***HYPOCALCAEMIA DR.Methaq A.M.Mhussein***

***CAUSES***

***1.HYPOPARATHYRODISM***

***2.vitamin D def.***

***3.chronic renal failure***

***4.acute pancreatitis***

***5.resp.alkalosis***

***6.metabolic alkalosis***

***7.hypoalbuminamia***

***8.malabsorption***

***9.hypomagnacamia:*** *magnesium depletion can cause hypocalcemia by producing PTH resistance, which occurs when serum magnesium concentrations fall below 0.8 meq/L (1 mg/dL or 0.4 mmol/L) or by decreasing PTH secretion, which occurs in patients with more severe hypomagnesemia*

*10****. DRUGS*** *:Calcium chelators ,,Bisphosphonates, Cinacalcet ,Chemotherapy, Foscarnet ,Fluoride poisoning.*

*11****. PSEUDOHYPOCALCEMIA****— Two of the commercially available forms of gadolinium-based contrast agents (used in magnetic resonance angiography), gadodiamide and gadoversetamide, may interfere with the colorimetric assays for calcium that are frequently used in hospital laboratories*

***Clinical feature***

*The hallmark of acute hypocalcemia is tetany, which is characterized by neuromuscular irritability. The symptoms of tetany may be mild (peri-oral numbness, paresthesias of the hands and feet, muscle cramps) or severe (carpopedal spasm, laryngospasm, and focal or generalized seizures, which must be distinguished from the generalized tonic muscle contractions that occur in severe tetany). Other patients have less specific symptoms such as fatigue, hyperirritability, anxiety, and depression, and some patients, even with severe hypocalcemia, have no neuromuscular symptoms*

 *S o, Tetany signs (****Trousseau's sign ,Chvostek's sign*** *) , other symptoms of hypocalcamia ,Seizures, Cardiovascular Papilledema ,Psychiatric manifestations*

***Mangment***

*1.treatment of resp.alkalosis by rebreathing expired air.*

*2.calcium gluconate slow i.v or i.m*

*3.Mg may be required.*

*4.active form of vitamin D in CRF.*

*5.treatment of underlying cause.*

***\*HYPERPHOSPHATAEMIA***

***CAUSES:***

*1.Acute and chronic renal failure*

*2.Rhabdomyolysis*

*3.acute haemolysis*

*4.tumour lysis syndrome*

***Clinical feature***

*1.pruritus 2.metastatic calcification 3.features of sec.hyperparathyrodism 4.features of underlying causues*

***Treatment DR.Methaq A.M.Hussein 2010***

*1.oral phosphate binders.*

*2.Dialysis.*

*3.Treatment of underlying cause.*

***\*hypophosphataemia caused by 3 mechanisms:***

***1.Internal redistribution***

 *Increased insulin secretion, particularly during refeeding*

 *Acute respiratory alkalosis*

 *Hungry bone syndrome*

***2.Decreased intestinal absorption***

 *Inadequate intake*

 *Antacids containing aluminum or magnesium*

 *Steatorrhea and chronic diarrhea*

 *Vitamin D deficiency or resistance*

***3.Increased urinary excretion***

 *Primary hyperparathyroidism*

 *Vitamin D deficiency or resistance*

 *Hereditary hypophosphatemic rickets*

 *Oncogenic osteomalacia*

 *Fanconi syndrome*

***Clinical features***  *th e manifestations depend in large part upon the severity and chronicity of the phosphate depletion, with the plasma phosphate concentration usually being below 1.0 mg/dL (0.32 mmol/L) in symptomatic patients****.***

 *The symptoms of hypophosphatemia are due to two consequences of intracellular phosphate depletion which impact virtually all organ system s* ***1.: Red cell 2,3-DPG (diphosphoglycerate) levels fall****, thereby increasing the affinity of hemoglobin for oxygen and reducing oxygen release at the tissue level.* ***2.Intracellular ATP levels fall*** *with severe hypophosphatemia and those cell functions dependent upon energy-rich phosphate compounds begin to fail.*

*So the clinical features:*

*-muscle weakness,-sever dyspnea,-cardiac arrythmia,-confusion and convulsion and coma,-haemolysis.*

***Treatment***

*1.Diet e.g milk .*

*2.oral phosphate suplement.*

*3.i.v theray not exceed 18 mmol/day*

***Hypermagnesaemia***

***Causes:***

1. *Renal failure 2.Magnesium infusion 3.Oral ingestion 4.Magnesium enemas 5. Miscellaneous such theopheline tox,and milk-alkali syndrome.*

***Clinical features Dr.Methaq A.M.Hussein***

 approximate relation between clinical manifestations and the degree of hypermagnesemia can be summarized as follows **1.plasma Mg concentration 4 to 6 meq/L (4.8 to 7.2 mg/dL or 2 to 3 mmol/L) —** nausea, flushing, headache, lethargy, drowsiness, and diminished deep tendon reflexes **.2. Plasma magnesium concentration 6 to 10 meq/L (7.2 to 12 mg/dL or 3 to 5** mmol/L) — somnolence, hypocalcemia, absent deep tendon reflexes, hypotension, bradycardia, and ECG changes **3.. Plasma magnesium concentration above 10 meq/L (12 mg/dL or 5 mmol/L) —** muscle paralysis, respiratory paralysis, complete heart block, and cardiac arrest. In most cases, respiratory failure precedes cardiac collapse.

**Treattment:**treatment of underlying cause

**Hypomagnesaemia**

**1.GASTROINTESTINAL LOSSES 2.RENAL LOSSES** Loop and thiazide-type diuretics ,Volume expansion ,Alcohol ,Hypercalcemia, Nephrotoxins ,Loop of Henle or distal tubule dysfunction ,Primary renal magnesium wasting like( Gitelman's syndrome ,Paracellin-1 mutation ,Na-K-ATPase mutation EGF gene mutation **3.MISCELLANEOUS**  foscarnet ,chronic use of omeprazole ,"hungry bone" syndrome **,**diabetes mellitus,

**Clinical feature**

 Hypomagnesemia is a common problem, occurring in nearly 12 percent of hospitalized patients. A higher incidence, as much as 60 to 65 percent, has been found among intensive care unit patients.Symptomatic magnesium depletion is often associated with multiple biochemical abnormalities such as hypokalemia, hypocalcemia, and metabolic alkalosis. As a result, it is often difficult to ascribe specific clinical manifestations solely to hypomagnesemia.

 tetany, positive Chvostek and Trousseau signs, and generalized convulsions . In addition, apathy, delirium and coma may occur.

**Treatment**:i.v Mg infusion slowly.