APPROACH TO HYPOMAGNESEMIA IN CHILDREN

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FORGOTTEN CATION

- Magnesium deficiency is relatively common among hospitalized children.
- Might remain asymptomatic despite low levels.
- Symptoms are non specific.
- Masked by symptoms of other co morbidities and overlooked.
- Magnesium is usually not a part of many basic metabolic panels
- Magnesium levels can be normal despite a marked decrease in total body magnesium content

CASE SCENARIO

- 2-year-old girl, 2nd born to 2nd degree consanguineous marriage
- Evaluated elsewhere, for complaints of increased frequency of micturition and frequent episodes of vomiting - past 2 months.
- History of death of elder sibling due to renal failure at the age of 8 years present, other details were not available.
- Work up for urosepsis was negative.
- USG abdomen done revealed nephrocalcinosis
- Child referred to tertiary care center for further evaluation.

CASE SCENARIO

- Weight- 9.5 kg between (3rd to 15th centile)
- Length - 85 cm (50th centile)
- Weight for height -on 3rd centile

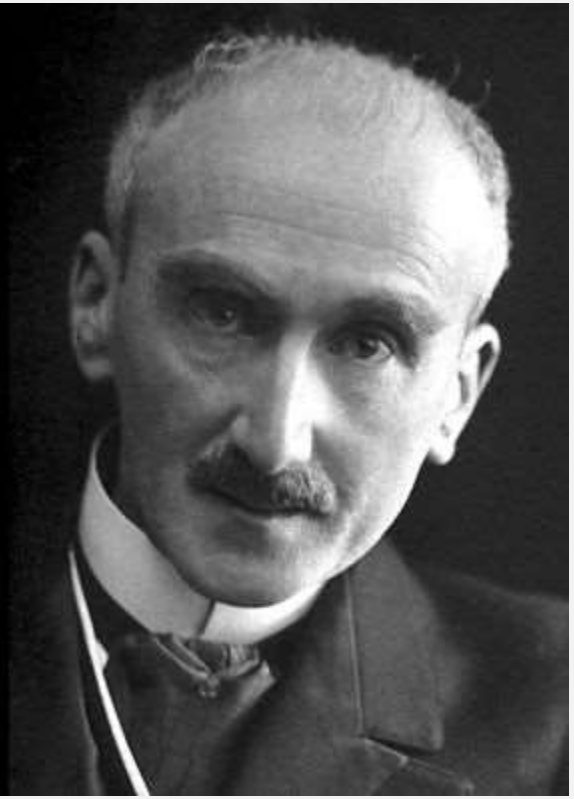
- Basic evaluation revealed the following.
- Bl urea 24 mg/dl
- Sr creatinine 0.6 mg/dl
- Sr sodium 141 Meq/l
- Sr potassium 4.1 Meq/l
- Sr calcium 9.8 mg/dl
- Sr phosphorus 4.1 mg/dl
- Sr vitamin D 24 ng/ml
- Sr PTH 570 pg/ml
- Sr Magnesium 0.8 mg dl
- Sr uric acid 4.5 mg/dl

QUESTIONS

- What additional investigations are needed at present?
- What is the step wise approach to evaluate for the cause of Hypomagnesemia?
- What will be the treatment for acute symptomatic and chronic hypomagnesemia?

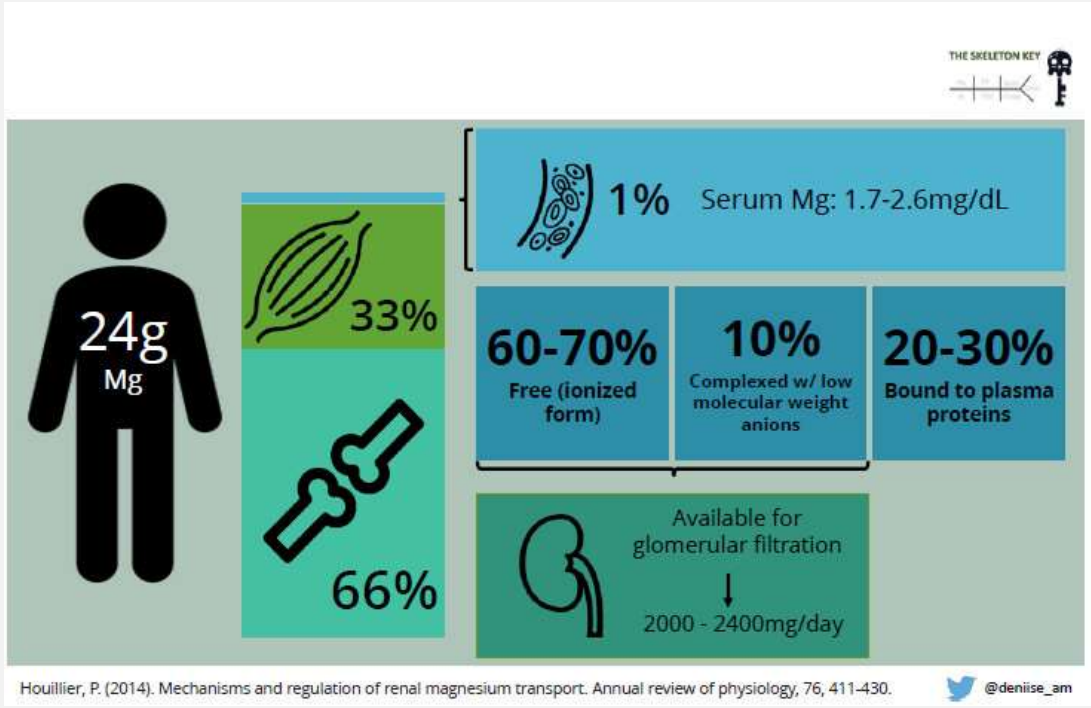
“The eye sees only what the mind is prepared to comprehend”

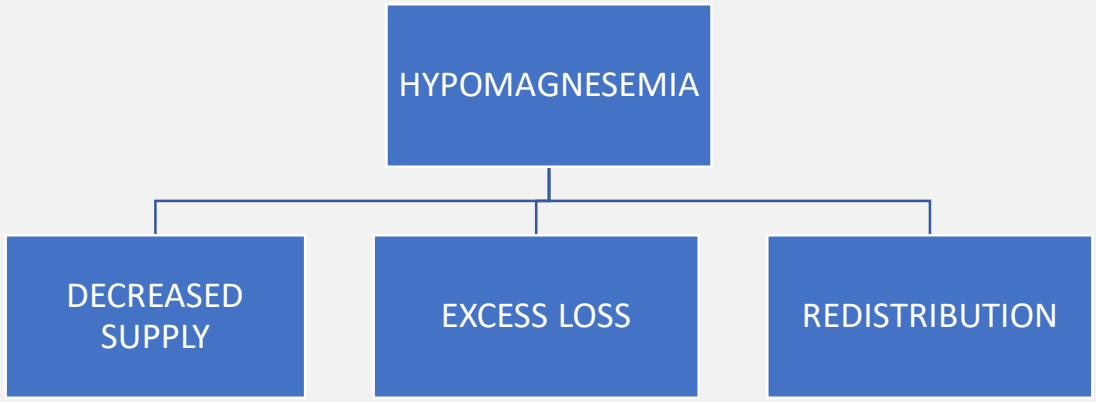
Henri Bergson
French philosopher,
Nobel Prize in Literature



HYPOMAGNESEMIA

- Normal magnesium level= 1.5- 2.7 mg/dl
- Fourth most common cation in the body.
- Only 1% of body magnesium is extracellular,(60%-ionized,15% complexed,25% protein bound)
- Magnesium is stored mainly in bones, but also in liver and muscles.
- Magnesium levels determined by intake, excretion and redistribution



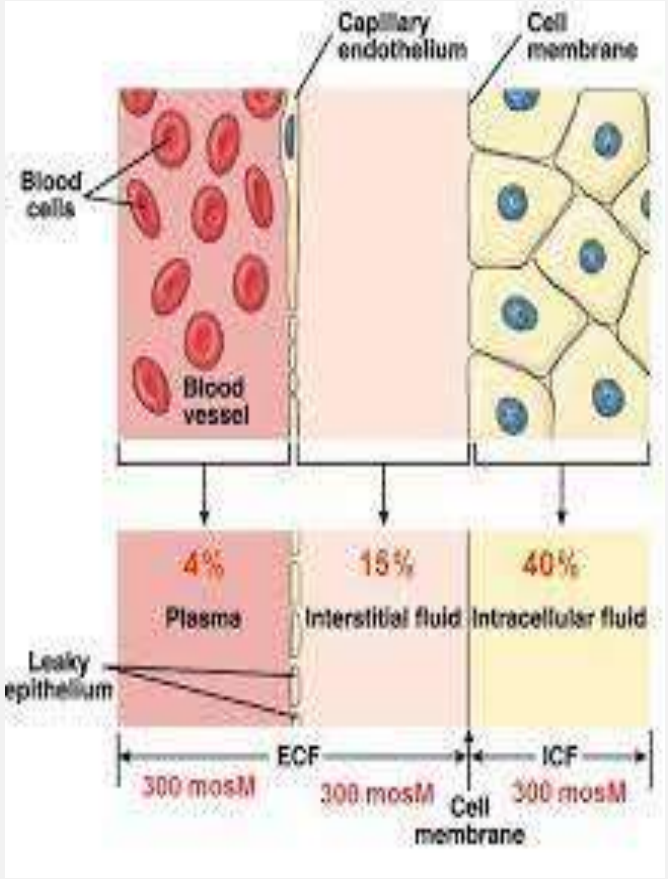

HYPOMAGNESEMIA
DECREASED SUPPLY
EXCESS LOSS
REDISTRIBUTION

HYPOMAGNESEMIA DUE TO REDUCED SUPPLY

- Low oral intake
- Malnutrition
- Parenteral nutrition with low magnesium content
- Free fatty acids, fibre, phytates, phosphate, oxalate, calcium can decrease bio availability
- Newborn of diabetic mother
- Newborn with IUGR

HYPOMAGNESEMIA DUE TO REDISTRIBUTION

- Refeeding syndrome
- Hungry bone syndrome
- Rapidly proliferating neoplasm
- Massive transfusion due to citrate effect
- Pancreatitis
- Insulin administration



HYPOMAGNESEMIA DUE TO EXCESS LOSS

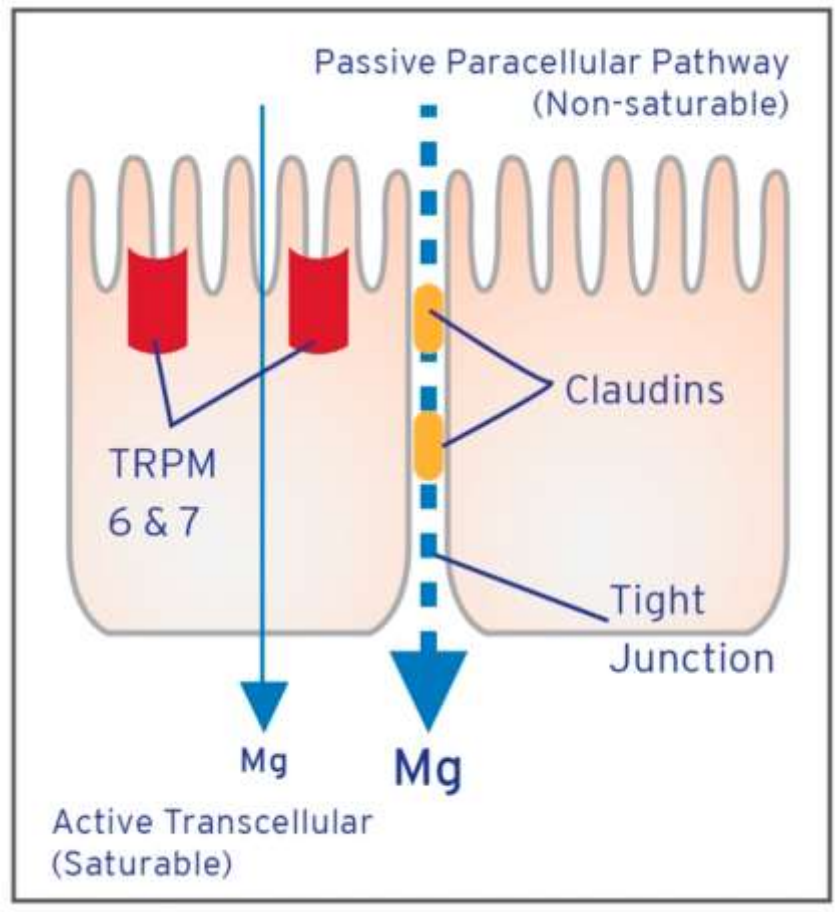
INCREASED INTESTINAL LOSS OF MAGNESIUM

Acquired

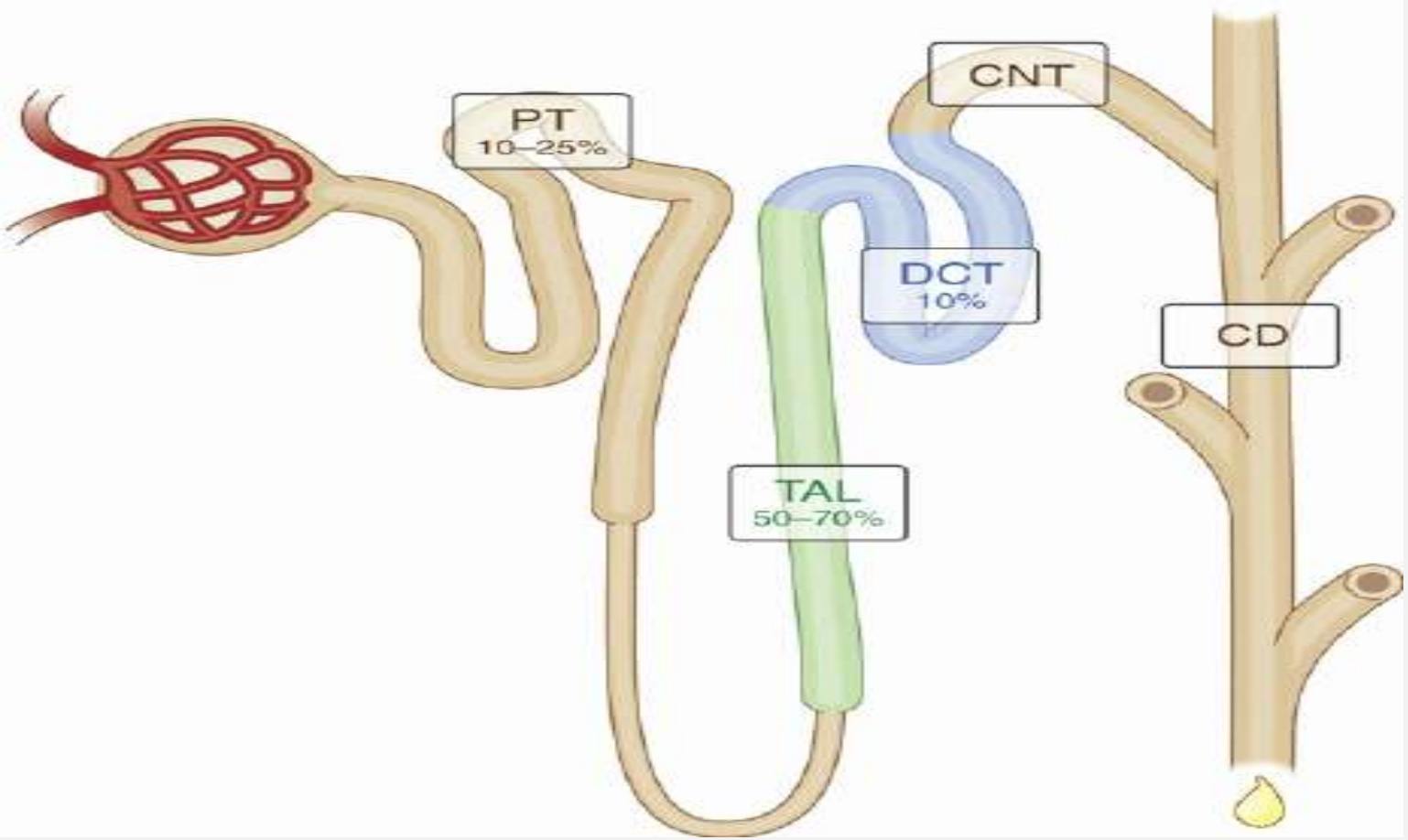
- Mal absorptive syndromes (celiac, IBD)
- Diarrhea
- Excess vomiting/NG aspiration
- Short bowel syndrome
- Bowel resection
- Drugs
- Proton pump inhibitors
- Laxative abuse

Genetic

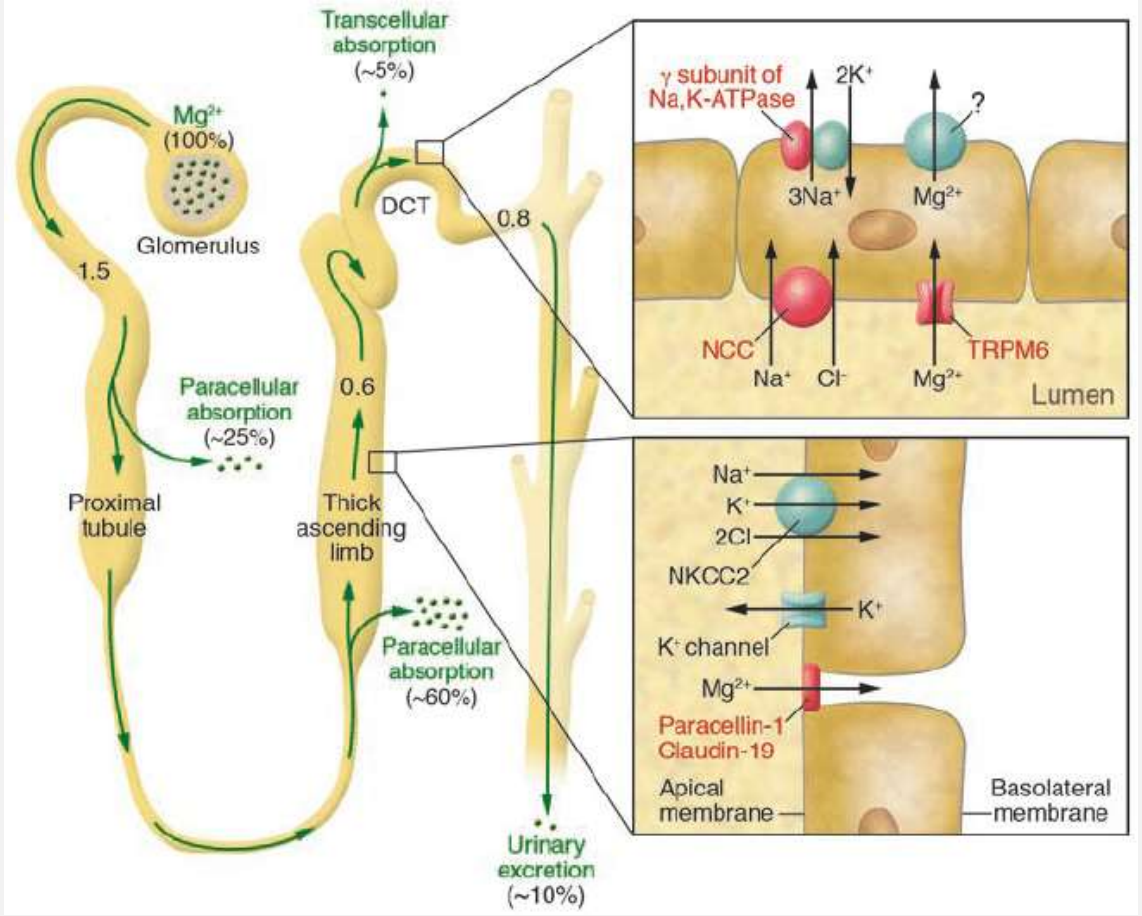
- Primary intestinal hypomagnesemia



Magnesium reabsorption in the kidneys



RENAL MAGNESIUM HANDLING



INCREASED RENAL MAGNESIUM LOSS

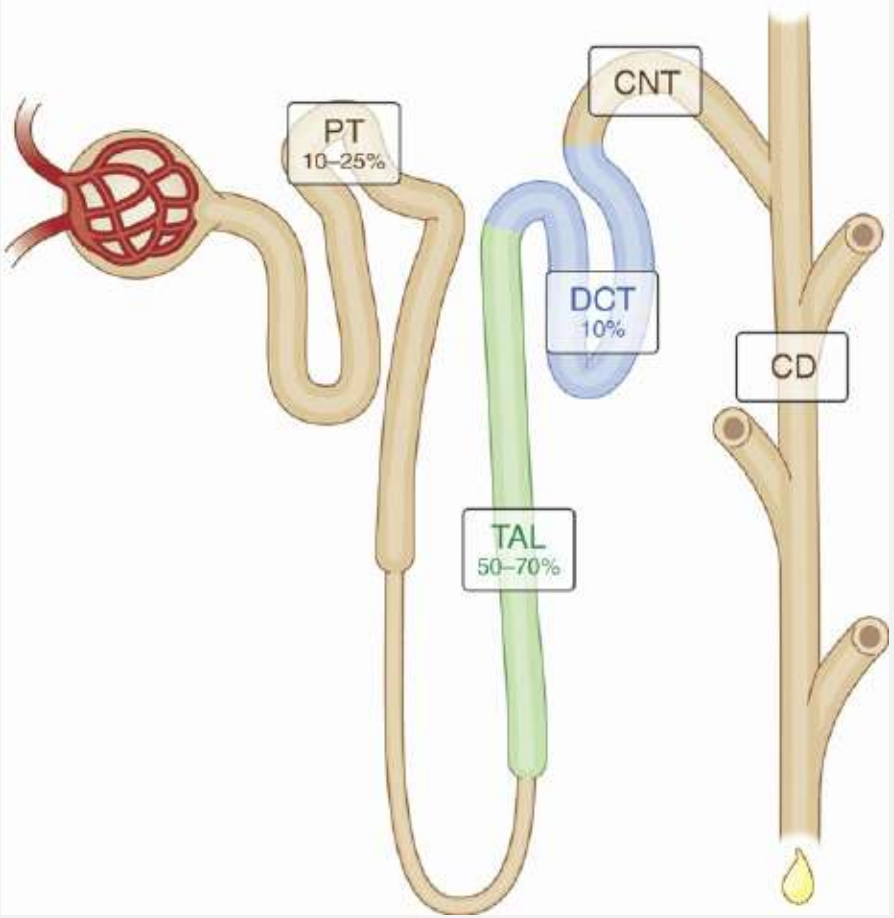
Genetic (tubular) causes

- Autosomal-dominant hypocalcaemia, hypomagnesemia, and hypercalciuria
- Familial hypomagnesemia, hypercalciuria, and nephrocalcinosis
- Isolated hypomagnesemia (autosomal-dominant, autosomal-recessive)
- Gitelman & Bartter syndromes
- EAST syndrome
- Autosomal dominant hypoparathyroidism

INCREASED RENAL MAGNESIUM LOSS

ACQUIRED CAUSES

- Diabetes
- Mannitol
- ATN- Recovery phase
- Post obstructive nephropathy
- Chronic kidney disease
- Hypercalcemia
- Volume expansion

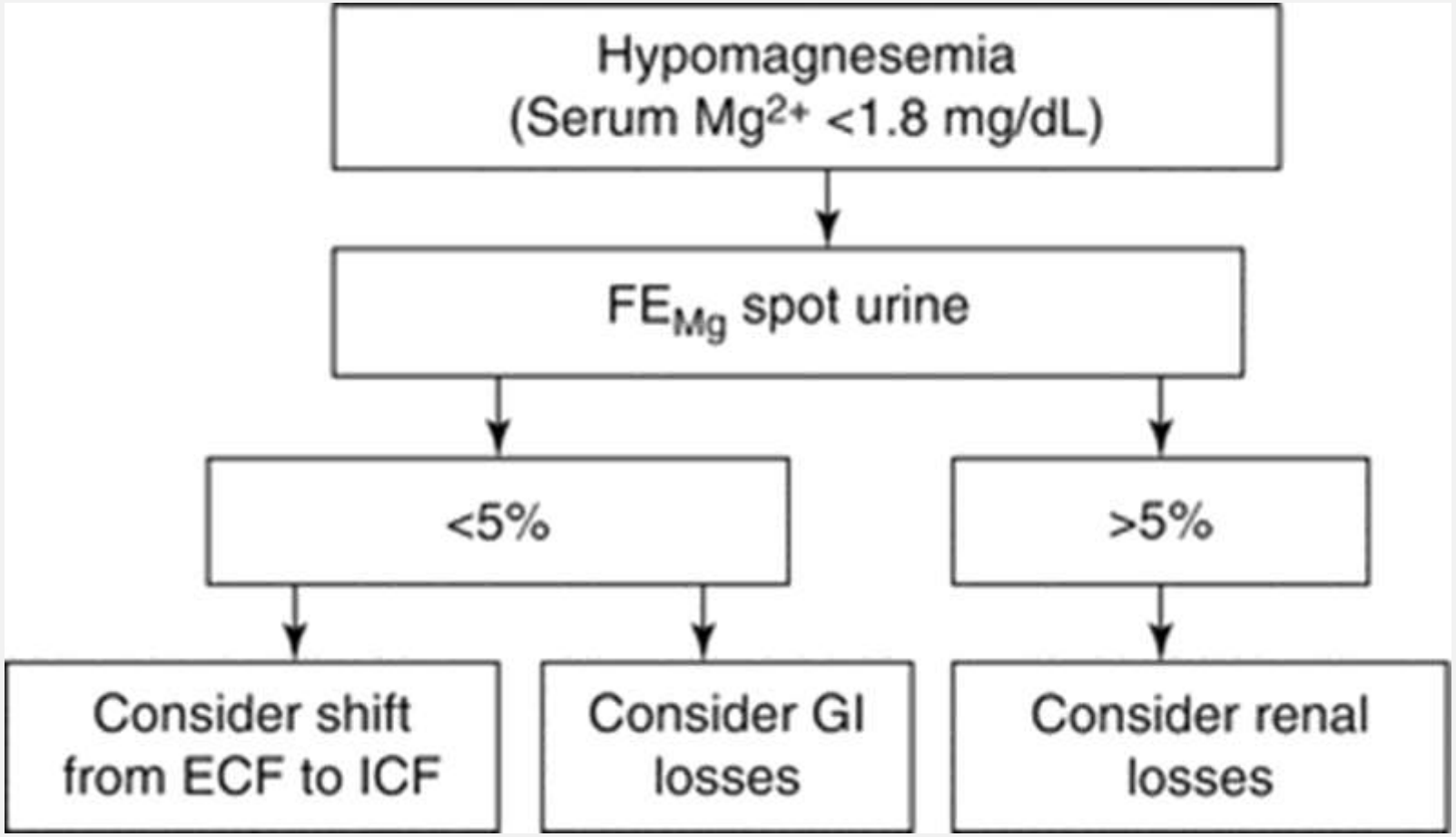


INCREASED RENAL MAGNESIUM LOSS

DRUG INDUCED

- Amphotericin
- Cisplatin
- Cyclosporin
- Loop diuretics
- Thiazides
- Pentamidine
- PPIs
- Aminoglycosides
- Epidermal growth factor inhibitor

HYPOMAGNESEMIA APPROACH



ADDITIONAL INVESTIGATIONS

In view of nephrocalcinosis, 24 hr urine analysis was done which revealed the following

- 24 hr Urinary calcium - 12 mg/kg/day
- Urinary citrate/creatinine ratio - 510 mg/g
- Urinary oxalate - < 0.5 mmol/1.73m^2/24 hrs
- Urinary cystine/ creatinine ratio - 40 mg/g -
- Urinary uric acid - 200mg/1.73m^2/24 hrs
- FeMg -13

ADDITIONAL INVESTIGATIONS

ABG

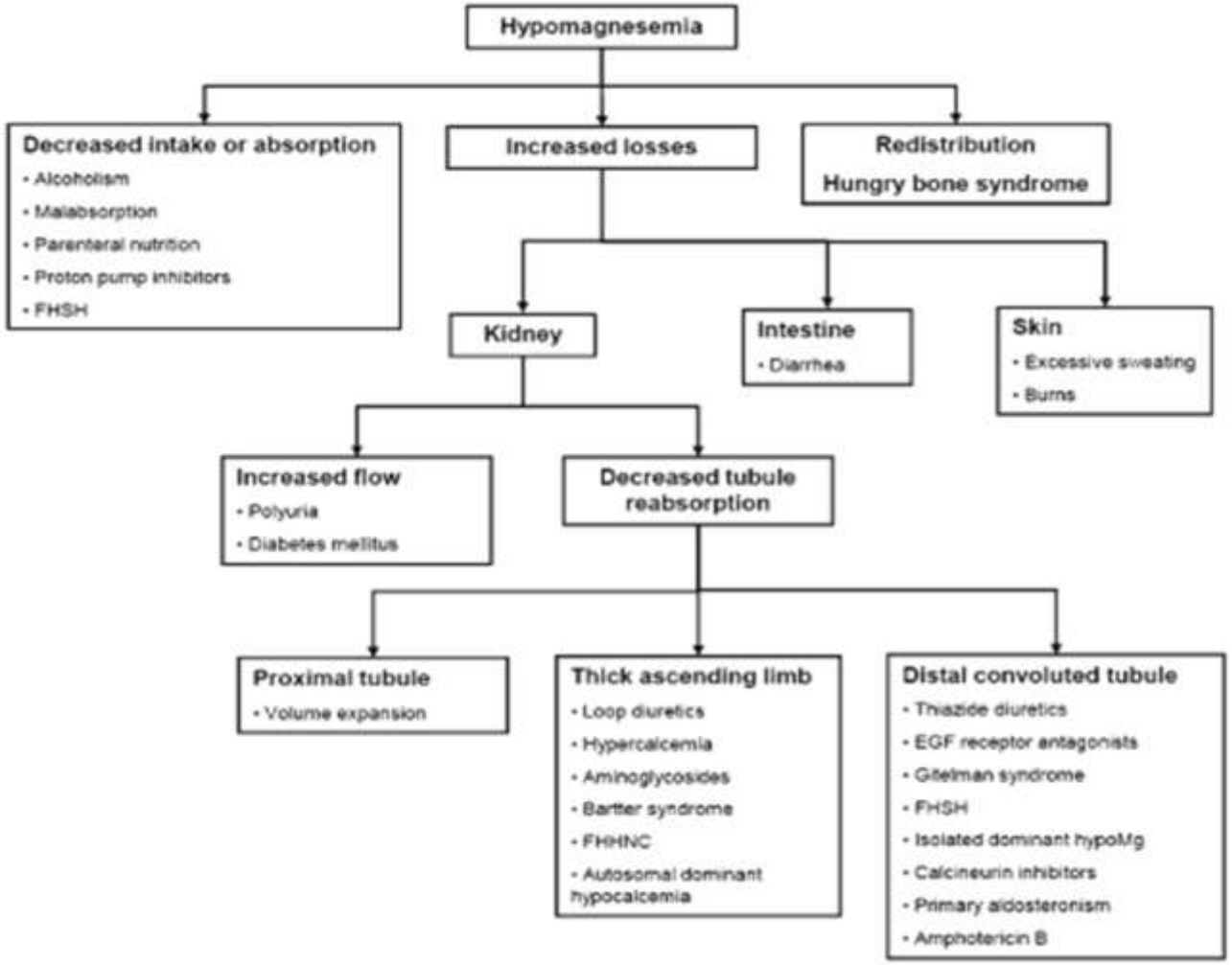
pH - 7.4

PCO_2 - 39 mm Hg

HCO_3 - 23 Meq/l

PaO_2 - 80 mm Hg

HYPOMAGNESEMIA APPROACH



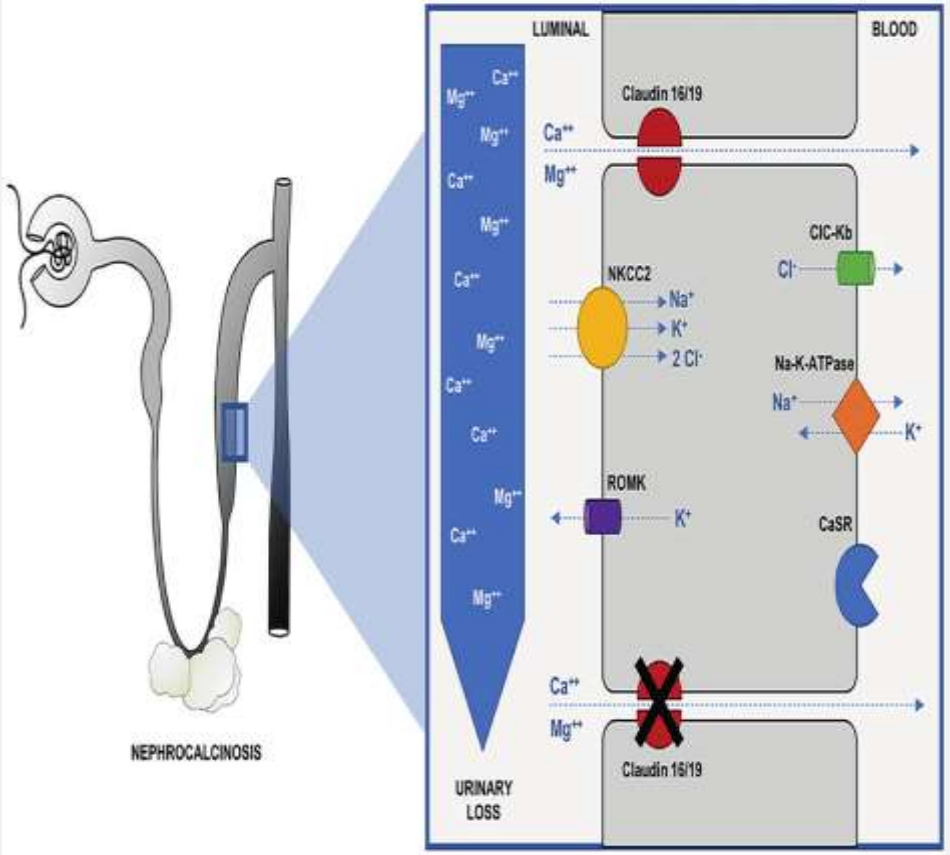
FHHNC familial hypomagnesemia with hypercalciuria and nephrocalcinosis,

IDH isolated dominant hypomagnesemia

FHSH
Familial hypomagnesemia with secondary hypocalcemia

FAMILIAL HYPOMAGNESEMIA,HYPERCALCIURIA WITH NEPHROCALCINOSIS

- Autosomal recessive inheritance
- Mutation in the gene for Claudin 16/19
- Severe renal calcium and magnesium wasting in TAL segment
- Serum calcium is normal
- Progresses to chronic renal failure
- Children with CLAUDIN 19 mutation have additional ocular involvement



TREATMENT

- Acute hypomagnesemia- parenteral magnesium sulfate(50%) 25-50 mg/kg (0.05-0.1 ml/kg) slow IV, repeated 4-6th hourly till stabilization.
- Long term therapy - magnesium sulfate, magnesium oxide or magnesium gluconate
  - Dose: 2.5 - 5 mg/kg (0.1 - 0.2 mmol/kg) 3 times daily orally
  - Increase to 10 - 20 mg/kg (0.4 - 0.8 mmol/kg) up to 4 times daily orally if required
  - Tolerance is better with smaller, more frequent dosing
- Treatment for hypercalciuria with thiazide diuretics and renal transplant for progressive renal failure


THANK YOU!