पाण्डु रोग

(ANEMIA)

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पाण्डु रोग

निरुक्ति

पाण्डु पाण्डुस्तु पीत भागार्धः केतकीधूलिसन्निभः

परिभाषा

सोल्परक्तोलपमेद्वास्को निःसारः शिथिलेन्द्रियः।

वैवर्ण्य भजतेः तस्य हेतू श्रणु सलक्षणम् ö

च.चि. १६/६

सम्प्राप्ति चक्र

पित्तादि प्रकोपक आहार विहार

वायु द्वारा समस्त देह में पित्त का प्रसर

कफ, वात, रक्त, का दुषण

रक्ताल्पता

त्वचा का श्वेतपीत वर्ण, विवर्णता

पाण्डु रोग

प्रकार

- वातज्,
- पित्तज्,
- कफज्,
- सनिपातज्,
- मृत्तिकाभक्ष्णजन्य,

(च.सू.वा.)

पूर्वरुप :-

हृदयस्पन्दनं रौक्ष्यं स्वेदाभाव: श्रमस्तथा।।

(च.चि. १६/१२)

सामान्य लक्षण :-

संभुतेस्मिन् भवेत् सर्वः कर्णक्ष्वेडी हतानालः।
दुर्बलः सदनोन्नाद्दिट् श्रम्रभ्रमनिपीडितः॥
गात्रशूल ज्वरश्वासगौरवारुचिमान्नरः।
मृदितेरिव गात्रैश्च पीडितोन्माथितैरिव॥
शूनाक्षिकुटो हरितः शीर्णलोमा हतप्रभः।
कोपनः शिशिरद्वेषी निद्रालू ष्ठीवनोल्पवाक्॥
पिण्डकोद्वेष्टकरपरूपादरूक्सदनानि च।
भवन्त्या रोहणासैविशेषाश्चास्य वक्ष्यते ॥

(च.चि. १६/१३–१६)

चिकित्सा सूत्र

तत्र पाण्ड्वामयी सिनगधस्तीक्ष्णैरूर्ध्वानुलोमकै।
संसोध्यो:.....:

(च.चि. १६/२५)

वातिके स्नेहभूयिष्ठं पैत्तिके तिक्तशीतलम्।
श्लेष्मिके कटुतिक्तोष्णं विमिश्रं सन्निपातिके॥

(च.चि. १६/१३६)

नाभ्यां समन्ततः शोथः श्वेताक्षिनखवक्रता।
पाण्डुरोगेग्निसादश्च श्वथुश्चाक्षिकूटयो:॥

का.सू.२५/३४

चिकित्सा

दाडिम घृत्

- कटूकाद्य घृत,
- पथ्या घृत,
- दन्ती घृत,
- द्राक्षा घृत,
- हरिद्रादि घृत,
- विशालादि फाण्ट,
- गौमूत्र हरीतकी,
- नवायसलौह,
- मण्डूर वटक,
- योगराज,
- शिलाजतु वटक,
- पुनर्नवा मण्डूर,
- घाट्यावलेह,
- बीजकारिक।

ANEMIA

DEFINITION

Anemia(an-without emia blood)

A decrease in the RBCs count, hemoglobin, and/or hematocrit values resulting in a lower ability for the blood to carry oxygen to blood tissue is called anemia.

PREVALENCE

- 90 % OF globally children

79% of indian children

71% of urban children

84% of rural children

pathophysiology

Decrease In RBCs, hb%,hct level

|

Diminised O2 carrying capacity

|

Hypoxia & hypoxia induced
effects on organ function

|

Signs & symptoms of anemia

Cuttoffs for Hb% HCT to define anemia

Age group	Hb(gm/dl)	HCT(%)
6 mo- 5 yr	<11	<33
5-11yr	<11.5	<34
12-13yr	<12	<36
Non pregnent women	<12	<36
Men	<13	<39

Red cell incidices in normal children`

Red cell incidies	At birth	0.5 – 2 yr	6 – 12yr	12 - 18 yr
MCV	108	78	86	90
MCH	34	27	29	30
MCHC	33	33	34	34

EVALUTION FOR ETIOLOGY

- Maternal infections
- Prematurity
- Blood loss (malaria , epistaxis)
- Jaundice
- G6pd deficiency
- Sepsis
- ABO/ Rh incompatibility
- Cephalohemotoma

Cont....

- Heminegiomas
- Chronic dirrohea
- Prior surgery
- Drug intake(eg. Anticonvulsants etc.)
- Renal disease
- liver disease
- Family h/o severe anemia
- h/o gall stone
- Recuurrent jaundice

SYMPTOMS

- Lassitude
- Easy fatigability
- Anorexia
- Irritability
- Poor school performance
- Dyspnea on exertion
- Tachycardia

Count….

- Dizziness
- Headache
- Tinnitus
- Lack of concentration
- drowsiness
- Palpitation

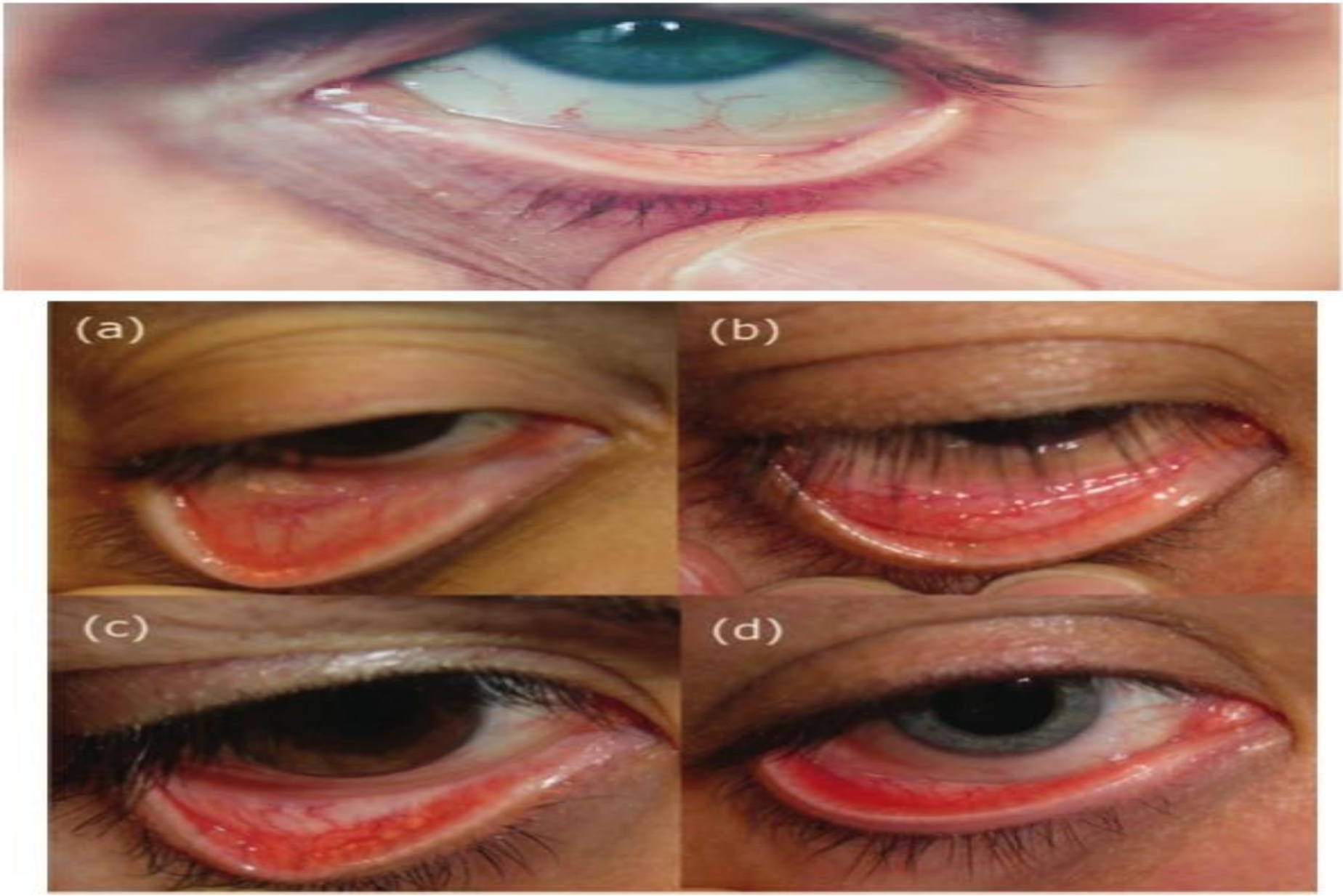
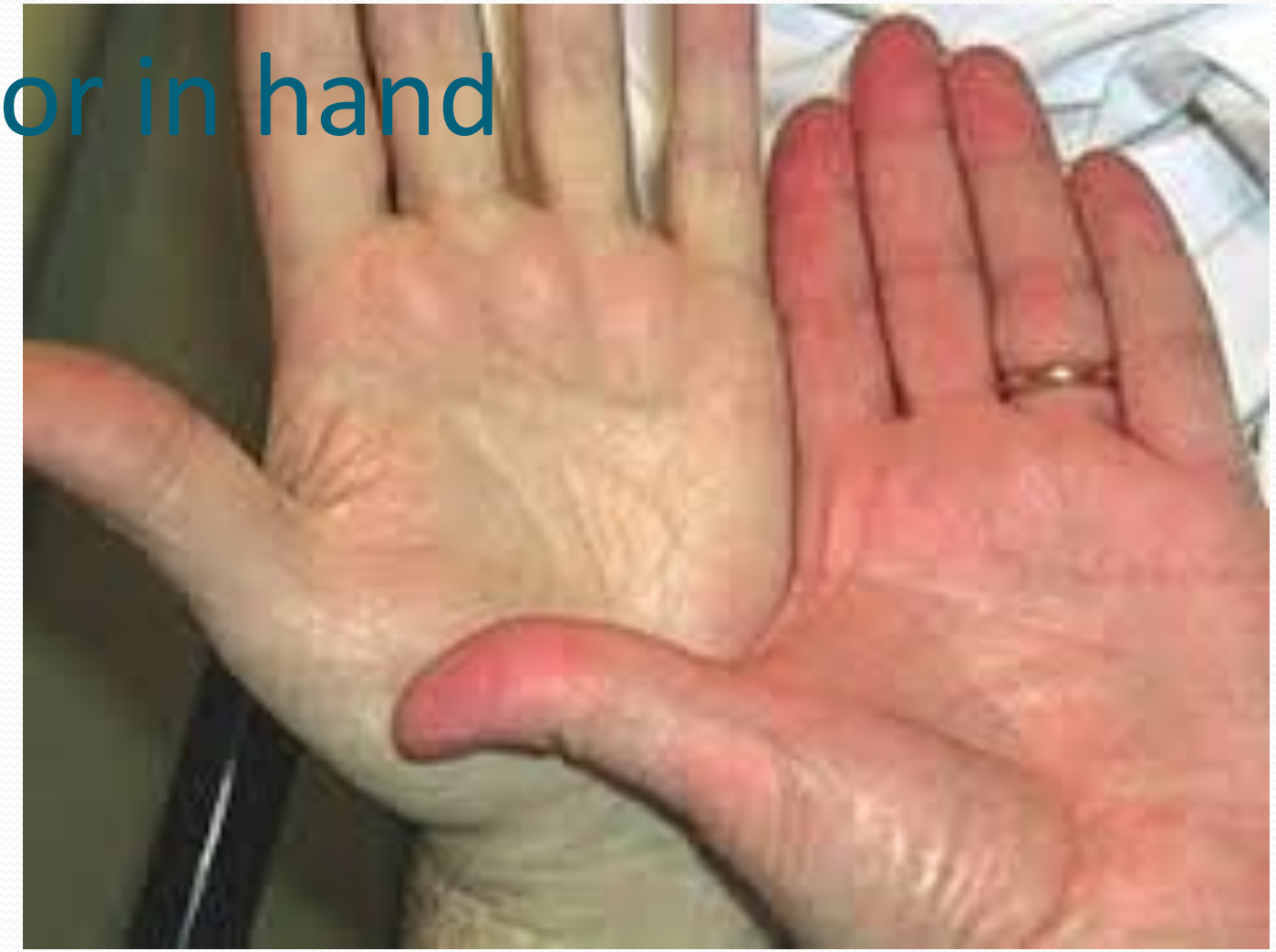


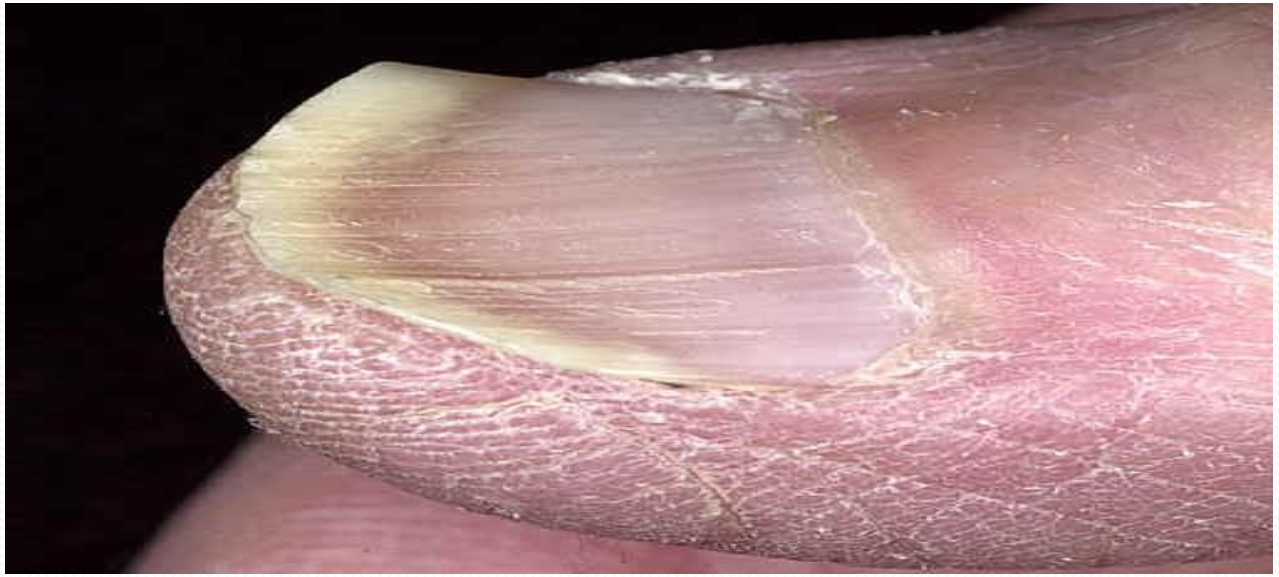
Fig. 2 Digital photographs of the palpebral conjuctiva of four patients with hemoglobin concentrations of (a) 7.3, (b) 12.7, (c) 14.0, and (d) 14.5 g/dL.

Pallor in hand

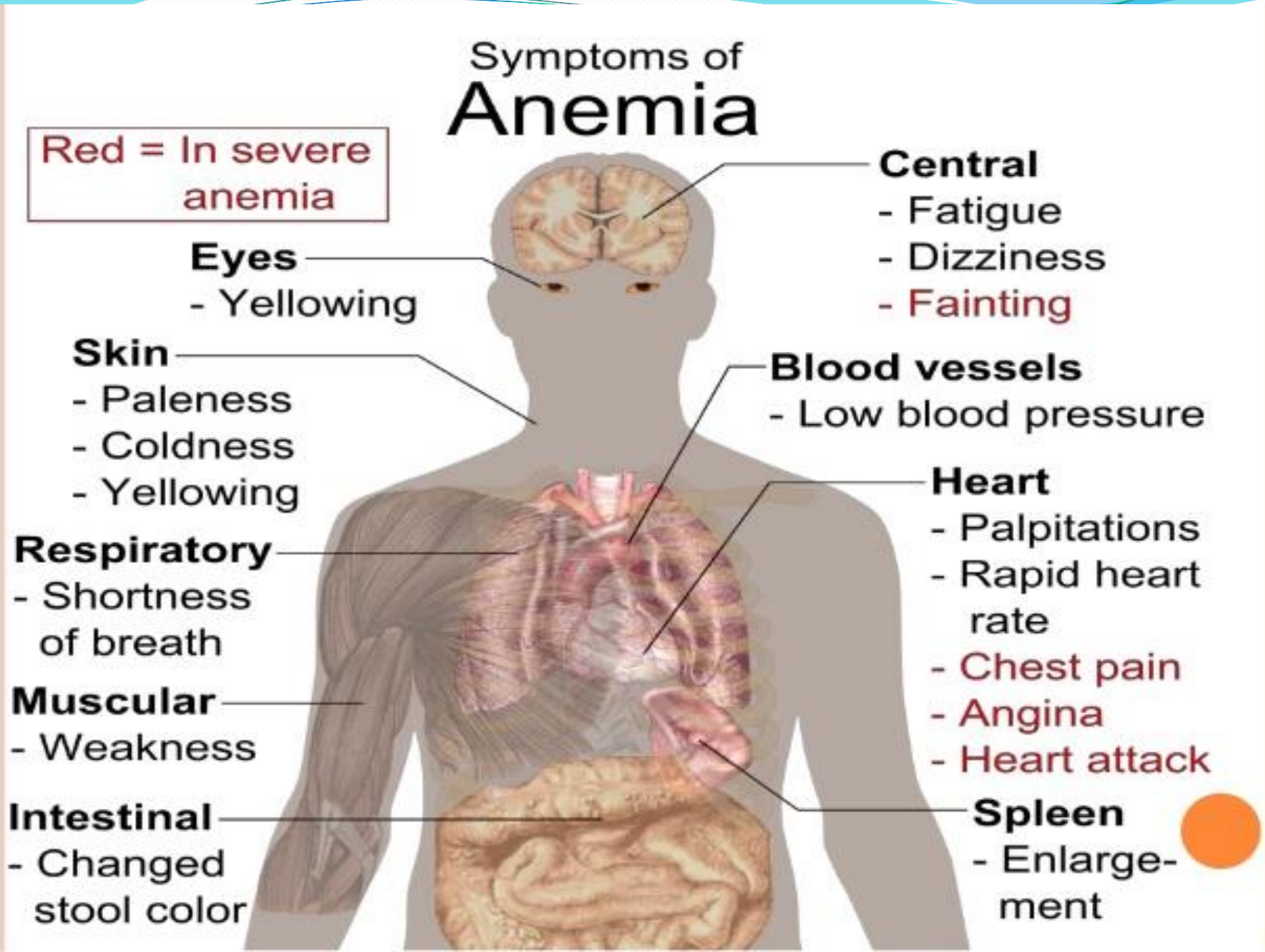


- Angular stomatitis, cheilosis

- Koilonochia



SYMPTOMS


Symptoms of
Anemia
Red = In severe anemia
Central
- Fatigue
- Dizziness
- Fainting
Eyes
- Yellowing
Skin
- Paleness
- Coldness
- Yellowing
Blood vessels
- Low blood pressure
Heart
- Palpitations
- Rapid heart rate
- Chest pain
- Angina
- Heart attack
Respiratory
- Shortness of breath
Muscular
- Weakness
Intestinal
- Changed stool color
Spleen
- Enlarge-ment

SIGN OF ANEMIA

- Pallor -nails bed,oral mucous membrane,conjunctivae
- Facial odema
- Mild systolic flow murmur(pulmonary area)
- Lack palmer creases in infants
- Spleenomegaly(in hemolytic anemia)
- Lymphodenopathy
- Systolic bruits

In modrete to severe anemia

- Systolic bruits(vascular murmur)
- Postural hypotension
- Collapsing pulse
- In ECG -

  depressed ST segment(Hb%<6gm/dl)

  flattened or inverted T waves

INVESTIGATION

CBC

Hb%

RBCs count

Hct

Mcv

Mch

Mchc

Rdw

PS for RBCs

TYPES OF ANEMIA

- Iron deficiency anemia
- Megaloblastic anemia
- Pernicios anemia
- Hemorrhagic anemia
- Aplastic anemia
- Hemolytic anemia

  Thalassemia

  sickle cell anemia

IRON DEFICIENCY ANEMIA

DEFINITION

Iron helps to multiple metabolic process, O2 transportation DNA synthesis and electron transportation.

So, iron deficiency anemia when the decrease in total iron body content is severe enough to diminish erythroposis and cause anemia.

CAUSES OF IDA

Increased requirments

- Growing infants & children
- Chronic renal diseases
- Chronic inflammetory diseases
- Pregnency
- Lactation

Increased loss

- GI bleeding
- Persistent hematuria
- Parasitic infection
- Regular blood donar
- menorrhogia

Countd..

Decreased intake

- Vagetarian diet
- Socioeconomic factors
- Anorexia

Decreased iron absorption

- Gastrectomy
- chrohn's disease
- (it is chronic inflammatory disease of intestine specially the colon & ileum associated with ulcer & fistula)

SYMPTOMS

- Irritability
- Anorexia
- Generalized weakness
- Fatigue
- Leg cramps
- Breathlessness
- Tachycardia
- Koilonchia
- Platynychia
- spleenomegaly

INVESTIGATION FINDINGS

- Low Hb %of above cutt off
- RDW – increased
- MCV & MCHC- decreased
- RBCs – reduced
- S. iron & S.ferritin – decreased
- TIBC – increased
- Ps for RBCs – microcytic, hypochromic red cells

MANAGEMENT

- Causes of anemia should be identified
- Dietary counseling
- Oral iron therapy given -

3-6 mg/kg/day of elemental iron in empty stomach

S/E – abdominal discomfort, vomiting, constipation, diarrhea

Duration- 3 month approx.

Parentral therapy

- INDICATION
- Intolerence to oral iron
- Malabsorption
- Ongoing blood loss

  sothat iv iron sucrose is safe and effective in children
- DOSE- 1-3 mg/kg b.wt diluted in 150 ml of NS
- The total dose of iron can be calculated by this formula
- Iron required(mg)=

  wt(kg) x 2.3x (15-pt. Hb in gm/dl)+500 to 1000mg

Blood transfusion

MEGALOBLASTIC ANEMIA

megaloblastic anemia is a distinct type of anemia characterized by macrocytic red blood cells and erythroid precusors that so nuclear dysmaturity.

This disorder caused by incomplete formation of RBCs resulting in large numbers of incomplete & immature Developed cells .These cells do not working like healthy RBCs.

CAUSES

- Vit. B12 deficiency
- Folic acid deficiency
- Drugs-

  phenytoin, phenobarbitone,PPI,zidovudine neomycin, metformin etc.
- Metabolic causes-

  congenital intrinsic factor deficiency

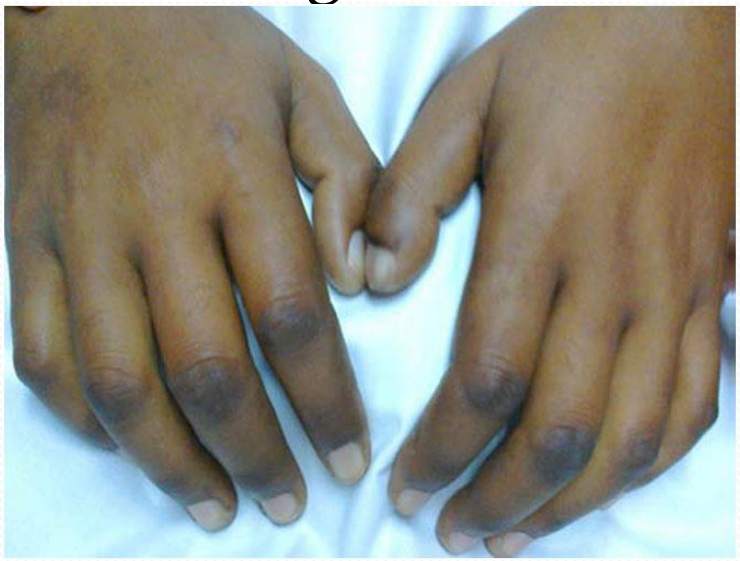
  congenital folate malabsorption

  Dihydrofolate reductase deficiency

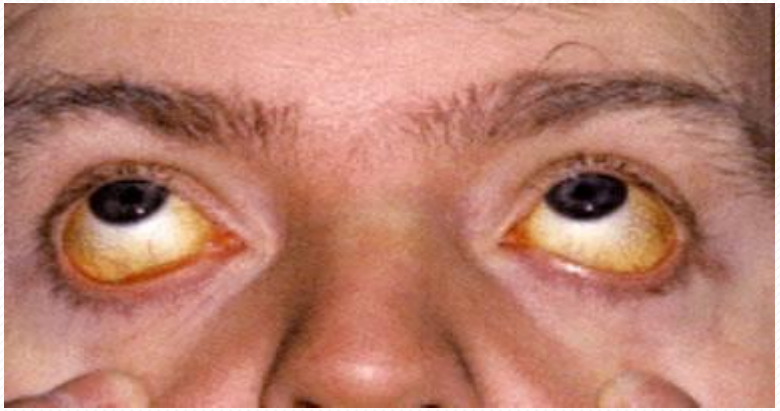
CLINICAL FEATURES

- Pallor
- Irritability
- Easy fatigability
- Glossitis
- Stomatitis
- Hyperpigmentation of skin On knuckles and terminal phalanges
- Hepato/ spleenomegaly may be present

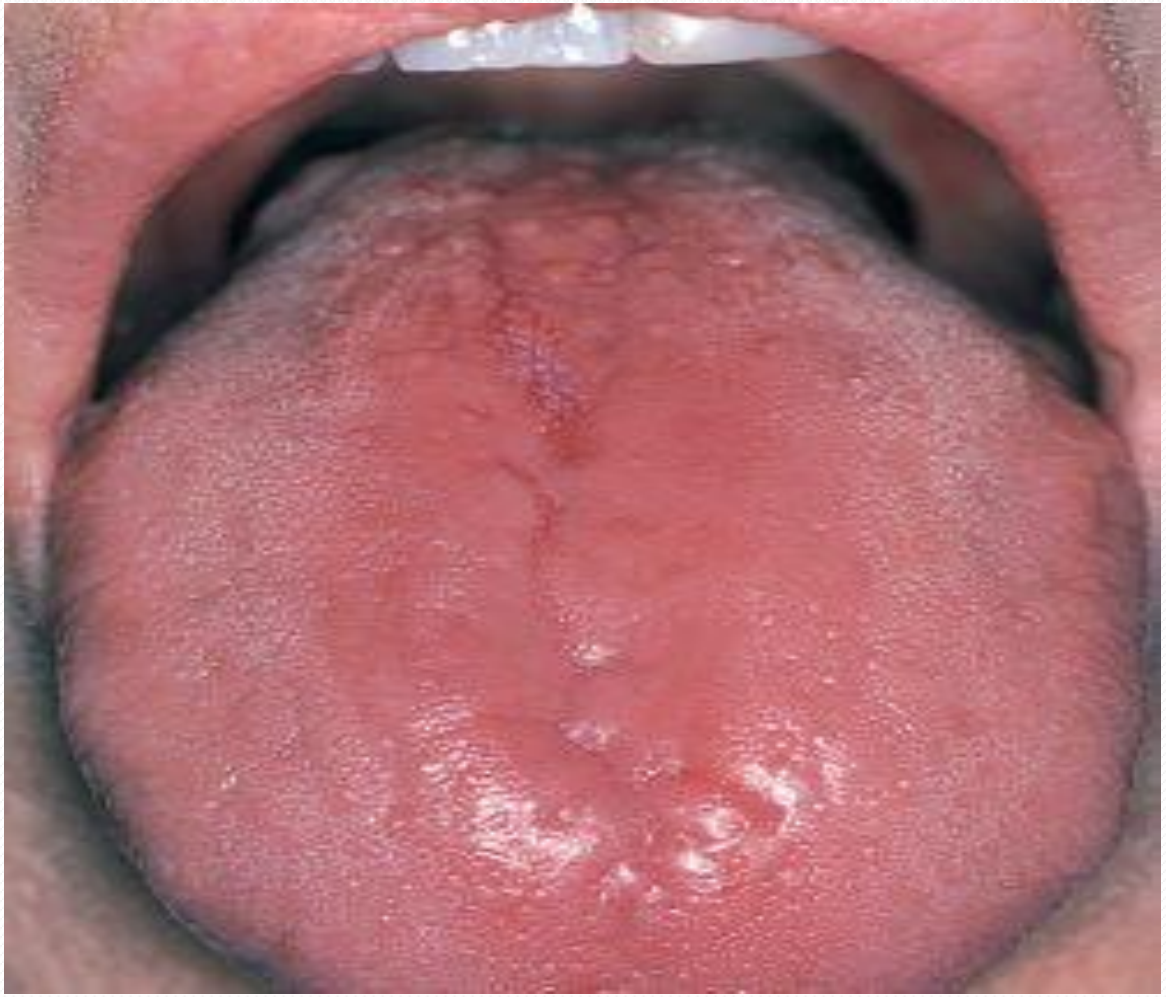
- Hyperpigmentation of knuckle in megaloblstic anaemia



- Presence of jaundice along with pallor would suggest – haemolytic anaemia.



- Glossitis



Investigation FINDINGS

- Low Hb%
- Macrocytic red cells
- Hypersegmented neutrophils(nucleus >6 lobes)
- S.B12 level
- S.follate level
- Schilling test

MANAGEMENT

- Management depends upon the causes.
- Tab.folate 1-5 mg daily for 3-4 weeks
- Vit.B12 administration -

  for therapeutic dose 250-1000mcg IM on alternate day for 1-2 weeks then weekly until blood count is normal

  maintenance dose 1000 mcg/2-4 month

HEMOLYTIC ANEMIA

Hemolytic anemia is a condition in which RBCs are destroyed and removed from blood stream before their normal life span is over.

normal life span of RBCs-

120 days in adults

90 days in neonates

Hemolytic anemia divided into two types-

1. Acquired
2. inherited

CAUSES

ACQUIRED CAUSES

- Hemolytic uremic syndrome
- Malaria
- Kala-azar
- Transfusion reaction
- Hemolytic disease of new born
- Physical injury i.e burn
- Chemical injury i.e snake bite, lead & arsenic toxicity
- Drugs- ceftriaxone, etc.
- March hemoglobinuria

Count…

INHERITED CAUSES

- Hemoglobinopathies e.g thalassemia, sickle cell anemia.
- Red cell membrane defect e.g G6PD deficiency.
- Porphyria
- Unstable hemoglobin
- Lipid membrane defect e.g abetalipoproteinemia
- Hereditary spherocytosis

CLINICAL FEATURES

- Generalized weakness
- Pallor
- Fatigue
- Jaundice
- Hemoglobinuria(in Intravascular hemolysis)
- Spleenomegaly (may be present)

Investigation FINDINGS

- The reticulocyte count is useful in detemine the rate of red cell distruction. Normal reticulocyte count –

  3.2±1.4% in newborn

  1.2±0.7% in children

- Sign of accelerated erythrocyte destruction-
- Fall in Hb level at> 1 g/dl per week
- Increased unconjucated s. bilirubin
- Increased urobilinogen excretion
- Increased s.lactate dehydrogenase
- Decreased erythrocyte life span

Count….

Sign of accelerated erythropoiesis-

- Macrocytosis
- Increase in nucleated red cells
- Reticulocytosis
- Increased plasma iron turnover
- Increased erythrocyte iron turnover

MANAGEMENT

- Maintain fluid balance and renal output during and after acute hemolysis.
- If Shock than managed by appropriate measures
- Blood transfusion in acute anemia
- Acute autoimmune hemolytic anemia is treated with corticosteroids (predinisolone 1-2 mg/kg/day) which are tapered

THALASSEMIAS

Thalassemia caused by defects in globin gene, are the most common monogenic disease . More than 200 mutations are described and the defects are inherited in an autosomal recessive manner .Thalassemia is a inherited blood disorder passed down through families in which the body makes an abnormal form of protein of Hb in RBCs

CLINICAL PRESENTATION

- Severe pallor
- Hepatomegaly
- Spleenomegaly
- Mild to moderate jaundice
- Chronic hepatitis
- Intolerance to exercise
- Irritability
- Cardiac murmur present
- Bony abnormalities such as frontal bossing, prominent facial bones.
- Failure to thrive

Spectrum of disease

B thalassemia traits

- Pt. Have mild anemia , abnormal RBCs and hemoglobin HPLC results with elevated level of of HBA2, HBF.
- In PS of blood film hypochromic , microcytic and presence of target cells.

Thalassemia intermedia

This condition may occur due to a compound heterozygous states . In this condition no needs to regular blood transfusion .

Count….

Thalassemia major

- This condition is characterized by transfusion dependent anemia, Spleenomegaly , Bone deformities, growth retardation hemolytic facies are present.
- In PS of blood shows severe hypochromia, microcytosis, marked ansiocytosis, fragmented red blood cells, polychromasia, nucleated red cells and occasionally immature leukocytes are present.

MANAGEMENT

- It is untreated or inadequately treated.
- Blood transfusion
- Bone marrow transplant
- Possible surgery to remove the spleen and gallbladder
- Hematopoietic stem cell transplant

Chelation therapy-

- It is capable to removing excess iron from the body.
- This usually occurs after 1-2 yr of transfusions when ferritin level is about 1000-1500 úg/l
- Given Deferoxamine 40-60mg/kg/day is infused over 8-12 hrs during the night for 5-6 days a week

APLASTIC ANEMIA

- aplastic anemia comprises a group of disorders of the hematopoietic stem cells resulting in the suppression of one or more of erythroid, myeloid and megakaryocytic cell lines. It condition may be acquired or inherited
- CAUSES-
- Acquired injury of viruses, toxins, chemicals.
- Abnormal marrow microenvironment
- Immunologic suppression of hematopoiesis
- Inherited bone marrow failure syndrome

Clinical features

- Severe anemia
- Congestive heart failure
- Petechiae
- Gum bleeding
- Thrombocytopenia
- Fever
- Pneumonia
- Sepsis
- Congenital physical anomalies

INVESTIGATION FINDINGS

- Ps of RBCs find macrocytosis, agranulocytosis.
- The corrected reticulocyte count is < 1% reduced red cell production
- Bone marrow aspirate and biopsy are essential for evalution of bone marrow cellularity
- Sucrose hemolysis test is positive
- ham's test may be positive

MANAGEMENT

- supportive care should be instituted with PCV for severe anemia
- Platelets transfusion for severe thrombocytopenia
- Antibiotic given for secondary infections
- Hematopoietic stem cell transplant

SICKLE CELL ANEMIA

- It is an autosomal recessive disease that result from the substution of valine for glutamic acid at position 6 of beta globin gene. Patient who are homozygous for the Hbs gene have sickle trait.

  sickle red blood cells are less deformable and obstruct the microcirculation, resulting in tissue hypoxia, which further promotes sickling. These red blood cells are rapidly hemolyzed and have a life span of only 10-20 days.

CLINICAL FEATURES

- Pain is the most common presentation of vaso occlusive crisis.
- Pain with acute chest syndrome if pleuritic in nature
- Arthritis or osteomyelitis if joints are involve.
- Shortness of breath
- Fever with dehydration
- Unilateral weakness
- Sudden increase in pallor
- Syncope
- Hepato/Spleenomegally
- Ictrus

INVESTIGATION FINDING

- Thrombocytosis
- Leukocytosis (>20000/mm)
- In PS of blood sickle shaped RBCs with target cells
- Indirect S. bilirubin level increase
- Hemoglobin Electrophoresis test must be done because it can differentiate individuals who are homozygous or hetrozygous

MANAGEMENT

- Hydration therepy
- Analgesic given for pain crisis
- Blood transfusion
- O2 supplimentation if pt.has hypoxia.

Preventive care

- All children require prophylaxis with penicillin or amoxicillin atleast until 5 yr of age.
- Pt should receive immunization with pneumococcal, meningococcal vaccine.
- Folate supplimentation

Special thanks to ……

Dr. D.B.CHAVAN

ASS.PROF.

GAC & H,NANDED

RESEARCH TOPIC

"To evaluate the efficacy of *pathya ghrita* in *pandu roga* in children w.s.r. to *iron deficiency anemia*."

Thank You
Thank You
Thank You!!!!