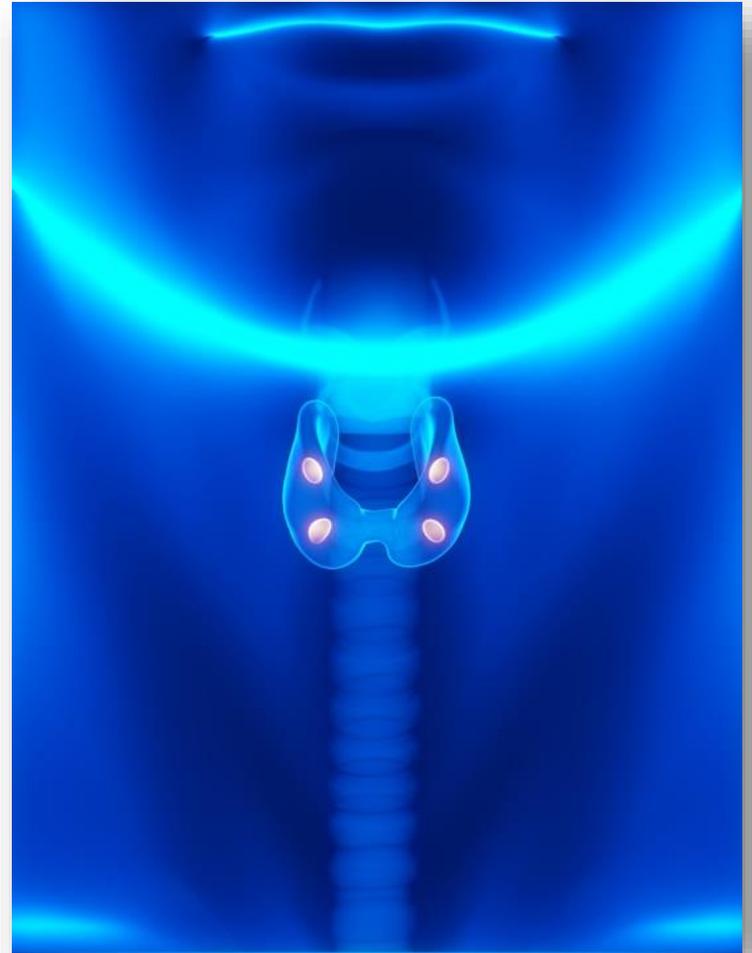


# Hyperparathyroidism



# Parathyroid hormone (PTH)

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The main actions of PTH are:

- Bone resorption
- Tubular calcium reabsorption and phosphate excretion in the kidney
- Formation of calcitriol (1,25 dihydroxy vitamin D) which increases gastrointestinal calcium absorption
  - Calcium and phosphate homeostasis



# Hyperparathyroidism types

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- **Primary**- Most often caused by parathyroid adenoma, causing elevated parathyroid hormone (PTH) and serum calcium.
- **Secondary**- most often due to Chronic Kidney Disease (CKD), especially with an estimated glomerular filtration rate (eGFR) below 60.
  - Can also be caused by vitamin D deficiency, poor PO calcium intake or gastrointestinal loss (i.e. malabsorption).
- **Tertiary**- due to autonomous production of PTH in setting of longstanding CKD.

# Normocalcemic primary hyperparathyroidism

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In **normocalcemic primary hyperparathyroidism** (PHPT), levels are elevated but serum calcium is normal.

In order to make this diagnosis, certain conditions must be met:

- In particular, all secondary causes for hyperparathyroidism must be ruled out, and ionized calcium levels should be normal.
- The most common explanation for the finding of an elevated PTH and normal serum calcium remains concomitant hypercalcemic primary hyperparathyroidism and vitamin D deficiency.

# Secondary hyperparathyroidism

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**Secondary hyperparathyroidism** (SHPT) occurs when the parathyroid gland appropriately responds to a reduced level of extracellular calcium.

- PTH concentrations rise, and calcium is mobilized by increasing intestinal absorption (via increase in calcitriol) and by increasing bone resorption.
- Thus, it is characterized biochemically by elevated PTH and normal or low serum calcium concentrations.

# Secondary hyperparathyroidism

- May occur in patients with renal failure and impaired calcitriol (1,25 dihydroxy vitamin D) production.
- Also in individuals with inadequate calcium intake or absorption.
- Can occur with vitamin D deficiency or with gastrointestinal diseases causing malabsorption.
- Assessment of renal function (serum creatinine), vitamin D status (25-hydroxyvitamin D, 25OHD), and calcium sufficiency (urinary calcium excretion) may help differentiate normocalcemic primary and secondary hyperparathyroidism.

# Secondary hyperparathyroidism due to CKD

- This is most common cause of SHPT
- Estimated prevalence in US of unrecognized/undertreated SHPT ranges from 2 to 5 million patients
- SHPT physiology starts prior to Stage III CKD, but PTH rarely starts rising prior to eGFR below 60
- Initially, elevation of PTH is appropriate, leading to calcium homeostasis (more calcium absorption and phosphate excretion)
- Adverse effects in the long-term include bone disease and pathologic calcification of tissues, including vasculature

# Pathophysiology of SHPT in CKD

CKD causes phosphorus retention and increased fibroblasts growth factor 23

Decline in 1,25 dihydroxy vitamin D (calcitriol)

Reduced levels of available calcium

Increasing PTH (appropriately maintains calcium homeostasis initially)

Can lead to osteitis fibrosa cystica, adynamic bone disease and vascular calcifications

# Testing frequency for SHPT

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**Frequency of checking intact parathyroid hormone (iPTH), calcium (Ca), and phosphorous (Phos)**

- **CKD 2- yearly**
- **CKD 3- every 6 months**
- **CKD 4- every 3 months**
- **CKD 5- every 3 month**

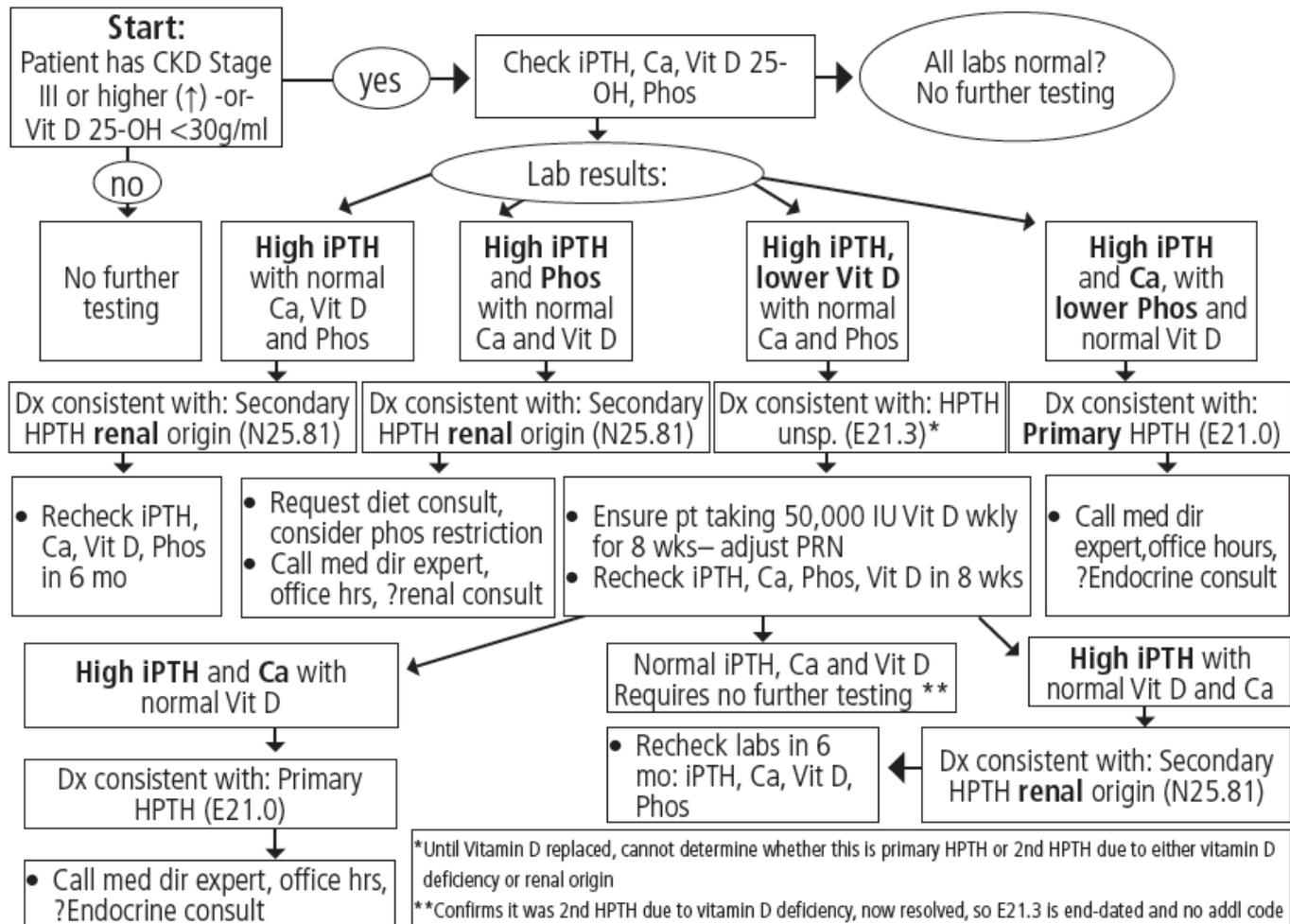
# Management and control of SHPT

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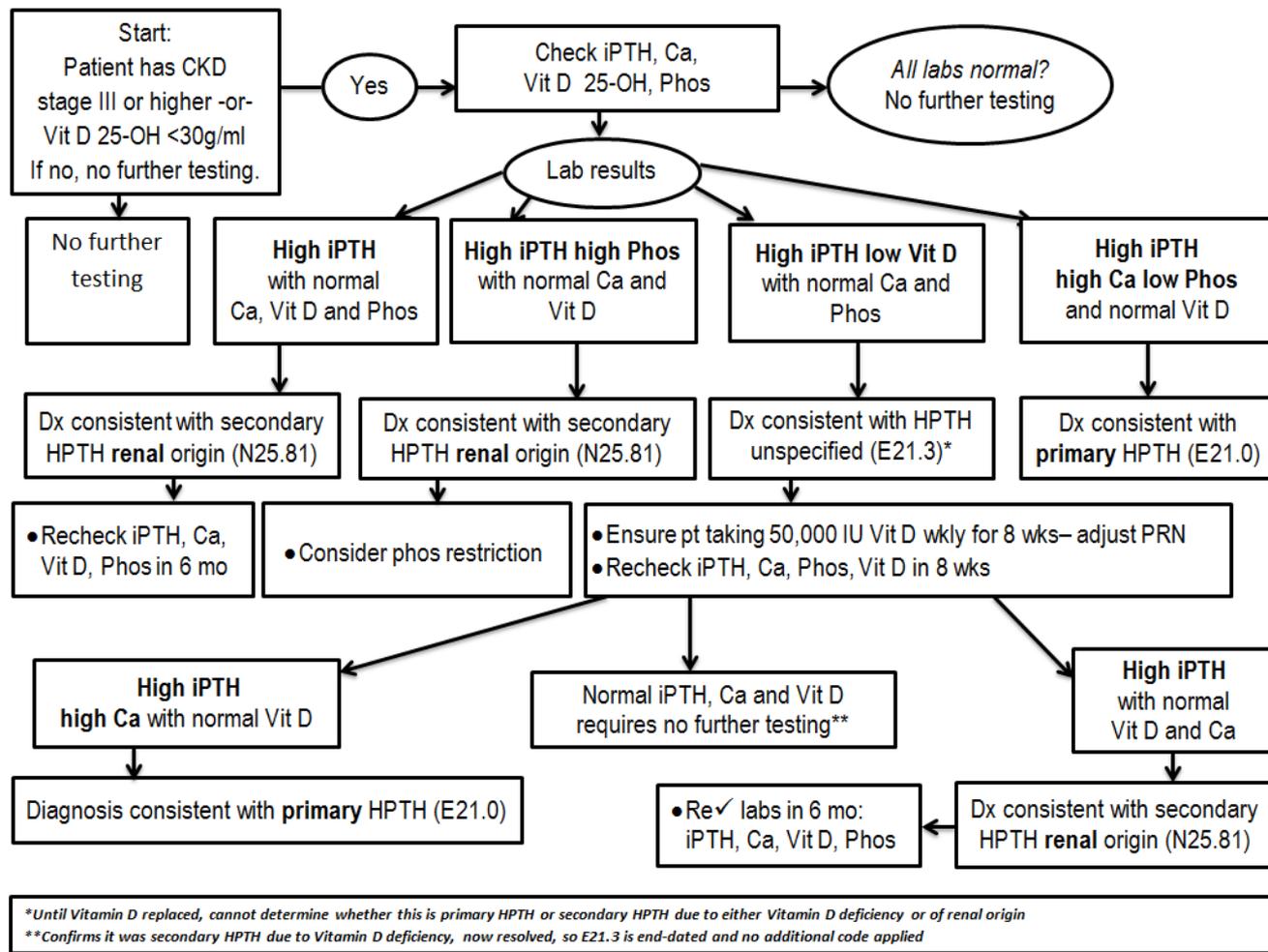
**Goal iPTH levels  
after SHPT  
diagnosis**

- **CKD 2: 35-70**
- **CKD 3: 35-70**
- **CKD 4: 70-110**
- **CKD 5: 200-300**

# CCM - secondary hyperparathyroidism decision tree



# Secondary hyperparathyroidism decision tree

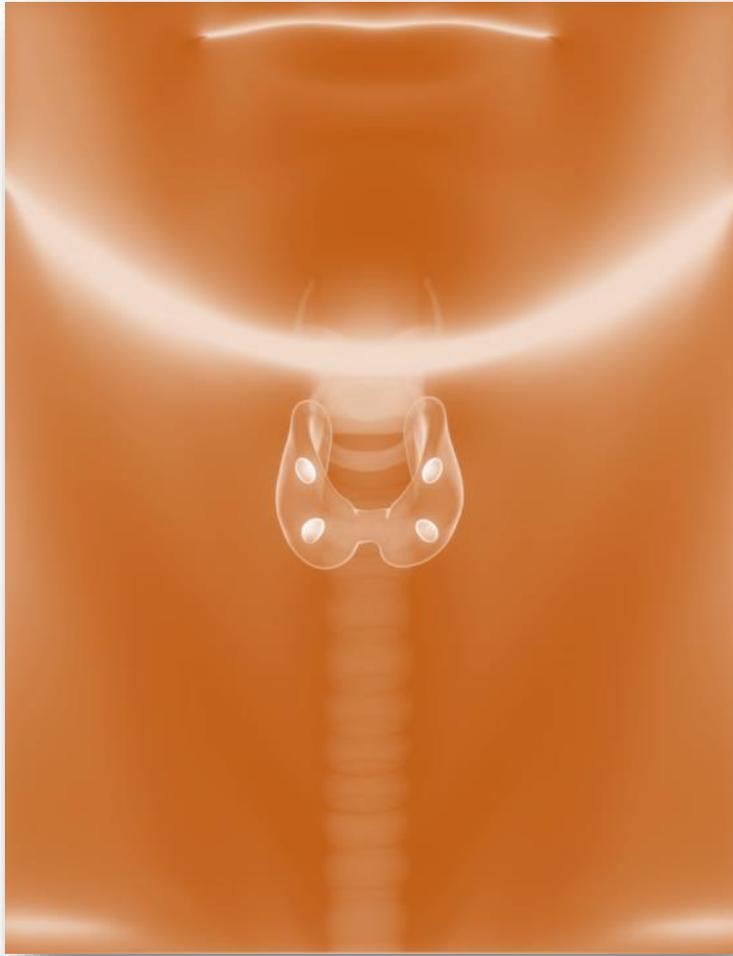


# Key points

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- In your patients with CKD, consider screening for secondary hyperparathyroidism (order vitamin D testing as well), especially in stage III or greater CKD patients.
- Treatment usually involves laboratory monitoring and possible dietary changes such as phosphorous restriction
  - Foods with higher phosphorous include most dairy, whole grains, peas, beans, processed meats, nuts, seeds, chocolate





ICD-10 and  
required  
specificity

# Parathyroidism disease codes

ICD-10-CM	Description
<b>E21.0</b>	Primary hyperparathyroidism
<b>E21.1</b>	Secondary hyperparathyroidism, NEC (excludes of renal origin)
<b>E21.2</b>	Other (tertiary) hyperparathyroidism
<b>E21.3</b>	Hyperparathyroidism NOS
<b>E21.4</b>	Other specified disorders of parathyroid gland
<b>E21.5</b>	Parathyroid disorder, unspecified
<b>N25.81</b>	Secondary hyperparathyroidism of renal origin

# Resources

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- International Classification of Diseases, Tenth Revision, Clinical Modification (ICD-10-CM). (2017, February 2). Retrieved January 1, 2017, from CDC/National Center for Health Statistics website: <https://www.cdc.gov/nchs/icd/icd10cm.htm>
- Quarles, L.D., & Berkoben, M. (2017, January 6). Management of secondary hyperparathyroidism and mineral metabolism abnormalities in adult predialysis patients with chronic kidney disease. Retrieved April 12, 2017, from UpToDate website: <https://www.uptodate.com/contents/management-of-secondary-hyperparathyroidism-and-mineral-metabolism-abnormalities-in-adult-predialysis-patients-with-chronic-kidneydisease>
- Secondary hyperparathyroidism. (2017, March 17). Retrieved April 12, 2017, from The National Kidney Foundation website: <https://www.kidney.org/atoz/content/secondary-hyperparathyroidism>