left-sided weakness. He had a ten year history of hypertension and was taking carvedilol, losartan, and hydralazine prior to presentation. On arrival, his blood pressure was 263/142 mmHg. He had 3/5 grade weakness in the left upper and lower extremities. Laboratory analysis showed a potassium level of 2.8 mmol/L (n = 3.5–5 mmol/L) and a bicarbonate level of 33 mmol/L (n = 21–29 mmol/L). Screening labs for PA were drawn after potassium repletion. CT Head without contrast revealed an acute 2.5-centimeter intracerebral hemorrhage of the right basal ganglia. He was admitted to the intensive care unit and was started on a nicardipine drip with an improvement of blood pressure. His weakness improved and he was discharged home on carvedilol, hydralazine, nifedipine, and losartan.
Screening for PA revealed a plasma aldosterone concentration (PAC) of 22.8 ng/dL (n < 16 ng/dL) and a plasma renin activity (PRA) of 0.1 ng/ml/hr (n = 0.2–1.6 ng/ml/hr). The PAC/PRA ratio was therefore extremely elevated at 228. The presence of spontaneous hypokalemia, very low renin, and PAC >20 ng/dL confirmed the diagnosis of primary aldosteronism. He underwent an adrenal MRI which revealed two left adrenal nodules, the largest measuring 10 mm, and a 7.3 mm right adrenal nodule, consistent with bilateral adrenal adenomas. The patient did not desire surgery, therefore adrenal vein sampling was deferred. His hypertension improved with the addition of a mineralocorticoid receptor antagonist. Eight weeks after his stroke the patient was readmitted due to chest pain. He was found to have severe multi-vessel coronary artery disease and underwent a four vessel coronary artery bypass.
Conclusion: Patients with PA have higher rates of adverse cardiovascular events compared to age-, sex-, and blood pressure-matched controls with essential hypertension. Studies demonstrate that aldosterone excess has blood pressure independent proinflammatory and profibrotic effects on the vessel wall which leads to endothelial dysfunction and thus accelerated atherosclerosis. Appropriate treatment can eliminate the excess cardiovascular risk associated with PA. This case highlights the importance of including PA in the differential diagnosis of secondary hypertension, particularly among patients presenting with spontaneous hypokalemia, severe uncontrolled hypertension and early onset cardiovascular or cerebrovascular disease.

Cardiovascular Endocrinology

CARDIOVASCULAR ENDOCRINOLOGY AND LIPIDS DISORDERS CASE REPORT

Bone Breaking Triglycerides

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A 40 yo African American female with pmhx of T2DM, DLD was admitted for worsening leg and arm pain that started a year prior but had worsened in the last 6 months. Pain started in the right arm and progressed to include the right leg and left leg. She had presented to the ER 3 times in the last 3 weeks with no diagnosis and prescribed anti-inflammatories. On ROS she had unintended weight loss of 50 lbs. Pain was not relieved with anti-inflammatories or narcotics. She was diagnosed with diabetes in the previous 5 years and had not been compliant with her medications. Plain x-rays showed OA of the hip. An osseus survey showed multiple expansile, bubbly, and lucent intramedullary lesions consistent with polyostotic fibrous dysplasia versus multiple myeloma. CT showed a radioluscent lesion of the left femur with absence of normal bone trabeculae. Her labs showed normal calcium, phosphorous, renal function, PTH and no evidence of monoclonal gammopathy. Vitamin D was low at 8.2 ng/ml (6.6–49 ng/ml). CT CAP showed no concern for malignancy in other organs. A lipid profile was done and showed elevated fasting triglycerides of 2617 mg/dL (<150 mg/dl) and LDL direct 54 mg/dl (<100 mg/dl). A1c was 11.2% on admission. She denied any use of alcohol, estrogens, SSRI's. No history of pancreatitis. On physical exam she did not have tendinous xanthomas, eruptive xanthomas, palmar xanthomas, or lipemia retinalis. Family history not significant for lipid disorders. Patient was fasted for 24 hours and then started on intensive insulin regimen as well as fenofibrate for hypertriglyceridemia. Triglycerides came down to less than 500 over 7 days. She was evaluated by ortho for her bone lesions and underwent bone lesion biopsy as well as prophylactic IMN of her bilateral femurs for prevention of impending fragility fractures. Bone biopsy was significant for xanthoma of the bone. Following discharge, she remained on fenofibrate and fish oil as well as a basal/bolus insulin regimen. Triglycerides remained controlled. She has not followed up outpatient for further workup. This case highlights an atypical presentation of triglyceride deposition in the setting of hypertriglyceridemia. It shows that hypertriglyceridemia should be included in the differential for lytic lesions when preliminary workup is negative. It also highlights that complications other than pancreatitis and cardiovascular disease can significantly alter a patient's life if triglycerides go untreated.

Cardiovascular Endocrinology

CARDIOVASCULAR ENDOCRINOLOGY AND LIPIDS DISORDERS CASE REPORT

Challenges in Managing Metabolic Complications in a Patient With Familial Partial Lipodystrophy Type 3

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Familial partial lipodystrophy (FPL) is a rare group of autosomal dominant genetic disorders which causes variable loss of subcutaneous fat from abdomen, thorax or extremities in addition to the numerous metabolic complications like insulin resistance, diabetes mellitus and dyslipidemia[1]. FPL type 3 was first characterized by Agarwal et al. in 2002[1], in which peroxisome proliferator-activated receptor-γ (PPARγ) gene was the molecular basis of this disorder. It is extremely rare and so far only 30 patients or so have been recognized with this mutation[2]. FPL3 is unique because it generally spares the loss of fat from trunk, face and neck region and also presents with more severe metabolic derangements. We report a case of a young female