

# A Case of Persistent Hyperparathyroidism

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## Background

Hyperparathyroidism is the third most common endocrine disorder diagnosed in the US and one of the most common causes of hypercalcemia. Most cases are due to a single adenoma or hyperplasia of the glands. Aberrant production of parathyroid hormone (PTH) leads to loss of feedback system in the PTH-calcium loop and causes elevated levels of calcium in the blood via increased kidney, GI, and bone resorption. This can cause symptoms including osteoporosis, nephrolithiasis, GI upset, and mood changes. We present an unusual case of persistent hyperparathyroidism.

## Description of Case

JD is a 50 yo male who presented to new PCP to establish care. Initial laboratory studies were ordered and returned unremarkable, except for elevated calcium. Subsequent labs were drawn to investigate etiology and demonstrated elevated PTH and decreased 25-OH-Vitamin D. Review of systems (ROS) was positive for fatigue and headache, but the remainder of ROS was negative. Most likely diagnosis was primary hyperparathyroidism. Patient was referred to endocrinologist and ENT surgeon for removal of culprit parathyroid. Two surgeries failed to locate parathyroid adenoma or hyperplasia and laboratory evidence continued to demonstrate primary hyperparathyroidism. Patient was referred to tertiary medical center for evaluation. Specialized work-up failed to locate adenoma or hyperplastic tissue.

## Lab Evaluation

### Initial:

Calcium: 11.2 mg/dL

Intact PTH (Normal Values 14-71 pG/mL):  
119.6 pg/mL

25-OH-Vitamin D (Normal values 30.0-100.0  
ng/mL): 29.6 ng/mL

### Current:

Ca 10.9 mg/dL

PTH: 105 pg/mL

24-hour Urine Calcium: 420 mg

## Imaging

11/17/20 Sestamibi (Without SPECT-CT)

Negative

6/14/21- Sestamibi/SPECT-CT

Intense uptake in mid to inferior right thyroid

DEXA

Normal

1/28/22 Sestamibi (Without SPECT-CT)

Negative

8/16/22 4D Parathyroid Scan

Negative

9/13/22 Venous Sampling

Venography demonstrated left sided IVC, the internal jugular veins did not demonstrate distinct thyroid veins to sample. Due to abnormal anatomy, procedure was aborted prior to completion of sampling. Of sampling completed, parathyroid hormone ranged from 105-220 pG/mL. The 220 pG/mL collection was located in the mediastinum, indicating possible adenoma there.

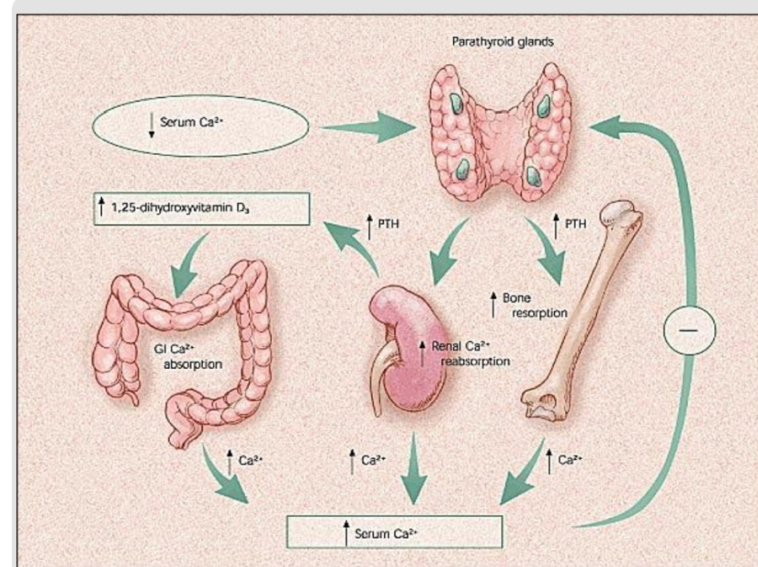


Figure 1. Parathyroid Hormone Feedback Loop

## Treatment

### Medications:

Vitamin D3 1,000u PO QD

3/25/21 Parathyroid Exploration, Left thyroid lobectomy

Left lower parathyroid not located, the others had normal appearance. Left thyroid lobectomy performed due to missing parathyroid in which biopsy demonstrated no parathyroid tissue.

Biopsy Parathyroid glands: Normocellular parathyroid tissue

Post-op PTH: 162

7/29/21 Parathyroid exploration, right thyroid lobectomy

Ectopic tissue, possibly ectopic parathyroid, near cricoid cartilage. This tissue was dissected and demonstrated probable hypercellular parathyroid tissue. Right thyroid excised and benign.

Post-op labs: PTH 130

## Discussion

Hyperparathyroidism, although often asymptomatic, can have injurious effects on the body, including osteopenia to osteitis fibrosa cystica and fragility fractures to kidney failure. When left unmanaged, these outcomes can be devastating and have significant morbidity and mortality. Most cases of hyperparathyroidism occur due to single adenoma or area of hyperplasia and surgical excision will cure the illness. However, in our case, exhaustive diagnostics have been done and the causative agent has yet to be found, therefore classifying as persistent hyperparathyroidism. Currently, our patient's final management is uncertain due to the unclear etiology of aberrant parathyroid hormone production. Familial syndromes have been ruled out. Our patient continues to undergo specialized workup.

## Conclusion

It is important to thoroughly evaluate patients with primary hyperparathyroidism to avoid devastating manifestations including bone loss, fractures, and kidney disease. This is especially true in patients of younger age without documented bone or kidney disease to prevent progression with age. Therefore, providers must be vigilant in detecting and pursuing primary hyperparathyroidism.

## Acknowledgements

Fig. 1- Hartsock, M. (2004). *Hyperparathyroidism*. American Family Physician.