

HYPOCALCEMIA

Hypocalcemia is defined as

Gestation	Total serum calcium	Ionic serum calcium
Preterm	< 7 mg/dL (1.75 mmol/L)	<4 mg/dL (1.0 mmol/L)
Term	< 8 mg/dL (2 mmol/L)	<4.8 mg/dL (1.2 mmol/L)

Clinical presentation

Asymptomatic: ENH is usually asymptomatic LNH and is incidentally detected .

Symptomatic: Neuromuscular: irritability – myoclonic jerks, jitteriness, exaggerated startle and seizures .

Cardiac: Tachycardia, heart failure, prolonged qt interval and decreased contractibility (More often non specific and not related to the severity of hypocalcemia.

Apnea, cyanosis, tachypnea, vomiting and laryngospasm are other symptoms that are noted.

Treatment of early onset hypocalcemia (ENH)

Asymptomatic hypocalcemia:

- 80 mg/kg/ day of elemental calcium (8 mL/kg/ day of 10% calcium gluconate; 1 mL= 9.4 mg of elemental calcium) for 48 hours.
- This may be tapered to 50% dose for another 24 hours and then discontinued.

Neonates tolerating oral feeds may be treated with oral calcium (IV preparation may be used orally).

Symptomatic hypocalcemia:

- A bolus dose of 2 mL/kg diluted 1:1 with 5% dextrose over 10 minutes under cardiac monitoring.

- When there is severe hypocalcaemia with poor cardiac function, calcium chloride 20 mg/ kg may be given through a central line over 10-30 minutes.
- Followed by continuous IV infusion of 80 mg/kg/day elemental calcium for 48 hours.
- Continuous infusion is preferred to IV bolus doses (1 mL/kg/ dose q 6 hourly). Calcium infusion should be dropped to 50% of the original dose for the next 24 hours and then discontinued.
- Normal calcium values should be documented at 48 hours before weaning the infusion. The infusion may be replaced with oral calcium therapy on the last day.

Late Onset Hypocalcemia(LNH) Causes and Treatment:-

Causes:

1. Increased phosphate load : cow milk , renal insufficiency
2. Hypomagnesemia
3. Vitamin D def
4. Maternal vit d def
5. Malabsorption
6. Hepatobiliary disease
7. PTH resistance
8. Transient neonatal pseudo hypoparathyroidism
9. Hypoparathyroidism(Primary and secondary)
10. Autosomal dominant hypocalcemic hypercalciuria
11. Iatrogenic

It usually presents at the end of first week of life

The initial treatment of LNH is same as that of ENH. This should be followed by specific management according to the etiology and may be life-long in certain diseases.

Investigations required in infants with persistent / late onset hypocalcaemia

First line	Second line
Serum phosphate Serum alkaline phosphatase Liver function tests Renal function tests X ray chest/ wrist Arterial pH	Serum magnesium (Mg) Serum parathormone 25-hydroxyvitamin D Urine calcium creatinine ratio Maternal calcium, phosphate, alkaline phosphatase

Interpretation of investigations

Disorder causing hypocalcaemia	Findings
Hypoparathyroidism	High: phosphate Low: SAP, PTH, 25-OH D
Pseudo-hypoparathyroidism	High: SAP, PTH, Phosphate Low: 25-OH D
Chronic renal failure	High: phosphate, SAP, PTH deranged RFT Low: 25-OH D, pH (acidotic)
Hypomagnesemia	High: PTH Low: phosphate, Mg, 25-OH D
VDDR*I	High: SAP, PTH Low: Phosphate, 25-OH D
VDDR*II	High: SAP, 25-OH D, PTH Low: Phosphate

1. **Hypomagnesemia:** Symptomatic hypocalcemia unresponsive to adequate doses of IV calcium therapy is usually due to hypomagnesemia.

It may present either as ENH or LNH.

The neonate should receive two doses of 0.2 mL/kg of 50% MgSO₄ injection, 12 hours apart, deep IM followed by a maintenance dose of 0.2 mL/kg/ day of 50% MgSO₄, orally for 3 days.

2. **High phosphate load:** These infants have hyperphosphatemia with near-normal calcium levels. This results from feeding of animal milk which contain high phosphate load (e.g. cow's milk). Exclusive breastfeeding should be encouraged and animal milk should be discontinued, if possible. Phosphate binding gels must be avoided

3. **Hypoparathyroidism:** These infants have high phosphate and low calcium levels in the blood and *normal renal function*. High phosphate levels in the absence of high phosphate intake (cow's milk) and normal renal functions indicate towards hypoparathyroidism.

If phosphate level is high, addition of calcium may lead to calcium deposition and tissue damage. Thus, reduction in the phosphate level must be attempted in order to keep the calcium and phosphate product (Ca x P) less than 55. These neonates should be supplemented with calcium (50 mg/kg/ day in 3 divided doses) and 1,25 (OH), vitamin D. Therapy may be stopped in hypocalcemia secondary to maternal hyperparathyroidism after 6 weeks.

4. **Vitamin D deficiency states:** These babies have hypocalcemia associated with hypophosphatemia due to an intact parathormone response on the kidneys. They benefit from 1, 25 (OH), vitamin D, supplementation in a dose of 30- 60 ng/kg/ day.