ORAL ASPECTS OF METABOLIC DISORDERS

DEPARTMENT OF ORAL AND MAXILLOFACIAL PATHOLOGY & ORAL MICROBIOLOGY

The dietary constituents of food are:

- Protein
- Fats
- Carbohydrates
- Minerals
- Hormones
- Vitamins
- Dietary fibres and
- Water.

PROTEIN

- Proteins are complex organic nitrogenous compounds.

- They are indispensible constituents of the diet because they are the only source of the essential amino acids which include valine,lysine,leucine,isoleucine,phenylalanine ,methionine,threonine,tryptophan,histidine and arginine.

Sources -

- Animal sources:milk,milk products, eggs,meat,fish etc.

- Plant sources-pulses,cereals,dry fruits,nuts beans etc.

- Animal proteins are called proteins of high biological value because proteins of animal tissue closely resembles those of human tissue and can be employed more economically for repair and growth.

- Plant proteins are called proteins of low biological value because the amino acid pattern in them is different from that of human tissue and can not be employed so economically.

- Proteins of high and low biological value are sometimes called first class and second class proteins respectively.

Functions -

- Build new tissues during the period of growth or pregnancy and lactation.

- Essential for repair and maintenance of worn out body tissues.

- Provides the raw material for the synthesis of certain substances like antibodies,enzymes, haemoglobin ,hormones and plasma proteins.

Daily requirement-

- 1 gm per Kg body weight for an adult,some of it in the form animal protein.

- An extra amount of protein (1.5-2 gm/Kg body weight)should be added in debilitating diseases,children and in pregnant and lactating women.

- Protein metabolism involves both digestion and absorption of protein.

Protein digestion

- The enzymes mainly involved in protein digestion are pepsin and pancreatic proteases.

- Several pancreatic proteases are trypsin,chymotrypsin,elastase, carboxypeptidase etc.

Absorption of protein

- The molecules of all the aminoacids are much too large to diffuse readily through the pores of the cell membrane.

- Hence they are absorbed by active transport using carrier protein.

- There are different carrier proteins for different groups of amino acids .

- Hence a congenital disorder can affect absorption of only one group of amino acids sparing others.

Disturbances in protein metabolism

- Protein energy malnutrition is the term used to refer to the broad spectrum of diseases which includes Kwashirkor at one end and nutritional marasmus at the other end with marasmic kwashirkor in the middle.

- The term Protein Energy Malnutrition is however misnomer because protein deficiency alone is very uncommon.

- The real deficiency is that of food energy which is measured in terms of calorie.

- Protein Energy Malnutrition should be therefore substituted by Protein CalorieMalnutrition.

Causes beyond Protein Calorie Malnutrition are-

- An inadequate diet,both in quantity and quality;primarily due to poverty.

- Infectious and parasitic diseases such as repeated diarrhoea and worm infestation.

- Poor environmental conditions.

- Adverse cultural practices .

Marasmus

- It is one of the Protein Calorie Malnutrition diseases.

Predisposing factors-

- Rapid succession of pregnancies.

- Early and often abrupt weaning followed by artificial feeding of infants in inadequate amount.

Some of the salient features of Marasmus are-

- Major causative factor-severe calorie malnutrition.

- Weight as percentage of normal-below 60%.

- Growth retardation both physical and mental-severe.

- Odema - absent.
- Mascular wasting-severe Appetite - usually good.
- Skin changes-loose with loss of elasticity.
- Hair - brittle(fragile)
- Face-small sunken (monkey like face)
- Diarrhoea - may be present.
- Anaemia-may be present.
- Liver enlargement-absent.


Characteristic features

kwashiorkor

- Major causative agent-gross protein deficiency.
- Weight as percentage of normal-60-80%.
- Oedema - present allover the body.
- Mascular wasting-occasionally seen.
- Growth retardation - less.
- Appetite -poor.
- Skin changes-depigmentation of skin all over the body.
- Hair - sparse,straight,greyish or reddish
- Face - swollen (moon like)

- Diarrhoea - often present
- Anaemia - present.
- Liver enlargement - frequent.
- Occurrence and outcome-less common occurrence with serious outcome,may be fatal.

The oral lesions, when apparent,include-

- A bright reddening of the tongue.

- Loss of the papillae.

- Bilateral angular cheilosis.

- Fissuring of lips.

- Loss of circumoral pigmentation.

- Xerostomia.

- Epithelium gets readily detached from the underlying tissue,leaving a raw bleeding surface .

KWASHIORKOR , results from gross protein deficiency

1. Generalized oedema *ie*. swelling present all ovei the body.
2. Swollen face (moon like)
3. Hair: sparse, straight with loss of pigment

4. Generalized skin changes:

i.)areas of skin pigmentation alternating with scaling

ii.)areas of decreased pigmentation

Effect of protein and protein energy deprivation on hormones and enzyme production-

- Many of the body's hormones and all enzymes are proteins (except Ribozyme).Hence any disturbance in protein metabolism will lead to reduced synthesis of hormones and enzymes leading to -

- Delayed puberty and amenorrhoea in girls.

- Loss of libido and impotence in males.

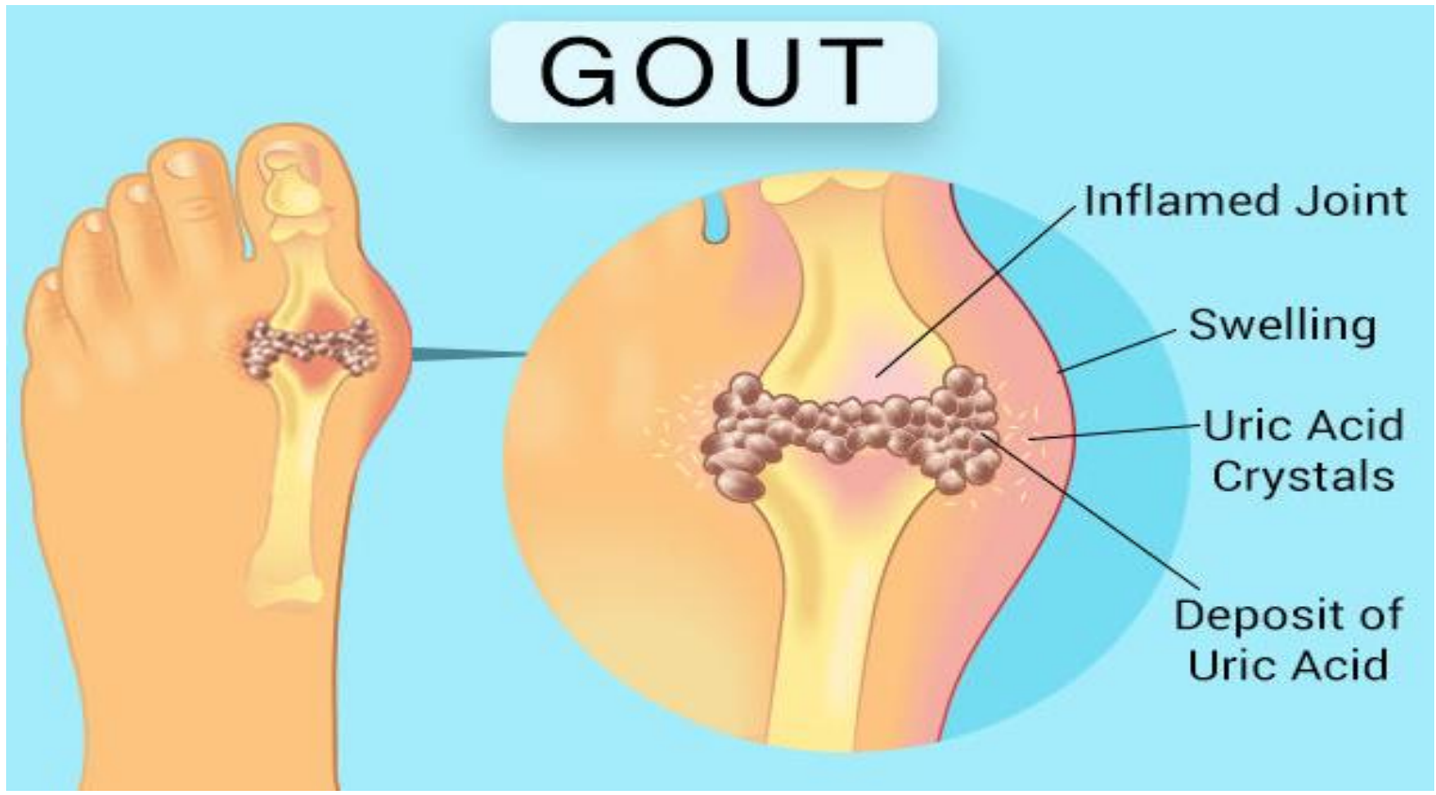
- Marked atrophy of the thyroid gland due to reduced secretion of thyrotropic hormone.

- Diarrhoea due to reduced formation of digestive enzymes.
- Marked bradycardia and reduced BMR due to thyroid deficiency.

Gout:

- A common disorder of protein metabolism, is characterized by excessive uric acid production, leading to the formation of urate crystals deposited in joints.

- Here the disorder is due to excessive intake of protein .



Amyloidosis

- Amyloid is an abnormal proteinaceous substance which is deposited between cells in tissue and organs of the body in a variety of clinical disorders.

- Two major classes of amyloid identified are-

- Amyloid light chain,composed of immunoglobulin light chain

- Amyloid associated,made up of nonimmunoglobulin protein.

- Under routine stains,amyloid is seen as intercellular pink translucent material by light microscopy.

Type A amyloid is a fibrilar protein of unknown origin, seen in

- Prolonged inflammatory diseases
- Genetic diseases and
- Syndromes such as familial Mediterranean fever.

Type B amyloid is commonly seen in patient with multiple myeloma and macroglobulinemia.

Type C amyloid is third type of amyloid include amyloid of aging, localized nonspecific amyloid and amyloid adjacent to APUD tumor

Types

- **Primary amyloidosis** -

  There is no evidence of preceding or existing disease.

- Amyloidosis associated with multiple myeloma.

- **Secondary amyloidosis-**

  Associated with variety of chronic inflammatory disease.

- Localized amyloidosis-

  It is characterized by small localized deposits of amyloid in the skin,bladder and respiratory tract.

- **Familial amyloidosis-**it is a rare condition. Examples are familial Mediterranean form or familial amyloidosis with polyneuropathy.

- **Hormone related amyloid-**it is associated with tumors of endocrine cells which secretes peptides hormones.

Etiology-

- Collagen diseases particularly rheumatoid arthritis
- Chronic infections like tuberculosis and osteomyelitis ,Ulcerative colitis.
- Malignant diseases like Multiple myeloma, Hodgkin's disease,Renal cell carcinoma.

Clinical features-

Site-

- Commonly affected organs are kidneys,heart,GI tract,liver,respiratory tract,skin,eyes,adrenals,nerves and spleen.
- There may be primary localized collection of amyloid.

Symptoms-

- The general symptoms are fatigue,weakness,ankle ,edema, dyspnea, paresthesia,orthostatic hypotension and weight loss.

- Signs-purpuric spots caused by hemorrhage resulting from amyloid deposits in the blood vessels.

- Myocardium - congestive cardiac failure is a common problem due to amyloid deposits on myocardium.

- GIT involvement-there is hepatomegaly,malabsorption or colitis may develop.

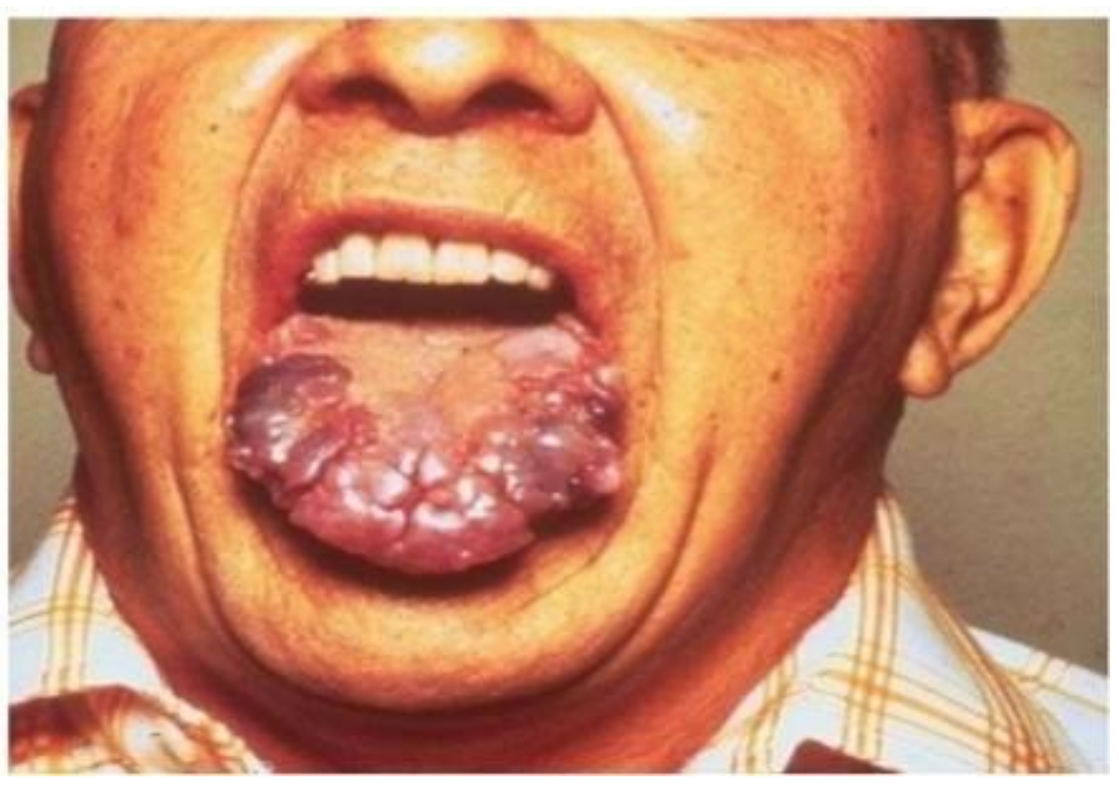
Oral manifestations-

- fibrous glycoproteins are deposited in submucosa as well as in deep muscular layer of tongue.

- Skin lesions may diffusely involve the face or may present as small elevated yellow nodules.

- Macroglossia- it is due to amyloid deposit.
- Seen in both primary and secondary form.
- Symptoms- there are difficulties in chewing,swallowing or talking.

- Appearance - tongue is enlarged and studed with small enlargements .



- Signs - motility of tongue is decreased. Yellowish nodules are present along the lateral border of the tongue and impression from teeth is also visible.

- Gingiva - it may be infiltrated and may be bluish,spongy and hypertrophied.

- Xerostomia may result from salivary gland involvement.

Histopathologic Features

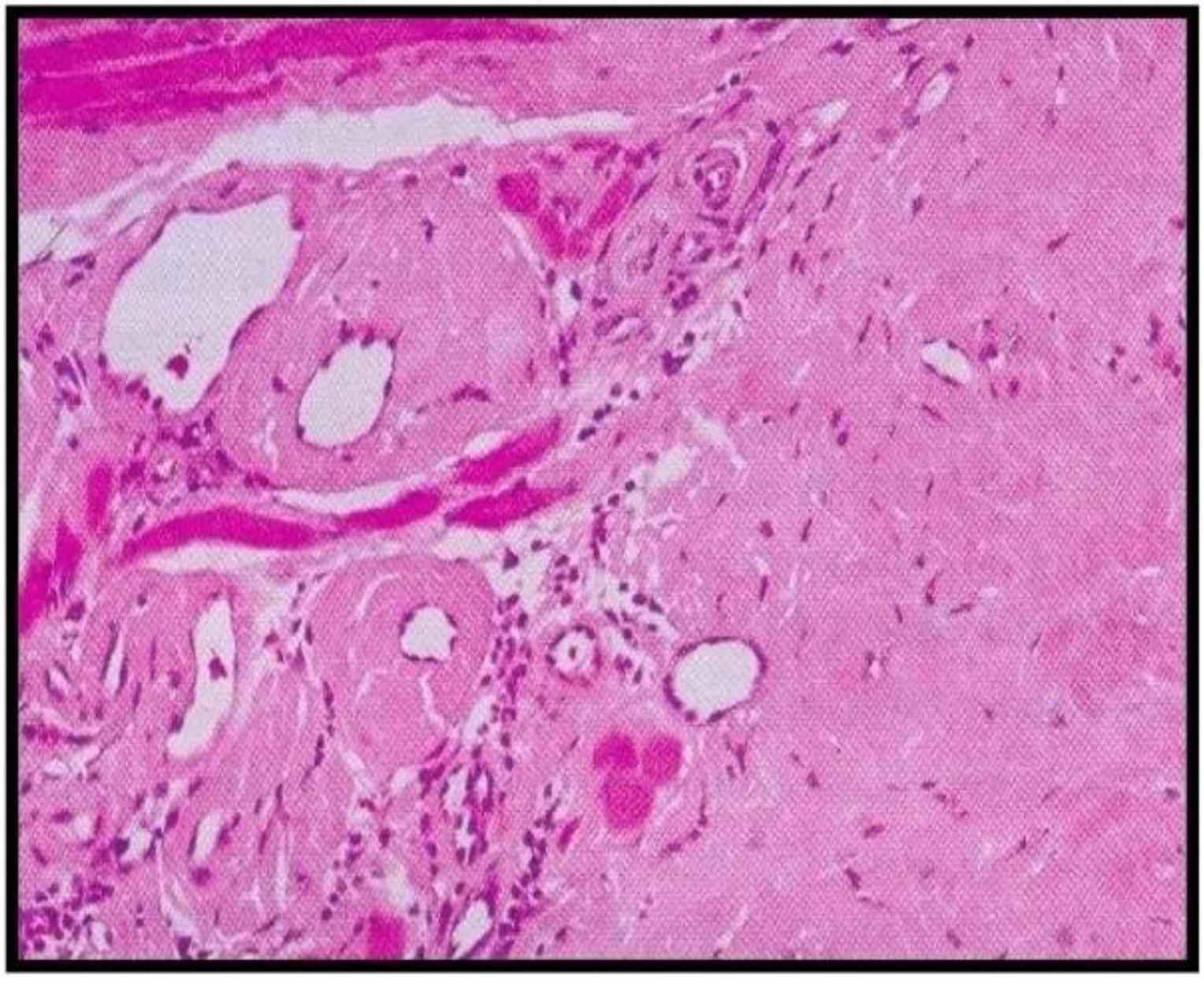
- Biopsy of rectal mucosa has classically been used to confirm a diagnosis of primary or myeloma-associated amyloidosis with up to 80% of such biopsy specimens being positive.

- Aspiration biopsy of abdominal subcutaneous fat.

- Alternative tissue sources: gingiva and labial salivary glands is a simpler procedure.

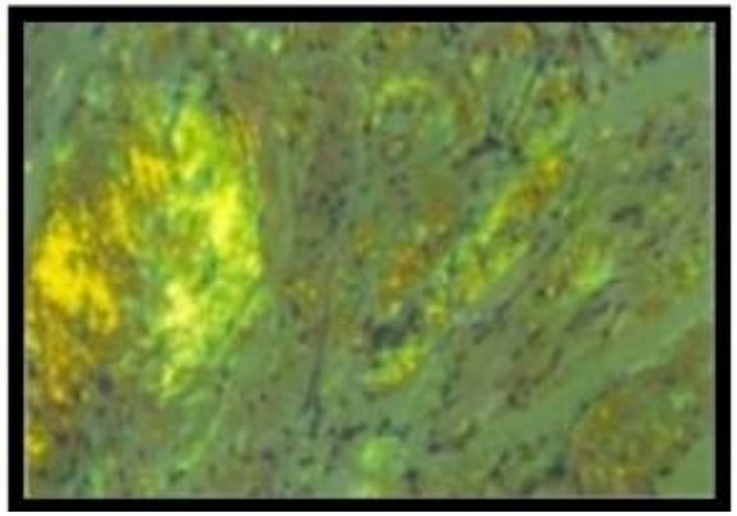
- Histopathologic examination of gingival tissue shows extracellular deposition in the submucosal connective tissue of an amorphous, eosinophilic material, which may be arranged in a perivascular orientation or may be diffusely present throughout the tissue.

•Labial salivary gland tissue shows deposition of amyloid in a periductal or perivascular location in more than 80% of the cases.



- A standard means of identifying amyloid uses the dye, Congo red, which has an affinity for the abnormal protein (**amyloid appears red).**

- When this tissue is viewed with polarized light, it exhibits an apple green birefringence.



- Crystal violet staining reveal a characteristic metachromasia, this purple dye appears more reddish when it reacts with amyloid.

- Staining with thioflavine T, a fluorescent dye, also gives positive results if amyloid is present

Treatment and Prognosis

- No effective therapy is available for amyloido sis .
- Surgical debulking of amyloid deposition in the tongue has met with limited success
- Most patients die of cardiac failure, arrhythmia, or renal disease within months to a few years after the diagnosis.

PORPHYRIA

- Porphyria refers to an inborn error *of* porphyrin metabolism and it is characterized by the overproduction *of* uroporphyrin and other related substance.
- Also results from certain infections or intoxication.

Porphyria

Erythropoietic porphyria

1)Urophorph yria

2) Protoporphyria

Hepatic porphyria

1)Acute intermittent porphyria

2) Porphyria variegata

3)Porphyria cutanea tarda

4)Hereditary coproporphyria

Erythropoietic uroporphyria

Also known as "congenital porphyria".

- Non sex-linked recessive character.
- Both genders being equally affected.
- First sign :red urine containing much uroporphyrin.
- May be noted at birth or during first years of life.
- Photosensitivity become apparent during first year of life.

- A vesicular or bullous eruption appears on the face,back of the hands and other exposed parts of the body.

- Ruptured vesicles heal slowly and leave depressed,pigmented scars.

Oral findings-

- The deciduous and permanent teeth may show a red or brownish discoloration.

- Deposition of the porphyrin in the developing teeth and bone is due to its physical affinity for calcium phosphate.

- The presence of porphyrin in the deciduous teeth indicates that the metabolic disorder may have been present during fetal life.

Hepatic porphyria

- It is multisystem disorder.

Lipids

- Lipids are heterogeneous group of organic compounds which are relatively insoluble in water but soluble in solvents such as ether,chloroform and benzene.
- **Classes of lipids-**
- Triglycerides,conjugated lipids,cholesterol and so on.

Sources-

Animal sources like ghee,butter, fish,oil etc.

- Plant sources like groundnut,mustard,cotton seed,rape seed and coconut oil .

Daily requriment-10-20 gms of fat per day for adults.

- Young children need 25%extra amount offat.

Functions-

- Fuel reserve of the body.
- Membrane structure and membrane permeability.
- Sources of fat soluble vitamin.
- Regulator of cellular metabolism.

Digestion of lipid

- Digestion of lipid involves mainly three enzymes namely lingual lipase,gastric lipase,and pancreatic lipase.
- Among them lingual lipase and gastric lipase have little role in man.
- The major part is digested in the small intestine.

ABSORPTION OF LIPID

- **Maximum absorption takes place in the last part of the duodenum and proximal portion of the jejunum and absorption of lipid is mainly a carrier mediated transport.**

- **Disturbances in lipid metabolism**
- It is concerned with the assimilation,utilization,replacement,and synthesis of the various fatty acids of the cell.
- Disturbances of lipid metabolism are rare.
- They are identified on the basis of the particular lipid involved.

GAUCHER'S DISEASE

- It is characterized by the deposition of glucocerebroside in cells of the macrophage monocyte system.
- Deficiency of lysosomal hydrolase, glucocerebrosidase which cleaves glucoserebroside to ceremide is held responsible for this disorder.

C/F-

- Divided into three clinical forms-

Type 1:chronic nonneuronopathic form-

- Often present in childhood with hepatosplenomeghaly,pancytopenia and skeletal disease.
- It has a striking predilection for occurrence amongst individuals of Ashkenazi Jewish descent.
- Radiographic evidence of skeletal involvement including an **Erlenmeyer flask** deformity of distal femur.

Type 2:Infantile or acute neuronopathic form -

It causes progressive neurovisceral involvement and results in death at infancy.

Type 3:Juvenile form or Norrbottnian form-

- The patients are juveniles presenting with systemic involvement. Progressive CNS involvement usually begins at teens or twenties.

- In all the three types of Gaucher's disease,examination of the biopsy of spleen or liver wiil reveal the typical Gaucher's cell.

- This is a round pale cell,measuring between 20 -80 microns in diameter,containing a small eccentric nucleus and a wrinkled or 'crumpled silk' cytoplasm.

Gaucher Disease: Clinical Signs and Symptoms

Pulmonary involvement

Progressive neurologic symptoms*

Hepatosplenomegaly

Thrombocytopenia and anemia

Skeletal involvement

* In neuronopathic subtypes only.

- **Prognosis and treatment-**
- Prognosis of Type II Gaucher's disease is very poor.
- In the other two types patients survive till sixth decade of life,when the patient die of some recurrent infection.
- Enzyme replacement therapy for Gaucher's disease is available.

NIEMANN PICK DISEASE

- It is the least common of the genetic disturbances of lipid metabolism .

- It is inherited as an autosomal recessive trait.

- It results from lysosomal accumulation of sphingomylin resulting from inherited deficiency of sphingomyelinase.

Signs and Symptoms of Niemann-Pick Disease

- Abdominal swelling within 3-6 months
- Loss of Appetite
- Loss of early motor skills
- Enlarged spleen & liver
- Jaundice at birth
- Irregular speech

- **Types -**
- Type A or the acute infantile type.
- Type B or the non-neurological form,less common.
- Type C or biochemically and genetically distinct form of the disease,characterized by intracellular cholesterol esterification.
- The mutant gene is localized to 18qll-12.

Niemann -Pick disease Type A-

- It is a severe infantile form with extensive neurologic involvement,marked visceral accumulation of spingomyelin and progressive wasting.
- Death occurs within first three years of life.

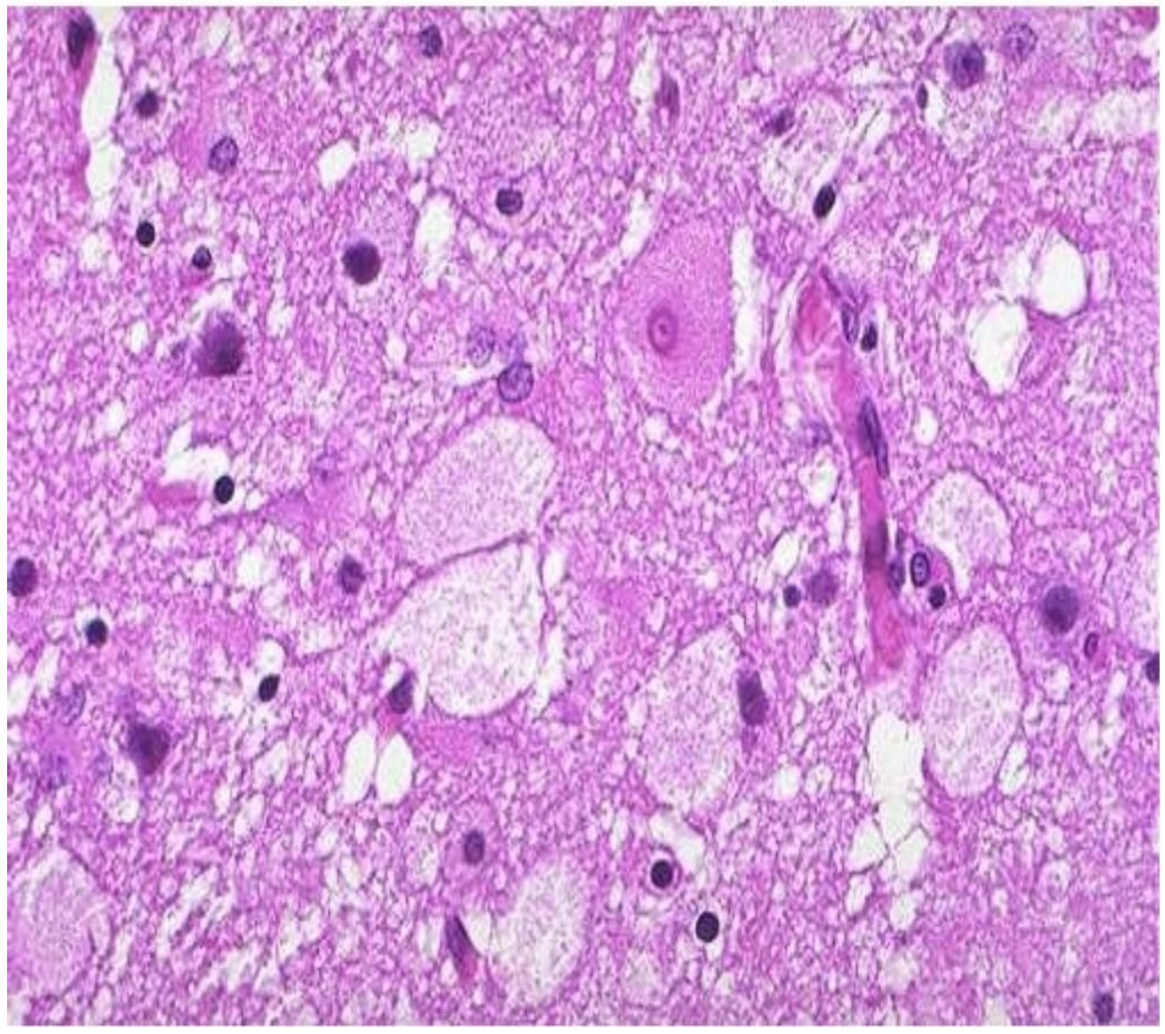
Niemann Pick disease Type B-

- In this type patient's organomegaly is seen commonly but no nervous system involvement is seen.

histopathologic feature

It is characterized by the presence of Niemann Pick cells .

- Niemann -Pick cell is the affected cell,which becomes extremely enlarged secondary to the distention of lysosomes due to the accumulation of sphingomylin and cholesterol.
- The cell shows foamy cytoplasm due to numerous vaculoes which stain for fat .



- **Prognosis and treatment-**
- Prognosis is poor as the vast majority of the patients die of the disease.
- Enzyme replacement therapy is currently being explored.

- # LETTERER-SIWE DISEASE
- It is an acute, aggressive form of histocytosis,which invariably occurs in infants usually before the age of three years.

C/F-

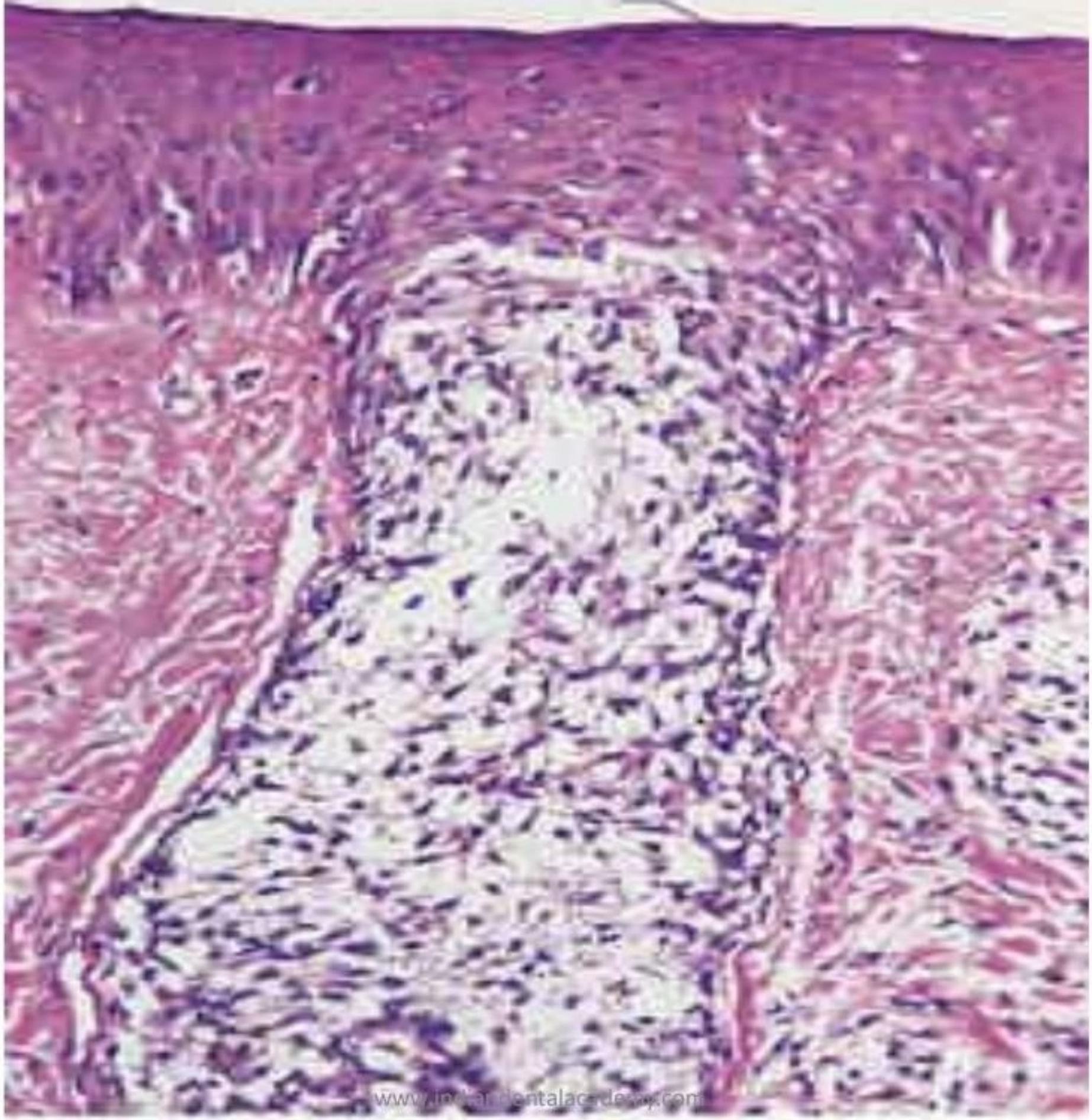
- The initial manifestation of the disease is often a skin rash involving the trunk, scalp and extremities.

- The rash may be erythematous,purpuric or ecchymotic,sometimes with ulceration.
- The patients commonly have a persistent,low grade spiking fever with malaise and irritability.
- Splenomegaly,hepatomegaly and lymphadenopathy are early manifestation.
- There is nodular or diffuse involvement of visceral organs,particularly the lungs and the GIT,later in the course of the disease.

- **Oral manifestation-**
- Ulcerative lesions along with gingival hyperplasia.

- Diffuse destruction of bone of the maxilla and mandible may occur causing loosening and premature loss of teeth.

- **Histologic Features-**
- There is basically a histocytic proliferation with or without eosinophils.
- These histocytes do not contain significant amount of cholesterol.
- Hence foam cells are not its features.
- Sometimes altered histocytes are present in sufficient numbers to resemble a histocytic lymphoma.

- 

Treatment and prognosis-

- Prognosis is extremely poor.
- In most cases course of the disease is rapid and terminates fatally in short time.
- Chemotherapy is effective in some patients,which is maintained in remission for years.

Disturbances in carbohydrate metabolism

Disturbances in carbohydrate metabolism

Classification:

Monosaccharide

Oligosaccharide

Polysaccharide

Fuunction:

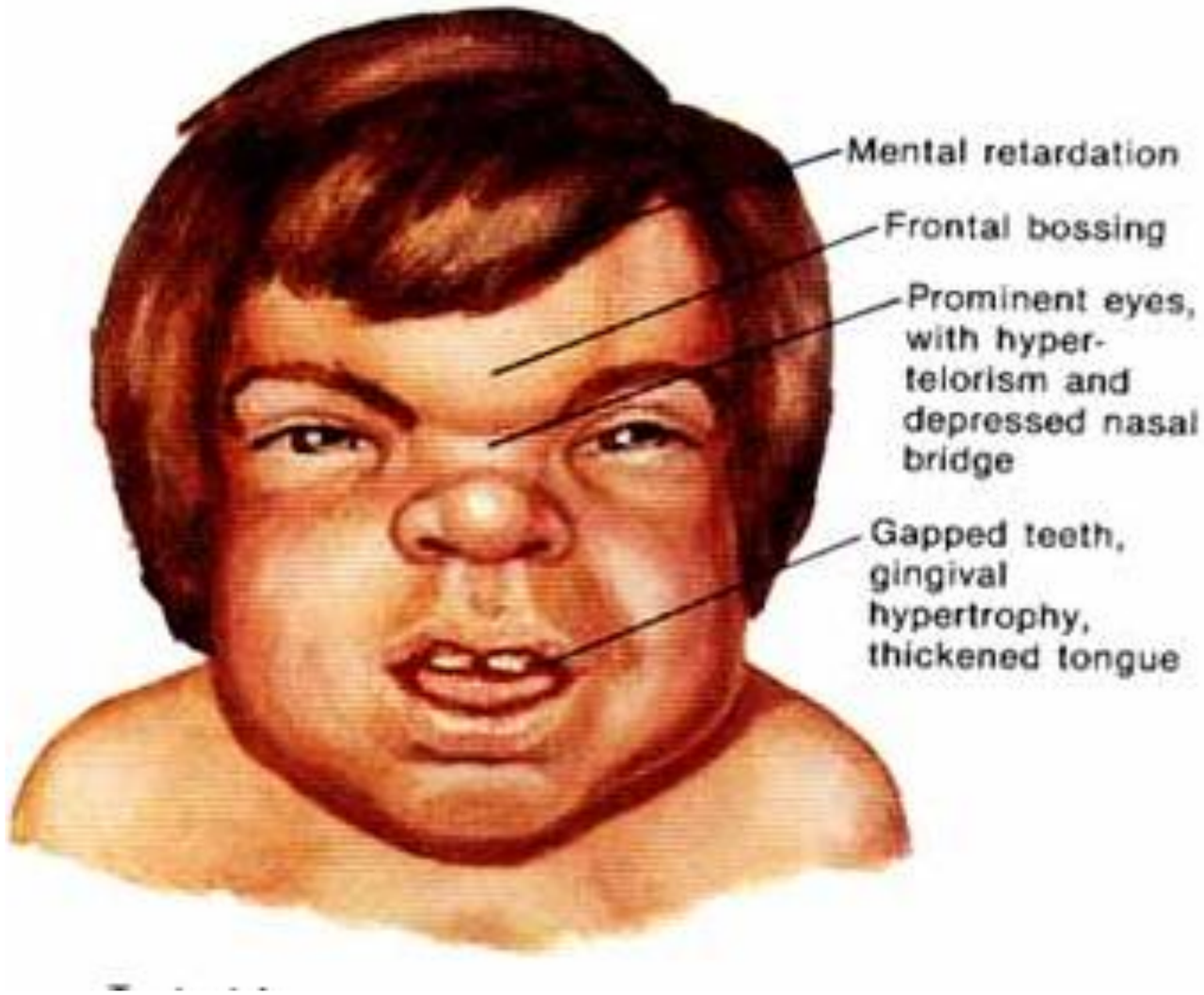
Energy source

Fuel for fetus

Cell membrane

Hurler's syndrome

- It is disturbance of mucopolysaccharide metabolism. characterized by elevated mucopolysaccharide excretion.
- Intracellular accumulation of chondroitin sulphate B and heparin sulphate in the tissues.
- It is inherited as an autosomal recessive trait.



Clinical Features:

Age:

First two years of life, progresses during early childhood and adolescence and terminates in death before puberty.

Signs:

- Head is large.
- Prominent forhead
- Broad saddle nose and wide nostrils.
- Hypertelorism and puffy eyelids with coarse bushy eyebrows.
- Thick lip.
- Large tongue.
- Open mouth and nasal congestion and noisy breathing

Abdomen

- Progressive corneal clouding.

- Hepato- splenomegaly resulting in protuberance of abdomen.

Claw hand

- A short neck and spinal abnormalities are typical, while flexion contractures result in the 'claw hand'.

Oral Manifestations

- ***Mandible***-*shortening* and broadening of mandible with prominent gonions and wide intergonial distance.
- ***Teeth***-*there* is typical spacing of teeth. Teeth are small and misshapen.
- ***Gingiva***-*there* may be gingival hyperplasia.

Radiological Features

- *Bone* destruction-localized areas of bone destruction in the jaws may be found which appear to represent hyperplastic dental follicles with large pool of metachromate material probably mucopolysaccharide.

- *Dentigerous cyst type* appearance-radiolucent area resembles a dentigerous cyst.

- *Laboratory diagnosis-there* is excessive accumulation of intracellular mucopolysaccharide in many tissues and organs throughout the body including liver, spleen, reticuloendothelial system, nervous system, cartilage, bone and heart.
- Abnormal deposits are found in many sites with involved fibroblasts assuming the appearance of clear (gargoyle) cells.
- There is elevated level of mucopolysaccharide in the urine.

Management

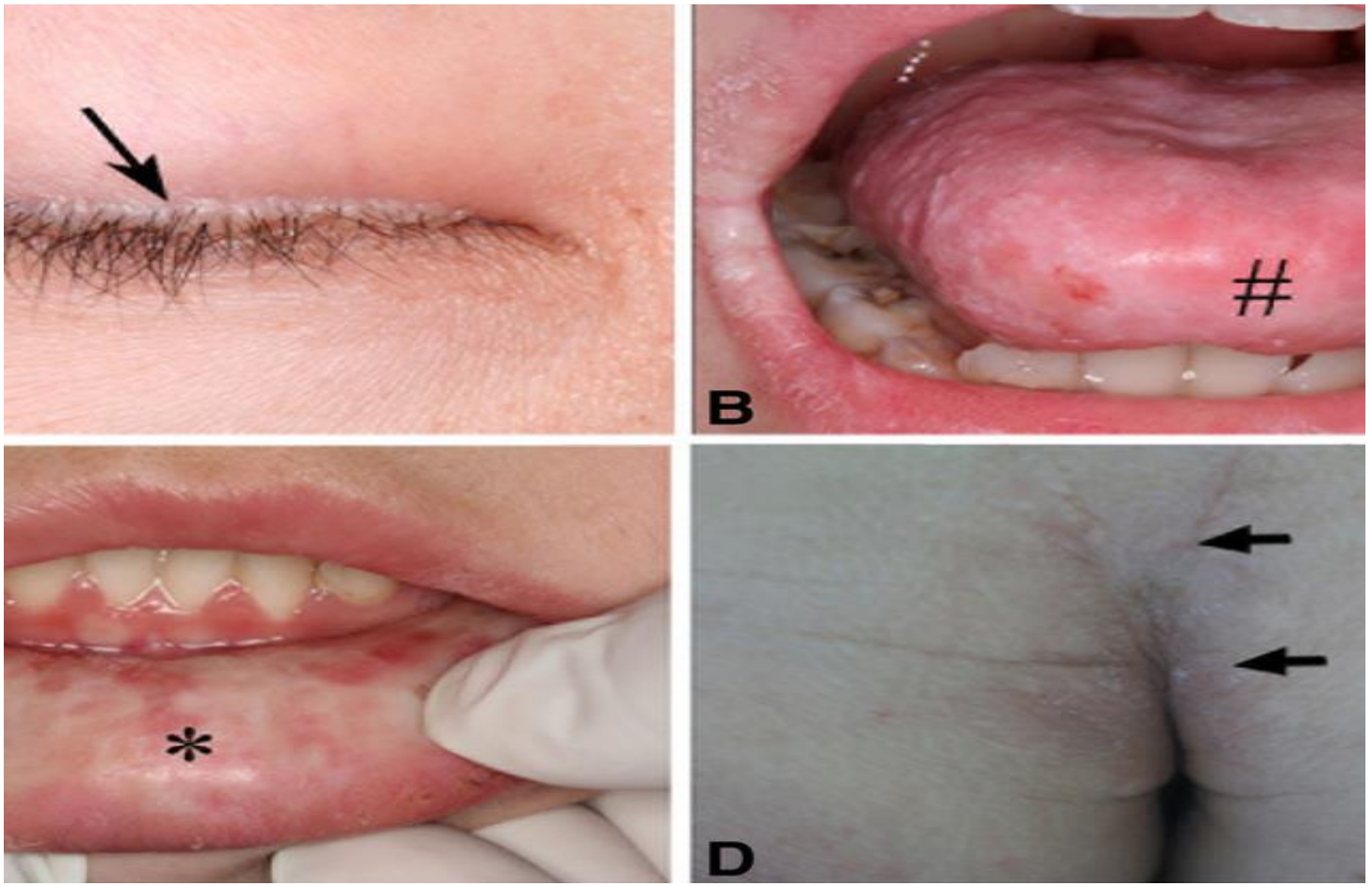
- Death usually occurs before the age of ten due to pneumonia and cardiac failure.

Lipoid Proteinosis

- Hereditary Fructose Intolerance
- It is transmitted as autosomal recessive trait. It results from a deficiency in fructose 1-phosphate aldolase. It is manifested by hypoglycemia and vomiting after ingestion of fructose containing foods. Affected individuals rapidly acquire an intense aversion to all sweets and fruits. There are fewer incidences of caries in these individuals.

Oral menifestation

- Oral mucous membrane showing yellowish white popular plaque.
- Thikening of tongue, frenum, lips .
- Blister , warty skin papules, scarring & dental anomalies found.
- Enamel hypoplasia may found.
- Recurrent painful parotitis may result involment of buccal mucosa .



A. Beaded papules along eyelid

B. Hypertrophic and stiff tongue

C. Hypertrophic and stiff lip

D. Waxy plaque on buttock

Hereditary fructose intolerance

- It is autosomal ressesive trait, manifested by hypoglysemia & vomitimg after injection of fructose containing food.
- It result from deficiency in fructose 1 phosphate aldose.

Oral manifestation :

- Caries score is less.

DISTURBENCE IN MINERAL METABOLISM

Zinc

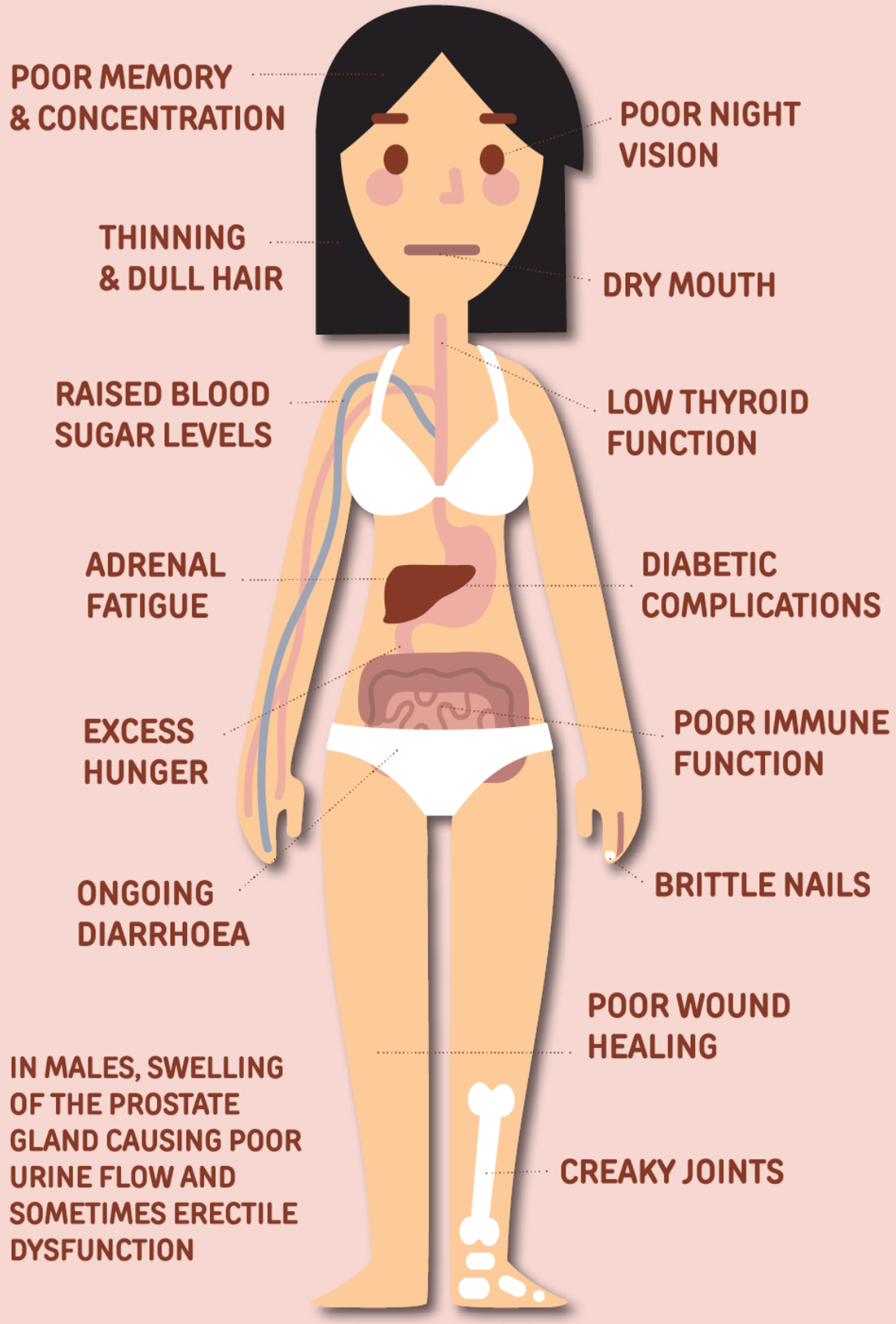
Zinc Deficiency :

- It is also called as *Acrodermatitis Enteropathica*. Rare disease of infancy and childhood, transmitted as autosomal recessive character. Since supplements appear to be curative even pathogenesis of the characteristic lesions involves a number of etiologic factors.

Clinical Features

- **Age-**it is usually occur in childhood after weaning.' Symptoms-the primary signs of the disorder are skin lesion, hair loss, nail changes and diarrhea.
- **Signs-**erythematous, pustular, moist erosions of the orofacial areas occur as an early manifestation. In fully developed conditions, the buttocks, elbows, fingers and toes are affected by vesiculobullous rash similar to that affecting the orofacial region. Retarded body growth and mental changes also occur with some frequency.

INDICATIONS OF A ZINC DEFICIENCY INCLUDE:



Oral Manifestations

- ***Site***-*the* buccal mucosa, palate, gingiva and tonsils.
- ***Candidiasis***-large number of children suffer from candidiasis.
- The perioral area usually being affected by weeping erosions, angular fissuring and spreading dermatitis.
- ***Papi/Joma***-there may be numerous small whitish papillomas on the buccal mucosa and borders of the tongue.
- The oral changes are sometimes described as 'stomatitis', 'glossitis' *and* 'stomatitis producing thrush like picture'.
- ***Signs***-buccal mucosa is present with red and white spots, erosions, ulcers and desquamation.
- ***Tongue lesion***-lesion on the tongue is sometime papillated and halitosis is often severe.

Diagnosis

- *Clinical diagnosis*-stomatitis, skin lesion, and hair loss will give clue to the diagnosis.
- *Laboratory diagnosis*-serum level of zinc is decreased.

Management

- *Zinc sulfate*-220 mg of zinc sulfate tds daily produces remarkable improvement.

Phosphorus

- Phosphates form an intermediate stage in the metabolism of fats and carbohydrates by their function in phosphorylation. They are used in building the more permanent organic phosphates including some catalysts essential to the structure and function of cells. They are utilized in the formation of phosphoproteins, such as milk casein and in the formation of the nerve polypeptides.

- Important or essential mineral.

- about 1 kg in adult body.

- About 80%in combination with ca
  10% in muscle & blood
  10% in vrious compounds.

- Serum level- 3-4 mg/dl.

Out of total blood phosphate
  40% in ionic form
  50% in combination with other cations
  10% bound to potiens.

Requirements

➢Daily dietary phosphorus intake

- Newborn infants : 240 mg
- Children & adult : 800 mg
- Adolescents & pregnant & lactating women : 1200 mg

Dietary sources

sea food : tuna, mackerel, salmon, sardines.

Meats : liver, rabbit, chicken, eggs, egg yolk.

Nuts & seeds : pinon nuts, sunflower seeds, walnuts, almonds, cashews, peanuts.

Vegetables : garlic, onion, soybeans.

Dairy : cheeses.

Grains : wheat, oats, oatmeal, rice bran, rye, wheat.

Absorption :

- 90% of average daily intake of phosphate is absorbed from small intestine.

Factors interfere with absorption of phosphorus :

- Excess of calcium, iron, aluminium.
- Long term antacid use.

Excretion

- Phosphorus is excreted primarily through the urine.
- Almost 2/3rd of total phosphorus that is excreted is found in the urine as phosphate of various cations.
- Phosphorous found in the feces is the non absorbed form of phosphorus.

- They provide the energy rich bonds such as adenosine triphosphate, which is important in muscle contraction and they form part of such coenzymes as pyridoxal phosphate, which is necessary in decarboxylation and transmission of certain amino acids such as tyrosine, tryptophan and arginine

- The suggested dietary intake of phosphorus ranges from 240 mg for infants to 800 mg for adults. It is increased in lactating and pregnant women by 50%.

Functions of Phosphates

- Formation of bones
- Like calcium, important component of teeth.
- Important constituent of cells
- Forms energy rich bonds in ATP
- Forms co-enzymes
- Regulates blood and urinary pH
- Forms organic molecules like DNA & RNA

Hypophosphatasia

- It is a hereditary disease transmitted as a recessive autosomal characteristic. There is deficiency of serum alkaline phosphate. It resembles rickets. There is low level of serum alkaline phosphatase activity and elevated urinary excretion of phosphorylethanolamine which result in formation of defective bone matrix.

Types

- ***Perinatal type*** - it is diagnosed at birth and death occurs within few hours of birth. Death occurs due to respiratory failure.

- ***Infantile type*** - it appears at 6 months of age.

- ***juvenile* type**-- this appears in childhood and has wide range of clinical expression .

- ***Adult type*** - *it* is mild form and appear late in life.

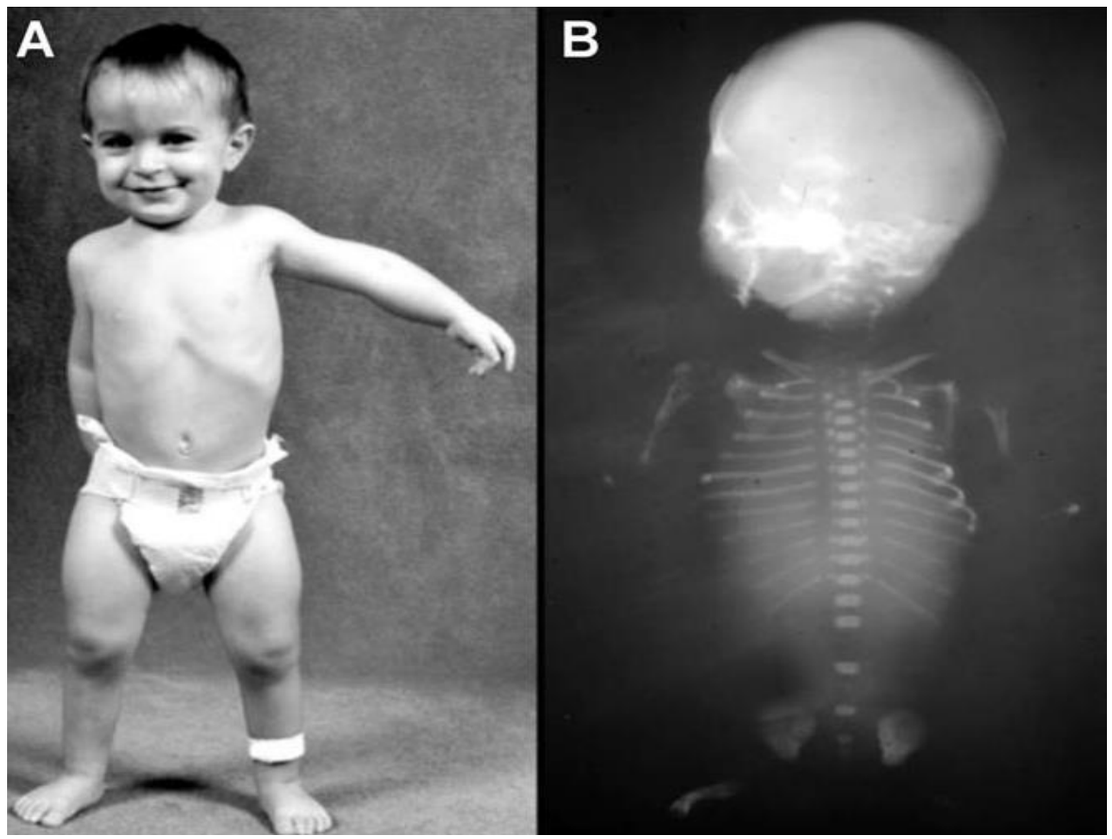
Clinical Features

- *Symptoms-there* is anorexia, irritability, persistent vomiting and mild pyrexia.

- *Infantile* form-severe hypocalcemia, bone abnormalities and failure to thrive manifest the infantile form. Most of the cases are lethal. Deformities of rib may be seen in these patients.

- *Juvenile* form-hypophosphatasia of childhood is characterized by increased infection, growth retardation and rochite-like deformities including deformed extremities, costochondral junction enlargement (rochite rosary) and pulmonary gastrointestinal and renal disorders.


A
B
C


A
B

- *Adults* form-the adult form includes fracture with a prior history of rickets and osseous radiolucency. Stress fracture of metatarsal bones of feet may be presenting sign of this condition.
- *Homozygous involvement-disease* with homozygous involvement begins *in utero* and patient die within 1st year. Bowed limb bone and marked deficiency of skull ossification.
- *Heterozygous involvement-in* heterozygous, there is milder effect with poor growth and fracture and deformities.
- *Skull-skull* suture close early resulting in bulging suture and gray marking on internal surface of skull which occur due to increased intracranial pressure. Shape of skull is brachiocephalic.

- ***Pseudohypophosphatasia-****disease* resembling classic hypophosphatemia butwith normal serum alkaline phosphatase level. There is osteopathy of long bones and skull.

There is **premature loss of deciduous teeth**.

Calcium

- Most abundant of all minerals in body.
- Total content in adult male is about 1- 1.5 kg.
- Out of this 99% is present in bones & teeth &1% outside skeletal tissue.

Blood calcium -

- Most of blood Ca is present in plasma.
- Normal serum Ca is 9-11 mg/di.
- About half of this (5mg/di.) is present in ionised state which is functionally most active.

Dietary requirements

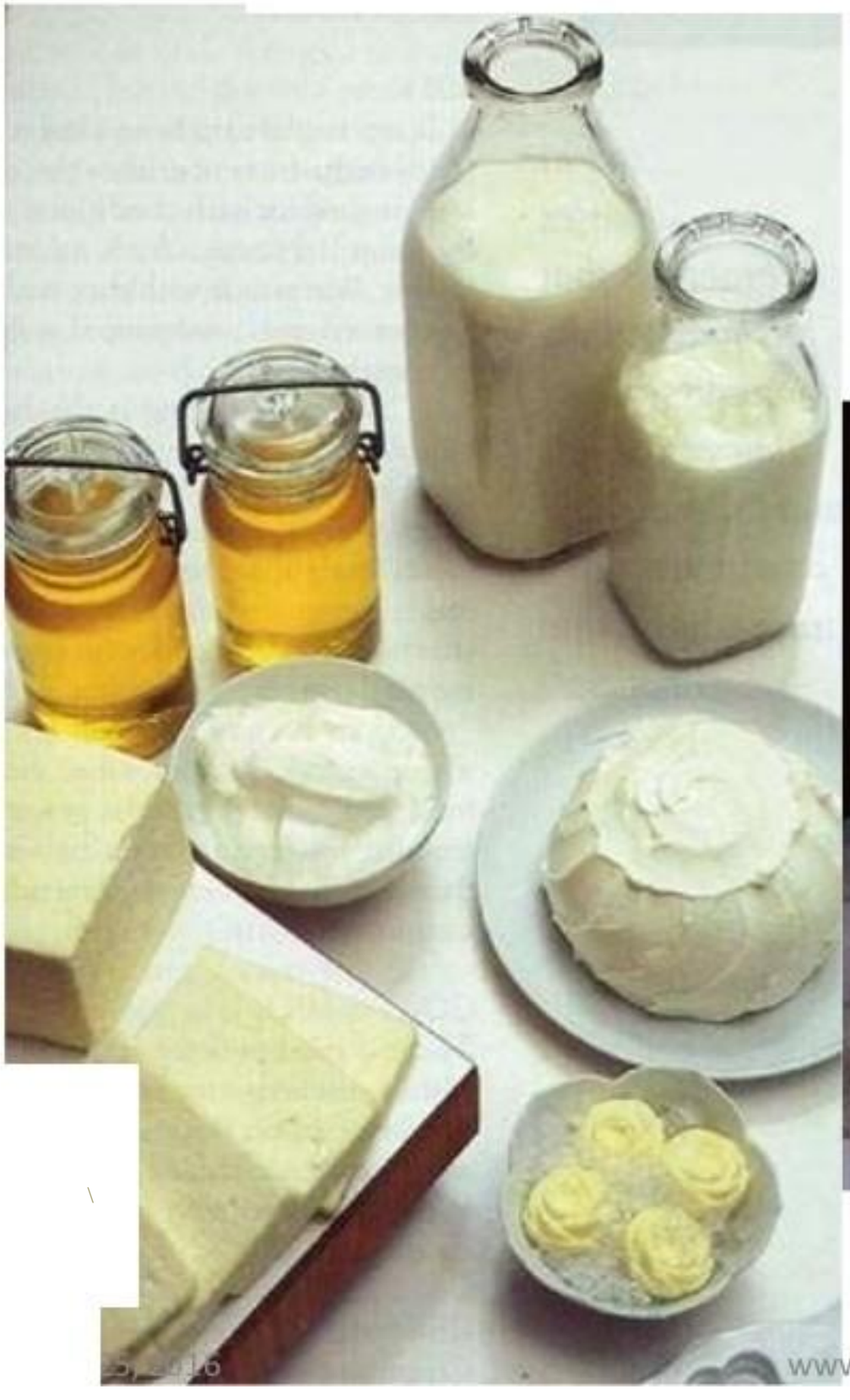
- Infants - 300-500 mg / day.

- Children (1 -18 yrs) - 0.8 - 1.2 g/ day.

- Adult men & women- 800 mg / day.

- Women during pregnancy, lactation & post menopause- 1.5 g/ day.

Best sources



Absorption

- Occurs in duodenum by active energy dependent process & passively in jejunum.
- It is influenced by several factors, either promoted or inhibited.

Factors interfere with calcium absorption:

- Phytic acid,
- Oxalic acid
- Hypochlorhydria or achlorhydria

Factors help in calcium absorption:

- Vitamin D
- Citrates (lower pH of intestinal tract)
- High protein diets

CALCIUM REGULATION

- PARATHORMONE
- CALCITONIN
- VITAMIN D
- GLUCOCORTICOIDS
- TESTOSTERONE
- ESTROGENS

Excretion of calcium

- It is excreted partly through kidney & mostly through feces.
- Feces - 80%
- Dermal losses - 50 mg/di

Functions:

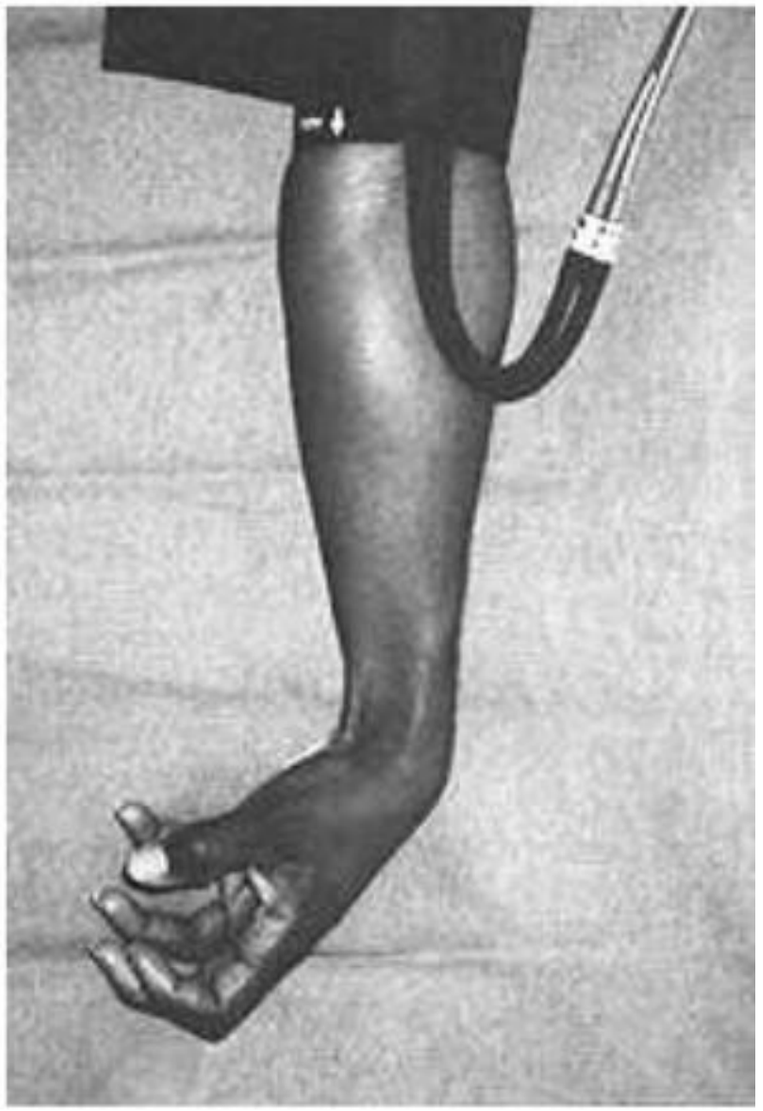
- Formation of bones & teeth
- Maintenance of skeletal & teeth structure
- Normal membrane permeability
- Normal heart rhythm
- Neuromuscular excitability
- Coagulation of blood
- Muscle contraction
- Messenger in hormone action

Pathologies related to Cametabolism

- Hypercalcemia
- Hypocalcemia
- Hyperparathyroidism
- Hypoparathyroidism
- Osteoporosis
- Rickets
- Osteomalacia
- Pathological calcification

TETANY

CHVOSTEK' S SIGN



TROUSSEAU'S SIGN

Fluoride

- Trace element
- Present in blood, bone & teeth

Sources of fluoride

- Food
- Water ingested daily
- Tea
- Rice
- Table salt
- Various spices

water fluoride level:

- 0.7-1.2 ppm fluoride--- optimum range
- 1 ppm fluoride--- serves daily requirement
- Lower concentration is recommended for warmer climates where water consumption is higher

Daily dietary fluoride intake:

- Should not exceed 3mg
- Because it is toxic element

Blood fluoride level

20 μg present in ionized form

Absorption

- From intestine
- Intakes of calcium above certain minimal levels will reduce absorption ofdietary fluoride

Excretion

- Urine

Functions:

- Helps in tooth & bone development
- Hardening of surface enamel (resisting acid dissolution)

Anticariogenic effect

- Fluoroapatite resists acid dissolution

Fluorosis

Excess of fluoride in drinking water or diet is harmful.

Characterized by

- Widespread calcification of tendons, ligaments and muscle sheaths
- Exostoses
- Osteosclerosis
- Collagen synthesis is affected
- ***dental fluorosis***

Dental fluorosis

Lusterless, white, and opaque enamel

Zones of yellow to dark-brown discoloration

Trace element	sources	Normal content	Related patholgy
IODINE	Present in sea food vegetables & milk	8- 12 *µg/dl* (blood level)	Hyper thyroidism, Hypothyroidism, Goitre.
COPPER	It is necessary for normal erythropoiesis as well as for iron absorption	100-150 mg (body content)	Microcytic hypochromic anemia, Wilson's disease, Menkes' syndrome.
IRON	Involved in normal metabolism of cells & storage iron as ferritin & hemosiderin.	2.3 -3.8 gm (body content)	Iron deficiency anemia, Bronze diabetes, Bantu siderosis
ZINC	Milk & dairy products, eggs, pulses, leafy vegetables.	1.4- 2.3 gm (body content)	Dwarfism, hypogonadism, disorders in bone growth, wound healing.

Trace element	sources	Normal body content	Related pathology
MANGANESE	Cereals , vegetables, fruit s, nuts & tea.	10- 18 mg (body content)	Alterations of bones, ataxia & inf ertilit y.
COBALT	An integral part of vitamin 812	5- 8 *µg* (body content)	Megaloblastic anemia, polycthemia
CHROMIUM	Have a role in carbohydrate & lipid metabolism	6- *20µg*	Malnutrition & total parenteral alimentation
SELENIUM	Prevents liver cell necrosis, supplementary to vitamin E	4- 10 mg (body content)	Liver cell necrosis, exudative diathesis, muscular dystrophies & myopathies.

VITAMIN

Introduction

- "vitamins have been defined as organic compounds which are required in minute amounts to maintain normal health of organisms'.

VITAMINS

Fat Soluble

- Vitamin A
- Vitamin D
- Vitamin E
- Vitamin K

WATER SOLUBLE

B COMPLEX

Energy-releasing

- Thiamine (Bi)
- Riboflavin (B_2)
- Niacin (B_3)
- Pyridoxine(B_6)
- Biotin (B_7)
- Pantothenic acid(B_5)

Hematopoitic

- Folic ci d(B_9)
- Vitamin B_{12} (cyanocobalamin)

Vitamin C

VITAMIN A

Is widely distributed in animal and plant foods

animals - pre-formed - Retinal.

plants - pro-formed - carotene

Dietary sources of vitamin A

PRE-FORM VITAMIN A

meat, liver & dairy products

PRO-FORM VITAMIN A

Yellow, red and Green vegetable and ftuit

Recommended dietary allowance :

- Men and women -600 mcg. Per day
- Pregnancy and lactation -950 mcg. Per day
- Infants -350 mcg. Per day
- Children -600 mcg. Per day

Absorption and storage:

- liver has enoromous capacity to store - in the form of retinol palmitate.

- under normal conditions a well-fed person has sufficient Vitamin A reserves to meet his need for 6 to 9 months or more.

- Free is highly active but toxic & therefore transported in blood stream in combination with retinol binding protein.

Physiological Functions of Vitamin A

- Epithelial cell "integrity'
  - Reproduction
  - Resistance to infectious disease
- Bone remodeling
- Growth and Vision

The signs of vitamin A deficiency

Ocular

- Night blindness
- Conjunctival Xerosis
- Bitot's spot
- Corneal xerosis
- keratomalacia

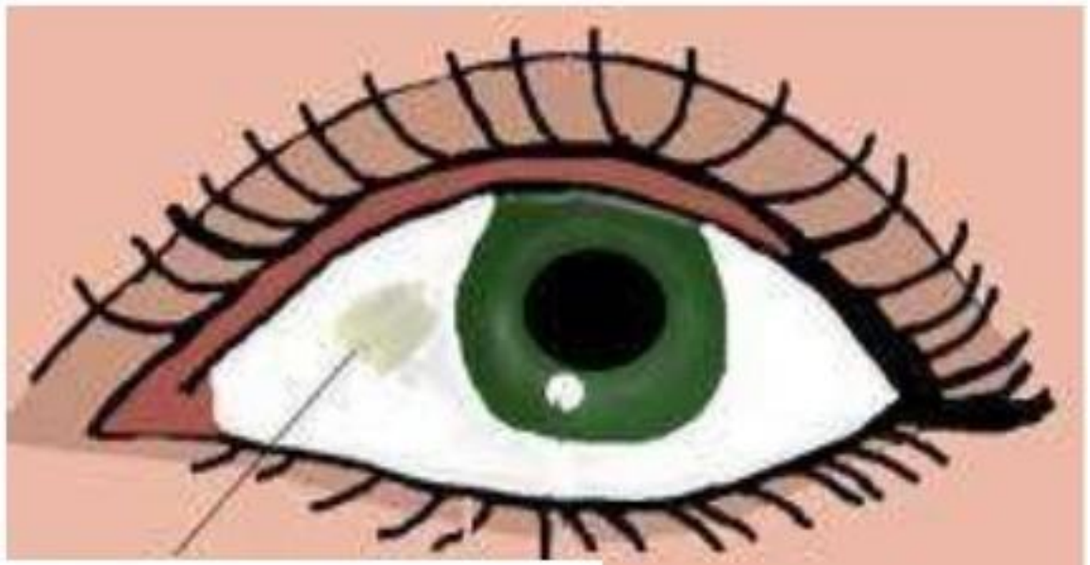
Extra ocular

Retarded Growth

Skin Disorder

Effect on reproductive organs.

Effect on bone



Conjunctiva l xerosis

Hyper Vitaminosis A

If the daily dose > **30,000 mcg**

toxic symptoms:-

- painful joint
- thickening of long bones
- Anorexia
- low grade fever
- rashes
- irregular menstruation
- fatigue
- loss of hair

ORAL MANIFESTATION OF VITAMIN A DEFICIENCY

- Teeth - vitamin A deficiency leads to defective formation of enamel.
- Hypoplasia of teeth - since enamel forming cells are disturbed.
- **Dentin - lacks** the normal tubular structure.
- Caries - there is increased risk for caries .
- Eruption - delayed in prolonged **deficiency.**

- retarted in alveolar bone formation rate .
- gingival epithelium becomes hyperplastic,in prolonged deficiency it shows keratinization .
- tissue is easily invaded by bacteria that may cause periodontal disease.
- Salivary gland - undergo typical keratinizing metaplasia .

TREATMENT

- vitamin deficiency should be treated urgently .

- depending upon deficiency symptoms it is given in the dose of 7,500 to 15,000 mcg per day for one month.

- nearly all of the early stages of xerophthalmia can b reverse;p by administration of a massive dose 2,00,000 IU or 110mg orally on two successive days.

- If Hypervitaminosis - restriction of diet.

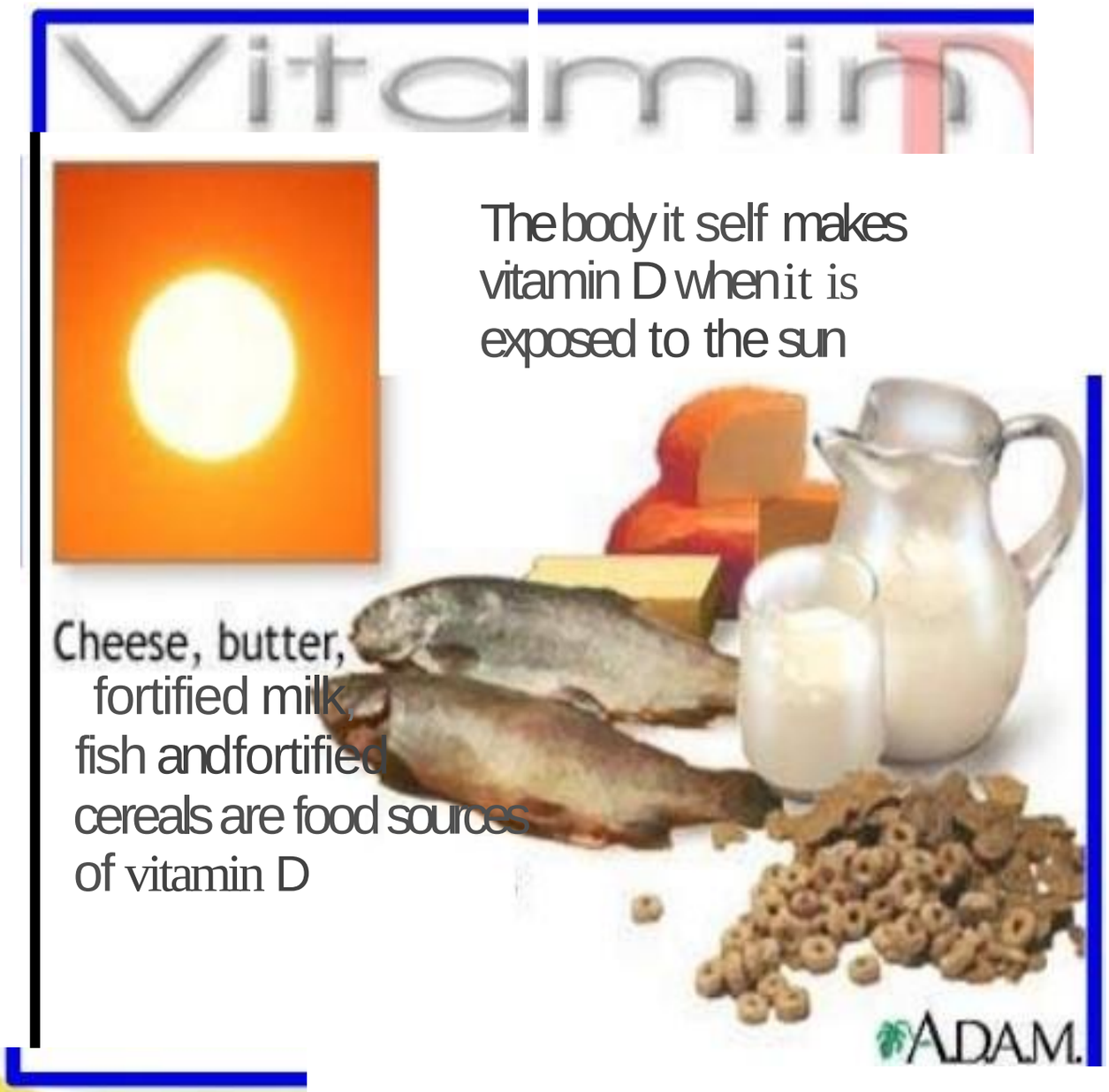
VITAMİN-D

- It is also called SUNSHINE VITAMIN.
- It is available in 2 forms

D3 - cholecalciferol

D2 - calciferol

Dietary Sources of Vitamin D



Chemical origins of vitamin D

- Precursors of vitamin D are found in both yeast and animal tissues. In yeast, a sterol precursor (**ergosterol)** is converted to vitamin D 2 **(ergocalciferol).** Ergocalciferol is the compound most commonly found as the additive to fortify milk.

- In the dermal tissue of animals, the precursor is 7- dehyd ro cholesterol which is converted first to a pre-vitamin D3, then to vitamin D3 (**cholecalciferol).**

- Vitamin D2and vitamin D3 are both converted to similar acti compotli ds (**calcidiol** and **calcitriol)** in the liver and kidn and D3 are sometimes referred to as

Recommended dietary allowance of Vitamin D

Daily requirement of vitamin-D is 400.

International Units of cholecalciferol. In countries with good sunlightQike India), RDA for vitamin D Is 200 international units per day

Absorption and Storage of vitamin- D

This depends upon the source of the vitamin **D**.

- Vitamin **D** that is taken into the gut (vitamin **D-**Containing foods or nutritional supplements) are absorbed by intestinal mucosal cells in the duodenum and jejunum and packaged into lipoproteins called **chylomicrons (CM).** The CM carry the vitamin to the liver or adipose for storage and eventual use.
- Vitamin D synthesized in skin through the action of sunlight is bound to a blood protein called (vitamin) binding protein **(DBP),** which transports it to the liver.

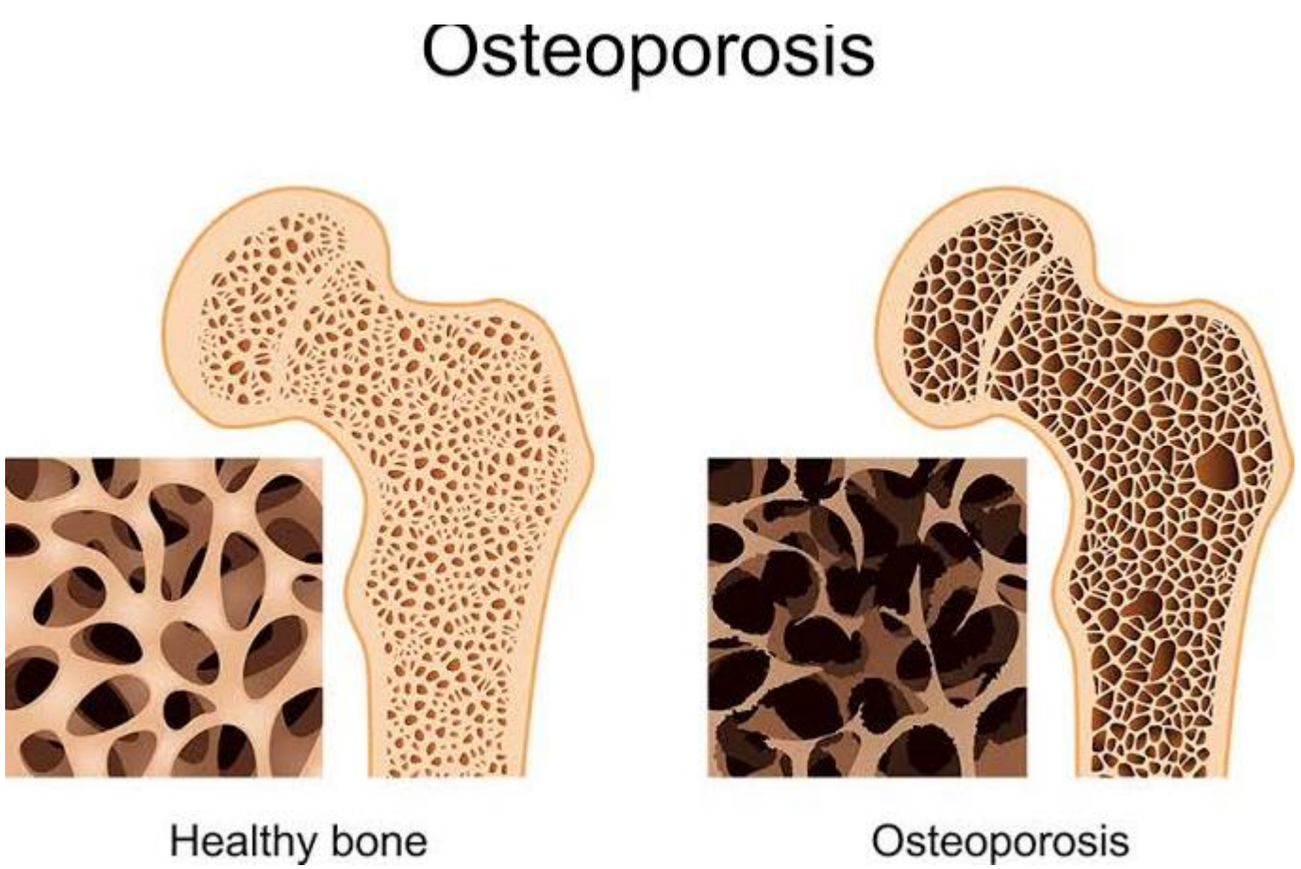
Biochemical functions of vitamin-D

- It regulates the plasma levels of calcium and phosphate . Calcitrol acts at 3 different levels to maintain plasma calcium(normal 9-11mg/dl)

- Action of calcitriol on intestine
- Action of calcitriol on bone
- Action of calcitriol on kidney

Vitamin D - Deficiency

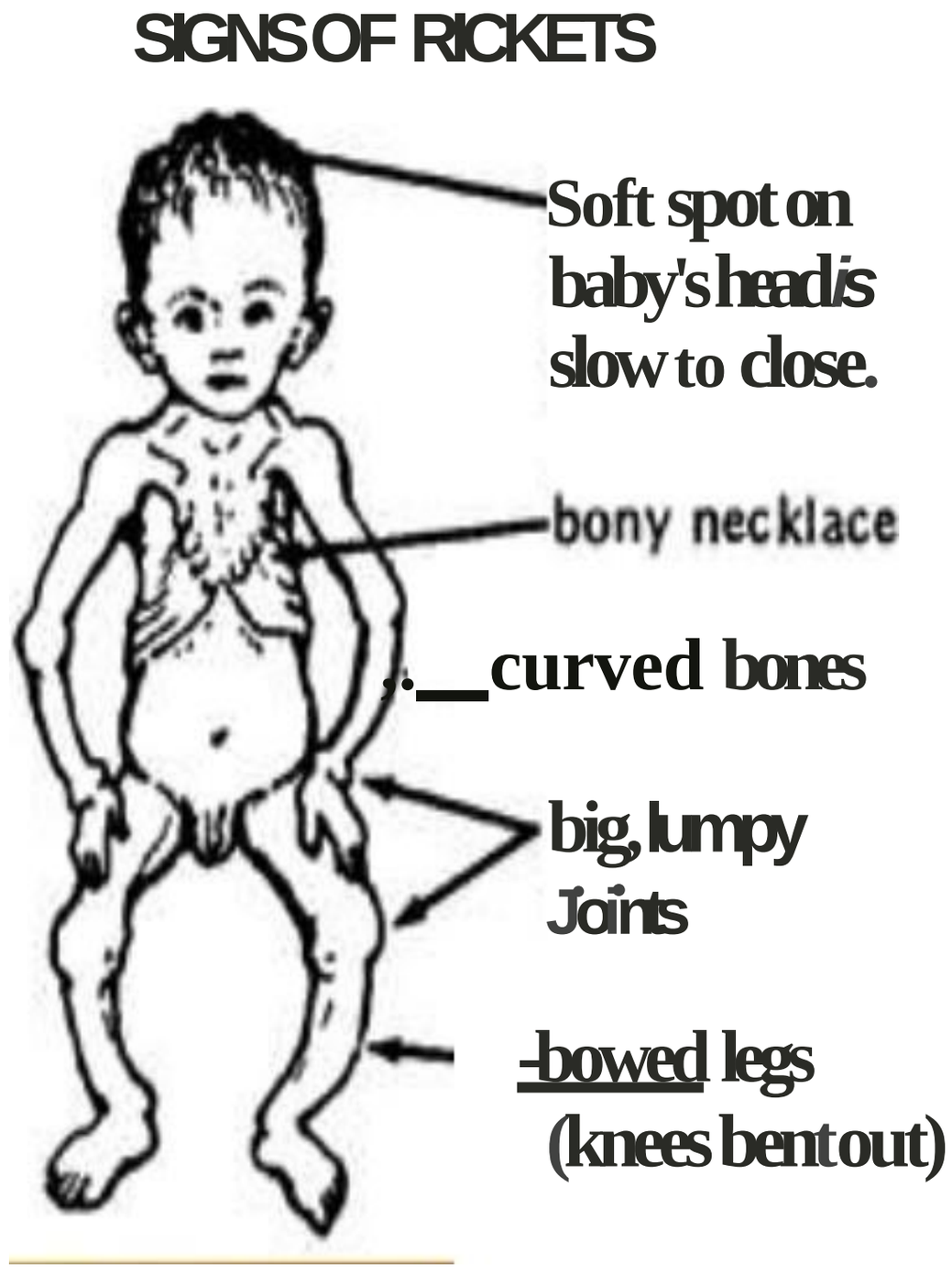
- RICKETS : Children's
- OSTEOMALACIA : Adults
- Increase the risk of **Osteoporosis**



Rickets

- The term is derived from the old English word for "twist"or"wrick,"
- Rickets is caused by a deficiency in **vitamin D.** During growth, human bone is made and maintained by the

interaction of **calcium, phosphorus,** and vitamin D. Calcium **is** deposited in immature bone (ostecld) in a process called calcification, which transforms immature bone into its mature and familiar form.

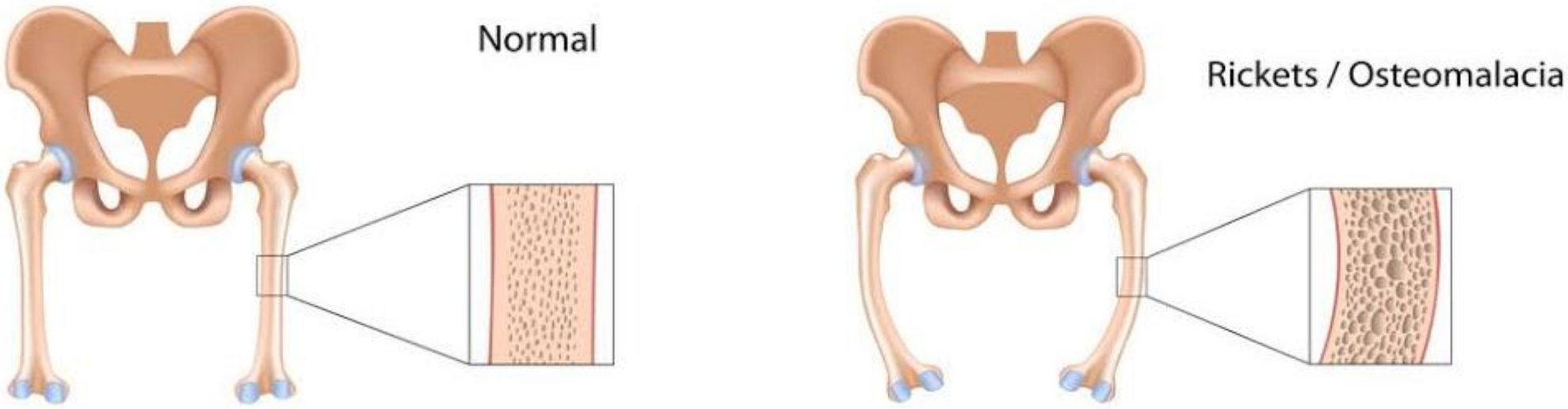


Osteomalacia

- Itis also known as adult rickets .flat bones and diaphysis of long bones are affected

- It is most commonly seen in post menopause female with history of low dietary calcium intake.

- Majority of patient have bone pain & muscle weakness..



Oral manifestation

- Teeth - developmental abnormalities of dentine & enamel.
- Caries - higher risk of caries
- Enamel - there may be hypoplasia of enamel, may be mottley, yellow gray in color
- Pulp - high pulp horns, large pulp chambr, delayed closure of root apices

MANAGEMENT

- Dietary enrichment of vitamin D in the form of milk
- Curative treatment includes 2000 to 4000 IU of calcium daily for 6 to 12 weeks.
- Patient with osteomalacia due to intestina malahsorption require larger dose of vitamin & calcium i.e. 40,000 to 1,00,000 IU **vitamin** D&15 to 20 gms of calcium lactate

VITAMIN-E

- Vitamin E is a naturally occurring antioxidant. It is essential for normal reproduction in many animals, hence known as *anti sterility vitamin.*

Dietary sources ofVitamin-E

- Many vegetable oils are rich source of vitamin E.
- Wheat germ oil, cotton seed oil, peanut oil, corn oil, sunflower oil.
- Italso present in meat, milk, butter and eggs

Recommended dietary allowance of vitamin E

A daily consumption of

- For men- 10 mg
- For women- 8 mg
- Vitamin E supplemented diet is advised for pregnant and lactating women.

Biochemical functions of vitamin E

- Vitamin E is essential for the membrane structure and integrity of the cell , hence it is regarded as membrane antioxidant .
- It prevents the peroxidation of polyunsaturated fatty acids in various tissues and membranes . Iy protects RBC from hemolysis by oxidizing agents .
- It preserves and maintains germinal epithelium of gonads for proper reproductive function and sterility .
- It protect liver from being damaged by toxic compounds such as carbon tetrachloride.
- It works in association with Vitamins A ,C and beta carotene , to delay the onset of cataract.
- It is believed that it prevents the oxidation of PDL , which have been implicated to promote heart diseases .

Deficiency of vitamin E

- Sterility
- Degenerative changes in muscle
- Megaloblastic anaemia
- Changes in central nervous system

Oral manifestations

- loss of pigmentation
- atrophic degenerative changes in enamel

Vitamin-K

It is available in 2 forms

- **Kl -** it is the form occurs in plant origin.
- **K2 -** is synthesized by intestinal bacteria.

Dietary sources of vitamin K

- Milk
- Meat
- Fish
- Spinach
- Cabbage
- Cauliflower
- Soya bean
- Wheat germ
- Carrots
- Potato
- Tomatoes

Recommended dietary allowance of vitamin K

- men and women - 70 -140 mcg. Per day
- children- 35 -75 mcg per day

Biochemical functions of vitamin K

- It is essential for the hepatic synthesis of coagulation factor II, V, VII, IX, X.

CLOTTING- it prevents hemorrhage only in cases when there is defective production of prothrombin

OXIDATIVE PHOSPHORYLATION it is as a co-factor in oxidative phosphorylation associated with lipid .

DEFICIENCY OF VITAMIN K

Causes

Decrease synthesis of factor 2,7,9,10

Increase clotting time

Prolonged bleeding time

Hemorrhagic condition

After **antibacterial** therapy,
Surgical **operations-**

Cholecystectomy
Conditions like Malabsorption, obstructive jaundice

Oral manifestation

- Spontaneous gingival hemorrhage
- Gingival bleeding after tooth extraction and minor surgical procedure .

Blood clotting

Bleeding in nose

Vitamin-C

- It is also called ascorbic acid and antibiotic vitamin.
- It is most active reducing agent.
- It is powerful antioxidant

Dietary sources of vitamin C

- Citrus fruits, gooseberry, guava, green vegetables, tomatoes, potatoes are rich in ascorbic acid.
- High content of vitamin C is found in adrenal gland and gonads.
- Milk is poor source of ascorbic acid

Recommended daily allowance of vitamin C

- Adults : 60-70 mg per day
- Additional intake (20-40 %o increase) are recommended for women during pregnancy and lactation

Biosynthesis and metabolism of vitamin C

Men cannot synthesize ascorbic acid due to the deficiency of single enzyme namely gulonolactone oxidase

Biochemical functions of vitamin C

1. Collagen formation
2. Bone formation
3. Iron and hemoglobin metabolism
4. Tryptophan metabolism
5. Tyrosine metabolism
6. Folic acid metabolism
7. Synthesis of corticosteriod harmones
8. s p a r action of other vitamins
9. Immunological function
10. Preventive action on chronic diseases

Vitamin C deficiency

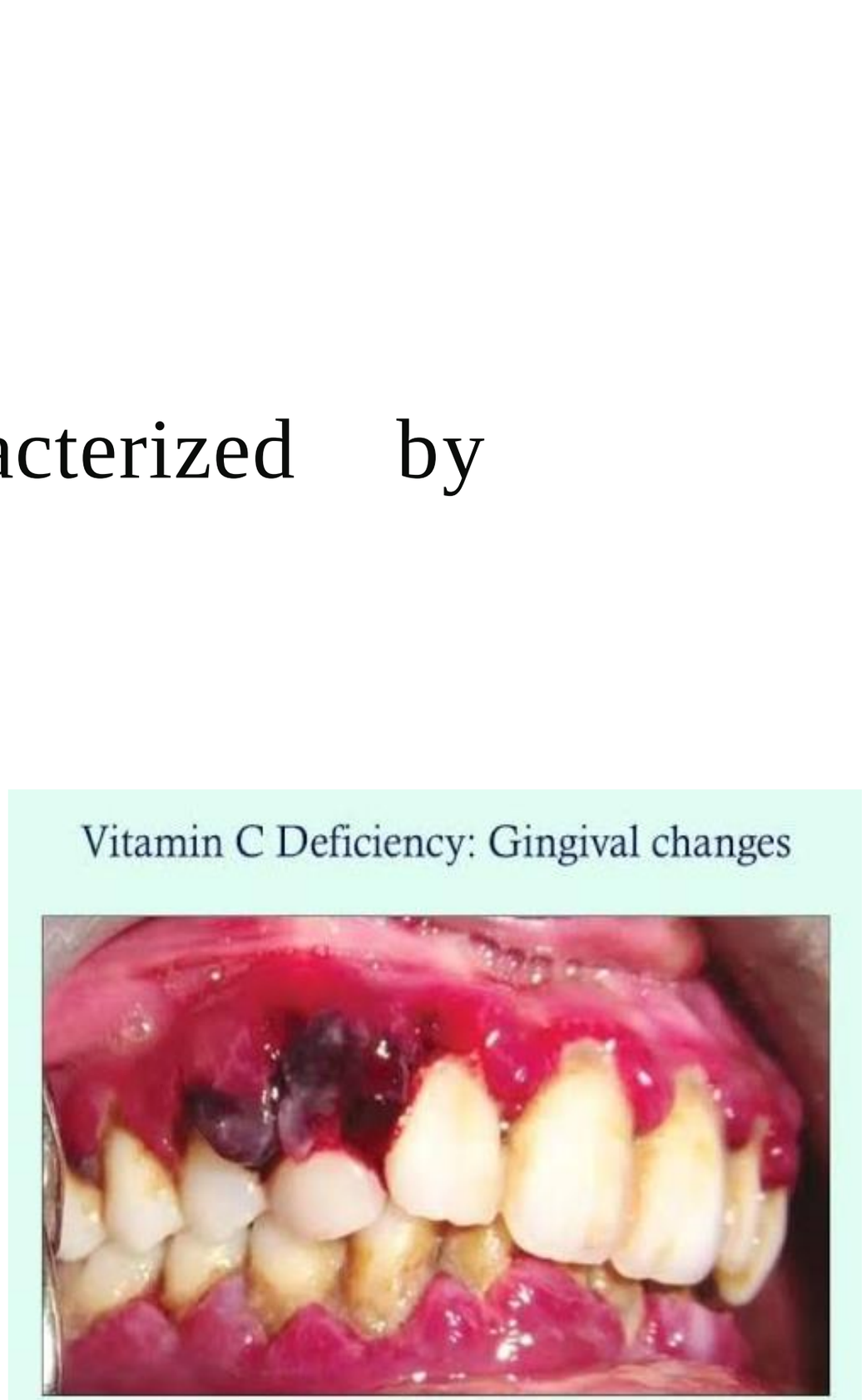
SCURVEY : this disease is characteized by

- Spongy and sore gums
- Loose teeth
- Anemia
- Swollen joints
- Decreased immunocompetence
- Delayed wound healing
- Hemorrhage
- Osteoporosis

Oral manifestations

Scorbutic gingivitis: characterized by

- Ulcerative gingivitis
- Rapid periodontal pocket development
- Tooth exfoliation



vitamins B complex

- Most B complex occurs in nature in the bound form within the cells of vegetables or animal tissues.
- digestion for the liberation of vitamins and its absorption is a result of breakdown of cellular structures in the gut.
- Excretion of vitamins occurs in the kidney

THIAMINE (VIT B1)

- It is also called **Anti Beri-Beri factor, Anti Neuritic factor and** also **Aneurin.**

- It has a specific coenzyme, thiamine pyrophosphate(TTP) which is mostly associated with carbohydrate metalolism

- Itis colorless basic organic compound

  Composed of a sulfated pyramiding ring.

Dietary source of thiamine

- cereals, Pulses, oil seeds, nuts, yeast. Polishing of rice removes about 80% of thiamine

- Animal: pork:, liver, heart, kidney, milk:

Recommended dietary allowance of vitamin B1

- Men - 1.3 mg per day
- women - 1.0 mg per day
- Pregnancy and lactation-2 mg .per day
- Children -1.lmg.per day

Biochemical functions of vitamin B_1

- The coenzyme, thiamin pyrophosphate or cocarboxylase is intimately connected with the energy releasing reactions in the carbohydrate metabolism.
- TPP plays an important role in the transmission of nerve impulse. It is believed that **TPP** required for acetylcholine synthesis and the· translocation of neural tissue

Deficiency of vitamin B1

- **Nervous disorders** - when cells cannot metabolize glucose, it affects the nervous system first, since it depends entirely on glucose for its energy requirement. & There is mental depression.

- **Digestive symptoms-** it occurs due to defecti hydrochloric acid production in the stomach complains of loss of appetite, poor digestion, loss weight

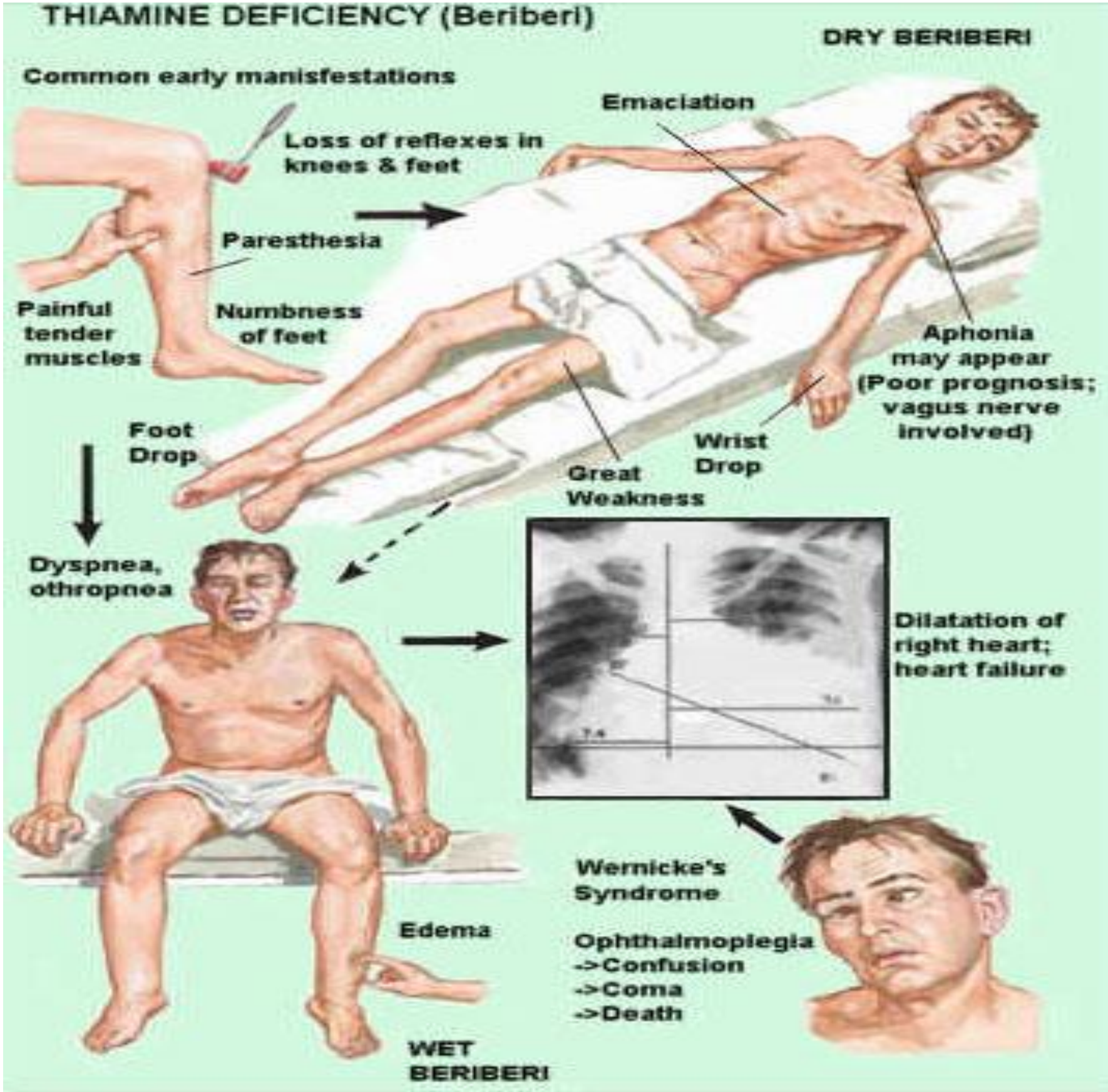
Deficiency of vitamin B1

- **BERI BERI**
  - DRY BERI BERI
  - WET BERIBERI..
  - INFANTILE BERI BERI
- Other diseases which can be associated with it are
  1. wernickes encephalopathy
  2. peripheral neuritis
  3. korsakoff 's psychosis

Oral manifestations

There is hypersensitivity of oral mucosa

Pain in tongue, teeth, jaw, and face


THIAMINE DEFICIENCY (Beriberi)
DRY BERIBERI
Common early manisfestations
Loss of reflexes in knees & feet
Paresthesia
Painful tender muscles
Numbness of feet
Emaciation
Aphonia may appear (Poor prognosis; vagus nerve involved)
Foot Drop
Wrist Drop
Great Weakness
Dyspnea, othropnea
Dilatation of right heart; heart failure
Wernicke's Syndrome
Ophthalmopiegia
->Confusion
->Coma
->Death
Edema
WET BERIBERI

Management

- Complete rest
- Thiamine 50 mg IM for 3 days then 10 mg 3 times daily by oral route.
- Infantile beriberi is treated via mothers milk:. The mother should receive 10,000 mcg twice daily, in additiop infant should be given thiamine in doses of 10,000 to 20,000 mcg IM once in a day for 3 day
- Thiamine antagonists:
  - Pyrithiamine and oxythiamine are the two important antimetabolites of thiamine

Riboflavin (Vit B2)

- Riboflavin through its coenzymes takes part in a variety of cellular oxidation reduction reactions.
- Coenzymes of riboflavin
- Flavin mononucleotide (FMN) and flavin adenine dinucleotide (FAD) are the two coenzyme forms of riboflavin

Dietary sources of vitamin B2

- Milk and milk products, meat, eggs, liver, kidney are rich sources. Cereals, fruits, vegetables and fish are moderate sources

Recommended dietary allowance of vitamin B2

- Adults: 1.2-1.7 mg/day
- Higher intakes (0.2-0.5 mg/ day) are advised for pregnant and lactating women.

Biochemical functions of vitamin B2

- The flavin coenzymes participate in many redox reactions responsible for energy production
- The coenzymes, FAD and FMN are associated with several enzymes involved in carbohydrate, lipid, protein, and purin metabolism

Deficiency of vitamin B2

CHEILOSIS

- Chapping & fissuring of lips

GLOSSITIS

- Inflammmed
- Red beefy tongue

Niacin (Vit B3)

- Niacin or nicotinic acid is also known as Pellagra preventive(p.p) factor of Goldberg.
- The coenzymes of niacin (NAD and NADP) can be synthesized by the essential amino acid, tryptophan.

Dietary sources of Niacin

Rich natural sources:

Liver

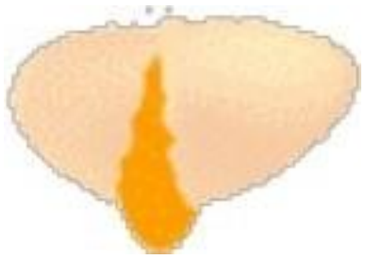
Yeast

Whole

grain

Cereals

Pulses



Moderate sources:

milk

Fish

Eggs

vegetables

Recommended dietary allowance of Niacin

- Adults : 15-20 mg/ day
- Children: 10-15 mg/ day
- 1 mg niacin= 60 mg tryptophan

Biochemical functions of Niacin

- The coenzymes NAD and **NADP** are involved in a variety of oxidation reduction reactions.
- A large number of enzymes belonging to the class oxidoreductases are dependent on NAD or **NADP.**

Deficiency of Niacin

- its deficiency results in **pellagra.**
- it is called disease of 3 Ds

DERMATITIS **DIARRHOEA**

DEMENTIA

- **skin -** in acute cases, skin lesions may produce vesiculation, cracking, exuda tion, crusting with ulceration & secondary infection
- Chronic ,ases - dermatitis occurs as roughening & thickening of skin.
- **If not treated may rarely lead to 4th D- death**

Oral manifestation

- Oral mucosa - becomes fiery red & painful & salivation is profuse.

- Tongue -epithelium of entire tongue is desquamated.

  The tongue becomes red swollen & beefy .

- In advaitced cases, the tongue loses all the papillae & reddening becomes intense.

Pantothenic Acid (Vit B5)

- Pantothenic acid (Greek: pantos- everywhere), formely known as chick anti dermatitis factor. Its metabolic role as coenzyme A is also widespread

Recommended dietary allowance of vitB5

Adults: **5-10** mg

Dietary sources of pantothenic acid

- It is one of the most widely distributed vitamins found in plants and animals.
- The rich sources are egg, liver, meat, yeast, milk

Biochemical functions of VitB5

- function of pantothenic acid are exerted through coenzyme A or CoA (A for acetylation).
- Co enzyme A serves as a carrier of activated acetyl or acyl groups. This is comparable with ATP which is a carrier of activated phosphoryl groups.

Deficiency of VitB5

- Burning feet syndrome: pain and numbness in toes, sleeplessness, fatigue

pyridoxin (VitB6)

- Vitamin B6 is used to collectively represent the three compounds namely pryidoxine, pyridoxol and pyridoxamine

Dietary sources of vitamin B6

Vegetable sources:

Wheat Corn Cabbage

Tubers

Animal souces: egg yolk , fish, milk, meat

Recommended dietary allowance of vitamin B6

- Adults : 2.2 mg /day
- During pregnancy, lactation and old age an intake of 2.5 mg/day recommended
- Isoniazide , deoxypyridoxine and methoxy pyridoxine are the antagonists of vitamin B6

Biochemical functions of pyridoxine

- Pyridoxal phosphate, thecoenzyme of vitamin B6 is found attached to the e- amino group of lysine in the enzyme .
- Pyridoxal phosphate participates in reactions like transamination, decarboxylation, deamination, transsulfuration, condensation

Deficiency of vitamin B6

- Neurological symptoms such as:

depression,irritability, nervousness confusion

- Convulsions and peripheral neuropathy are observed in severe deficiency.
- These symptoms are related to the decreased synthesis of biogenic amines

(GABA,seratonin,epinephrine,norepinephrine)

- Demyelination of neurons

Oral manifestation

Pellagrous stomatitis , Angular cheilosis

SYMPTOMS OF VITAMIN B6 DEFICIENCY

SORE GLOSSY TONGUE

CRACKED AND SORE LIPS

SKIN RASHES

Biotin (Vit B7)

- Biotin (formerly known as anti egg white injury factor, vitamin B7 or vitamin H) is a sulfur containing B- complex vitamin. It directly participates as a coenzyme in the carboxylation reactions.

Dietary sources of biotin

The rich sources are liver, kidney, egg yolk, milk, tomatoes, grains

Recommended dietary allowance of biotin

- Men -women - 100- 300 mcg/day
- Children - 50-200 mcg./day
- Infant - 35 mcg/day

Biochemical functions of Biotin

- Biotin serves as a carrier of CO_2 in carboxylation reactions.
- As a coenzyme , biotin is involved in various metabolic reactions
- Gluconeogenesis and citric acid cycle
- Fatty acid synthesis

Deficiency of Biotin

- Loss of appetite
- Anaemia
- Nausea
- Dermatitis
- Glossitis
- Biotine deficiency is uncommon as it is well distributed In foods and also supplied intestinal bacteria

Folic Acid (Vit B9)

- Folic acid or folacin (latin: folium-leaf) is abundantly found in green leafy vegetables.
- It is required for synthesis of certain aminoacids, purins and pyrimidine- thymine.

Dietary sources

- Adults : 200 mcg
- Higher intakes are recommended during pregnancy and lactation: 400 mcg

Recommended dietary allowance of folic acid

- Rich sources are green leafy vegetables, whole grains, cereals, liver, kidney, yeast and eggs.
- Milk is rather a poor source of folic acid

Biochemical function

1. Aminopterin and methopterin are structural analogues of folic acid . They competetively inhibit dihydrofolate reductase and block formation of THF .
2. Aminpterin and methotrexate are used in treatment of many cancers.
3. Tetra hydrofolate, the coenzyme of folic acid is actively involved in the one carbon metabolism

Deficiency of folic acid

- Macrocytic anemia
- Folic acid deficiency in pregnant women may cause neural defects in the fetus.Hence,high doses of folic acid are recommended during pregnancy to prevent birth defects.
- Formiminoglutamate (FIGLU) in urine is us to assess folic acid deficiency.

Oral manifestation

Defective keratinization and increase susceptibility of infection in oral mucosa

Gingivitis and oral ulceration

Atrophy of filiform and fungiform papilla of tongue with glossitis

Cyanocobalamine (Vit B12)

- Most of the therapeutic preparations contain cyanocobalamine. It is sensitive to light. It is heat liable.

Dietary sources

- It is mainly present in animal sources. Liver, kidney, brain, meat, fish and eggs are good souces.
- Milk and milk products are fair sources. curd is better source than milk.
- Since, it Is absent in plant foods, vegetarians ar likely o develop Vit B12 deficiency.

Recommended dietary allowance of Vit B12

- Adults : 3 mcg per day
- Children : 0.5-1.5 mcg per day
- During pregnancy and lactation : 4 mcg per day

Biochemical functions of Vit B12

- Vitamin B12 acts as the prosthetic group or coenzyme. Vitamin B12 enzymes are called cobamide enzymes.
- About ten enzymes requiring Vit B12 have been identified, most of them are found in bacteria.
- Synthesis of methionine from homocystein
- Isomerization of methymalonyl Co A

Deficiency of Vit B12

- Perinicious anemia: it is characterized by low hemoglobin levels, decreased number of erythrocytes and neurological manifestations.
- The excretion of methylmalonic acid in urine and estimation of serum B12 level are used to assess B12 deficiency.

oral manifestations

- Glossitis
- Glossodynia
- Recurrent ulcer
- Cheilitis
- Lingual paresthesia
- Burning sensation