Hypocalcemia & Hypoparathyroidism

Confirm it, then let the PTH split the differential · and check the magnesium before calling it refractory

Check the Mg
every time

CONFIRM IT FIRST *a large share of "hypocalcemia" is an artifact of low albumin*

CORRECT FOR ALBUMIN

Corrected Ca = measured Ca + 0.8 × (4.0 – albumin g/dL). Roughly half of serum calcium is protein bound, so a hypoalbuminemic inpatient can look hypocalcemic with a **normal ionized calcium and no disease at all**.

IONIZED IS DEFINITIVE

Measure ionized calcium when the albumin is abnormal, in critical illness, in **acid-base disturbance**, or after **massive transfusion**. **Alkalosis increases protein binding** and lowers ionized calcium at an unchanged total, which is why hyperventilation produces tetany.

THEN GRADE IT

Severity is driven by **how fast it fell**, not only the number. A rapid drop after thyroidectomy is symptomatic at a level a patient with chronic hypoparathyroidism tolerates comfortably.

RECOGNIZE IT *neuromuscular irritability, and one ECG change*

Sign	Detail
Paresthesia	**Perioral and fingertip tingling** is usually the earliest symptom, then cramps and carpopedal spasm
Trousseau sign	Carpal spasm after inflating a blood pressure cuff above systolic for **3 minutes**. **More specific** than Chvostek
Chvostek sign	Facial twitch on tapping the facial nerve. **Present in up to about 10% of normal people**, so a positive result on its own proves little
ECG: prolonged QT	The change that matters, because it carries a risk of **torsades**. **Get an ECG in any severe or symptomatic hypocalcemia**
Severe and late	**Laryngospasm, bronchospasm, seizures, and hypotension with reduced cardiac contractility.** These are the emergency presentations

THE PTH SPLITS THE DIFFERENTIAL *one test, two branches*

PTH result	What it means, and the usual causes
PTH LOW or inappropriately normal *hypoparathyroidism*	**The gland is the problem. Post-surgical is by far the commonest** (thyroidectomy, parathyroidectomy, neck dissection), then autoimmune, infiltration, radiation, and congenital causes such as **DiGeorge**. **Magnesium depletion belongs here too**: it causes functional hypoparathyroidism by impairing both PTH secretion and PTH action
PTH HIGH *appropriate response*	**The gland is working; something else is driving calcium down. Vitamin D deficiency**, **chronic kidney disease** (reduced 1-alpha hydroxylation plus phosphate retention), **acute pancreatitis** (saponification), **tumor lysis** and **rhabdomyolysis** (phosphate binding), **citrate** from massive transfusion or apheresis, **hungry bone syndrome** after parathyroidectomy, and drugs including **bisphosphonates and denosumab**. **Pseudohypoparathyroidism** is PTH resistance: high PTH with low calcium and a characteristic phenotype

NOW READ THE PHOSPHATE *it separates the two branches further, at no extra cost*

LOW Ca + HIGH PO_4

Points to **hypoparathyroidism** (no PTH means phosphate is retained) or **CKD**, and to the acute phosphate loads: **tumor lysis and rhabdomyolysis**. **Here you treat the phosphate as well**, and give calcium only for symptoms.

LOW Ca + LOW PO_4

Points to **vitamin D deficiency or malabsorption**, where secondary hyperparathyroidism is driving phosphate out in the urine. Also **hungry bone syndrome**, where calcium *and* phosphate are consumed by remineralizing bone.

ALSO CHECK

Magnesium in everyone, **25-hydroxy vitamin D**, creatinine, and **alkaline phosphatase**, which is high in vitamin D deficiency and in hungry bone but normal in uncomplicated hypoparathyroidism.

TREATMENT *route decided by symptoms, not by the number alone*

Situation	What to give
Symptomatic or severe *tetany, seizure, long QT*	**IV calcium gluconate**, typically 1 to 2 g diluted, infused over 10 to 20 minutes with cardiac monitoring, then an infusion if it recurs. **Prefer gluconate to calcium chloride peripherally**: chloride has three times the elemental calcium but is far more sclerosant and **causes tissue necrosis if it extravasates**, so reserve it for central access in arrest
Always, alongside	**Replace magnesium**, and do it first or simultaneously. **Calcium will not correct while the magnesium is low, no matter how much you give**: this is the single commonest reason hypocalcemia looks refractory
Chronic hypoparathyroidism	**Oral calcium plus an ACTIVE vitamin D analogue (calcitriol or alfacalcidol)**. Plain vitamin D is not sufficient, because **PTH is required for 1-alpha hydroxylation** and the patient cannot activate it
Autosomal dominant hypocalcemia *activating CaSR mutation*	A trap worth knowing: the calcium-sensing receptor is **over-active**, so the body defends a low calcium on purpose. **Treating to a normal calcium causes marked hypercalciuria and nephrocalcinosis**. Suspect it with hypocalcemia, low or normal PTH and a **high urine calcium**, and treat only symptoms
The target is deliberately low	Aim for a calcium at the **low end of normal** with the patient symptom free. Normalizing it fully causes **hypercalciuria, nephrolithiasis and nephrocalcinosis**, because without PTH the kidney cannot reabsorb the filtered calcium. **Monitor urine calcium, not only serum**

PEARLS & PITFALLS

- **Check magnesium in every case, and treat it before concluding anything is refractory.** It is the fastest fix available and the most commonly overlooked.
- **Give calcium and phosphate binders, not calcium alone, in hyperphosphatemic hypocalcemia** (tumor lysis, rhabdomyolysis). Giving calcium into a high phosphate **precipitates calcium phosphate in tissues**, so treat only symptomatic hypocalcemia there.
- **Post-thyroidectomy calcium is a scheduled check, not a symptom-driven one.** Hypocalcemia typically appears within 24 to 72 hours, and **hungry bone syndrome** after parathyroidectomy for severe hyperparathyroidism can be profound and prolonged, needing large sustained replacement.
- **Correct the calcium before blaming the QT on a drug**, and remember that in citrate-based apheresis or massive transfusion the patient is not calcium deplete, the calcium is chelated, so it returns once the citrate is metabolized.

References: Endocrine Society and international consensus guidance on the management of hypoparathyroidism · standard references on calcium and magnesium disorders.