Fatigue and Primary Hyperparathyroidism: A Case Report

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Introduction

Fatigue is a common complaint among patients and a presenting symptom in one-fifth of family medical visits.[1] There are multiple causes of fatigue, and usually a complete history and physical examination will help determine causation and/or further work-up that is needed. Routine blood screening is indicated; however, laboratory results will affect treatment management in only 5% of patients presenting with fatigue.[1]

Primary hyperparathyroidism (PHPT) results from inappropriate overproduction of parathyroid hormone from one or more of the parathyroid gland(s) and presents with hypercalcemia, which can be seen on a basic chemistry panel. It is the third most common endocrine disorder affecting 0.3% of the general population and 1% to 3% of postmenopausal women.[2] Intracellular and extracellular calcium is tightly regulated by physiological processes in our bodies, and central to this regulation are the parathyroid glands. When chief cells in our parathyroid glands sense that calcium in our blood is too low, parathyroid hormone is released, resulting in an increase of serum calcium through multiple means: receptor-mediated tubular reabsorption of calcium in the kidney, release of skeletal calcium stores, upregulation of 1-α-hydroxylase leading to increased 1,25-dihydroxy-vitamin D production, and increased calcium reabsorption from the gastrointestinal tract.[2] The rise of calcium in primary hyperparathyroidism will usually result without symptoms and occur as an incidental laboratory finding; however, nephrolithiasis is the most common symptom and occurs in 15% to 20% of newly diagnosed hyperparathyroid cases.[2] Most patients in developed countries have asymptomatic forms of PHPT or nonspecific symptoms such as fatigue, mild depression, and cognitive impairment.[3]

We report the case of a Caucasian female who presents with a 5-year history of fatigue.

Case Presentation

A 59-year-old woman had been presenting to our out patient clinic for over 5 years, seeing multiple providers with varying complaints. Fatigue remained an underlying secondary or tertiary symptom throughout her visits. In as early as 2007, iron deficiency anemia was diagnosed with a ferritin of 3.89 ng/ml, normal range (12–263 ng/ml). She was treated with 100 mg of iron citrate daily, but it is uncertain how compliant the patient was with treatment. She was still being treated for iron deficiency anemia until 2011, when her ferritin was 135 ng/ml, and the use of iron citrate was discontinued.

Through a routine chemistry screen in 2010, an elevated calcium of 11.4 mg/dl (normal range 8.5–10.5 mg/dl) was discovered. Further work-up was encouraged at that time, such as another calcium level, phosphorous, and PTH, but the patient did not maintain contact. Two years later, in 2012, she was seen with the main complaint of fatigue, and a chemistry screen was run, revealing another elevated calcium of 12.5 mg/dl.

The patient then followed up a month later with a repeat calcium of 12.2 and low phosphorous of 2.4 mg/dl (normal range 2.5–5.0 mg/dl). After this result the patient was strongly encouraged to discontinue self-prescribed supplemental calcium and vitamin D. Shortly after, another calcium was drawn with a PTH, revealing 10.7 mg/dl of calcium and 410.1 pg/ml of PTH (normal range 15–65 pg/ml). At this point, a referral was put in for an endocrinology consult. We considered starting the patient on a loop diuretic to increase the urinary excretion of calcium if the referral took longer than expected.

Conclusion

The history of this patient's low ferritin would contribute to fatigue but, in this case, was not the only cause. In fact, the fatigue persisted despite gaining adequate levels of stored iron. She did not have the typical history or symptomatology associated with PHPT of osteopenia or nephrolithiasis, resulting from calcium excretion from the bones and into the

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