CLINICAL VIGNETTE

An Elevated Creatinine Kinase in a Patient with Parathyroid Adenoma

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Case Summary

A 63-year-old female was seen by Rheumatology about 4 years prior for elevated creatinine kinase (CK). Her past medical history includes hypertension, hyperlipidemia, fatty liver disease, osteoarthritis of the knees and spine and ductal breast carcinoma in-situ. At that time, she was taking red yeast rice for about 6 months and started to develop diffuse muscle aches, fatigue, and decreased energy. Physical exam at that time revealed normal muscle strength and no stigmata of inflammatory myopathy. Labs included CK 468 U/L (38-282 U/L). Aldolase 7.0 U/L (2.3-10.3 U/L). Antinuclear antibody (ANA) was negative. The reason for the elevated CK was thought to be red yeast rice which contains lovastatin as a naturally occurring component.[1] Suspicion for an inflammatory myositis was low and she was advised to avoid statins and monitor CK. She was also treated for osteoarthritis of the knees with corticosteroid and hyaluronic acid injections without much improvement and she was eventually referred to orthopedics for possible knee replacement surgery.

Orthopedics recommended knee replacement surgery. Her preop assessment, noted to a persistently elevated calcium 10.7mg/dL (8.6-10.4 mg/dL). A parathyroid hormone level was also elevated 90pg/mL (11-51 pg/mL). She was referred to endocrinology and neck ultrasound demonstrated a parathyroid adenoma.

Patient returned to Rheumatology for persistently elevated CK, joint pain, and muscle weakness despite avoiding red yeast rice and statins. Patient described chronic joint pain localized to the knees, thumbs and low back for many years. The joint pain was achy in character and associated with morning stiffness lasting 15 to 20 minutes. She had intermittent swelling in the knees and thumb joints. She denied any rashes, oral ulcers, Raynaud's phenomenon or photosensitivity. She did report difficulty swallowing certain foods without water and struggled with chronic constipation. She had weakness in the thighs which worsened with activity and experiencing difficulty getting into the bathtub or walking. In the past, she would do aquatic therapy but for the last couple months was not able to participate due to weakness. Overall, she acknowledged worsening fatigue and feeling deconditioned over the past couple years.

On physical exam patient's vital signs were stable and she was not in any acute distress. Her heart and lung exam was normal. There were no rashes or gottron papules. There was tenderness to palpation of the carpometacarpal joints bilaterally but otherwise joint exam was unremarkable. Her muscle strength was 5/5 in bilateral upper and lower extremities without any proximal muscle weakness and no edema.

Labs included elevated CK 405 U/L (38-282 U/L), Sedimentation Rate 47 mm/hr (<25mm/hr), and C-Reactive Protein 0.8 mg/dL (<0.8mg/dL). The complete blood count was normal. TSH was normal. The calcium was 10.8 mg/dL (8.6-10.4mg/dL) but the rest of the basic metabolic panel was normal. Alkaline phosphatase was 150 U/L (37-113 U/L) without significant elevation of transaminase, except for GGT of 137 U/L (7-68). PTH was elevated 90 pg/mL (11-51pg/mL) but both vitamin D, 25-hydroxy 40 ng/mL (20-50ng/mL) and vitamin D 1,25-diOH 39.1 pg/mL (19.9-79.3 pg/mL) were normal. Phosphorous was 3.0 mg/dL (2.3-4.4 mg/dL). Chest x-ray revealed no acute cardiopulmonary abnormality. X-ray of the knees showed severe osteoarthritis in both knees. X-ray of the hands showed severe bilateral carpometacarpal osteoarthritis worse on the right compared to the left and mild osteoarthritis of the distal interphalangeal joints. Bone density scan was normal. Ultrasound of the thyroid-parathyroid revealed a hypoechoic nodule that could represent a parathyroid adenoma along the inferior left thyroid measuring up to 9mm.

Based on the patient's symptoms, labs concerning for hyperparathyroidism, and ultrasound consistent with a parathyroid adenoma it was thought the reason for her persistently elevated CK was due to hyperparathyroidism rather than an inflammatory myositis. Patient was recommended to follow-up with endocrinology and underwent a parathyroidectomy. After parathyroidectomy her CK normalized (253 U/L) for the first time and her symptoms of muscle weakness improved.

Discussion

Myopathies can be associated with a variety of conditions, including endocrine diseases, malabsorption, electrolyte disturbances, neuromuscular disorders, infectious diseases, drug and toxin-induced, and rheumatic conditions. It is important to consider a wide differential diagnosis when considering muscle weakness.

Although muscle weakness is common with disorders of calcium and phosphorous metabolism, hyper- and hypoparathyroidism are frequently overlooked as causes of muscle dysfunction because of their non-descript presentation.[2]

Patients usually present with mild proximal muscle weakness, muscle aches and pain, and CK is usually normal or slightly elevated.[3] EMG is usually normal.[4] Muscle biopsy can show atrophy of muscle fibers usually not associated with degeneration.[4] Primary hyperparathyroidism can cause muscle weakness due to decreased energy production and skeletal muscle catabolism.[2] If hyperparathyroidism is left untreated, hypercalcemia and hypomagnesemia can cause a necrotizing myopathy with rhabdomyolysis.[2]

When identified and treated, complete resolution of symptoms can occur and normalization of CK as seen with removal of an adenomatous parathyroid gland for hyperparathyroidism as in our patient.[4]

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