In The Name Of Allah

We will talk about hematological disorders ((anemia))

Anemia is decreased in Hb and RBCs that diseases depend on age and gender.

So Hb in males : 13\_16 and in females : 12 \_15

Anemia very common classified according to size and morphology of RBCs to:

1. Microcytic (small size) mcv <78fl
2. Macrocytic (large size) mcv >99fl
3. Normocytic (normal size) mcv 78\_99fl

The size of RBCs measured by mcv ((mean cell volume))..

**There’s two causes of anemia :**

1. **Decrease RBCs and Hb production :**

\*Haematinics deficiency (*e.g*. *Iron, B12, Folate*)

\*Bone marrow failure (e.g. *a plastic anaemia*)

\* Bone marrow replacement (e.g. *leukaemia, lymphoma*)

\* Hypothyroidism

\*Chronic renal failure (e.g. *erythropoietin deficiency*)

1. **Increase destruction or excessive loss of RBCs :**

\*Acute haemorrhage

\*Chronic blood loss (e.g. *haemorrhoids, menorrhagia, peptic ulcer, cancer)*

\* Haemolytic anaemia (e.g. thalassemia and sickle cell anemia which maybe *congenital or acquired*) .

Signs of anemia depend on : severity ,causes and rapidity with which it develops.

**\*\*clinical features :**

* **General**: pallor, weakness, malaise, tiredness, vertigo
* **CNS**: lack of concentration, decreased memory, syncope, seizures
* **CVS**: palpitation, dyspnoea, angina, congestive heart failure
* **GI**: anorexia, nausea, taste disturbance
* To assess the type, severity, and cause of anaemia we request the following :

Hb

MCV

RBC count

Haematinics

Disease specific investigations (e.g. if the Dr suspect that the pt has thalassemia we do Hb electrophoresis ) .

**\*\*Anemia deficiency three types :**

A\_ iron deficiency

B\_ vb12 deficiency

C\_ folic acid deficiency

**A\_ Iron deficiency anemia :**

The most common cause of anemia worldwide specially in developing countries due to malnutrition .

**CAUSES :**

* chronic blood loss (menstruation, peptic ulcer, haemorrhoids, oesophageal varices, GI malignancy)
* nutritional deficiency (inadequate diet)
* Increased demand (pregnancy, adolescence)
* Malabsorption ( gastrectomy, Crohn`s disease.)

**\*\* clinical features :**

* koilonychia (spoon-shaped nails)
* Dysphagia due to post cricoid webs ( Plummer-Vinson syndrome) we find atrophic glossitis ,dysphagia , post cricoid webs with malignant transformation susceptibility ( it’s very rare ).

**\*\* Diagnosis :**

* Clinical features
* Decreased Hb, decreased microcytic anaemia, decreased serum ferritin, high TIBC (total iron binding capacity )
* GI endoscopy

**B\_ VB12 deficiency :**

* Due to Poor intake (vegetarians)
* Malabsorption (pernicious anaemia, Crohn`s disease , gastrectomy, intestinal resection)

**\*\* clinical features :**

* Signs of anemia plus Premature greying of hair

And Neurological manifestations such :

Paresthesia (of the extremities and in the oral cavity sometimes )

Ataxia

Psychosis

Forgetfulness

\*\* **Diagnosis :**

* Clinical features
* Decreased Hb, macrocytosis,decreased B12
* In pernicious anaemia the cause is ((Auto-antibodies against gastric parietal cells and intrinsic factors which responsible for VB12 absorption .
* Schilling test (*radiolabeled* *B12 absorption test*)

**C\_ Folate deficiency :**

* Due to Inadequate diet (leafy vegetables)
* Alcohol consumption
* Increased demand (pregnancy)
* Malabsorption (coeliac disease)

\*\*((Folate deficiency during pregnancy predispose to neural tube defects ( spina bifida) and facial defects ))

**\*\*Diagnosis :**

* Clinical features
* Decreased Hb, macrocytosis, decreased serum level of red cell folate .

Dental aspects :

* Patients with low Hb level are poor candidate for GA
* Elective oral surgical procedures should be avoided in severely anaemic patients( so if Hb <10 g/dl) because of increased risk of infection and impaired wound healing.
* Patients presenting with oral signs of deficiency anaemia should have their FBC and haematinics examined.
* Oral manifestations of deficiency anaemia include:
* Glossitis
* Angular chelitis
* Aphthous ulcers
* Burning/sore tongue
* Candidiosis
* Pallor of oral mucosa

(( the dr showed us photo of girl with multiple aphthous ulcers appeared recently she suffered of VB12 deficiency her tongue appear red and depapilated )). The tongue of patient with iron deficiency is depapilated but not red . some have angular chelitis and candidiasis so if there’s no local cause of this manifestations so we do blood test to exclude anemia .

Till here included in the second exam for us ( 2012 batch )

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Haemolytic anemias :

* Is heterogeneous group of disorders that are characterised by increased destruction of RBCs with a reduction of their circulatory life span (normally 100-120 days).
* Increased destruction of RBCs is accompanied by compensatory bone marrow hyperplasia and increased production of reticulocytes (immature RBCs).
* In addition, hyperbilirubinaemia causes jaundice which occurs because of increased haemoglobin

**\*\* The two main types of haemolytic anemia are :**

* **Sickle cell anaemia**
* **Thalassemia**

**1\_ Sickle cell anaemia :**

* It’s common in Jordon and it’s an autosomal recessive disorder that results in the formation of haemoglobin with abnormal physical and chemical properties called (HbS) .
* In the deoxygenated state, HbS undergoes polymerisation within the erythrocytes which become rigid, sickle shaped and unable to pass through capillaries .
* It exists in two forms; a homozygous form (*sickle cell anaemia*) and a heterozygous form (*sickle cell trait*) in which only one chromosome carries the abnormal gene .

\*\***Clinical features** :

Symptoms usually appear in the first year of life which include:

* Anaemia
* Failure to thrive
* Sickle crisis( clustering of destructed RBCs inside the blood vessels and it becomes painful ), infarction (fever, malaise, acute pain)
* Chronic haemolysis

\*\* **Diagnosis :**

* Family history
* Clinical features
* Decreased Hb, increased reticulocytes, sickled erythrocytes
* Hb electrophoresis (HbS)
* **Dental aspect :**
* Elective oral surgical procedures should be avoided in severely anaemic patients because of increased risk of infection and impaired wound healing
* Sickle crisis can be induced by hypoxia, dehydration and infection
* Postoperative pain is best managed by paracetamol not aspirin or NSAIDs (because NSAIDs when metabolized will cause acidosis and this acids can trigger sickle cell crisis ).
* Patients with SCA may had repeated blood transfusion so they’re at risk of blood born infections (HIV, HBV) and therefore represent a cross infection hazard .

\*\***Oral manifestations :**

* Delayed eruption and dental hypoplasia
* Increased bone radiolucency and formation of gross trabecular pattern due to marrow hyperplasia
* Bone trabeculae in the alveolar bone between the roots of the teeth appear as horizontal rows, creating a step ladder appearance.
* Thickened skull diploe with vertical trabiculations (hair on end appearance)
* Isolated radiopaque areas representing areas of past bony infarction
* sickle crisis can lead to orofacial pain in the absence of dental pathology. Infarcts occurring in the mental vessels can lead to mental nerve neuropathy manifesting as temporary parasthesia of the lower lip .

**2\_ Thalassemia :**

More important than SCA every one before marriage must do test for it .

* Its inherited disorders characterised by reduced rate of production of the alpha or beta globin chain in the haemoglobin molecule (( it’s qualitative problem but in SCA it’s quantitative ))
* Depending on the affected chain, there are two types of thalassemia; alpha and beta

Beta thalassemia is the most common type and exists in two types; a homozygous form (thalassemia major) it’s the disease and a heterozygous form (thalassemia minor) it’s the trait .

* Symptoms of thalassemia are variable and depend on the severity of the disease
* Thalassemia minor is usually asymptomatic or presents as mild anaemia
* Thalassemia major is characterised by failure to thrive, skeletal abnormalities, iron overload .

\*\***Diagnosis :**

* Clinical features
* Decreased Hb, microcytosis
* Haemoglobin electrophoresis shows high HbF ( fetal Hb)

**\*\*Dental aspect :**

* Oral surgical treatment in thalassemia patients is influenced by three major problems ; anaemia ,susceptibility to infection, and associated organ damage due to iron overload may cause diabetes , pancreatic damage ,kidney failure and xerostomia due to the accumulation of iron in the salivary glands .
* Routine dental procedure can be carried out safely in thalassemia minor patients
* Elective oral surgical procedures should be avoided in severely anaemic patients because of increased risk of infection and impaired wound healing .
* Antibiotics prophylaxis before surgical procedures is indicated in splenectomised patients and in patients with hypersplenism .
* Oral surgery can be complicated by the associated organ damage (*e.g. liver dysfunction, diabetes, cardiac disease, etc*)
* Patients who had repeated blood transfusion are at risk of blood born infections (HIV, HBV) and therefore represent a cross infection hazard .
* **Oral manifestations :**
* Teeth discoloration due to iron deposition in developing teeth
* Characteristic facial appearance described as “chipmunk faces”
* Malocclusion; anterior open bite, spacing, and protrusion of upper labial segment
* Thickened skull diploe with vertical trabiculations (hair on end appearance)
* Increased bone radiolucency and formation of gross trabecular pattern due to marrow hyperplasia
* Facial nerve palsy can happen due to widening of diploe bone
* Pain, swelling, and impaired function of parotid gland caused by iron deposition
* Retarded pneumatisation of the maxillary sinus .

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SORRY FOR ANY MISTAKES

HAVE A NICE HOLLIDAY ^^