ONLINE NAVIGATE – Medico CME program
1st June 2023

Anaemia – a practical approach in the Indian context

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Flow of discussion

- Anaemia definition
- Implications
- Signs and symptoms
- Diagnosis- take a good look at the CBC
- Treatment - including special groups

How should we define ANAEMIA?

Anaemia is the state in which there is a decreased production of RBCs or a decreased concentration of hemoglobin in the RBCs.

AN – without **HAIMIA-** **blood (Greek)**

ANAEMIA-

Hb < 14 gm% for male **<12 gm% for female**

Range – 14-18 gm% **Range – 12-16 gm%**

Haemoglobin in the rbc

contains the red pigment **haemoglobin**

span: 60-120 days

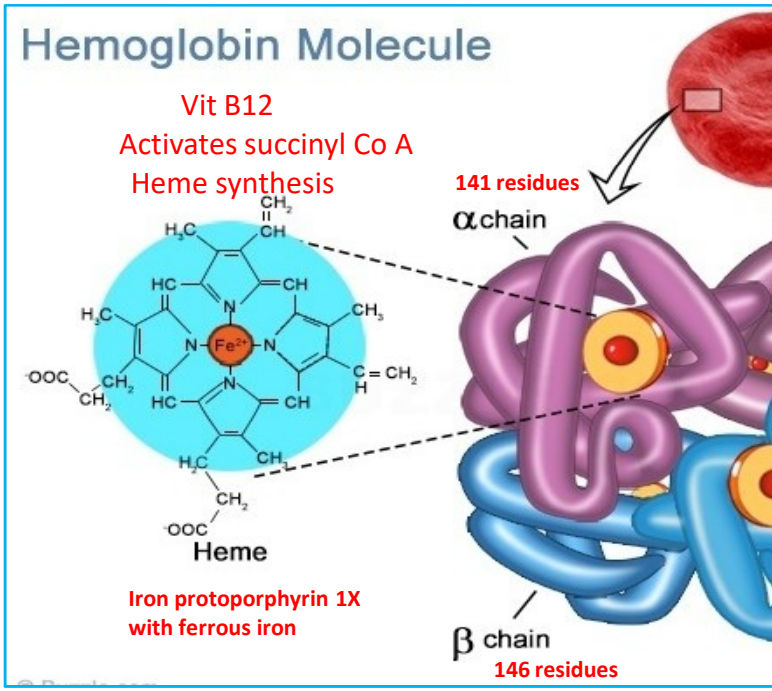
ions :

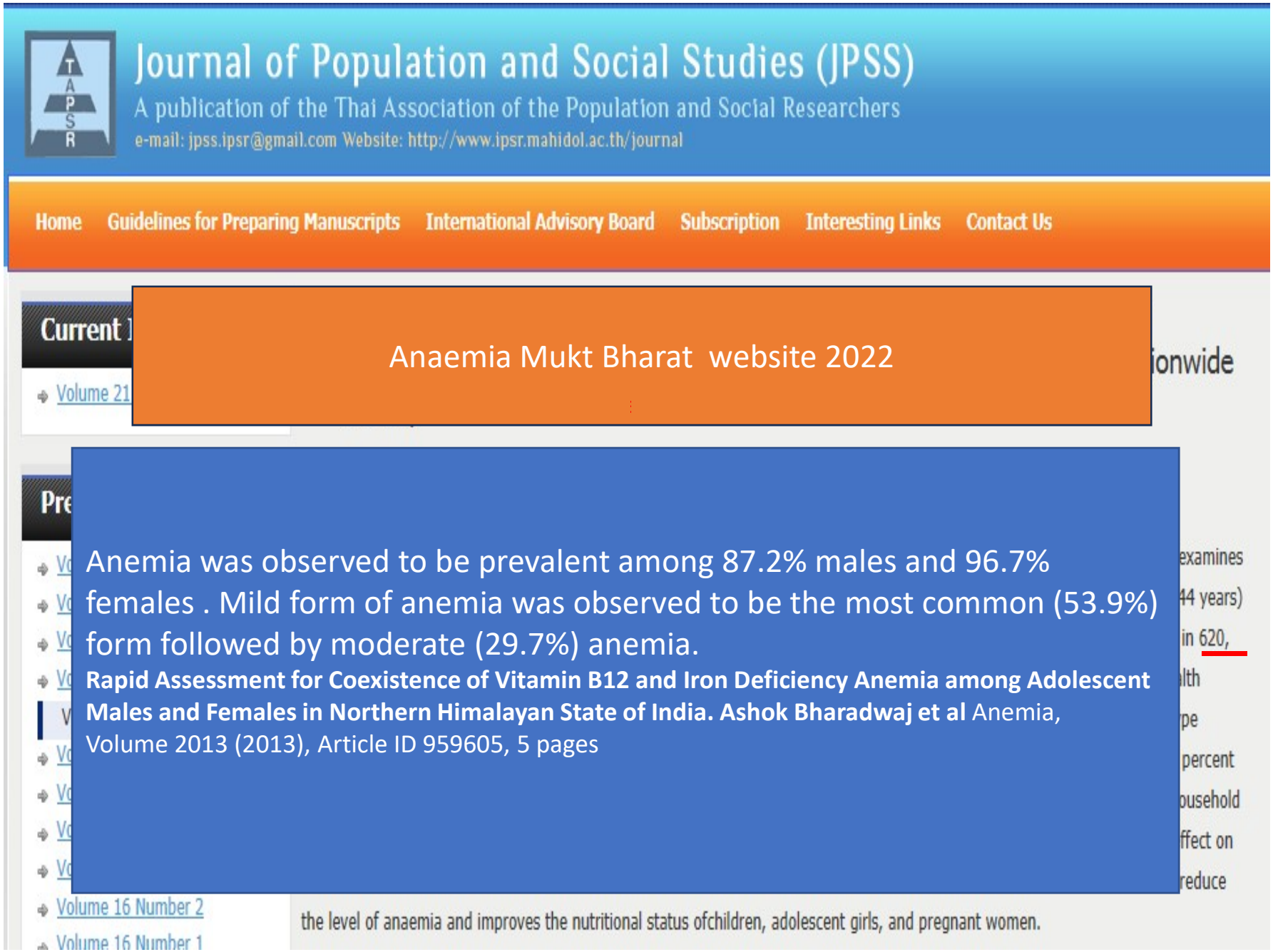
Transport of **O2** with the help of HB

To pick up oxygen from the lungs and deliver it to tissues elsewhere

To pick up **carbon dioxide** from other tissues and unload it in the lungs

Folic acid is r
for maturatio




Journal of Population and Social Studies (JPSS)
A publication of the Thai Association of the Population and Social Researchers
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Volume 21
Anaemia Mukt Bharat website 2022
ionwide
Anemia was observed to be prevalent among 87.2% males and 96.7% females . Mild form of anemia was observed to be the most common (53.9%) form followed by moderate (29.7%) anemia.
Rapid Assessment for Coexistence of Vitamin B12 and Iron Deficiency Anemia among Adolescent Males and Females in Northern Himalayan State of India. Ashok Bharadwaj et al Anemia, Volume 2013 (2013), Article ID 959605, 5 pages
examines
44 years)
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reduce
Volume 16 Number 2
Volume 16 Number 1
the level of anaemia and improves the nutritional status ofchildren, adolescent girls, and pregnant women.

Prevalence in India

IRON DEFICIENCY IS THE COMMONEST CAUSE OF ANAEMIA

Over half of all women and children in India are anaemic, a figure that has risen in the last three years.

The most recent National Family Health Survey (NFHS-5) data show rates increasing of anaemia from 53 per cent to 57 per cent in women and from 58 per cent to 67 per cent in children in 2019-21.

Efforts for anaemia mukt Bharat

Anaemia Mukt Bharat Programme:

Surakshit Matritva Aashwasan (SUMAN):

Janani Suraksha Yojana (JSY):

Janani Shishu Suraksha Karyakram (JSSK):

Pradhan Mantri Surakshit Matritva Abhiyan (PMSMA):

Anaemia in pregnancy

- **In Asia, Anaemia (irrespective of the severity) is the 2nd leading cause of maternal death accounting 12.8% independent of deaths due to postpartum haemorrhage.**
- **In India, prevalence of Anaemia found to be 58.7% in pregnant women and 63.2% in breastfeeding mothers .**
- **In India, About 20% of maternal deaths are caused by Anaemia**
- **Anaemia is additional risk factor in contribution of 50% of all maternal deaths.**

Khaskheli MN et al. Pak J Med Sci. 2016;32(3):630-634
Fathi Najafi T et al. Journal of Midwifery and Reproductive Health. 2013;1(2): 89-96
Patil RR et al. Indian Journal of Maternal and Child Health. 2013 Jul-Sep;15(3):1-7

Causes of High Burden of Anemia- especially iron deficiency

Low Iron Stores

During pregnancy in anemic mothers

Poor iron stores from infancy, childhood deficiencies and adolescent Anemia

Dietary

- Excessive consumption of 'Iron Inhibitors' (tea, coffee, calcium-rich foods) and low intake of 'Iron Enhancers' (Vitamin C etc.)
- Low bioavailability of dietary iron
- 50% of the population is consuming < 50% RDA

Iron Loss

- Due to parasitic load (malaria, intestinal worms)
- Poor environmental sanitation, unsafe drinking water and inadequate personal hygiene

Maternal Anem

- Increased iron requirement due to tissue, blood formati and energy requirem during pregnancy
- Iron loss from post-partum hemorrhage
- Repeated pregnanci with less than 2 yea interval

ANAEMIA

OT DIAGNOSIS BUT AN EVENT OR AN FFECT

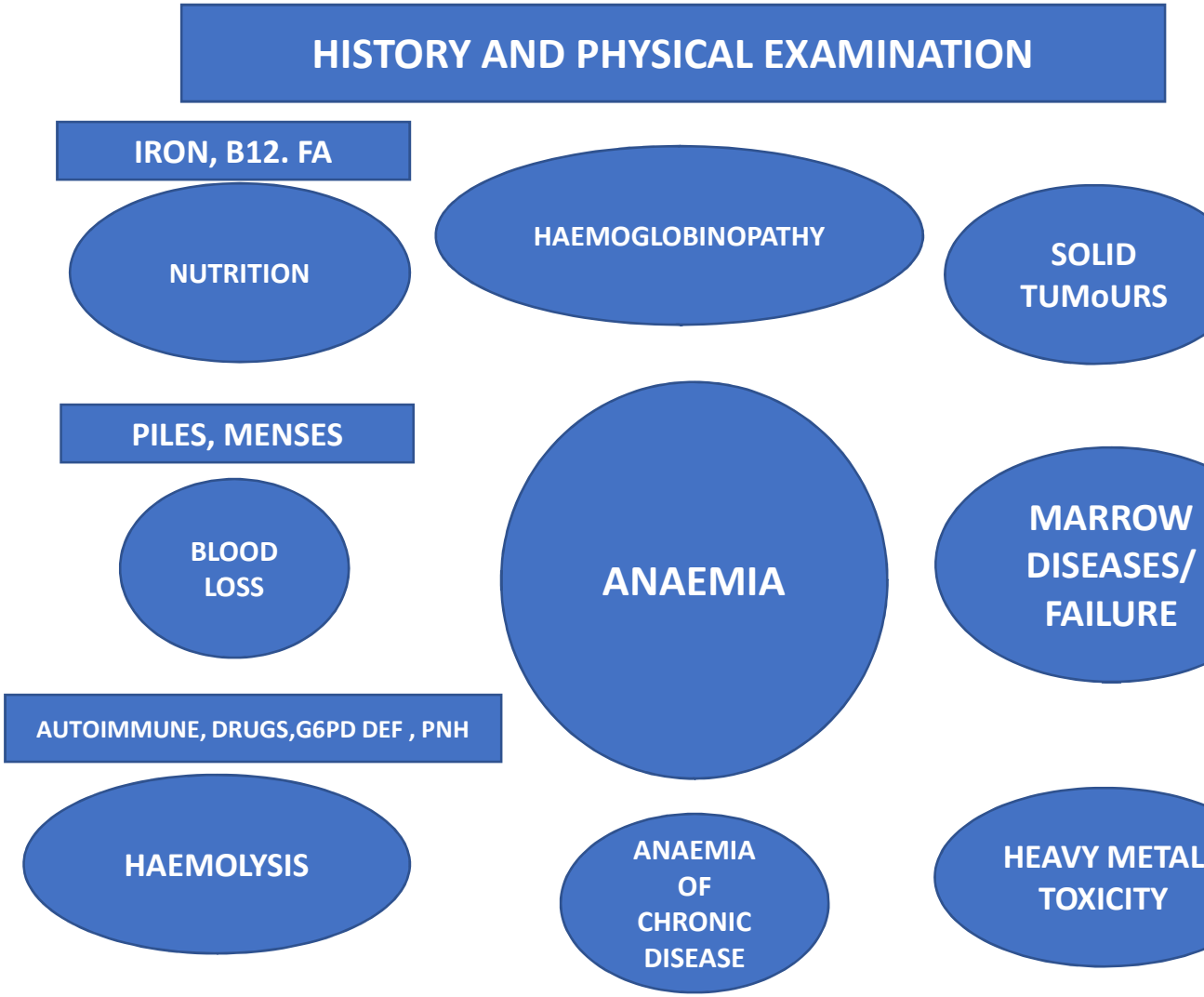
'S CAUSE OR AETIOLOGY SHOULD BE ETERMINED

WHAT MAY APPEAR LOW RISK MAY ARBOUR SOMETHING SINISTER

L PALOR WILL ONLY MAKE US SUSPICOUS OF ANAEMIA- NOT GIVE A DIAGNOSIS

HE LAB TESTS- ESPECIALLY THE PERIPHERAL SMEAR

ENCE OF JAUNDICE, FEVER, LYMPHADENOPATHY, ORGAN NLARGEMENT CAN SUGGEST MORE SINISTER REASONS



Common Causes of Anemia in the Elderly

TABLE 1

ause of anemia	*Percentage of cases*
nemia of chronic disease	30 to 45
on deficiency	15 to 30
osthemorrhagic	5 to 10
itamin B_{12} and folate deficiency	5 to 10
hronic leukemia or lymphoma	5
Myelodysplastic syndrome	5
o identifiable cause	15 to 25

l 1992;38:111–7

Why diagnose anaemia ?

To avoid unnecessary blood Transfusions

TEMPORARY

INFECTIONS

REACTIONS

IMMUNOSUPPRESSIVE

SHORT SUPPLY

RARE BLOOD GROUP

- INEVITABLE TRANSFUSION NEED
  - CARDIAC COMPROMISE
  - ELDERLY
  - RAPID DROP IN HB- G6PD, AIHA
  - TRAUMA
  - INTRA / POST OP BLEEDING

DELINES FOR TRANSFUSION OF RED CELLS

1. Do not transfuse if Hb > 10 gm/dl
2. Transfusion indicated if Hb < 7 gm/dl
3. Transfusion essential if Hb < 5 gm/dl
4. Hb 8- 10 gm/dl safe even if cardioresp probs
5. Symptomatic patients should be transfused.

NOTE: Wound healing and 02 delivery not compromised unless PCV < 18%

AVOID WHOLE BLOOD

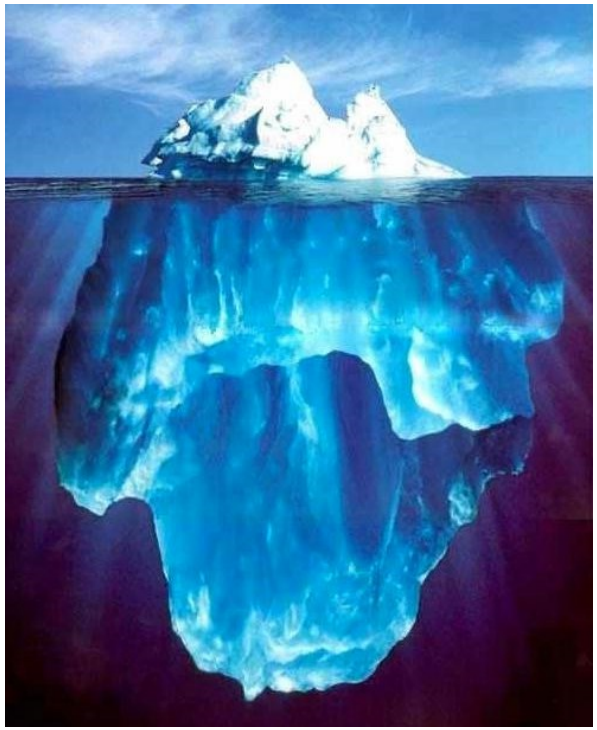
IMPLICATION OF ANAEMIA

Cause can vary from benign to malignant !

EVENT

AETIOLOGY

Quality of life



CAUSE VS EFFECT

Implications of anaemia in pregnancy

Causes can vary from SIMPLE to COMPLEX !

partum complications	Intrapartum complications	Postpartum complications	Fetal outcome
risk of preterm delivery	Prolonged labor	Postpartum hemorrhage	Low birth weight
e rupture of membranes	Increased rates of operative delivery and induced labor	Purperal sepsis	Prematurity
npsia	Fetal distress	Lactation failure	Infections
ne Death	Abruption	Pulmonary thromboembolism	Congenital malformation
nt infection		Subinvolution of uterus	Neonatal Anaemia
um hemorrhage		Postpartum depression	Abnormal cognitive development
e Heart Failure			Increased risk of Schizophrenia

Symptoms of Anaemia

SYMPTOMS

SOB / Leg cramps / fatigue / irritable

Poor diet Nutritional

BLEEDING GI blood loss, menstrual loss

MEDICATION Antiplatelet / NSAIDS

Constitutional symptoms- fever, weight loss

SIGNS

PALOR NAILS SKIN ICTERUS

Cardiac status

Respiratory status

Fever lymph nodes organomegaly

Haemodynamics

edema feet

DURATION Childhood, long term, recent onset

Blood loss during surgical intervention

REVIEW PAST RECORDS

WHAT IS THE STARTING POINT OF ANAEMIA APPROACH?

CBC

Haemoglobin / PCV — quantity

MCV, MCH, MCHC, RDW — quality

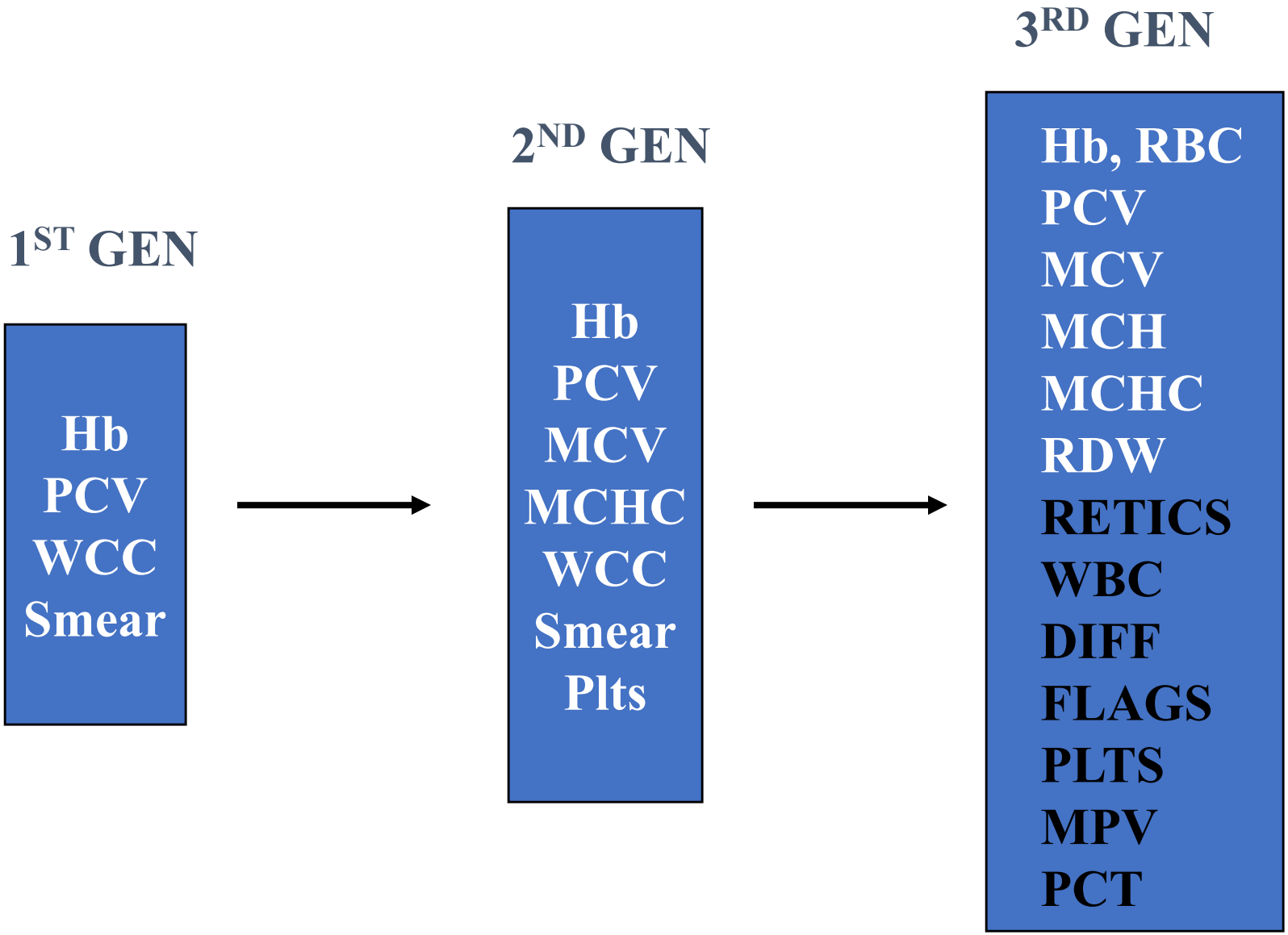
White cell count (Leucocytes) — quantity

Differential count — quality

Platelet count / PCT — quantity

MPV — quality

CBC- AUTOMATION



Stained sl
Reticuloc

STS- METHODOLOGY
rameters provided by automated hematology analyzers

asured parameters

emoglobin (**Hb**)
ematocrit (**HCT**)
BC count (**RBC #**)
latelet count (**PLT #**)
/BC count (**WBC #**)
/BC diff. (**WBC %**)

Derived parameters

- **MCH** (Hb/RBC #)
- **MCV** (HCT/RBC #)
- **MCHC** (Hb/HCT)
- **RDW** (RBC volume)
- **MPV** (Plt TV/PLT

RAPID, RELIABLE, REPRODUCIBLE CBC TEST AUTOMATION

and histograms (RBC, WBC & PLT)

CLASSIFICATION ON INDICES

S	MCV	MCH	
icro	LOW	LOW	IDA/ THAL/ Sideroblastic/ ACD/ Lead toxicity
ytic	HIGH	N	B12/ folate/ MDS/H'Lysis/ Myeloma/ Alcohol/
	N	N	Combination defect /Bld loss/ haemolysis

STEPS - APPROACH TO ANAEMIA

SYMPTOMS AND SIGNS

LABORATORY PARAMETERS

CPC – CLINICAL PATH CORRELATION

ASSIMILATION OF EVENTS AND “PATTERN OF RECOGNITION”

THERAPY

MONITORING AND ASSESSMENT

CLINICAL CLASSIFICATION OF ANAEMIA

Fe, B12, Fol, drugs, BM disorders

Jaundice, spleen

GI, Menses

PRODUCTION VERSUS DESTRUCTION VERSUS LOSS

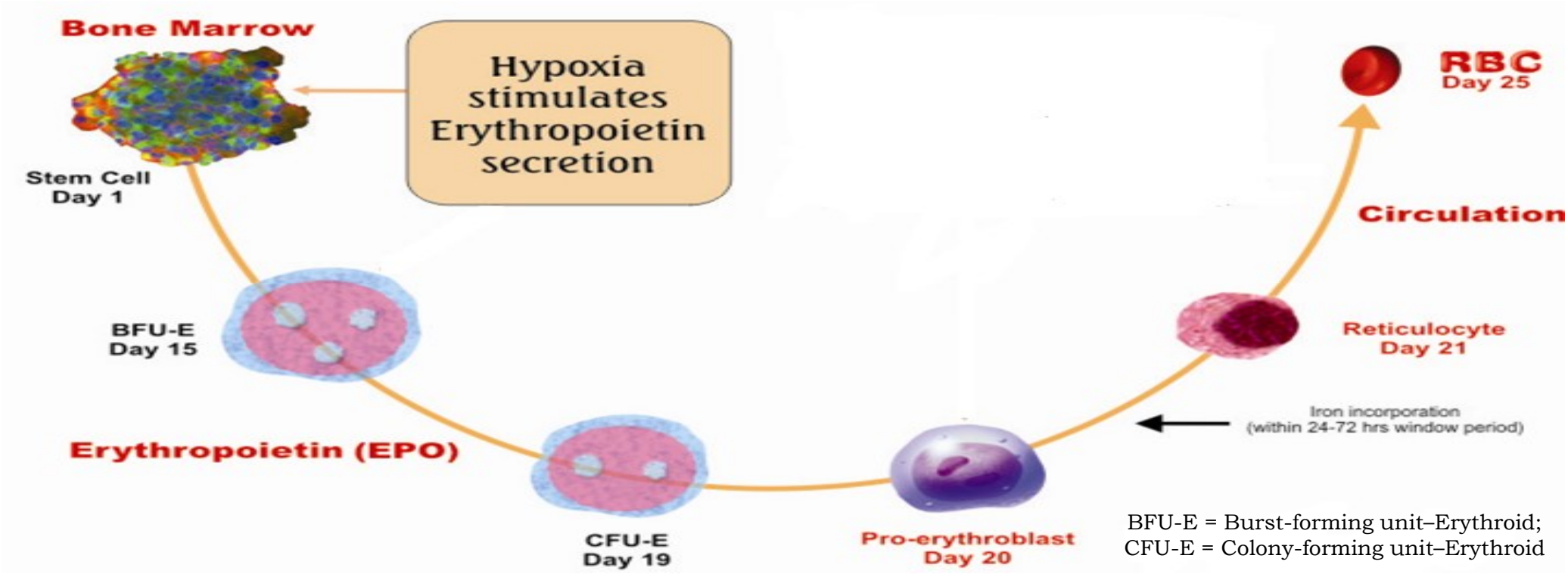
Infective

immune , non immune,
Sickle, Spherocytes ,
Elliptocytes ,
Ovalocytes

G6PD

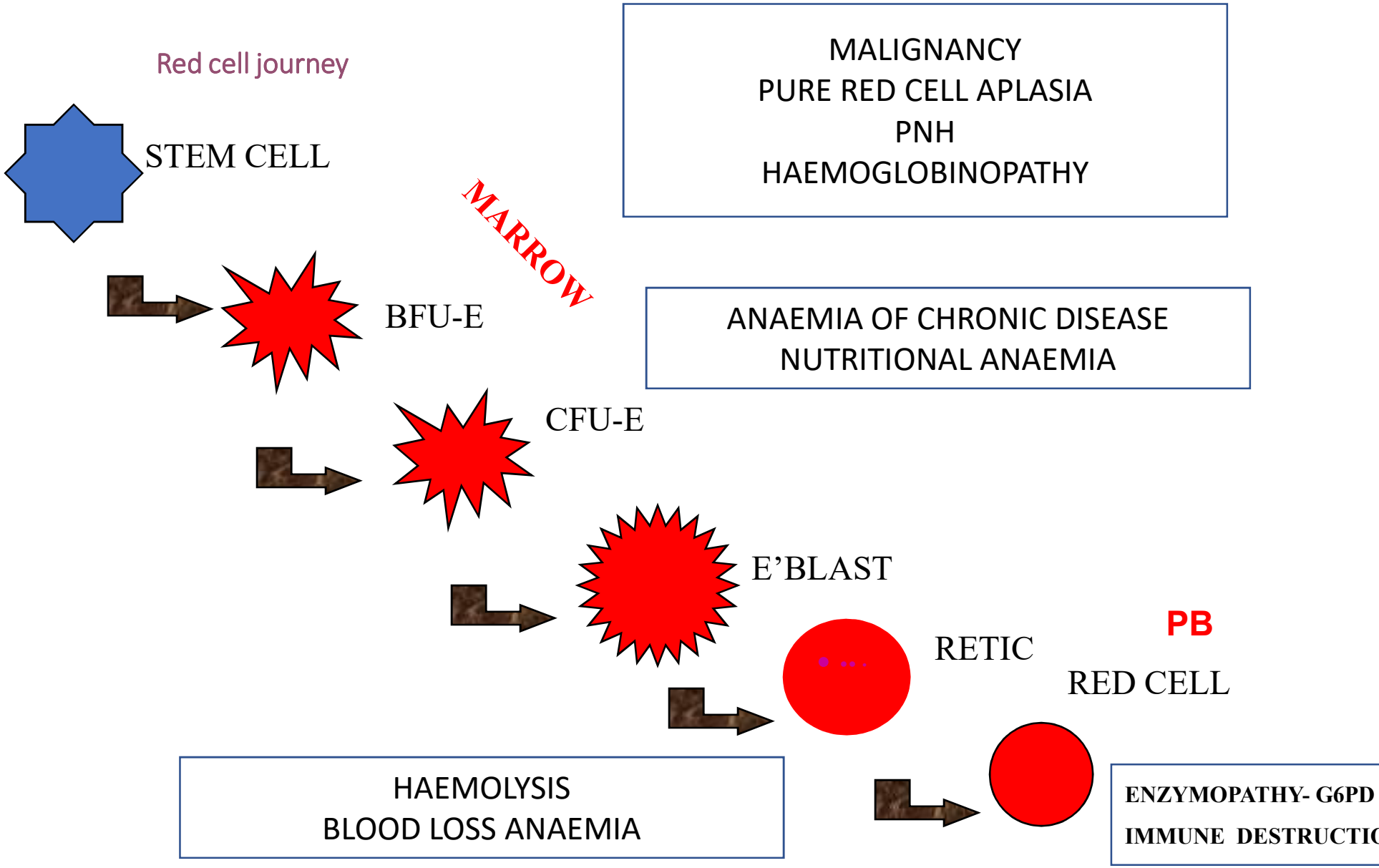
Formation of RBCs (Erythropoiesis)

Approx. 200 billion new erythrocytes generated each day, requiring 20–25mg of iron for Hb production



Body absorbs only 1-2mg of new iron per day. So, 90%-95% of the iron used in this process comes from the recycling of old and inactive RBCs

Anaemia is a late indicator of Iron deficiency, so estimated prevalence of Iron deficiency is **2.5 times that of anaemia**.


Red cell journey
STEM CELL
MARROW
BFU-E
CFU-E
E'BLAST
RETIC
PB
RED CELL
MALIGNANCY
PURE RED CELL APLASIA
PNH
HAEMOGLOBINOPATHY
ANAEMIA OF CHRONIC DISEASE
NUTRITIONAL ANAEMIA
HAEMOLYSIS
BLOOD LOSS ANAEMIA
ENZYMOPATHY- G6PD
IMMUNE DESTRUCTIO

SSMENT OF PERIPHERAL SMEAR

Peripheral smear- Diagnostic- Give a direction for investigations and therapy

C morphology

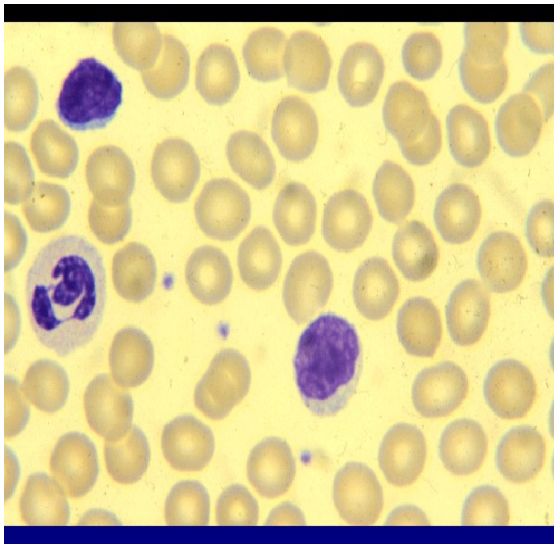
ence of nrbcs

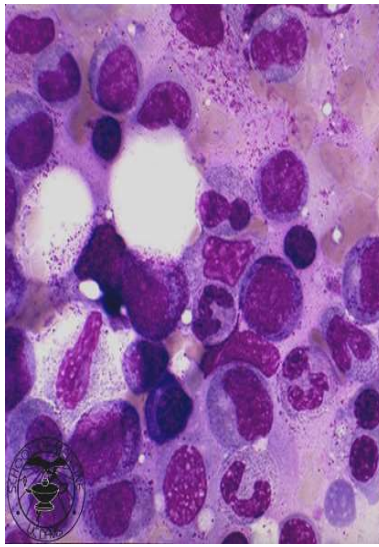
mented red cells, sickle cells

ction like malaria

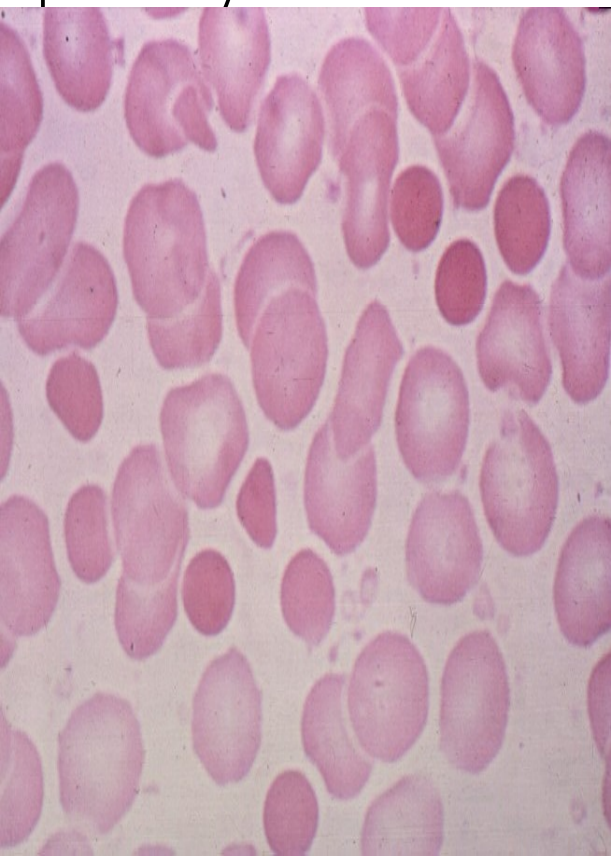
ormal leucocytes

molysis

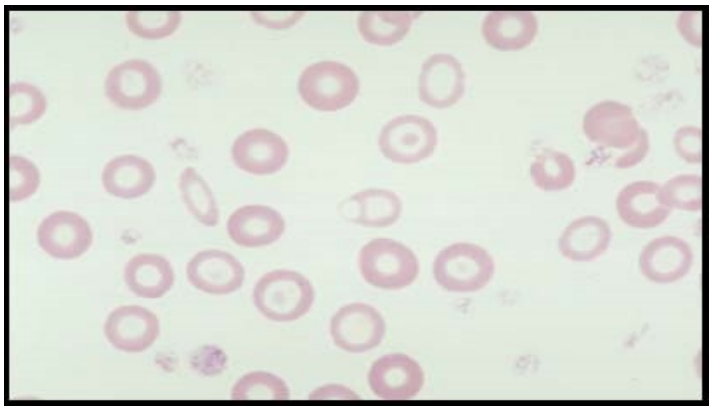




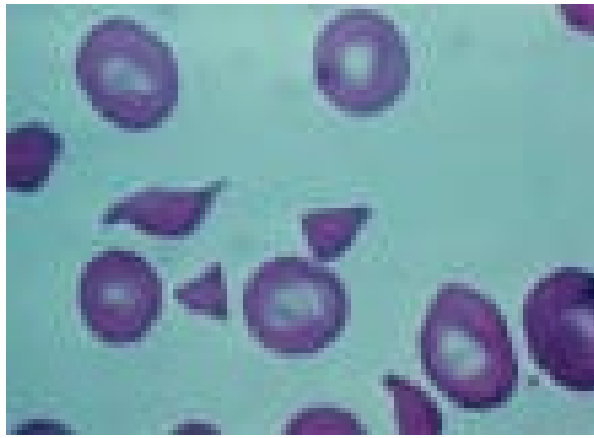
Spherocytes



BITE CELL ANAEMIA



MICROANGIOPATHY



SICKLE CELLS

APPROACH TO ANAEMIA

OCHROMIC - MICROCYTIC ANAEMIA

CV AND MCH

D CELL COUNT

L RDW

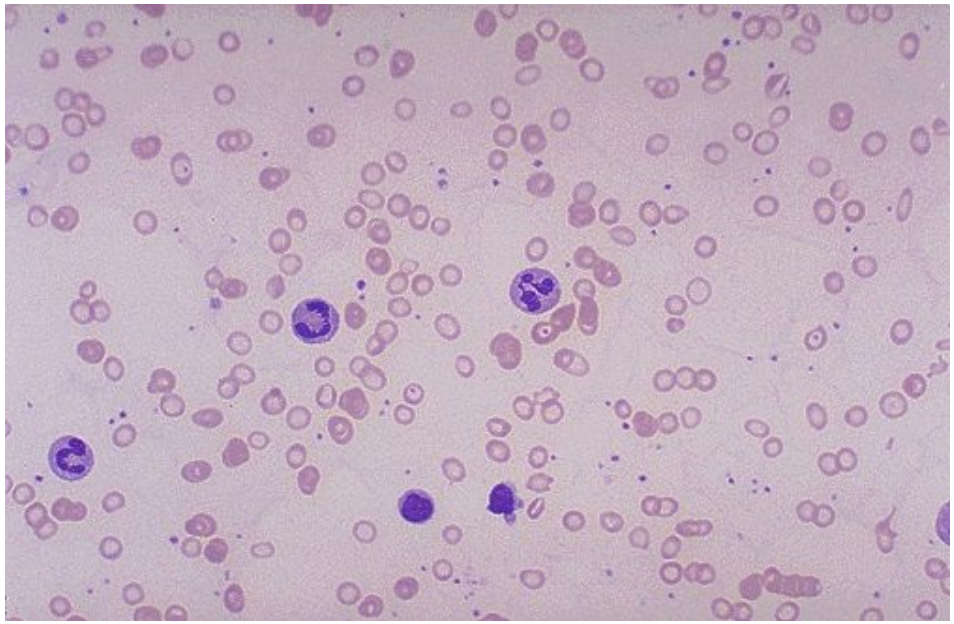
FICIENCY

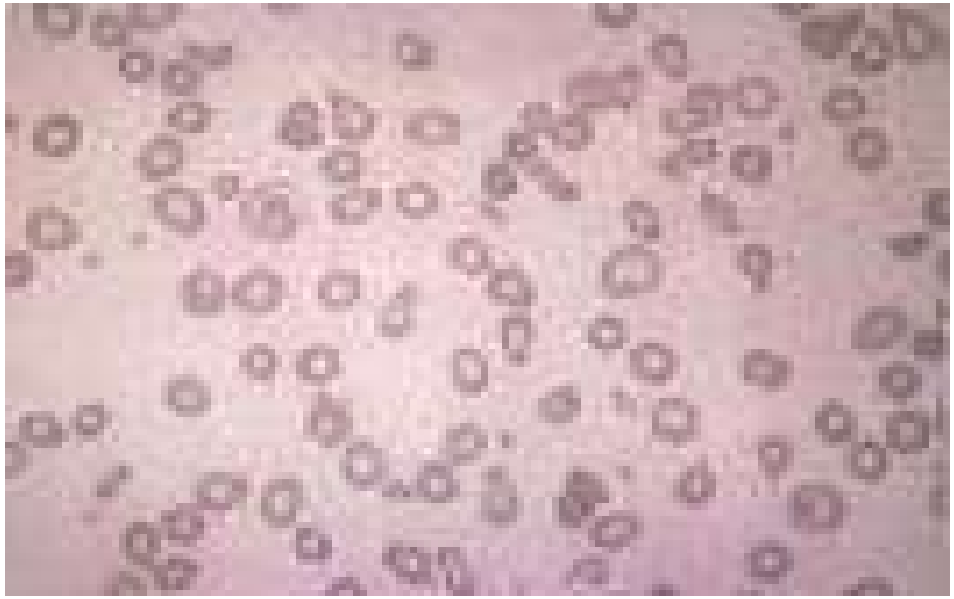
IAL. TRAIT (MINOR)

BLASTIC

A OF CHRONIC DISEASE

OXICITY .





APPROACH TO ANAEMIA

NORMOCHROMIC- NORMOCYTIC ANAEMIA

OMBINATION OF MICRO + MACRO

EMOLYSIS

EMORRHAGE

OLE OF RDW

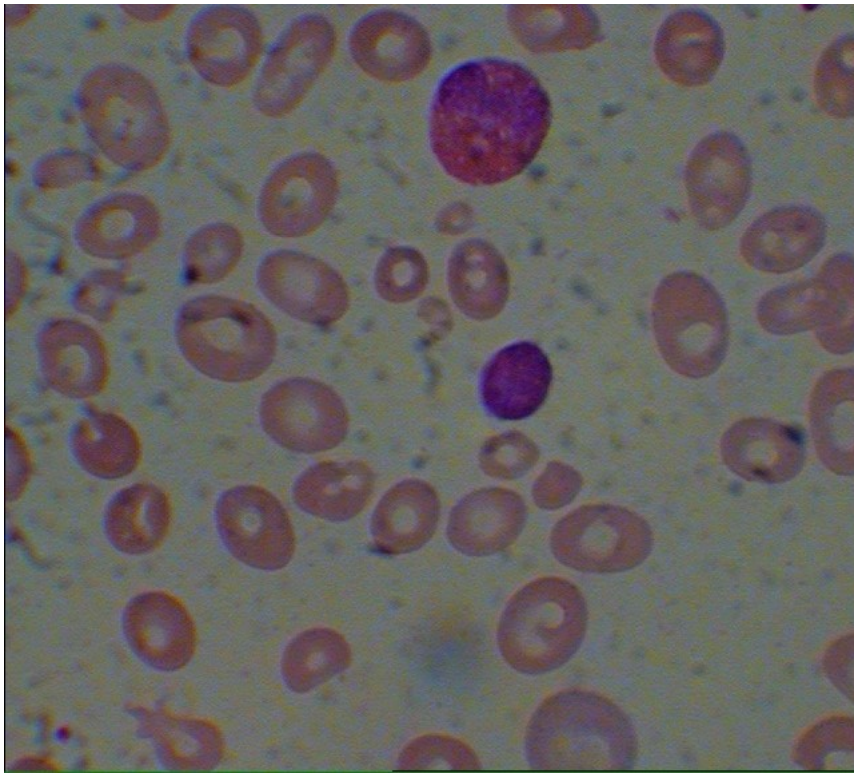
UALLY NEEDS MORE WORK UP INCLUDING BMA AND
MBX

APPROACH TO ANAEMIA

MACROCYTIC ANAEMIA

HIGH MCV
LOW RBC COUNT
USUALLY LOW RETIC

Megaloblastic
Hypothyroid
Liver disease
MDS
Malignancy
Haemolysis



Additional tests for diagnosis

Reticulocyte count

S. LDH

Direct and Indirect Coombs test

G6PD

S. Ferritin

S. Vit B12 , S Folate levels

Haemoglobin Electrophoresis

Thyroid function

S. ANA

S. Erythropoietin levels

ESR

- S iron studies
- Soluble transferrin receptor
- Lead levels
- DNA analysis
- Unstable haemoglobin
- RBC enzyme analysis
- PNH by flow cytometry

- Bone marrow examination

NDICATORS FOR BONE MARROW EXAMINATION

POINTERS FROM HISTORY AND EXAMINATION

RAISED ESR, ROULEAUX, HOT SPOTS ON SCAN

BLASTS ON SMEAR, HYPOGRANULAR NEUTROPHILS

OR ABNORMAL CELLS

NORMOCHROMIC-NORMOCYTIC ANAEMIA OR UNEXPLAINED ANAEMIA

Deficiency Anaemia – Global Scenario

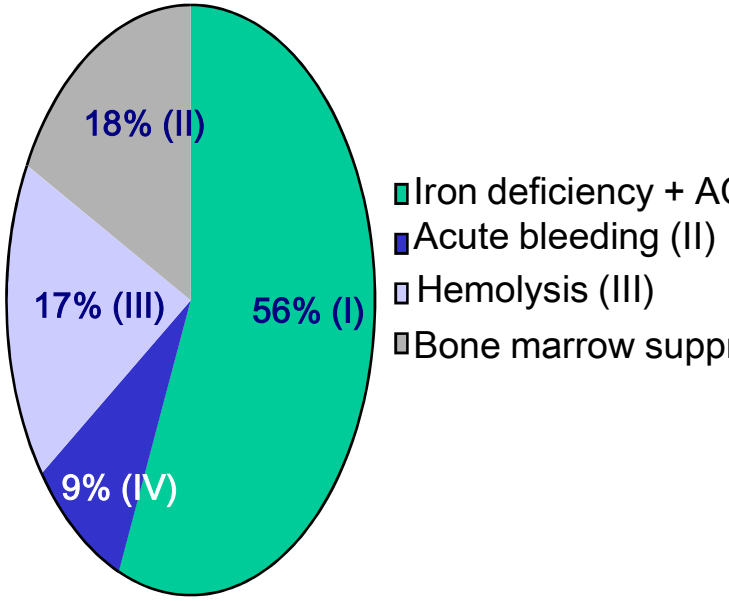
IRON DEFICIENCY IS THE COMMONEST CAUSE OF ANAEM

Almost half of the world population is anemic –

- 9-14% in developed countries
- 43-51% low developed countries
- 65-75% in India alone

In absolute number, anaemia- 1.62 billion people globally (24.8%)

- 293 million children of Pre-school age – 47%
- 56 million pregnant women – 42%
- 468 million non-pregnant women – 13%
- Nearly 50% of women of reproductive age and
  26% of men in age group of 15-59years are anemic



ron Deficiency Anaemia: Three Stages

n depletion is the onset of iron deficiency with a reduction in storage iron still with normal iron level for erythropoiesis.

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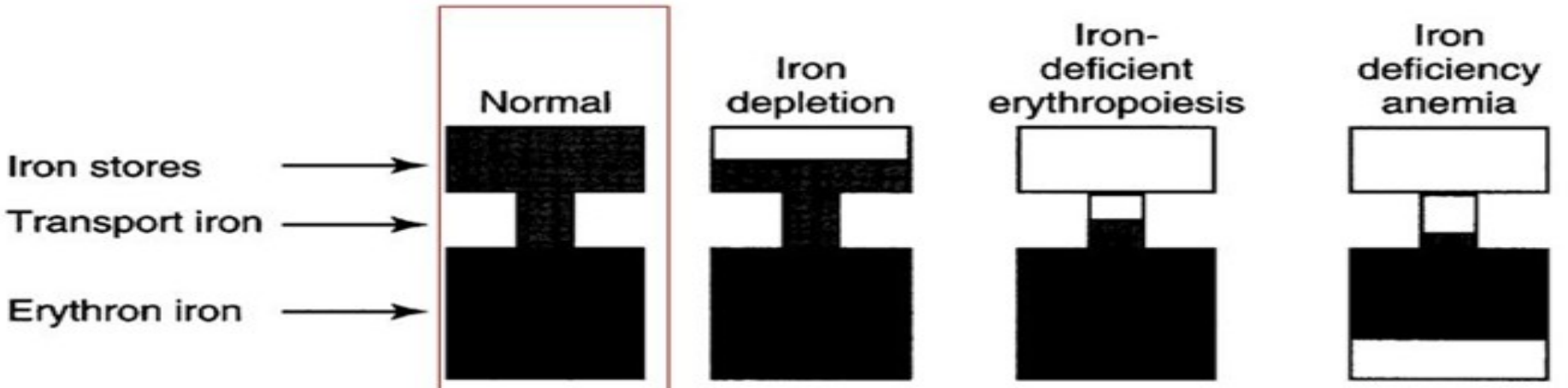
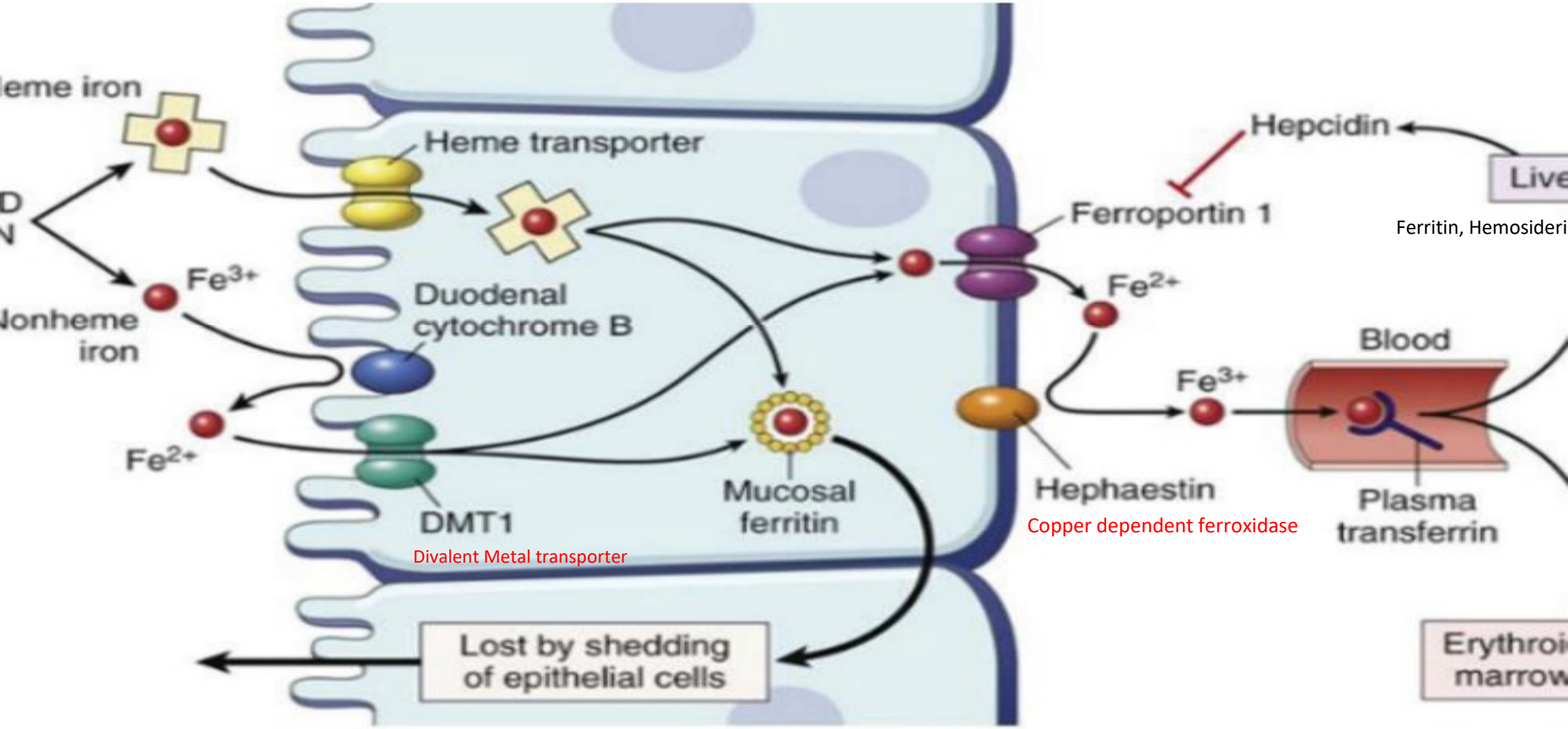


	Normal	Iron depletion	Iron-deficient erythropoiesis	Iron deficiency anemia
Marrow iron stores	2–3+	0–trace	0	0
Plasma ferritin (ng/mL)	10–200	< 20	10	< 10
Plasma iron (µg/dL)	33–150	< 115	< 60	< 40
Transferrin saturation (%)	25–50	< 30	< 15	< 10
Erythrocyte morphology	Normal	Normal	Normal	Microcytic, hypochromic

Iron Absorption & Storage



Agric Sc Food Technol. 2018;4(1):024-030

Therapeutic solutions to address IDA

ORAL IRON PREPARATIONS – SEVERAL SALTS

INTRAMUSCULAR IRON PREPARATION

INTRAVENOUS IRON PREPARATIONS

APPROACH TO IRON THERAPY

Oral Tablets	Ferrous sulfate, fumarate, succinate, gluconate. Several other salts
	60-100 mg of elemental iron per day.
	Concept of alternate day therapy
	After food intake of iron rather than fasting
	Inexpensive. Easily available. Good bioavailability.
Problems:	SIDE EFFECTS NON-COMPLIANCE

IRON RICH DIET

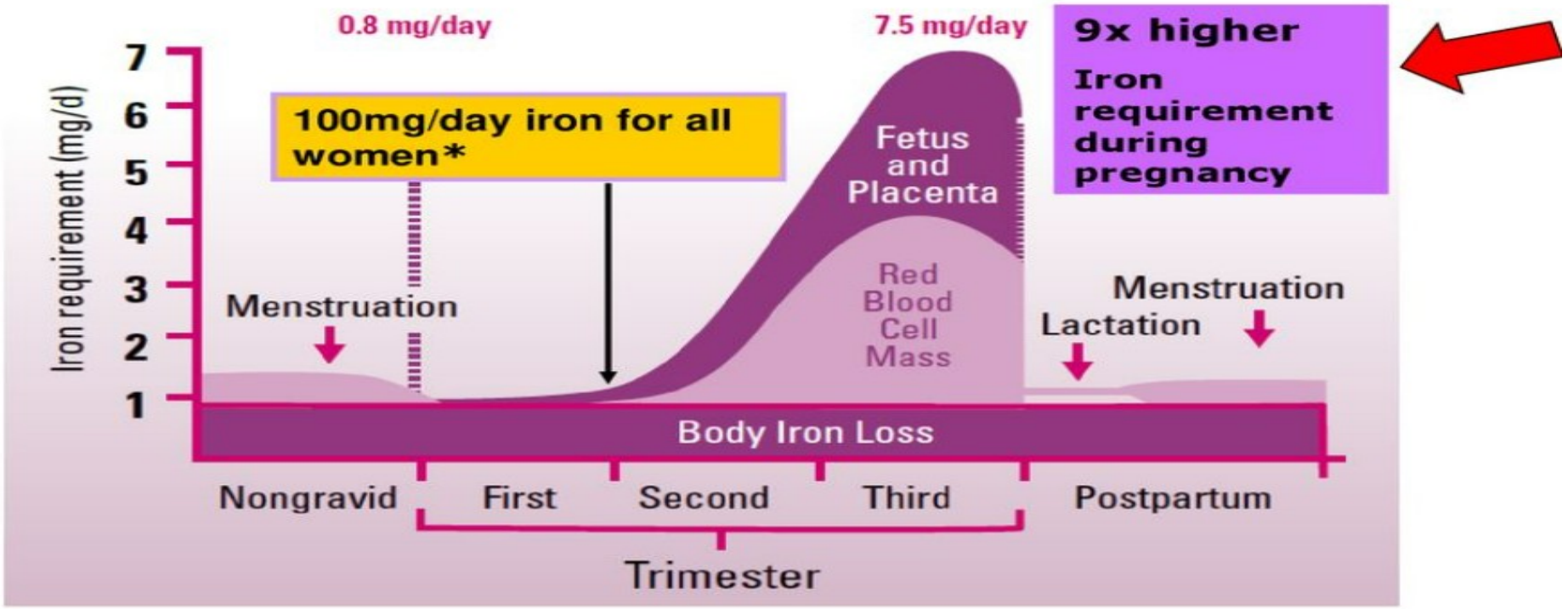
PARENTERAL THERAPY

More options for parenteral

newer, safer, less toxic and more effective

- Intolerant to oral iron therapy
- GI malabsorption syndrome
- Intestinal resection surgery
- 3rd and 4th trimester pregnancy
- Post operative to reduce transfusion need
- Iron deficiency with heart failure to improve cardiac function
- Iron deficiency in renal failure patient
- Religious belief

ron needs during pregnancy



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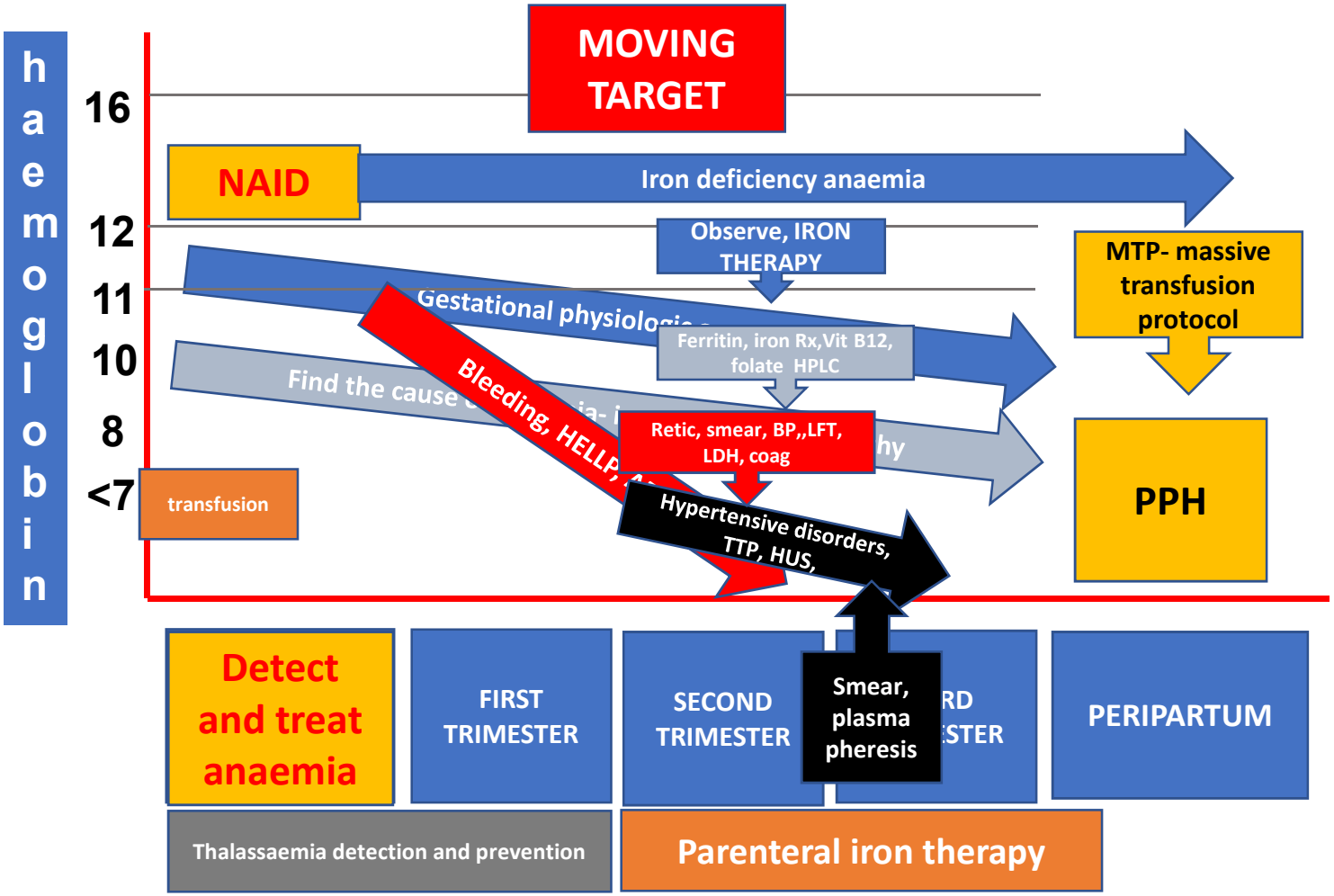
Birds eye view of anaemia in pregnancy

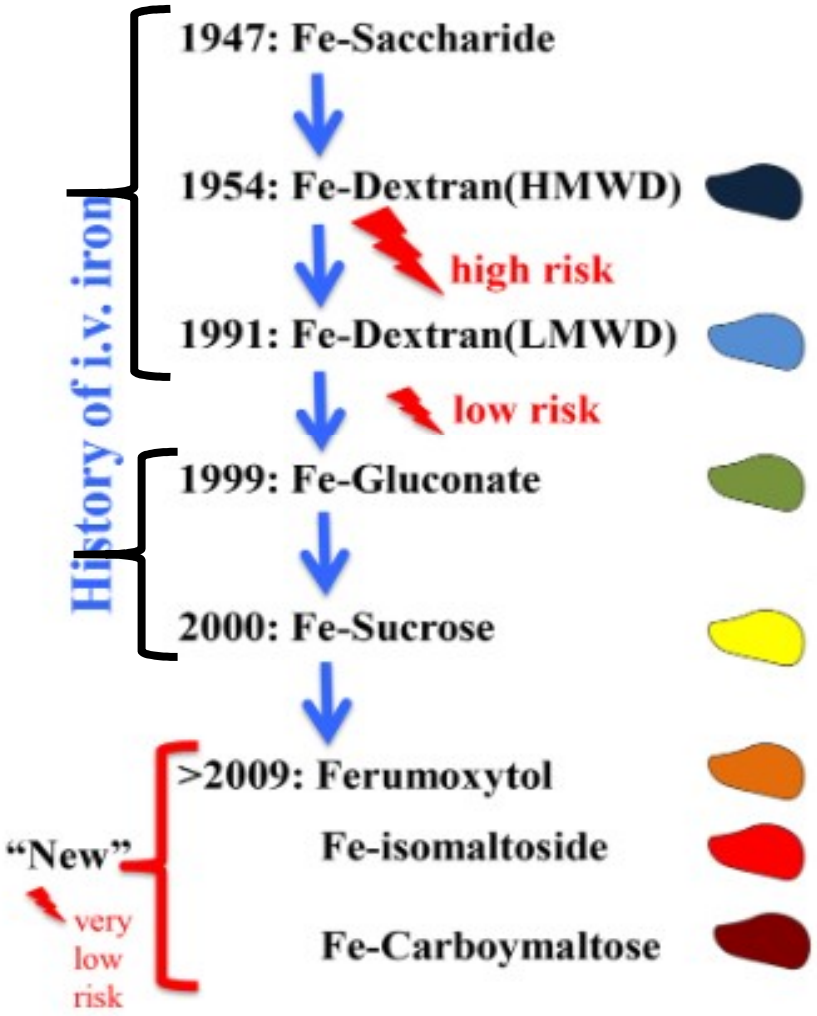


Parenteral Iron preparations - old to new

First generation

Second generation

Third generation



First Gen.IV iron--Cases of severe hypersensitivity reactions (eg.dextran-induced anaphylactic reactions, led to extreme caution within medical community

2nd Gen .IV IRON – Lesser ADR But Posology limitations

3rd GEN -Circumvent toxicity issues inherent with earlier preparations & posology limitations of iron sucrose products.

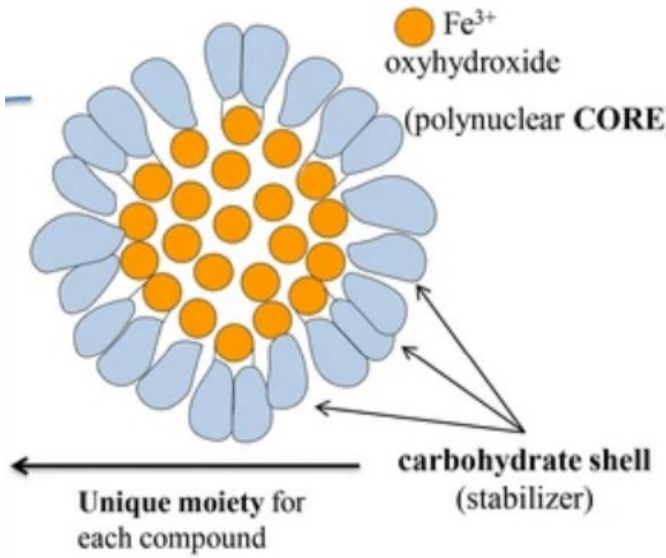
2018, 11, 82

Bioengineering of IV Iron

IV iron preparations are bioengineered as iron-carbohydrate complexes and consist of colloidal suspensions of iron oxide nanoparticles with a polynuclear Fe(III)-oxyhydroxide/oxide core surrounded by a carbohydrate ligand

Physicochemical differences between the IV irons include mineral composition, crystalline structure, conformation, size and molecular weight

key point of difference between IV iron products is the carbohydrate ligand, which influences complex stability, iron release and immunogenicity, and is a unique feature of each drug



Key differences in:
- ✓ Immunogenicity
- ✓ Strength of stabilization

2018, 11, 82

Dose calculation

Iron Sucrose

Calculated using Ganzoni formula:

Cumulative iron deficit [mg] = body weight [kg] x (target Hb - actual Hb) [g/dl] x 2.4 + iron storage depot [mg]

Thus, for **Iron Sucrose** Inj. multiple visits are required

Day	Dose
Mon	200mg
Tue	-
Wed	200mg
Thu	-
Fri	200mg
Sat	-
Sun	-
Mon	200mg
Tue	-
Wed	180mg

Ferric Carboxymaltose

1000 mg can be administered in single visit in over minimum 15 mins

Weight/Hb	≥10 gm/dL	<10 gm/dL
Body weight ≥ 35 kg and <70 kg	1000 mg	1500 mg
Body weight ≥ 70 kg	1500 mg	2000 mg

In most of Indian pregnant women 1000 to 1500 mg is well suited

ltose summary of Product Characteristics. Available from: https://www.medicines.org.uk/emc/product/5910/smpc/print

Dilution for infusion

syndrome (hyperphosphaturic hypophosphatemia triggered by high fibroblast growth factor 23 that ses hypovitaminosis D, hypocalcemia and secondary hyperparathyroidism).

System	Common (>1%- <10%)	Uncommon (>0.1% - <1%)
Immune system		hypersensitivity
Nerv		
Vascu		
Gastr		ulence
Skin		
Musc		
Gene		malaise, oedema peripheral
Investigational	transient blood phosphorus decreased, alanine aminotransferase increased	aspartate aminotransferase increased, gamma-glutamyltransferase increased, blood lactate dehydrogenase increased

Contraindications

known hypersensitivity to compound or to any of its excipients
anaemia not attributed to iron deficiency
evidence of iron overload or disturbances in utilization of iron
in pregnancy in the first trimester
in children below 14 yrs

ltose summary of Product Characteristics. Available from: https://www.medicines.org.uk/emc/product/5910/smpc/print

Postpartum anemia- the 4th Trimester !

A neglected health issue

In India, more emphasis and vigilance is given to the antenatal period

The entire focus shifts to the newborn

Most common maternal

Causes for PPA

- Peripartum hemorrhage
- Anemia during pregnancy
- Inadequate iron intake during pregnancy
- Closely spaced pregnancies
- Multiple births
- Following delivery, women lose some amount of iron through breastfeeding and lactation

ROLE OF IV IRON THERAPY

m Care. 2019 Aug; 8(8): 2703–2707

Ann Hematol (2012) 91:143–154

Latest Update: Expert Opinion on FCM use in Pregnancy and PPA

Ferric Carboxymaltose for the Treatment of Anemia during Antenatal and Postpartum Period: Expert Opinion

PC Mahapatra[1], Sanjay Gupte[2], Narendra Malhotra[3], PM Gopinath[4], Suchitra N Pandit[5], Sunita Tandulwadkar[6], Mahesh Gupta[7], Sheela Shenoy[8], Vidya V Bhat[9], Arun M Boruah[10], Kawita Bapat[11], Milind R Shah[12], Jaideep Malhotra[13], Neharika Malhotra[14], Swami Onkar C[15], Ruchika Garg[16]

IV FCM may be preferred over oral iron and IV iron sucrose for all severities of anemia in the second and third trimesters of **pregnancy (optimal time 12-32 weeks)**

FCM should be the choice of iron supplementation in **PPA** to rapidly and effectively correct iron deficiency, improve iron stores, and raise Hb to optimal levels

Within 6 weeks of FCM treatment, one can expect a rise in Hb by nearly 3–4 gm/dL with a significant rise in ferritin and replenishment of iron stores

Asian Federation of Obstetrics and Gynaecology, Volume 14 Issue 3 (May–June 2022)

Anemia Mukt Bharat Guidelines 2018

Anemia management protocol for Pregnant women

Mild anemia (Hb 10-10.9g/dL) & Moderate anemia (7-9.9 g/dL)

First level of treatment	100 mg elemental iron and 500 mcg folic acid daily for 6 months
	Parental iron (IV Iron Sucrose or FCM) may be considered as the first line of management in pregnant women who are detected to be anemic late in pregnancy or in whom compliance is likely to be low (high chance of lost to follow-up).
If no improvement, after first level of treatment	▪ Referral to higher health facility ▪ The case may be managed with IV Iron Sucrose/Ferric Carboxymaltose

Anemia Mukt Bharat Guidelines 2018

Anemia management protocol for Pregnant women

Severe anemia (Hb 5-6.9 g/dL)

First level of treatment	Immediate hospitalization if it is the third trimester of pregnancy where round-the-clock specialist care is available The treatment will be done using IV Iron **Sucrose/Ferric Carboxymaltose** by the medical officer

FOGSI GCPR 2017 recommendations

In postpartum anemic patients, parenteral intravenous (Iron sucrose/ ferric carboxymaltose) may be the preferred alternative over oral iron for ensuring compliance and faster response **(Grade A, level 3)**

Ferric carboxymaltose has an advantage of administration as a bolus dose in the postpartum period for correction of anemia and restoration of iron stores **(Grade A,level2)**

What is BETA THALASSAEMIA trait?

ETHNIC COMMUNITY

USUALLY IRON OVERPRESCRIBED

LOW HB – usual Hb 9-11gm% but may vary

HB ELECTROPHORESIS RAISED A2

DNA ANALYSIS FOR GENE MUTATION

GENETIC COUNSELLING

Should be given iron if proven iron deficiency

Thalassaemia trait

Hb Low
Normal / raised RBC
Low MCV
Low MCH
Normal MCHC

IDA

Low Hb
Low RBC
Low MCV
Low MCH
Low MCHC

WHAT IS THE SIGNIFICANCE OF THALASSAEMIA?

NOR	NORMAL LIFE, MILD ANAEMIA	HB 9-11 GM%
TERMEDIA	SHORT STATURE, JAUNDICE, ABNORMAL FACIES, HEPATOSPLENOMEGALY	HB 7-9 GM%
AJOR	TRANSFUSION DEPENDENT	HB <7 GM%

Table 4. Interpretation of haemoglobinopathy carrier testing results

MCH (pg)	Ferritin	Haemoglobin electrophoresis	Interpretation
≥27	Normal	Normal	Thalassaemia unlikely but one gene deletion α-thalassaemia not excluded
	Normal	HbS present	Carrier for sickle cell disease
	Low	Normal	Reduced iron stores or iron deficiency, thalassaemia unlikely but one gene deletion α-thalassaemia not excluded
<27	Normal	HbA_2 increased HbF increased	Carrier for β-thalassaemia
		HbA_2 normal HbH present	Carrier for α-thalassaemia
		HbS present	Carrier for sickle cell disease
			Possible co-existent thalassaemia carrier state
		Normal	Possible carrier for α-thalassaemia
			DNA testing indicated
<27	Low	Normal	Iron deficiency Thalassaemia may coexist If woman is pregnant, seek advice about DNA testing; test partner for full haemoglobinopathy screen

Thalassaemia Screening in Ante natal Clinic

- Aim- identify couples in which both partners have thalassaemia minor and/or a haemoglobinopathy and who are at risk of having a baby with serious disease.

- This allows timely prenatal diagnosis and/or early diagnosis and treatment of affected children.

- Detailed genetic counseling and family studies are important for future pregnancies

emoglobinopathy and iron deficiency

REMEMBER- YOU COULD ALSO BE RESPONSIBLE FOR THE BIRTH OF A THALASSAEMIA MAJOR !!

- Known haemoglobinopathy - serum ferritin
- Give oral supplements if their ferritin <30 ug/l
- Unknown haemoglobinopathy status – Look at the indices – hypo microcytic -trial of oral iron (1B)
- Do haemoglobinopathy after at least 3 weeks of oral iron therapy
- If no response to iron therapy and no rise in HB- look for Vit B12/ Folate(1A)
- CHECK SPOUSE THAL STATUS

MEGALOBLASTIC ANAEMIA

EXTREMELY COMMON

History, Skin hyperpigmentation, bald tongue

Macrocytic anaemia, macro-ovalocytes, raised LDH, raise ind. bilirubin

CBC. RDW, ACTIVE B12, S. FOLATE, S.LDH, RETICS, S. IND BIL,

IM REPLACEMENT

IV REPLACEMENT

SUBLINGUAL / oral REPLACEMENT OF B12

REPLACE FOLATE SIMULTANEOUSLY

IFA. APCA

OTHER AUTOIMMUNE PROFILE (DM,HYPOTHYROID,VITILIGO)

HAEMOLYTIC ANAEMIA

AUTOIMMUNE HAEMOLYTIC ANAEMIA

DIRECT COOMBS TEST

INDIRECT COOMBS TEST

S. Bil, LDH, S.HAPTOGLOBIN

NON-AUTOIMMUNE HAEMOLYTIC ANAEMIA

Sickle Cell Anemia Clinical Features

omal Recessive Disease

AS (sickle cell trait) is
tomatic; Not serious
aturia, Isosthenuria
papillary necrosis)

Four principle genotypes of sickle cell disease:

	Genotype	Abbreviated For
	Homozygous sickle cell disease	**SS disease**
	Sickle cell-hemoglobin C disease	**SC disease**
	Sickle cell-beta^{+}-thalassemia	**S beta^{+}-thalassemia**
	Sickle cell-beta^{0}-thalassemia	**S beta^{0}-thalassemia**

Sickle Cell Anemia Clinical Features

equences of Vaso-Occlusion:

nful crisis

oke

ute Chest syndrome

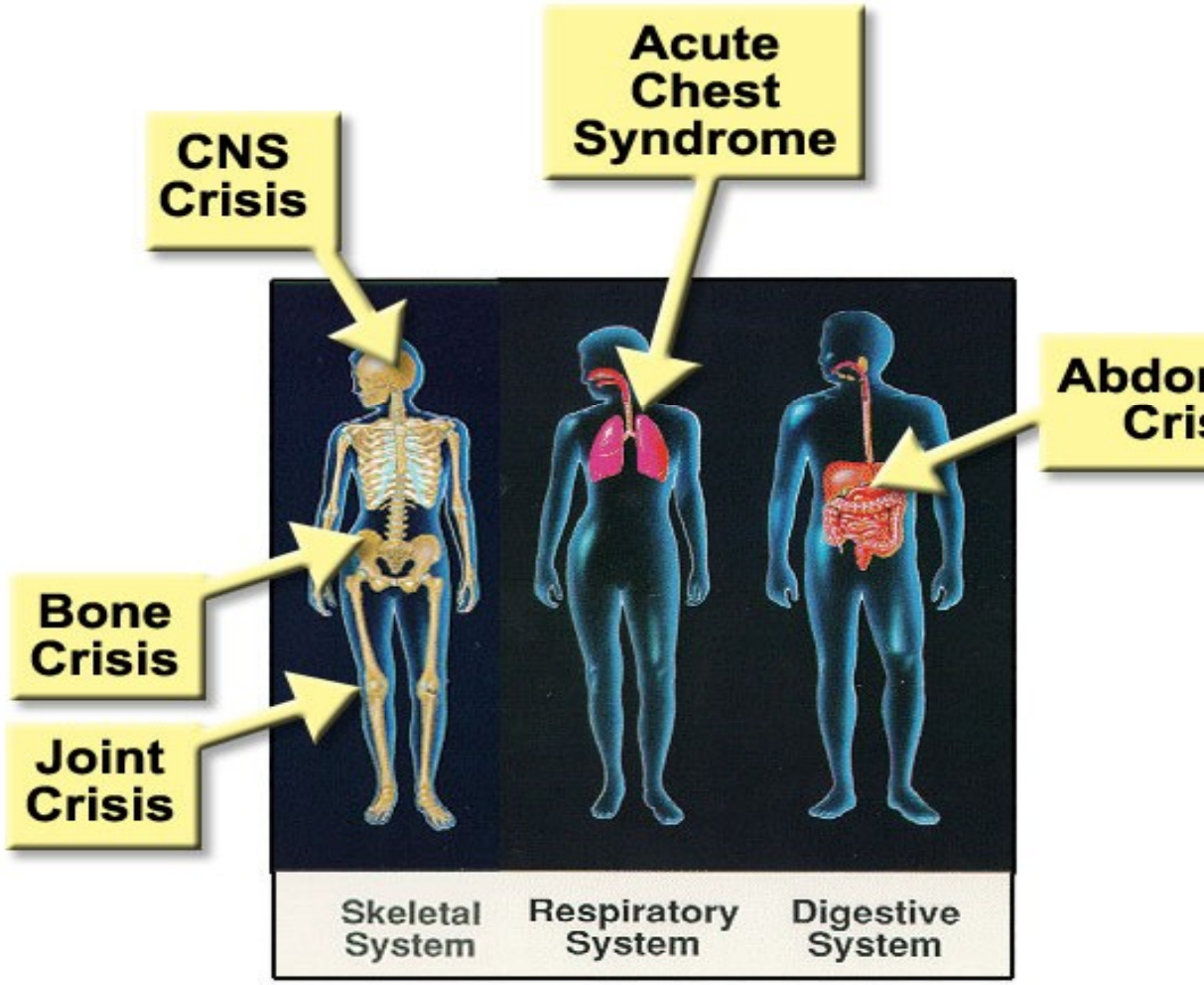
ctylitis

enic infarction

nal Disease

rly Death

HYDRATION,
PREVENTION OF ACIDOSIS,
PREVENTION OF INFECTION
OXYGENATION



COMBINATION THERAPY

IRON + B12

IRON + B12 + TRANSFUSIONS

IRON + B12+ TRANSFUSIONS + ERYTHROPOIETIN

ER INHERITED / STEM CELL DISORDERS AS CAUSES OF ANAEMI

G6PD DEFICIENCY

HEREDITARY SPHEROCYTOSIS

OTHER RBC MEMBRANE DEFECTS

WHAT IS REFRACTORY ANAEMIA?

ANAEMIA NON-RESPONSIVE TO STANDARD THERAPY OR CAUSE NOT RELATED TO COMMON CAUSES OF ANAEMIA.

MDS (myelodysplastic syndromes)

Anaemia of systemic diseases

Anaemia of cancers

Normal a2 thalassaemia

Alpha thalassaemia
Aplastic anaemia
Pure red cell aplasia
Granulomas
Glycogen storage disease

BONE MARROW EXAMINATION

ANAEMIA OF CHRONIC DISEASE

Low Hb, Low Indices,

No e/o thalassaemia or iron deficiency or lead poisoning or blood loss

Mal-utilisation of existing iron

ERYTHROPOIETIN COULD BE DRUG OF CHOICE

IDA-	**TSat- <15%**	**S.Ferritin < 12 ng/ml**
ACD-	**TSat- <20%**	**S.Ferritin < 100 ng/ml**

Ret He and soluble transferrin receptor

Laboratory findings in differential diagnosis of IDA

t	Iron deficiency anemia	Alpha/beta thalassemia	Anemia of chronic disease
noglobin	Decreased	Decreased	Decreased
an Corpuscular Volume	Decreased	Decreased	Normal-decreased
l Cell Distribution Width	Increased	Normal	Normal-decreased
throcyte Protoporphyrin	Increased	Normal	Increased
al iron-Binding Capacity	Increased	Normal	Decreased
nsferrin Saturation	Decreased	Normal	Decreased
um Ferritin	Decreased	Normal	Increased
nsferrin Receptor	Increased	Normal	Increased

ASSESSMENT OF THERAPY

CBC - Indices, reticulocyte count, haemoglobin

RISE IN Hb 1-1.5 GM/DL AT 10 -14 DAYS

Improvement in performance scale

Inadequate response-need for further evaluation

CASE STUDY 1
FEMALE 19 YEARS

- **SYMPTOMATIC ANAEMIA**
- **NO FEVER / CONSTITUTIONAL SYMPTOMS**
- **LOW HB SINCE ATLEAST 5 YEARS**
- **MULTIPLE IRON THERAPY**
- **RECENTLY VITAMIN B12 THERAPY**

• **HB**	**9 GM %**
• **MCV**	**59**
• **MCH**	**19**
• **MCHC**	**32**
• **RBC COUNT**	**5.9**
• **WBC**	**5900**
• **PLTS**	**476,000**

HB Electrophoresis

Hb A 98%, A2 1.8%
Iron Studies Normal
Normal B12
No bleeding
No drugs

alpha thalassaemia

CASE STUDY 2
MALE 39 YEARS

HB Electrophoresis normal
S Ferritin /Iron Studies normal
Normal B12
No bleeding
No drugs

- **SYMPTOMATIC ANAEMIA**
- **NO FEVER / CONSTITUTIONAL SYMPTOMS**
- **LOW HB SINCE ATLEAST 3 MONTHS**
- **RECEIVED IRON AND B12 THERAPY**
- **NO RESPONSE**

HB	**7 GM %**
MCV	**91**
MCH	**28**
MCHC	**32**
RBC COUNT	**1.3**
WBC	**6800**
PLTS	**357,000**

Bone marrow examination

PURE RED CELL APLASIA

THYMOMA ASSOCIATION

ase study 3
nale 72 years

Retired profes

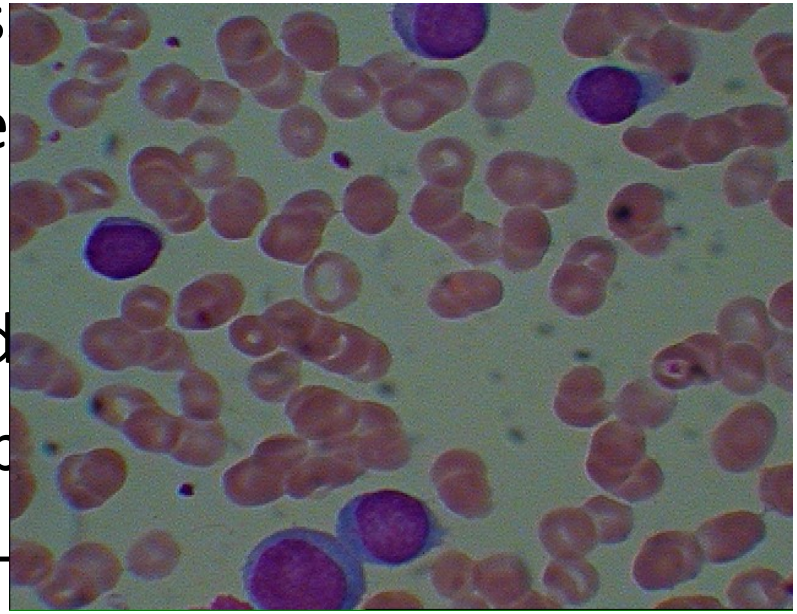
Severe fatigue

Dark urine

Appears jaund

Admitted for b

X match sent -



Hb 6.9 gm%
WBC 65400/cumm
Plts 132,000
MCV 98
ALC 47,000/cumm

DCT positive
ICT negative
S IgG 400 MG/L
RETIC 12%

FLOW CYTOMETRY S/O Chronic Lymphatic Leukaemia

Autoimmune haemolytic anaemia
Responded to steroid + IVIgG therapy

No palpable LNs
No organomegaly

ase study 4

32 YEAR OLD MAN
Asymptomatic other than fatigue
Recurrent iron deficiency anaemia
Responds well to oral iron
But then recurs

What is you next step?

A	**Continue replacement therapy**
B	**Observe**
C	**Endoscopy evaluation**
D	**Blood tests for malabsorption**

Case study 4

32 YEAR OLD MAN
Asymptomatic other than fatigue
Recurrent iron deficiency anaemia
Responds well to oral iron
But then recurs

What is you next step?

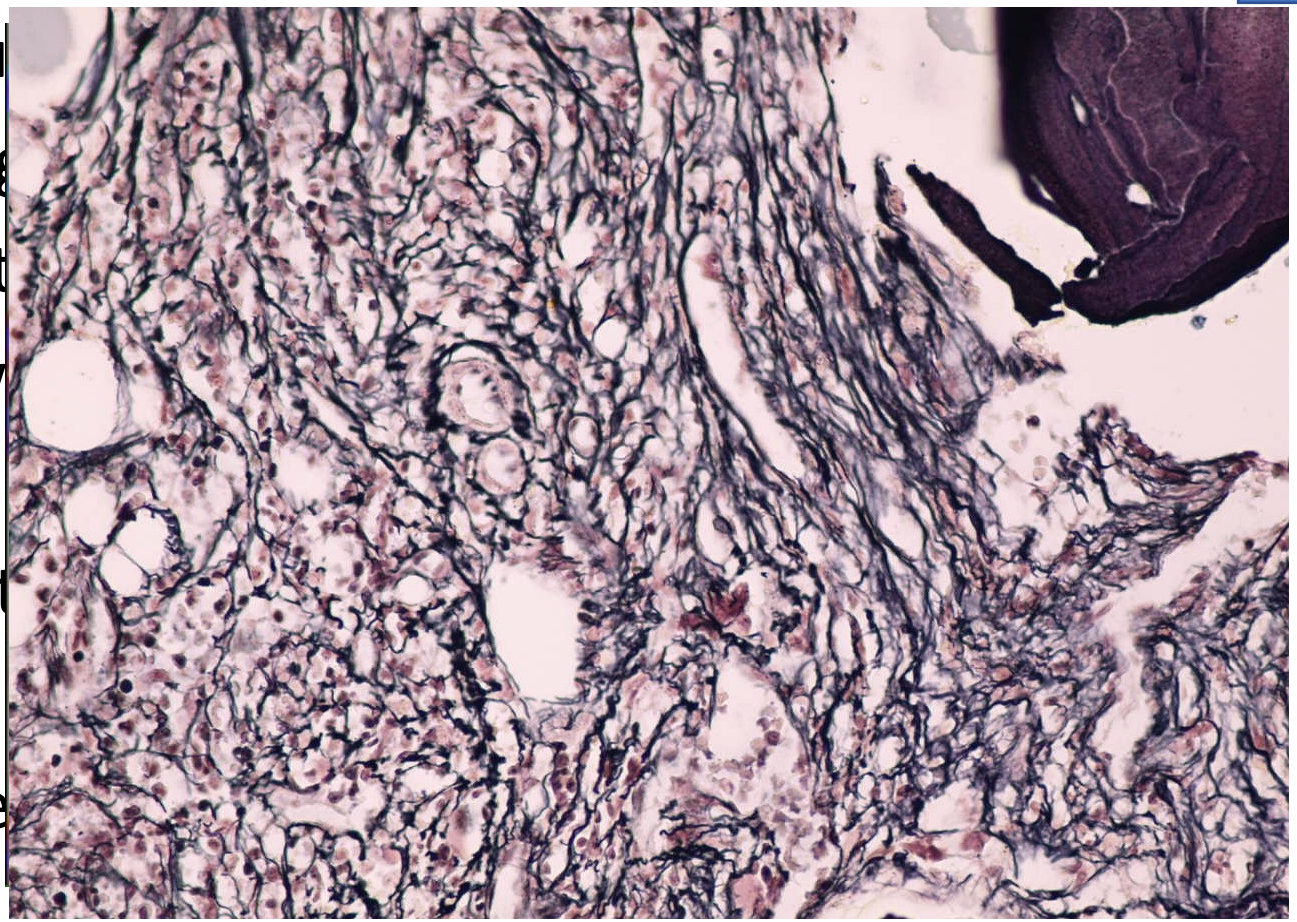
CA Colon
Resected
No chemotherapy

A	**Continue replacement therapy**
B	**Observe**
C	**Endoscopy evaluation**
D	**Blood tests for malabsorption**

ase study 5
emale 63 years

Primary myelofibrosis

Hou
Fatig
No t
Low
Red
Bilat
Abd
Sple



Hb 9.3 gm%
WBC 4500/cumm
Plts 153,000
nRBCs 10/100WBC
MCV 95
Normal Diff

Normal LFT, creat

ASE 6 Male 68 years

- Weight loss 10 kg over 6 months of the lockdown
- No blood loss anywhere
- Drop in Hb from 11.8 gm% to 6.5 gm%
- Transfused for symptomatic anaemia
- Post transfusion Hb 10gm%
- Stool occult blood positive
- Colonoscopy repeated

Angiodysplasia in the colon

COMMUNICATION / DISCUSSION/ opinions

CONCLUSIONS:

ia is an event for anaemia – not a diagnosis

specialty faces anaemia- iron and vit B12 deficiency are the commonest

miss a haemoglobinopathy – thalassaemia or sickle cell disease

ise approach with an automated CBC and well stained peripheral smear

o assess cause of anaemia pathophysiology

with relevant investigations (EBM)

blood transfusion unless specific indication

ement therapy for iron / vit b12 / folate or EPO
narrow examination as per indication

t underlying cause

response at intervals

Parenteral iron is gaining importance in IDA

Detecting beta thalassemia is extremely importa
prevent thalassaemia major birth- HB electropho
CVB, Amniotic cells

Using an algorithm for assessment of underlying
thalassaemia is necessary

In areas endemic to sickle cell or patients coming
such areas check for sickle cells

Thank you

"Approach to anaemia - A Clinician's view" can be viewed on the Jaypee Publishers website