



Case Report

Severe Weight Loss: An Unusual Cause

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ABSTRACT

Primary hyperparathyroidism is a relatively frequent condition that predominantly affects older women and is usually associated with asymptomatic mild to moderate chronic hypercalcemia. We report a case of hyperparathyroidism due to parathyroid adenoma leading to severe weight loss in a young male. It can be easily missed as patient had no other symptoms but once diagnosed it is easily treatable and complete recovery occurs over a period of time.

Key-words: Hyperparathyroidism, calcium, weight loss

INTRODUCTION

Primary Hyperparathyroidism often presents with unusual clinical manifestations. Inability to locate the adenoma in an ectopic parathyroid gland may delay the diagnosis of these cases. The main effects of parathyroid hormone (PTH) are to increase the concentration of plasma calcium by increasing the release of calcium and phosphate from bone matrix, increasing calcium reabsorption by the kidney, and increasing renal production of 1,25-dihydroxyvitamin D-3, which increases intestinal absorption of calcium. We present an unusual case of young patient with severe weight loss in the background of primary hyperparathyroidism and severe PTH elevation.

CASE HISTORY

24 years old male presented with severe anorexia, generalised weakness and loss of weight (around 20 kg) since last 6 weeks. On examination he had average built, BMI 22 kg/m², pulse 80/minute, blood pressure 150/100 mmHg, no thyromegaly but he had small lump on left side of neck. Other systemic examination was normal. Investigations showed Hb 11.0 gm/dl (14-18), WBC count 9000/mm³ (4000-11000), Platelets count 356000/mm³ (150000-400000), Total bilirubin 0.50 mg/dl (0.0-1.0), ALT 22 IU/L (0-40), Alkaline phosphatase 1692 IU/L (110-310), Serum Creatinine 1.5 mg/dl (0-1.4), Blood urea nitrogen 29 mg/dl (05-21), Serum uric acid 6.6 mg/dl (3.5-7.2), Serum Calcium 16.2

mg/dl (8.8-10.2), Serum Phosphorus 4.0 mg/dl (2.5-5.0), Serum Sodium 136 mEq/L (135-148), Serum Potassium 3.5 mEq/L (3.5-5.0), T4 3.9 mcg/dl (4.5-12.0), TSH 2.25 mIU/L (0.30-5.5), fasting blood sugar 71 mg/dl, ECG normal, Ultrasound abdomen nephrolithiasis. In view of severe hypercalcemia, patient further investigated and it showed Serum PTH >1900 pg/ml (14.0-72.0), Protein electrophoresis normal. Ultrasound neck showed large solid nodule posterior to right lobe of thyroid (Figure.1). Parathyroid adenoma suspected and patient undergone parathyroidectomy. Excised parathyroid section showed small parathyroid acini, cells containing small oval nuclei and clear cytoplasm with intact fibrous capsule (features suggestive of parathyroid adenoma). Serum calcium level decreased to 9.1 mg/dl and Serum PTH 70 pg/ml postoperatively. Patient discharged in stable condition after 5 days of surgery. One month after discharge, patient gained 10 kilogram weight and serum calcium, PTH remained normal.

DISCUSSION

Primary hyperparathyroidism is an endocrine condition causing metabolic bone disease characterized by hypercalcemia and diffuse bone resorption. A large number of cases are usually asymptomatic and are detected incidentally with hypercalcemia. ⁽¹⁾ In the clinically manifest disease, it is known to present with subtle and protean manifestations, leading to misdiagnosis in the early stages of the disease. ⁽²⁾ Excess PTH leads to involvement of skeletal system and the kidneys in majority of cases. Skeletal involvement is mainly as a result of increased bone resorption leading to characteristic manifestations like osteitis fibrosa cystica, subperiosteal resorption of distal phalanges, bone cysts and 'brown tumours'. Renal involvement is seen in more than 15% of cases of primary

hyperparathyroidism and is mainly due to hypercalciuria leading to nephrocalcinosis and nephrolithiasis. ⁽³⁾

Diagnosis of primary hyperparathyroidism in a clinically suspected case is suggested by hypercalcemia, hypophosphatemia, raised levels of bone specific alkaline phosphatase and raised PTH levels. Anterior neck mass may occasionally be palpable in a case of parathyroid tumour. Ultrasonography is a convenient and economical localizing modality and has an acceptable sensitivity and specificity of 73% and 100% respectively. ⁽⁴⁾ Fine needle aspiration cytology studies may help supplement the diagnosis in such cases. Surgical excision of the abnormal parathyroid glands offers the only permanent, curative treatment for primary hyperparathyroidism. ⁽⁵⁾ Treatment with a bisphosphonate such as alendronate can be considered in patients of primary hyperparathyroidism with low bone mineral density who cannot, or will not, undergo surgery. ⁽⁶⁾

CONCLUSION

Primary hyperparathyroidism, with its varied manifestations and indolent course, is a condition well known to pose a diagnostic dilemma to the clinician as seen in our patient who presented with severe weight loss at young age. An ectopic location of the parathyroid gland, albeit uncommon, may further complicate the issue. Once diagnosed, disease can be cured with simple surgery.

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