Parathyroid adenoma presenting as tetraparesia

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Abstract

OBJECTIVE: Parathyroid adenomas (PA) are benign tumors. We report an unusual case of delayed diagnosis of a PA and concomitant Vitamin D deficiency presenting as tetraparesia.
METHODS: A 30-year-woman was admitted to our clinic with an inability to walk. Our examination revealed tetraparesia and common, severe muscular atrophy.
RESULTS: Laboratory investigations showed the following: creatinine phosphokinase (CK): 37 IU/L (15–130), calcium (Ca): 11.5 mg/dL (9–11), phosphorus (P): 1.5 mg/dL (2.5–5), parathyroid hormone (PTH): 736.1 pq/mL (15–65), 25-hydroxyvitamin D (25-OHvit D): 4 ng/mL (11–43), and alkaline phosphatase (ALP): 1029.5 lU (64–300). Parathyroid scintigraphy revealed PA. A year after Vitamin D replacement, the patient's neurological status and laboratory findings improved.
CONCLUSION: We suggest that the physicians always keep in mind the primary hyperparathyroidism (HPT) and concomitant Vitamin D deficiency in the differential diagnosis of hypercalcemia when facing atypical neurological symptoms such as tetraparesia.

CASE REPORT

INTRODUCTION

The most common cause of primary (HPT) is parathyroid adenoma (PA). Hypercalcemia is responsible for the clinical presentations (Huang *et al.* 2007).The most common clinical features are nephrolithiasis, bone pain, arthralgias, muscular aches, peptic ulcer, pancreatitis, fatigue, depression, anxiety, and other mental disturbances (Tejwani *et al.* 2006). Vitamin D deficiency also is usually associated with primary HPT (Redman *et al.* 2009).

Studies previously have reported brown tumor cases related to primary or secondary HPT (Azria *et al.* 2006; Mustonen *et al.* 2004; Kaya *et al.* 2007). In these reports, the cause of myelopathy is spinal cord compression resulting from a brown tumor. In primary HPT, general malaise, muscular weakness, and musculoskeletal complaints without spinal cord compression have been reported before (Huang *et al.* 2007; Tejwani *et al.* 2006). We found only one report of primary HPT with tetraparesia in the literature (Heyman *et al.* 1986).

We aim to report this interesting case with the delayed diagnosis of primary HPT and concomitant Vitamin D deficiency due to PA presenting as tetraparesia, with common severe muscular atrophy as a complication.

CASE REPORT

A 30-year-old woman was admitted to our department with complaints of inability to walk for two years, anorexia, and general weakness. Her complaints had started 10 years before and increased gradually. Finally, the patient had been bedridden for two years. Except for this complaint, her medical history was unremarkable. The familiar history of HPT was not present in our patient. Her neurological examination revealed tetraparesia and the presence of bilateral indifferent plantar response. Bilateral severe muscular atrophy was also present. Laboratory investigations showed the following: CK: 37 IU/L (15–130), Ca: 11.5 mg/dL (9–10.5), P: 1.5 mg/dL (2.5–5), PTH: 736.1 pg/mL (15–65), ALP: 1029.5 lU (64–300), and 25-OHvit D: 4 ng/mL (11–43). Erythrocyte sedimentation rate was 30 mm/h. DEXA investigation showed T scores of –7.34 at the L1–4 region and –5.48 at the left femoral neck. Cervical MRI revealed central protrusion at the C3–4 and C4–5 regions. Collagen tissue disease markers were negative. Electromyelography (EMG) and thyroid scintigraphy were normal. 99mTc sestamibi parathyroid scintigraphy revealed PA. The patient was diagnosed as primary HPT due to PA. Vitamin D replacement was done for her Vitamin D deficiency, and then the patient underwent general surgery for removal of PA.

A year after her operation, the patient was examined in an outpatient clinic. Her clinical results had improved substantially and she could walk without help. Laboratory investigations showed Ca: 9.5 mg/dL (9–10.5), P: 3.1 mg/dL (2.5–5), PTH: 45.1 pg/mL (15–65), ALP: 28.3 lU (64–300), and 25-OHvit D: 26 ng/mL (11–43).

DISCUSSION

Calcium ion is one the most important minerals in neurotransmission, blood clotting, muscular contractions, and other metabolic events. There is a constant balance between calcium and phosphate in circulation. Increased calcium and decreased phosphate levels suggest primary HPT. The most common cause of primary HPT is PA and usually results in hypercalcemia (Hussain *et al.* 2008).

Mild and moderate elevations in calcium levels usually have nonspecific causes. Severe hypercalcemia can result in coma and cardiac arrest (Jouan *et al.* 2008). Long-term hypercalcemia can cause proximal muscular weakness. Complications of long-term hypercalcemia are uncommon today (Georges *et al.* 2008).

The clinical presentation of primary HPT is characterized by bone and renal diseases (Jouan *et al.* 2008; Georges *et al.* 2008). However, local neurological signs are rare. To our knowledge, there are only two reports of paraparesia in primary HPT. The first report suggested that paraparesia is caused not only by lithium-induced primary HPT, but also partly by lithium itself, which has peripheral neuropathic side effects at toxic levels (Leutgeb 1995). The second report revealed a 65-year-old female with paraparesis. After parathyroidectomy, all her symptoms completely resolved (Olukoga 1998). Heyman *et al.* (Heyman *et al.* 1986) reported a case of primary HPT presenting quadriplegia. In this case, all neurological deficits disappeared following the removal of a PA.

It is well-known that Vitamin D deficiency usually is associated with primary HPT (Redman *et al.* 2009). Vitamin D deficiency usually results from poor dietary Vitamin D intake and/or the lack of exposure to sunlight, similar to the present case. Parathyroid hormone, which is strongly linked with Vitamin D status, also might play a role in muscular function. Muscular weakness in Vitamin D deficiency can present with no specific pattern (Ceglia 2008). An interesting feature of our case is the presence of muscular weakness as tetraparesia.

Despite the current trend, some patients might still present late, with some exhibiting neurological manifestations of the primary HPT. In our case, we did not think that the tetraparesia was caused by the central protrusions at the C3–4 and C4–5 levels because there were no signs of spinal cord compression, such as increased deep tendon reflexes or the presence of an extensor plantar reflex. We also ruled out muscular and collagen tissue disorders because EMG investigation and collagen tissue disorders' markers were normal.

Bone involvement is frequent in patients with asymptomatic primary HPT (Bilezikian *et al.* 1991). Plasma 25-OHvit D levels also are reduced in severe primary HPT. In these cases, parathyroid sestamibi scanning is likely to show positive results for PA (Kandil *et al.* 2008). In our case, a patient with advanced primary HPT, bone involvement and reduced 25-OHvit D levels were present and the diagnosis of primary HPT was much delayed. However, a year after her operation and Vitamin D replacement, the patient could walk without help. Finally, we agreed that the cause of tetraparesia was muscular weakness resulting from long-term hypercalcemia and Vitamin D deficiency in our case.

In conclusion, the diagnosis of primary HPT is more likely delay, because general symptoms were anorexia, muscle weakness, and depression. We suggest that physicians always keep in mind primary HPT and concomitant Vitamin D deficiency in the differential diagnosis of hypercalcemia when facing atypical neurological symptoms such as tetraparesia.

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