortical disease. The hormone is released in pulses, particularly in the early hours of the morning, and measurement of isolated samples is of limited value. Inappropriately normal to slightly elevated ACTH levels despite adequate glucocorticoid replacement in patient with adrenal insufficiency could be related to an altered pituitary sensitivity to cortisol suppression. Extremely elevated ACTH levels despite