Anemia
10 Mehr 1399

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Anemia : case presentation:

77 years old female reported for weakness and anemia of 2 years duration On 11,06 1390 . She was pale

With few skin ecchymosis quite alert, BP has been high and treated with Norvasc (amlodipine 5 mgs) Besylate 2 times per day , lymph nodes not palpable , spleen was palpable 4 cen.. Below costal margin, . liver 2 cen. below costal margin.

Anemia

CBC: WBC 7000 with normal differential , RBC 4 500 000 per ml . Hct 30 per cent, hemoglobin 10.5 gm. per dl . MCV 100 fl . Platelet 520000 .creatinine 1.4 (1.2 ml) FBS 87 Ferritin 350 thyroid test normal . Reticulocyte count 0.4 per cent , LDH 600 unit .Si 223 TIBC 380 Ferritin 400 bc.r abl and Jack 2 and other genetic test negative

Peripheral smear: Macrocytosis, Tear drops. Diverse population high plantlets count some very large .

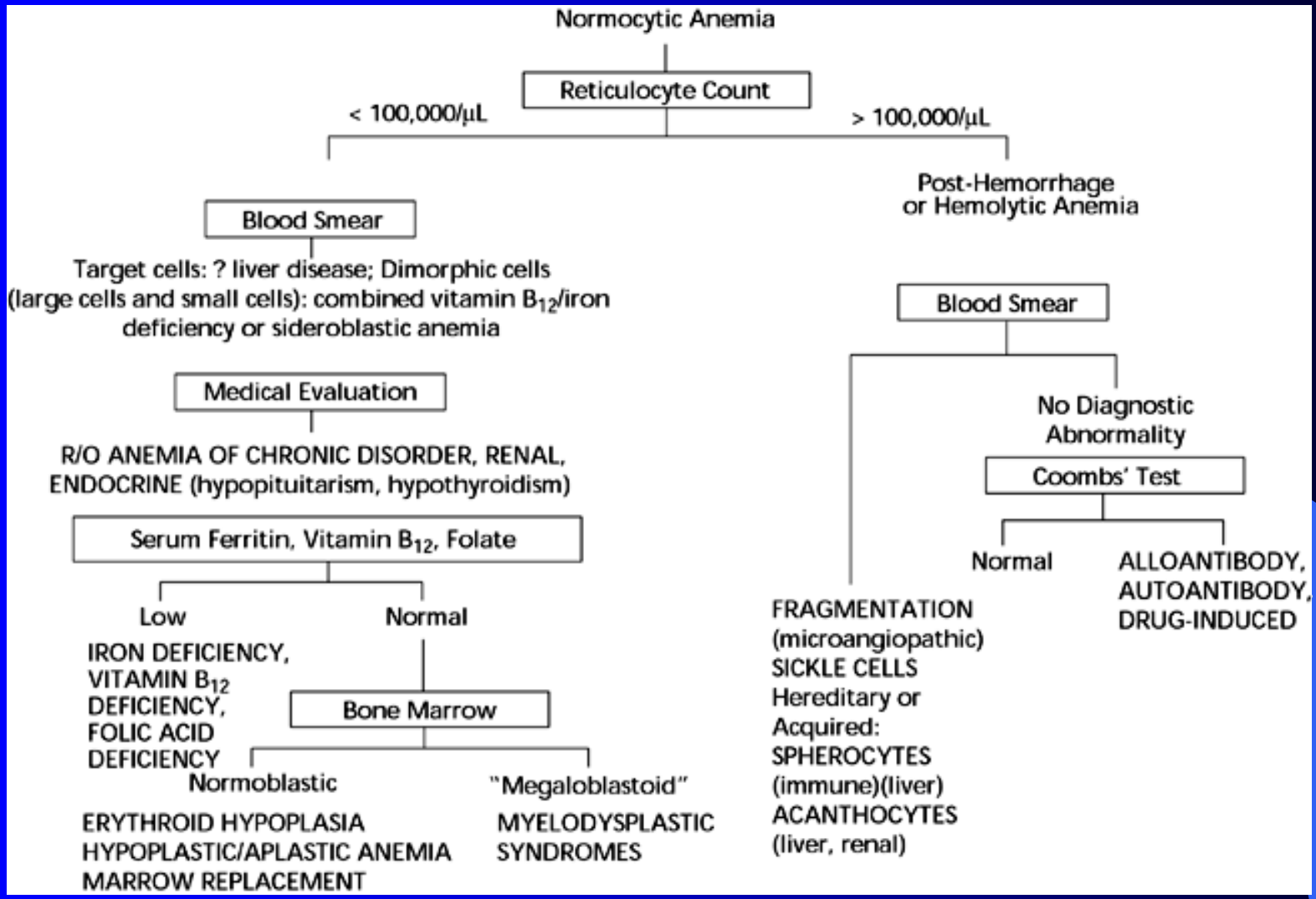
ESR 30 -60 ,

Differential diagnosis

Myelofibrosis . Myelodisplasia (MDS) , Myeloprolipheratife disease .

S 900 16 31 Bone marrow

pan myelosis and dysplastic in both erythroid and myeloid series suggestive for MDS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Reticulocyte Count
< 100,000/µL
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Post-Hemorrhage or Hemolytic Anemia
Blood Smear
Target cells: ? liver disease; Dimorphic cells (large cells and small cells): combined vitamin B12/iron deficiency or sideroblastic anemia
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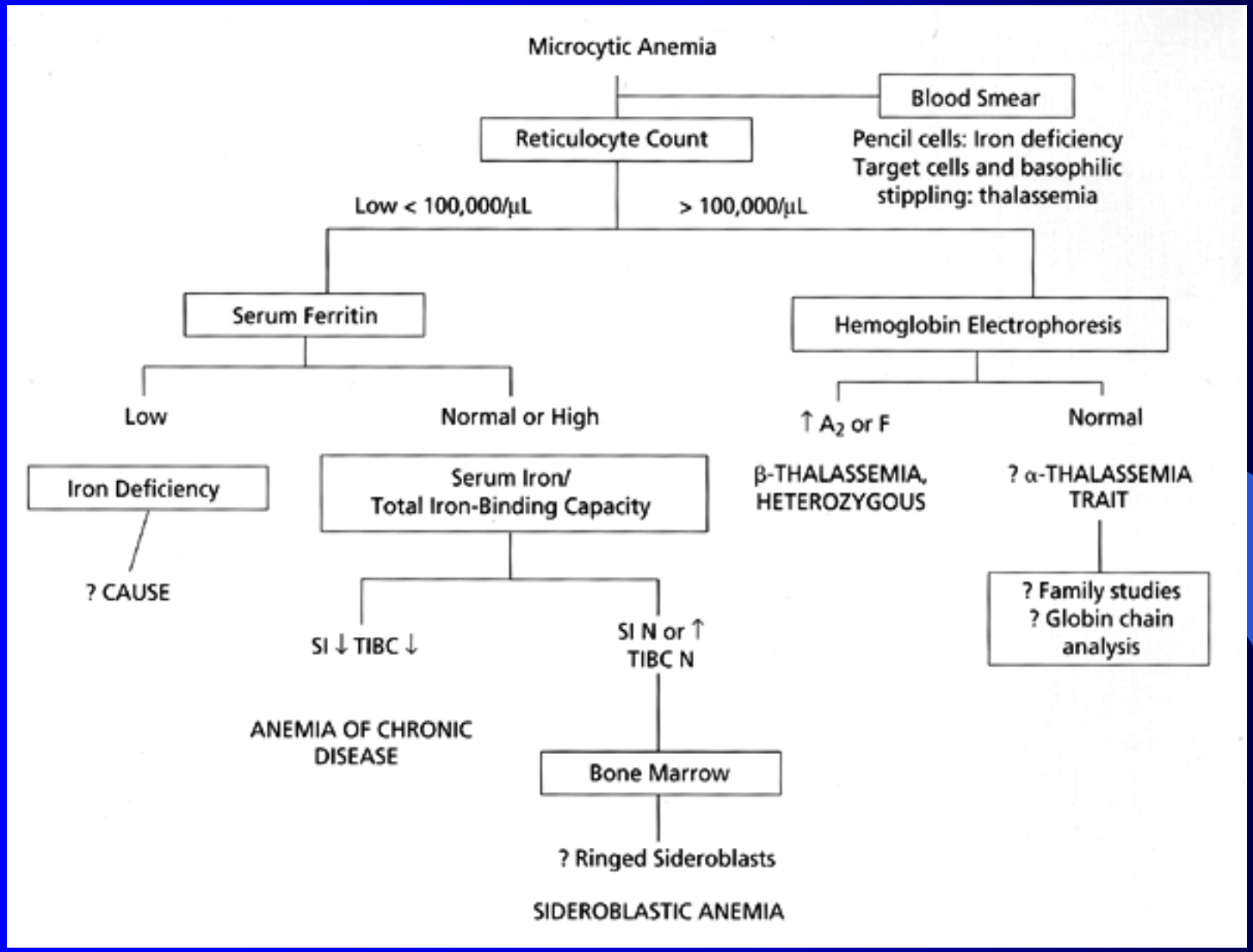
ALLOANTIBODY, AUTOANTIBODY, DRUG-INDUCED
FRAGMENTATION (microangiopathic) SICKLE CELLS Hereditary or Acquired: SPHEROCYTES (immune)(liver) ACANTHOCYTES (liver, renal)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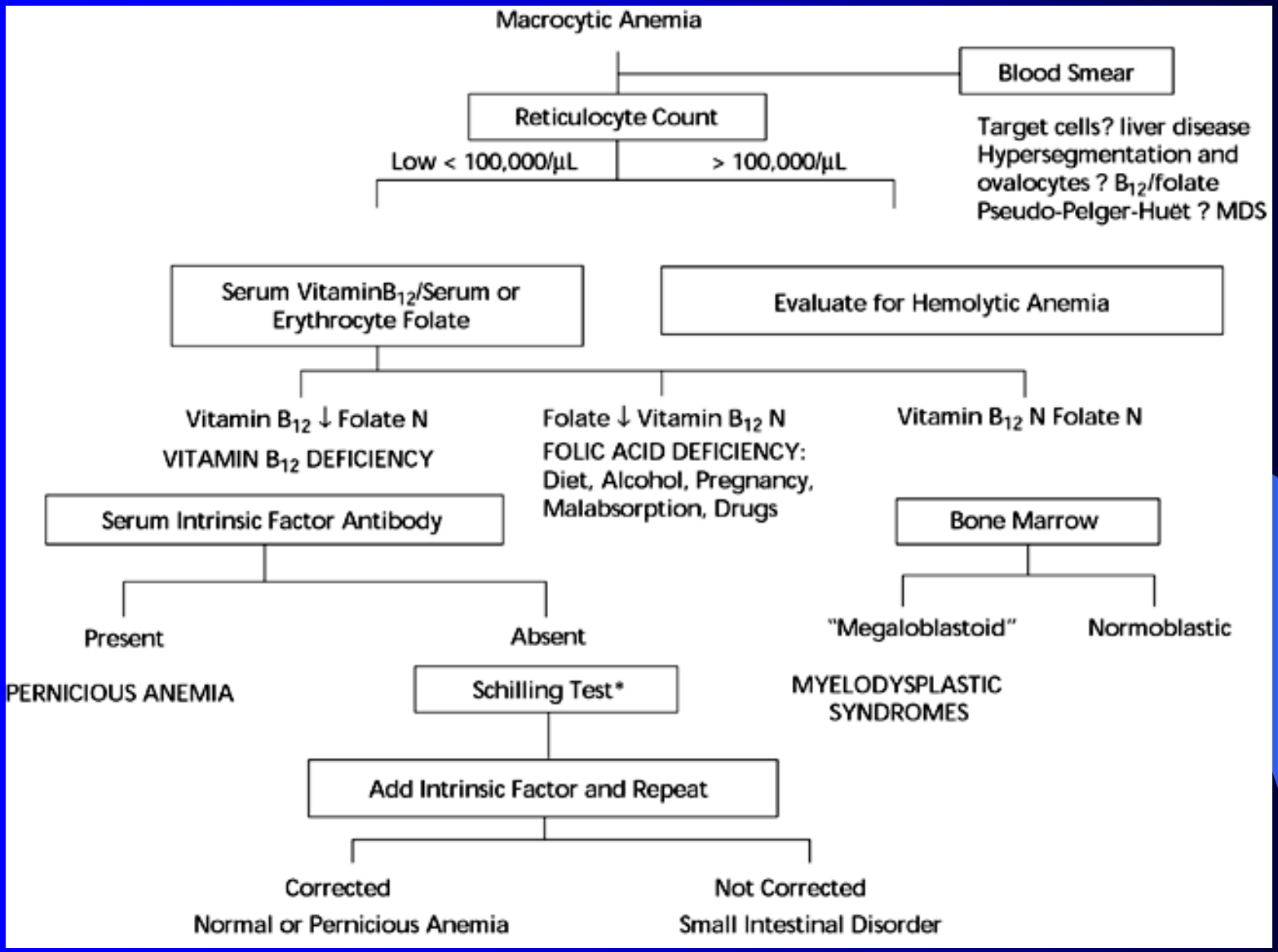
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Vitamin B12 ↓ Folate N
VITAMIN B12 DEFICIENCY
Folate ↓ Vitamin B12 N
FOLIC ACID DEFICIENCY:
Diet, Alcohol, Pregnancy,
Malabsorption, Drugs
Vitamin B12 N Folate N
Serum Intrinsic Factor Antibody
Bone Marrow
Present
Absent
"Megaloblastoid"
Normoblastic
PERNICIOUS ANEMIA
Schilling Test*
MYELODYSPLASTIC
SYNDROMES
Add Intrinsic Factor and Repeat
Corrected
Normal or Pernicious Anemia
Not Corrected
Small Intestinal Disorder

Treatment course

23.06.1390 . folic acid 1 Mg , B 12 1000 mg weekly b6 40 mg/ for times per day for a month. no improvement,

02,08.1390 : WBC 6900 RBC 303.000 Hb 10.5 gm .100 m; MCV : 101 fl platelet 576000 ml

Anemia

کم خونی زمانی که هموگلوبین به کمتر از مقدار طبیعی برای سن و جنس رسید وجود دارد:

۱-۲ ساله g ۱۱ در 100cc

۳-۵ ساله g ۱۱.۲در 100cc

۶-۱۱ ساله g ۱۱.۸در 100cc

Anemia

12-15	12.6g
16-19	13.6g
20-29	13.7g
30-49	13.7g
50-69	13.3g
>70	12.4g

Anemia

خانم ۱۲ تا ۱۵سال ۱۱.۹ آقایان کمتر از 14g در 100cc

خانم ۱۶ تا ۶۹ سال ۱۲ آقایان 12g در 100cc

خانم بالاتر از ۷۰ سال <۱۱.۸ آقایان کمتر از 12g در 100cc

Treatment

folic acid 1 mg b 12 1000 per week and b6 40 mg 4 times per day

02.10.1390 erythropoietin (cinapoetin) 10000 per week:

26,10, 1390 : Hydrea 500 cap , day and aspirin 80 mg per day

19,01 ,91 : cinapoetin (erythropoietin 10000 unit) pne per week Hydroxiurea 500 mg 0ne per day b12 10000 per week 2 times b 6 40 mg 4 times per day

03,01.91 : WBC 6700 , hem 12.4 gm platlet 283000 . erythropoietin 10000 per week .

05.11.91 : WBC Platelet 359000 hemoglobin 14.2 hct 43.1

Continuation of treatment

07.12.91 : treatment repeated

09.13.91: WBC 6200 hemoglobin 13.6 hct 39. Hydrea dermatology consult

02.07.92 : WBC 7100 hct 38 hb 13.2 .7 erydropoetin and hydrea

04,19.92 , hydrea 500 5 days per week erythropoietin 10000 per week

25.06.92 : wbc 6500 platlet 463

05.09,92 . wbc 6400 heglobin 13 hct 32 MCV 102

11.15.92 urinalysis normal

Fallow up

26.01 93 : Kidney stone noted hct 39 mcv 104 WBC 5600 heh 13.2 pla 203000 hydrea 500 aspirin 80

06,02 .93 : hydrea 500 aspirin 80 b6 erythropoetin 10000 WBC 6500 hct 40 plct 380000 MCV 104

10.15.93 : WBC 5600 hct 38 heglobin 13

12.18 93 : Brain MRI for head ache : Mengioma

Patient traveleed to paris continued the same treatment

22.09.95 : 299000 wbc 8400 hct 38 ciclosporin 100 per day cinapoetin folic acid b6 methl prednisone

18.11.95 : wbc 10000 platelt 322000 hct 38 hemoglobin 12.8

12.01 95 : Plat 328000 hct 28 CHF liver large

23.12.95 : wbc 12000 .platlet 304000 hct 27.2

28.01 .96 lleg edema sprinolacton and furesamide and captopril 25 6.25 qid

13.02.96 wbc 11.200 platlet 222000 hemoglobin 11.3 hct 33 amlodipine 5 daily

3.3.96 : wbc 12.600 hemglobin 10 hct 29

Continuation of treatment

28.03 .06 : wbc 10500 plate,et 220000 Hydrea 500 cinapoetin 10000 methyl prednisone hct ee22 one unit packed cell b6 folic acid

06,04,96 : hemoglobin 8,5 hvy 25 ,1 plat 8340000

11.6.9

> wbc 13200 plat 728000 hem 11.4 mcv 86 severe arthrosis and limping

25.06 .96 : He 8.1 wbc 7800 plar 69000

15.07 .96 : platelet 138000 hemog 7l5 packet cell 2 unit in 2 days

Continuation of treatment

08.08.96 : ych 28 hem 9 wbc 6800 plat 49000 cinapoetin methyl prenisone sprinolacton

22 .00.96 : plat 9400000 cinapoetin furosemide

16.10.90 Liver and spleen enlarged, good esprit encouraged for treatment

07.11.96 : liver enlarged ,heglobin 9.8 hct 28.8 wbc 9800 p lat 74000

07.12.96: heglobin 10 wbc 9900 plat . concern about weight loss , 84 years advised eating yougurt and honey , rippe of lamb . lamb feet soup (Pache and sirabi)

19 .01 97 : hemoglobin 12.9 hct 35.6 platlet 471000 wbc 7400

Wight loss concern: Pamidronate ciclosporin

Anemia in daily practice

- Anemia is decreased proportion of red cells to total blood volume . It is the most prevalent disease in nature
- We would learn the significance of anemia in medical practice and the best management.
- Every day about 50 per cent of your patients have anemia.

Diagnostic Criteria for anemia

- In men Hct. <40 or Hb < 14 g/dl or Red cell mass < 4.5 X 10/12 /L
- In woman Hct <37% OR Hb < 12 g/dl

Or Red cell mass < 4.0 X 10, 12 /L

When to suspect anemia

- Symptom: dizziness ,exertional dyspenea, fatigue,Headaches, loss of libido, mood disturbances ,sleep disturbances ,tinnitus, weakness .
- Signs: Glossitis,Jaundice ,Neurologic findings, Pallor ,Orthostatic hypotension,Peripheral edema,Retinal hemorrhages splenomegaly ,Cariac ejection murmurs,thachycardia,thachypnea ,venous hum

Systematic approach to the diagnosis of anemia

- History and physical examination ,family history ,diet, vitamin use ,pica excessive menstrual bleeding ,heart burn, tarry stool
- blood cell counts, peripheral smear .
- MCV (Red cell size evaluation):the most efficient organized approach to diagnosing causative factors or specific clinical disorders: microcytic,normocytic and macrocytic .leukocytes and differential reticulocyte count, LDH.morphologic abnormalities may suggest specific disorders

What do we see in peripheral blood and what it means

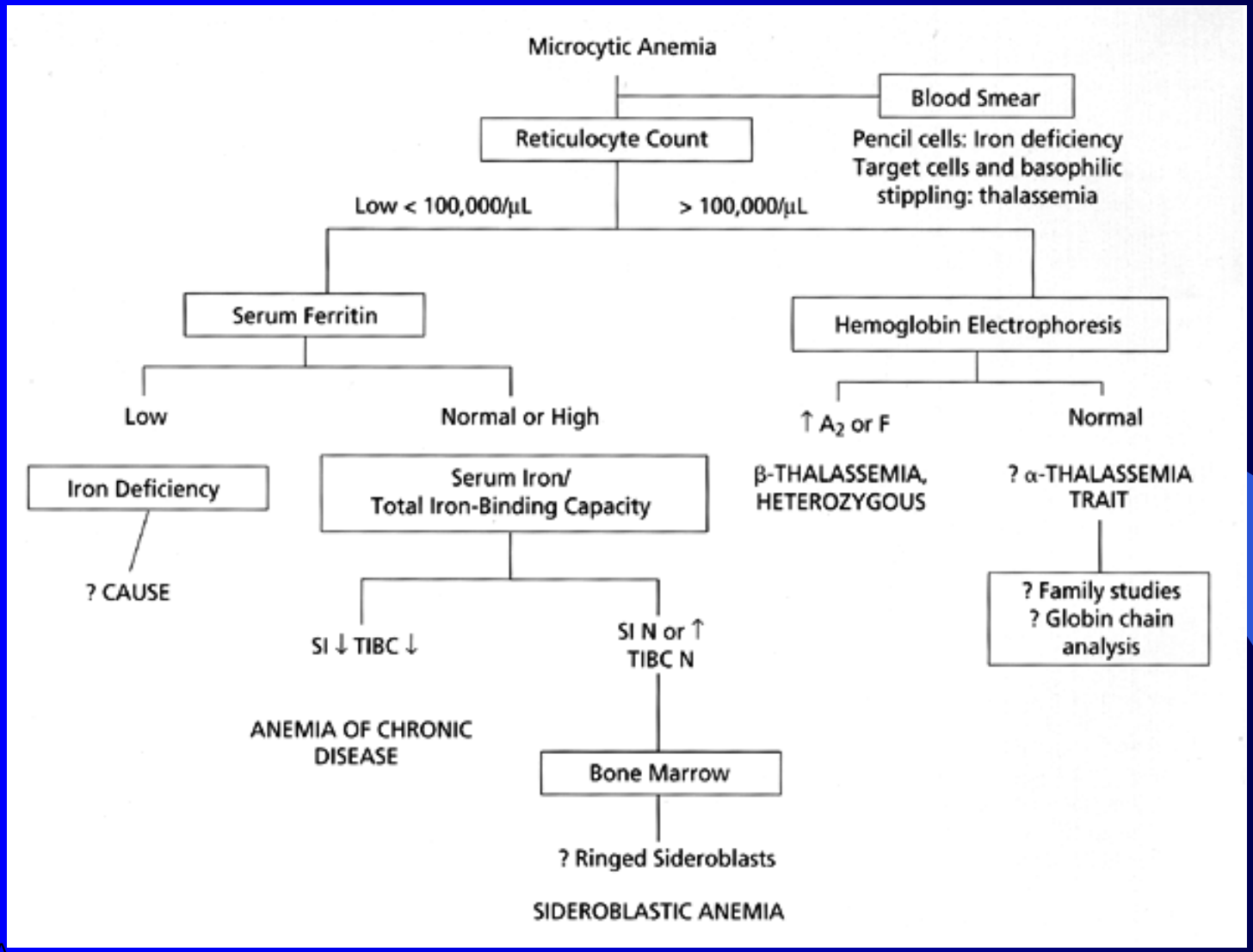
- Hypochromia(minor thallasswemia and Iron deficiency or inflamation .
- Fragmented RBC(Schistocytes) Microangiopathic or traumatic hemolysis.
- Macro-Ovalocytes,Hypersegmented neutrophils (Vitamin B12 or folate deficiency .Tear drop cells, NRBC : Hemolysis thallassemia,myelofibrosis,megaloblastic
- Echinocytes (burr cells) Renal failure ,Iron deficiency ,
- Spur cells : Liver disease
- Low platelets ,blasts , low white count ETC

Medication that cause certain anemias

- Macrocytic: chemotherapy of cancer, Cephalosporin,sulfasalazine, zidovudine
- Sulfanamides
- Hemolytic anemia: Cephalosporin, Dapsone,Methyldopa ,Sulfonamides Primaquine ,Quinidine ,Procainamide,
- naphthalene

Microcytic Anemia

- The most common cause of microcytic anemia is iron deficiency usually related to menstrual or gastrointestinal bleeding.
- frequent other cause of microcytosis in Iran is minor alpha or betha thallassemia
- Anemia of chronic disease and sideroblastic anemia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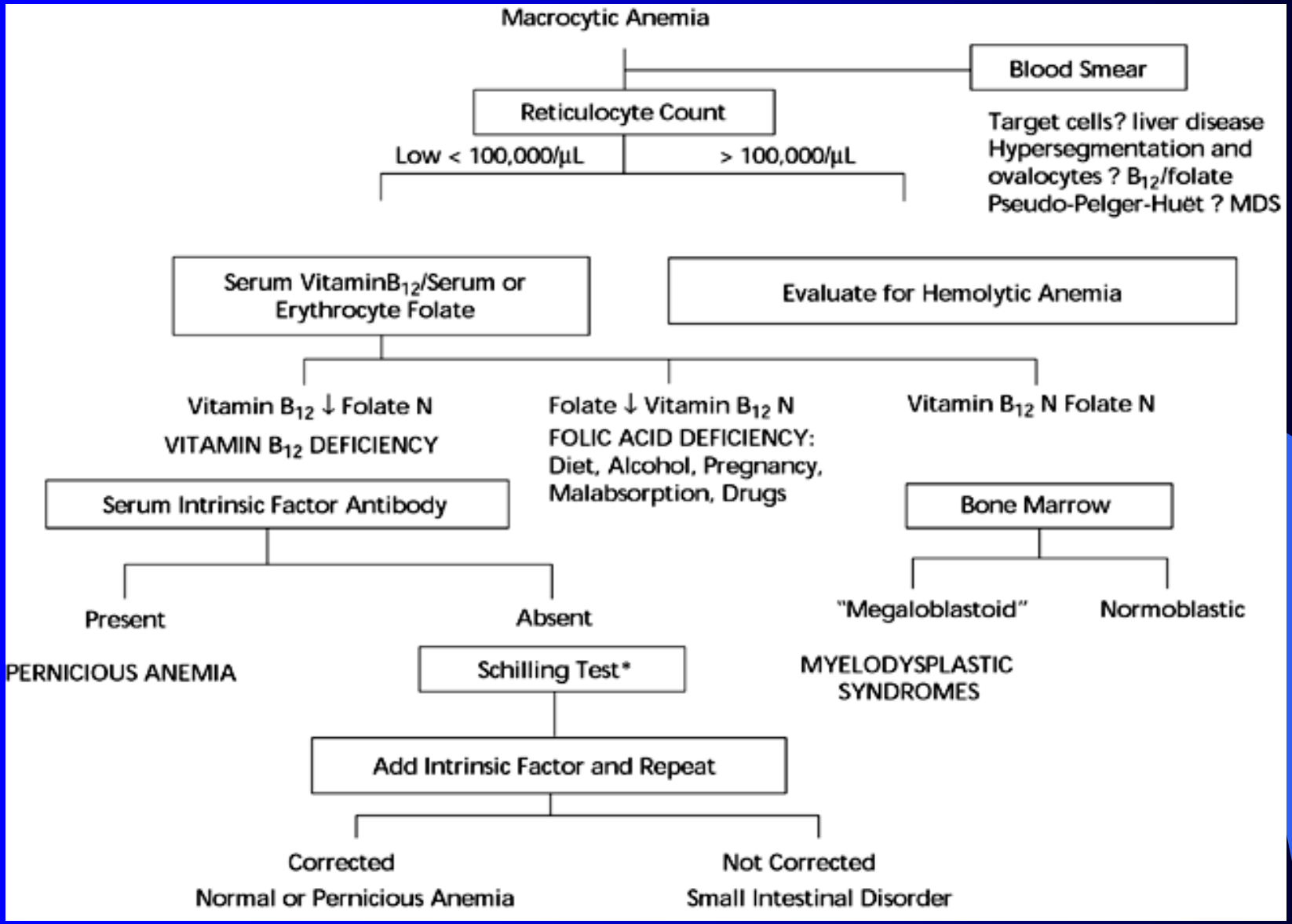
A

Most common causes of microcytic anemia

- Iron deficiency anemia,
- Some thalassemias
- Anemia of chronic disease
- Sideroblastic anemias

Macrocytic anemia with megaloblastic erythropoesis

- Copper deficiency,Drugs(Chemotherapy agent ,antivirals, anticonvulsants, oral contraceptives,folate deficiency ,Primary bone marrow disorders ,B 12 deficiency ,Vitamin C deficiency ,Hemolysis , infection ,trauma,congenital erythopoietic
- Anemia and purpura ,Hypophosphatemia,Hemoglobinopathies , G6PD deficiency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Causes of Vitamin B 12 and folate deficiencies

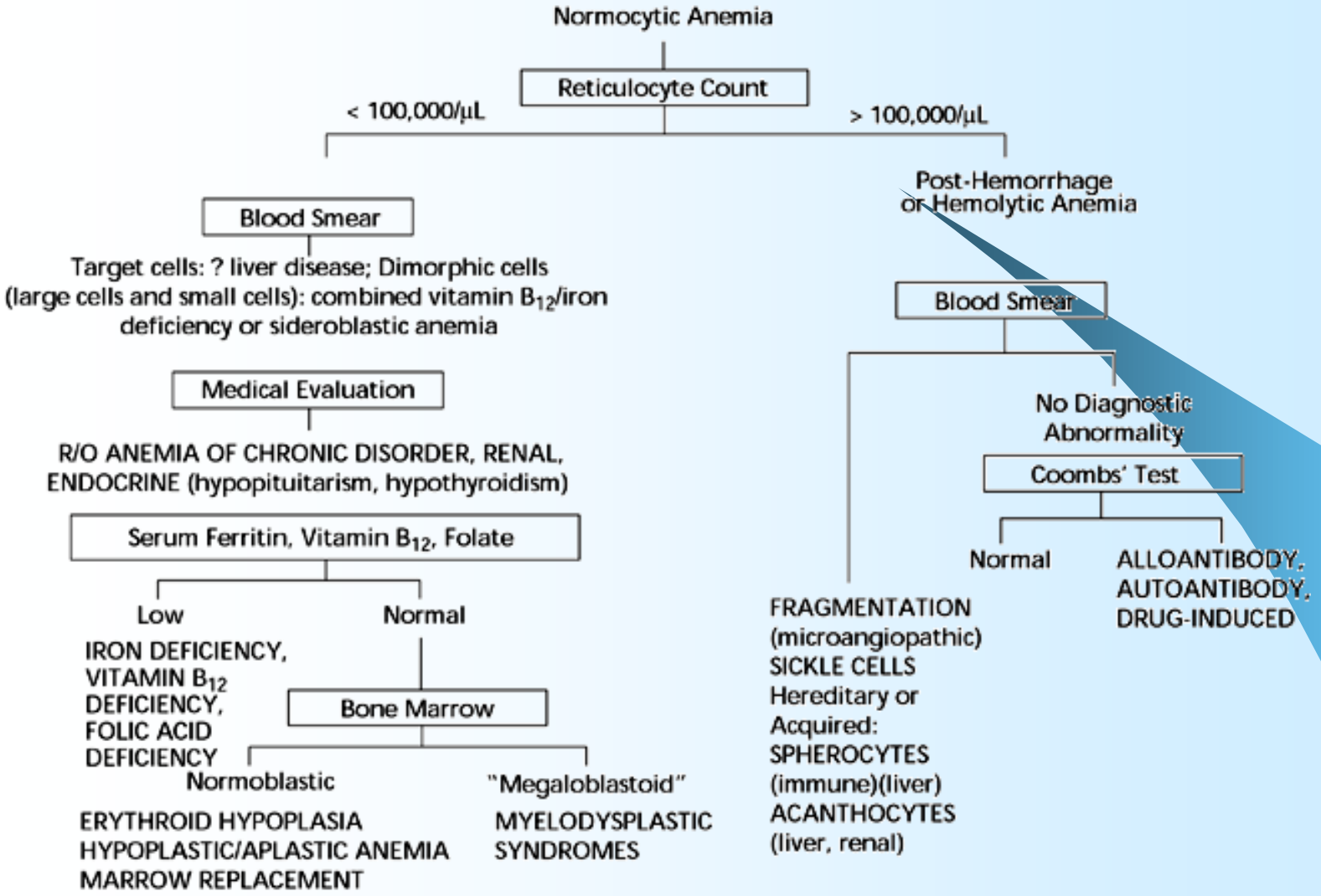
- Bacterial infestation of bowel diverticula
- Chronic pancreatitis insufficiency ,Chron's disease,Gastrectomy ,Hyperthyroidism ,Ileal resection ,Vegetarianism and malnutrition ,pregnancy and lactation .
- Malabsorption ,medications parasitic infestation ,perniciuous anemia ,sprue ,alcoholism

Macrocytic anemias

Clinical Finding	Pathogenesis	Peripheral smear
Pernicious anemia	Cobalamin deficiency	Oval macrocyte ,hypersegmentation
Nutritional deficiency malabsorption,pregnancy,drugs	Folic acid deficiency	Oval macrocyte ,hypersegmentation
Hemolytic anemia	Erythrocyte destruction ,compensatory reticulocytosisis (Reticulocytes are largers cells)	Round macrocytes,polychromasia
Liver disease	Increased erythrocyte membrane	Round macrocytes, ,target cells
Myelodysplasia	Dysplastic erythroid maturation , chromosomal abnormalities	Round macrocytes, target cells, pseudo-pelger –Huet cells (Bilobled neutrophils

Most common Normocytic normochromic anemia

- Anemia of chronic diseases .
- Hypopastic anemias ,Myelodysplasia ,
- Blodd loss , other bone marrow failure and
- Compensated hemolytic anemia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No Diagnostic Abnormality
Coombs' Test
Normal
ALLOANTIBODY, AUTOANTIBODY, DRUG-INDUCED
FRAGMENTATION (microangiopathic)
SICKLE CELLS
Hereditary or Acquired:
SPHEROCYTES (immune)(liver)
ACANTHOCYTES (liver, renal)

Classification of Hemolytic Anemias

Inherited Intracellular Defects Membrane abnormalities Hereditary spherocytosis Hereditary elliptocytosis Hereditary stomatocytosis Hereditary pyropoikilocytosis Enzyme abnormalities Deficiency of erythrocyte glycolytic enzymes Deficiency of enzymes in the pentose-phosphate pathway and glutathione metabolism Hemoglobinopathies **Acquired Intracellular Defects** Paroxysmal nocturnal hemoglobinuria **Extracellular Defects** Immune hemolytic anemias Alloimmune Autoimmune Drug-induced Traumatic (microangiopathic) hemolytic anemias Hemolytic anemias due to chemical and physical agents

Hereditary spherocytosis

- Autosomal dominant disease characterized by mild anemia ,spherocytosis ,splenomegaly and increase red cell osmotic fragility fragility , Severity of disease may vary in families.The disease may be complicated by aplastic crisis,chronic leg ulcer and Gall stones, reticulocyte is high and MCV usually normal .Treatment is splenectomy.

Autoimmune hemolytic anemia(warm Autoimmune)

- This hemolytic anemia is associated with lymphoproliferative disorder ,systemic lupus erythrematosus ,ulcerative colitis , congenital immunodeficiency disorders and AIDES ,
- Symptoms are anemia ,jaundice ,dark urine,abdominal pain and fever , . Bone marrow is necessary only to R/O other causes . Aplastic crisis is rare and comb's test is positive is 50 % of cases Treatment is prednisone 1 mg/kilo and if more than 15 mgs is needed for maintenance splenectomy is advised. And there is more than 50 % good response.

Hemolytic anemia (Cold Autoimmune)

- This is caused by Ig M and has specifity for “I or i) red cell antigen .This has the same etiology as warm auto immune . Infectious mononucleosis and mycoplasma pneumonia are also responsible . Raynaud phenomena and livedo reticularis and old age is typical.Treatment with prednisone and splenectomy is not effective .Plasmaphoresis may be effective

Hemolytic anemia (Drug induced)

- Drug may induce the production of auto antibodies . (auto antibody mechanism) {Like methyl dopa)
- The drug binds to erythrocyte membrane and an antibody bind to drug –erythrocyte complex .(The hapten mechanism.Pencillins)
- A drug –antibody complex may bind to erythocyte and cause complement activation with overt hemolysis(innocent bystander)

Common infections and inflamation causing hemolysis

- Autoimmune hemolytic anemia,
- Collagen vascular disorder,Malignancy medication ,microangiopathic hemolytic anemia,DIC Meningococcal septicemia rocky montain spotted fever TTP,Vasculitis ,Gas gangrene septicemia with streptococci, malaria mycoplasma

Enzymes defect: Glucose-6-Phosphate Dehydrogenate Deficiency

- A 33 years old man present to the emergency room with urinary tract symptoms and is given a sulfa drug .He is asked to see his primary care physician for complete evaluation. Over the next few days he experience fever ,fatigue and dark urine .On a return visit his hemoglobin is 7 .0 g/dL ,and his reticulocyte count is 6.) .His peripheral blood show polychromatophilia and blister cells .An erythrocyte G6PD assay is low normal.

Enzyme Defects, G6PD deficiency

- Embden Meyerhoff pathway : Pyruvate Kinase deficiency ,autosomal recessive. Normal osmotic fragility and has autohemolysis ,Splenectomy would help.
- The hexose monophosphate shunt :G6PD is the critical enzymes . This is a x-linked polymorphic enzymes ,very common (100 million)
- Acute hemolysis in Mediterranean (Very low enzymes
- Mild hemolysis in black
- Chronic hemolytic anemia (Non -Mediterranean

Hemoglobinopathies congenital

- Molecular anomaly of the alfa or betha chain .

- Hemoglobin S: 20 % of people in west and central Africa (Substitution of Glu with Val in position 6.
- Hemoglobin M: Oxidation of the heme iron (methhe --moglobin : Alteration of histidine residue)
- Unstable hemoglobin : Productiobn of unstable tetramer due to substitution of amino acid in in the alfa or betha chain
- Hemoglobin with altered oxygen affinity : anemia or erythrocythemia : Alteration of oxygen biding

Hemaglobinopathies, Aquired

- ***Increased F*:** pregnancy ,myelomonocytic leukemia,acute leukemia,CML,Myelofibrosis,MDS,Megaloblastic anemias,Trisomy 21, myeloma and brochogenic carcinoma,
- ***Increased Hemoglobin H*** erythroleukemia ,
- ***Decreased in Hb A2, :*** Iron deficiency,Sideroblastic anemia,erythroleukemia

Alfa and betha thallassemia

- β-Thalassemias are inherited disorders of β-globin synthesis rather than amino acid substitutions. Thus, in most patients with thalassemia, the globin structure is normal, but the rate of production is reduced
- The α-thalassemias result from the loss of α-globin genes, causing an imbalance of α-chain production of hemoglobin. There are normally four genes for α-globin production,

Refractory anemia :.
Refractory anemia with ring sideroblasts:
Refractory anemia with excess blasts:
Refractory cytopenia with multilineage dysplasia:
AML).
Refractory cytopenia with unilineage dysplasia:
Unclassifiable myelodysplastic syndrome:.
Myelodysplastic syndrome associated with an isolated del(5q) chromosome abnormality:
Chronic myelomonocytic leukemia (CMML).

Hereditary persistence of fetal hemoglobin (HPFH, BrE: Hereditary persistence of foetal haemoglobin) is a benign condition in which significant fetal hemoglobin (hemoglobin F) production continues well into adulthood, disregarding the normal shutoff point after which only adult-type hemoglobin should be produce

Normochromic normocytic anemia regularly develops in chronic renal failure when the glomerular filtration rate drops below 20-30 ml/min. ... The predominant cause of inadequate erythropoiesis is a failure to increase erythropoietin (EPO) production in response to the developing anemia.

\The type of anemia that is common with RA is called anemia of chronic disease. RA-related anemia can be treated directly, but keeping RA symptoms under control is the best way to address anemia. Rheumatoid arthritis (RA) is an autoimmune disorder that causes chronic inflammation of the joints, as well as joint pain.Aug 24, 2016

Hemoglobin level was not related to the site and severit of disease. CONCLUSION: Gastrointestinal diseases that do not usually cause bleeding are frequently associated with iron deficiency anemia in patients without gastrointestinal symptom or other potential causes of gastrointestinal bleeding .

Splenomegaly, which is usually caused by portal hypertension in patients with chronic liver disease, may lead to secondary hemolysis, an increase in plasma volume, macrocytosis and megaloblastic anemia. Alcohol, a common etiologic factor of chronic liver disease, is toxic to the bone marrow.Oct 7, 2009

Iron deficiency anemia is a common type of anemia — a condition in which blood lacks adequate healthy red blood cells. Red blood cells carry oxygen to the body's tissues. As the name implies, iron deficiency anemia is due to insufficient iron.Nov 11, 2016

Megaloblastic anemia (or megaloblastic anaemia) is an anemia (of macrocytic classification) that results from inhibition of DNA synthesis during red blood cell production. When DNA synthesis is impaired, the cell cycle cannot progress from the G2 growth stage to the mitosis (M) stage.

- Other cause of megaloblastic
- Anemia in other deficiency state.
- Hemolytic anemias
- Sckiling disease
- Hb S Modifing therapy
- Scike cell trait
- Other scickeling disorder
- Other heamogloninopathy
- Unstable hemoglobin
- Thallassemia

- Thallasssemia Hemoglobin bart syndrome
- B thalassemia
- Other Thallassemia
- Hereditary persistent hemoglobin F
- Hemoglobin patterns in Hemoglobin disorder
- Non Immune Hemolysis
- Heredithary elliptocytosis
- G6PD deficiency
- PYRUVATE KINASE DEFICIENCY.
- Other red cell enzymopathies

- Drug induced Hemolytic anemia
- Microangiopathic memolytic anemia
- Achantocytosis
- Autoimmune hemolytic anemia
- Cold hemoagglutinin disease
- Leukoerythroblastic anemia
- Aplastic anemia
- Paroxismal nocturnal hemoglobinuria
- Pure Red cell Apasia
- Iron Overload
- Transfusion hemocyderosis

Anemia in Pregnancy
Red cell mass 30 per cent
Plasma volume more than 60 per cent
Hemoglobin lower to 11 gms per dc.lit
1000 mg extra iron is needed
Increased folate requirement .
Prophylaxis40-60 mg elemental iron

Anemia in Pregnancy

Folate requirements

prophylaxis

Thrombocytopenia in Pregnancy

- Non-immune thrombocytopenia
- Immune thrombocytopenia
- Women with a platelet count <20×10/L
- Further reading

Prolonged bleeding after surgery

History and clinical assessment

Investigation

Treatment

Further reading

Anaemia in renal disease

Laboratory features

Manegement

Side effects EPO

Further reading

Anemia in endocrine disease

Pituitary disorders

Thyroid disorders

Adrenal disorders

Further reading

Anaemia in joint disease

Anaemia of chronic disorders (ACD)

Typical features of ACD

Management

Bleeding and Fe deficiency

Further reading