

Hypercalcemia

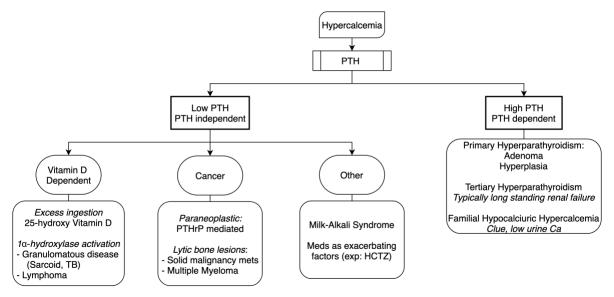
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- 1. Calcium Physiology (reference range typically 8.6 10.2)
 - Total Ca = ionized (free & regulated by PTH) + protein bound
 - PTH increases calcium by acting on:
 - o Bones: increases bone resorption by osteoclasts
 - \circ Kidney: increases Ca reabsorption + increases PO₄ excretion, increases activity of 1α -hydroxylase which is the enzyme that activates vitamin D
 - o GI: indirect increase in Ca absorption via vitamin D activation
- 2. DDx: Also see Penn Frameworks and CPSolvers schema

Outpatient: primary hyperparathyroidism = most common. Inpatient: think malignancy



- 3. History: Pearl: typically, don't get any symptoms until Ca > \approx 11.5, severe symptoms at > \approx 13
 - Bones (pain, fragility), stones (nephrolithiasis), groans (abdominal pain, constipation), psychiatric overtones (fatigue, cognitive dysfunction, depression).
 - Polyuria/Polydipsia: due to a nephrogenic DI like state

4. Evaluation:

- 1st: Repeat test and <u>calculate corrected Ca</u> for albumin level. *Note:* ionized calcium has rapid sample processing requirements
- Initial workup: PTH, PO₄, Albumin, 25-hydroxyvitamin D Pearl: <u>Chloride/PO4 ≥ 33</u> is suggestive of a PTH-mediated process
- If PTH suppressed (appropriate): PTHrP, calcitriol (1,25-dihydroxyvitamin D), lytic lesions (SPEP, bone imaging), granulomatous disease (CXR, ACE levels)
- If PTH elevated: consider FHH vs. primary Hyperparathyroidism. Imaging of neck is useful if patient is surgical candidate

5. Management:

- Indications for surgery: symptomatic, criteria from American Association of Endocrine Surgeons
- 1/3 of patients initially not meeting surgical criteria will develop an indication in the next 10 years. Follow-up is key!
- Medical management: Fluid intake, avoid excess Ca intake, avoid contributing meds
 - For poor surgical candidates: cinacalcet (calcimimetic), bisphosphonates, denosumab
 - Severe: Ca > ≈14 consider IVF, bisphosphonates, calcitonin, denosumab