Lab results in primary health care

Prepared by
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Data station

- Hematology (CBC).
- Liver profile
- Lipid profile
- Hyperuricemia
- FBS/RBS/HbA1c/IGT/IFG
- Thyroid profile
- renal profile, eGFR and UMA
- Hormonal therapy
- Vit d AND vit B12/hair fall
- Immunology tests/urine /stool analysis
ASK 7

- Why did you want it?
- Who ordered it for you?
- Where did you do it?
- When did you do it?
- What do you know about this test?
  - Was it first time?
  - How did you do it?
CBC

• 28 years old lady complains of easy fatigability, hair loss and palpitation since 3 month.

• Her CBC showed

  RBC 4.28 LOW  MCV 69.9 LOW
  HGB 9.7 LOW  HCT 29.9 LOW
  MCH 22.6 LOW  MCHC 32.2 LOW
  RDW 18.4 HIGH
CASE 1

• What is the most likely Diagnosis?
  a. Iron deficiency anemia
  b. Thalassemia Trait
  c. Both iron deficiency anemia on top of thalassemia trait
  d. Chronic disease anemia
CASE 2

- A 9-years old male
- WBC 4.2 (3.5-10.5)
- HB 9.0 g/dl (10.3-14.6 g/dl)
- RBC 4.5 x 10^9 (4.0-5.2)
- Hct 32 % (32-42)
- MCV 45 fl (82-98)
- MCH 24 pg (26-34)
- MCHC 26 g/dl (31-37)
- RDW 13% (11.5-14.5)
- Plt 360 x 10^3 (150-400)
• Serum iron 160 g/dl (50-120)
• Serum ferritin 169 ng/ml (7-140)
• Iron saturation 50% (0-5)

Q- what is the interpretation?

• Microcytic hypochromic anaemia with increased iron stores
• Diagnosis: Thalassemia
<table>
<thead>
<tr>
<th></th>
<th>Iron deficiency Anemia</th>
<th>Thalassemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb</td>
<td>Low</td>
<td>Low (100-115)</td>
</tr>
<tr>
<td>MCV</td>
<td>&lt; 85: Consider Fe deficiency</td>
<td>Low (60's)</td>
</tr>
<tr>
<td></td>
<td>&lt; 75: Fe deficiency</td>
<td></td>
</tr>
<tr>
<td>RBC Count</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>RDW</td>
<td>High</td>
<td>Normal</td>
</tr>
<tr>
<td>Ferritin</td>
<td>Low (&lt;30)</td>
<td></td>
</tr>
</tbody>
</table>
CONT

**MCV/RBC**

- If more than 13 likely to be IDA.
- If less than 13 likely to be thalassemia trait.
<table>
<thead>
<tr>
<th></th>
<th>IDA</th>
<th>Anemia of chronic inflammation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum ferritin</td>
<td>Decreased</td>
<td>Normal or decreased</td>
</tr>
<tr>
<td>Serum iron</td>
<td>Normal or decreased</td>
<td>Normal or decreased</td>
</tr>
<tr>
<td>Total iron binding capacity or transferrin</td>
<td>Increased</td>
<td>Normal or decreased</td>
</tr>
<tr>
<td>% iron saturation</td>
<td>Decreased (&lt;10%-15%)</td>
<td>Normal or decreased</td>
</tr>
<tr>
<td>MCV</td>
<td>Decreased</td>
<td>Normal or decreased</td>
</tr>
<tr>
<td>RDW</td>
<td>Increased</td>
<td>Normal</td>
</tr>
<tr>
<td>sTfR/log ferritin ratio</td>
<td>&gt;2</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Hepcidin (not currently clinically available)</td>
<td>Suppressed</td>
<td>Increased</td>
</tr>
</tbody>
</table>

IDA = iron deficiency anemia; MCV = mean corpuscular volume; RDW = red blood cell distribution width; sTfR = serum-soluble transferrin receptor.
Microcytic hypochromic anemia

• Differential diagnosis:
  1. Iron deficiency anemia.
  2. Lead poisoning.
  3. Thalassemia trait.
  4. Sideroplastic anemia.
Iron deficiency anemia

• Look at ferritin level.
• Treatment:
  1. Ferrous sulfate 200mg bd on empty stomach (2hrs prior and 4 hours after antacid).
  2. If not tolerated → decrease the dose or give ferrous gluconate.
  3. Ascorbic acid(vit c) 250-500mg bd with iron.
  4. Hgb should increase by 2g/100ml over 3-4 wks
  5. Confirm the response 2-4w after starting.
  6. Continue treatment for 3 months after correction to replenish the iron store.
  7. Once normal, monitor every 3 months for 1 year then annually.
CASE 3

• 48 years old Indian patient. Not known to have any previous medical problems. Presented with easy fatigability, weight loss, and numbness in both upper and lower limb.

• His CBC showed:
CONT

<table>
<thead>
<tr>
<th>WBC</th>
<th>12.1</th>
<th>H</th>
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<tbody>
<tr>
<td>NE</td>
<td>71.1</td>
<td>H 8.5</td>
</tr>
<tr>
<td>LY</td>
<td>15.9</td>
<td>L 1.9</td>
</tr>
<tr>
<td>MO</td>
<td>3.3</td>
<td>0.5</td>
</tr>
<tr>
<td>EO</td>
<td>0.5</td>
<td>L 0.1</td>
</tr>
<tr>
<td>BA</td>
<td>8.7</td>
<td>H 1.1</td>
</tr>
<tr>
<td>RBC</td>
<td>2.69</td>
<td>L</td>
</tr>
<tr>
<td>HGB</td>
<td>10.6</td>
<td>L</td>
</tr>
<tr>
<td>HCT</td>
<td>31.6</td>
<td>L</td>
</tr>
<tr>
<td>MCV</td>
<td>117.6</td>
<td>H</td>
</tr>
<tr>
<td>MCH</td>
<td>39.6</td>
<td>H</td>
</tr>
<tr>
<td>MCHC</td>
<td>33.7</td>
<td></td>
</tr>
<tr>
<td>RDW</td>
<td>14.1</td>
<td></td>
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<tr>
<td>PLT</td>
<td>578</td>
<td>H</td>
</tr>
<tr>
<td>MPV</td>
<td>7.2</td>
<td>L</td>
</tr>
</tbody>
</table>
CONT

• What is the most likely diagnosis?
  a. Multiple Myeloma
  b. B12 Deficiency
  c. Folate Deficiency
  d. Acute leukemia
Macrocytic hyperchromic anemia

- **Differential diagnosis:**
  1. Vit B12 deficiency.
  2. Folate deficiency.
  3. Alcoholic liver disease.
  5. Reticulocytosis.
  7. Cytotoxic drugs.

**Vitamin B12 deficiency DDx:**
  1. Dietary (vegans).
  2. Low intrinsic factor (pernicious anemia/gastrectomy).
  4. Ilial resection.
  6. Metformin.
  7. H.pylori.
  8. PPI/Ranitidine.
B12 deficiency

• Treatment:
  1. Parenteral B12 (hydroxycobalamin), IM initially 1mg 3X/w for 2 weeks then every 2-3 m.
  2. If dietary → oral B12 supplement.
  3. Check response by repeating after 8 weeks.

Folate deficiency:
  1. 5mg folic acid for 4 months.
  2. If malabsorption → increase the dose to 15mg.
CASE 4

- 20 years female complain on heavy periods and easy bruising in the last 1 month. These symptoms proceeded by a viral illness.
- Her CBC showed
<table>
<thead>
<tr>
<th>Test Description</th>
<th>Result Value</th>
<th>Reference Range Unit</th>
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<tbody>
<tr>
<td>Sample No: 1400265199</td>
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<td></td>
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<tr>
<td>WBC</td>
<td>5.63</td>
<td>3.7–10</td>
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<tr>
<td>RBC</td>
<td>5.17</td>
<td>4.5–5.5</td>
</tr>
<tr>
<td>Hb</td>
<td>144</td>
<td>130–170</td>
</tr>
<tr>
<td>Hct</td>
<td>0.435</td>
<td>0.4–0.5</td>
</tr>
<tr>
<td>MCV</td>
<td>84.1</td>
<td>83–101</td>
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<tr>
<td>MCH</td>
<td>27.9</td>
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<tr>
<td>MCHC</td>
<td>331</td>
<td>315–345</td>
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<tr>
<td>RDW</td>
<td>13.3</td>
<td>11.6–14.6</td>
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<tr>
<td>NRBC#</td>
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<td></td>
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<tr>
<td>PTT</td>
<td>48</td>
<td>130–430</td>
</tr>
<tr>
<td>MPV</td>
<td>11.6</td>
<td>7–11</td>
</tr>
<tr>
<td>WBC DIFFERENTIAL</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
CONT

• What is the most likely diagnosis?
  a. ITP
  b. TTP/HUS
  c. Dilution thrombocytopenia
  d. Inherit ant Thrombocytopenia
Manifestations of thrombocytopenia

• Petechial hemorrhage.
• Easy bruising.
• Mucosal bleeding
  • e.g. _ epistaxis.
  • _ gum bleeding
Causes of Thrombocytopenia

- Decreased production
- Congenital disorders
- Radiation or chemotherapy
- Vitamin B12 or folate deficiency
- Drugs
- Systemic lupus erythematosus
- Aplastic anemia
- Acute leukemia
- Lymphomas
- Alcohol abuse
- Viral infections, including HIV
- Splenic sequestration
CASE 5

• 50 years lady found on routine blood test to have a high platelet count. She is a symptomatic and history was not significant.

• What blood test you will initially ask for?
  a. CRP
  b. ESR
  c. Ferritin
  d. Blood film
  e. All of the above
Conditions Associated with Thrombocytosis

• Iron deficiency (overactive reaction)
• Acute blood loss
• Inflammatory disorders (IBD)
• Malignancies
• Splenectomy
• Myeloproliferative disorders
• Essential thrombocythemia
• Polycythemia
• Myelofibrosis
Causes of pancytopenia

- cancers that affect the bone marrow, such as:
  - leukemia
  - multiple myeloma
  - Hodgkin’s or non-Hodgkin’s lymphoma
  - myelodysplastic syndromes
  - megaloblastic anemia, a condition in which your body produces larger-than-normal, immature red blood cells and you have a low red blood cell count
- aplastic anemia, a condition in which your body stops making enough new blood cells
- paroxysmal nocturnal hemoglobinuria, a rare blood disease that causes red blood cells to be destroyed
- viral infections, such as:
  - Epstein-Barr virus, which causes mononucleosis
  - cytomegalovirus
  - HIV
  - hepatitis
  - malaria
  - sepsis (a blood infection)
diseases that damage bone marrow, such as **Gaucher disease**
damage from **chemotherapy** or **radiation** treatments for cancer
exposure to chemicals in the environment, such as radiation, arsenic, or benzene
bone marrow disorders that run in families
vitamin deficiencies, such as lack of **vitamin B-12** or **folate**
enlargement of your spleen, known as **splenomegaly**
liver disease
excess alcohol use that damages your liver
autoimmune diseases, such as **systemic lupus erythematosus**
In about **half of all cases**, doctors can’t find a cause for pancytopenia. This is called idiopathic pancytopenia.
CAUSES OF POLYCYTHEMIA

• Polycythaemia can cause **blood clots**. These put you at risk of life-threatening problems such as:
  
  • **pulmonary embolisms** – a blockage in the blood vessel that carries blood from the heart to the lungs
  
  • **deep vein thrombosis (DVT)** – a blockage that forms in the blood vessels in your leg before moving elsewhere in your body
cont

• Polycythaemia also increases your risk of heart attack and stroke. Seek emergency medical help if you think that you or someone you're with is having a heart attack or stroke.
• Apparent polycythaemia" is where your red cell count is normal, but you have a reduced amount of a fluid called plasma in your blood, making it thicker.

• Apparent polycythaemia is often caused by being overweight, smoking, drinking too much alcohol or taking certain medicines – including diuretics (tablets for high blood pressure that make you pee more).

• Apparent polycythaemia may improve if the underlying cause is identified and managed. Stopping smoking or reducing your alcohol intake, for example, may help.

Relative polycythaemia

• This is similar to apparent polycythaemia. It can happen as a result of dehydration.
Absolute polycythaemia

"Absolute polycythaemia" is where your body produces too many red blood cells. There are 2 main types:

primary polycythaemia – there's a problem in the cells produced by the bone marrow that become red blood cells; the most common type is known as polycythaemia vera (PV)

secondary polycythaemia – too many red blood cells are produced as the result of an underlying condition
• Secondary polycythaemia

• Secondary polycythaemia is where an underlying condition causes more erythropoietin to be produced. This is a hormone produced by the kidneys that stimulates the bone marrow cells to produce red blood cells.

• Health conditions that can cause secondary polycythaemia include:

  • chronic obstructive pulmonary disease (COPD) and sleep apnoea – these can cause an increase in erythropoietin, due to not enough oxygen reaching the body's tissues

  • a problem with the kidneys – such as a kidney tumour or narrowing of the arteries supplying blood to the kidneys
<table>
<thead>
<tr>
<th>PROCEDURE</th>
<th>RESULTS</th>
<th>REFERENCE RANGE</th>
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</thead>
<tbody>
<tr>
<td>ALK. PHOSPHATASE</td>
<td>99 (H)</td>
<td>0-62</td>
</tr>
<tr>
<td>ALT (SGPT)</td>
<td>241 (H)</td>
<td>28-76</td>
</tr>
<tr>
<td>AST (SGOT)</td>
<td>80 (H)</td>
<td>5-55</td>
</tr>
<tr>
<td>CK</td>
<td>1058 (H)</td>
<td>64-440</td>
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<tr>
<td>GGT</td>
<td>2</td>
<td>1-7</td>
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<tr>
<td>AMYLASE</td>
<td>733</td>
<td>500-1500</td>
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<tr>
<td>LIPASE</td>
<td>118</td>
<td>10-195</td>
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<tr>
<td>ALBUMIN</td>
<td>2.8</td>
<td>2.4-4.1</td>
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<tr>
<td>TOTAL PROTEIN</td>
<td>6.9</td>
<td>5.9-8.5</td>
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<tr>
<td>GLOBULIN</td>
<td>4.1</td>
<td>3.4-5.2</td>
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<tr>
<td>TOTAL BILIRUBIN</td>
<td>0.2</td>
<td>0.0-0.4</td>
</tr>
<tr>
<td>DIRECT BILIRUBIN</td>
<td>0.0</td>
<td>0.0-0.1</td>
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<td>BUN</td>
<td>17</td>
<td>15-34</td>
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<td>CREATININE</td>
<td>0.8</td>
<td>0.8-2.3</td>
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<td>CHOLESTEROL</td>
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<tr>
<td>GLUCOSE</td>
<td>98</td>
<td>70-150</td>
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<tr>
<td>CALCIUM</td>
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<td>7.5-10.8</td>
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<tr>
<td>PHOSPHORUS</td>
<td>5.9</td>
<td>3.0-7.0</td>
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<td>TCO2 (BICARBONATE)</td>
<td>11 (L)</td>
<td>13-25</td>
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<td>CHLORIDE</td>
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<td>POTASSIUM</td>
<td>4.6</td>
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<td>SODIUM</td>
<td>155</td>
<td>147-156</td>
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<td>A/G RATIO</td>
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<td>0.6-1.5</td>
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<td>B/C RATIO</td>
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<tr>
<td>INDIRECT BILIRUBIN</td>
<td>0.2</td>
<td>0-0.3</td>
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<tr>
<td>NA/K RATIO</td>
<td>34</td>
<td></td>
</tr>
<tr>
<td>ANION GAP</td>
<td>32 (H)</td>
<td>13-27</td>
</tr>
</tbody>
</table>
• Abnormal LFT (AST fatty liver, ALT autoimmune/viral, GGT alcohol)
• Don’t miss hormonal supplements in adults!!!!
• High LFT once \(\rightarrow\) repeat
• \(\geq 3\times\) upper limit of normal: Repeat after 1 week
• < 3X upper limit of normal: Repeat after 1 month
• If still high LFT results \( \rightarrow \) refer for US and hepatitis screen.
• If ALT < 2X ULN (upper limit of normal) + normal US + -ve Hep. Screen \( \rightarrow \) repeat after 3-6 months.
• If US shows fatty liver
  a. Alcohol consumer \( \rightarrow \) stop alcohol then repeat after 3-6 months.
  b. Non alcohol consumer \( \rightarrow \) treat metabolic syndrome then repeat after 3-6 months.
ALP

- Elevation of alkaline phosphatase is seen in normal childhood and adolescence, as well as pregnancy.
- Nonspecific and can be seen with liver or bone disorders or can be related to medication.
GGT

• GGT is a microsomal enzyme that is inducible by alcohol and certain drugs, including warfarin and some anticonvulsants.
Hepatitis Serology

- Hepatitis A virus (HAV), hepatitis B virus (HBV) and, less often, hepatitis C virus (HCV) are the usual causes of acute viral hepatitis.
- A person with symptoms of acute hepatitis should have the following three hepatitis serologies performed:
  - IgM anti-HAV
  - hepatitis B surface antigen (HBsAg)
  - anti-HCV (PCR)
- Presented in microbiology section
Hyperurecemia

- Deposition of monosodium urate monohydrate in the synovium.
- Uric acid > 450 Mmol/l.
- Management (RAPRIOP):
  R reassurance: resolves within 2 weeks.
  A advice: reduce alcohol intake/loose weight if obese/avoid food rich in purines (liver, yeast, kidney, seafood, oily fish e.g. sardines)/Aspirin at low dose increase gout (safe dose 75-150mg).
  P prescription:
  1. NSAIDs.(It’s not an option in uncontrolled hypertensive patients)
  2. Intraarticular cortisone injection. .(It’s not an option in uncontrolled hypertensive patients)
  3. Colchicine (lower onset of action) → SE diarrhea 500Mcg bd max 6mg.(only can be used with uncontrolled hypertensive patients)
  4. Oral steroids 15mg od.
  5. If no response → allopurinol (zyloric 100mg)or febuxostat(adenouric 80mg od). (start after 2 weeks of settling of symptoms→ increase dose of zyloric till uric acid < 300Mmol
  R referral: if hyperurecemia with ureate stone and recurrent UTI → urology.
investigations:
1. Uric acid.
2. ESR (high).
3. WBS (high).
5. X-ray (soft tissue swelling/erosion if severe).

O observation: after 2 weeks of NSAID to see response.

P prevention: (indication for allopurinol)
1. Recurrent attacks >/= 1
2. Tophi.
3. Renal disease.
4. Uric acid renal stones.
5. Prophylaxis if on cytotoxic or diuretics.
6. Radiological findings of chronic disease.
7. Urine uric acid > 1100 mg/dl (6.5mmol).
FBS, RBS, HbA1c, IFG, IGT

- To diagnose diabetes *(TWO READING OR ONE READING WITH SYMPTOMS)*
  1. HbA1c $\geq 6.5\%$
  2. FBS $\geq 7\text{ mmol/l}$
  3. RBS $\geq 11.1 \text{ mmol/l}$

**Prediabetes cut points:**
  1. IFG 5.6-6.9 mmol/l
  2. IGT 7.8-11 mmol/l
  3. HbA1c 5.7-6.4%
CONT

• GESTATIONAL DM (OGTT)
  • FBS 5.1
  • AFTER 1 HR 10
  • AFTER 2HR 8.5
Prostate specific antigen

- 3 Ng/ml from 50-59y
- 4 Ng/ml from 60-69
- 5 Ng/ml > 70 y
In older men the prevalence is:
50% at the age of 50 y.
80% at the age of 80 y.

Voiding symptoms/storage symptoms (irritation)/ post micturition.

Complications:
1. Recurrent UTI.
2. Retention of urine.
3. Obstructive uropathy.

Cases of raised PSA:
1. BPH.
2. Prostatitis and UTI (1 m after treatment).
3. Ejaculation (48hr).
4. Exercise (48hr).
5. Urinary retention.
6. Instrumentation of the urinary tract.
Management:
Watchful waiting
Alpha antagonist (tamsulosin (Omnic 0.4mg)/alfuzosin (xatral 10mg) → decrease symptoms.
SI: retrograde ejaculation, postural hypotension
5 alpha reductase (finasteride/Proscar 5mg) → decrease progression (needs 6 m to show proper effect, decrease PSA by 50%)
Subclinical hypothyroidism

- High TSH & normal T4.
- Presence or absence of mild symptoms of hypothyroidism.
- Risk of progression to overt hypothyroidism is 2-5% per year (higher in men).
- Risk increased by presence of thyroid autoantibodies (thyroid peroxidase AB) thyroglobulin AB used for thyroid cancer follow up

- Treat subclinical hypothyroidism patient if:
  1. TSH > 10.
  2. Thyroid autoantibodies +ve.
  3. Other autoimmune disorders.
  5. Symptomatic. (wt gain, cold intolerance, bradycardia or constipation)
<table>
<thead>
<tr>
<th>FT3</th>
<th>FT4</th>
<th>TSH</th>
<th>Comment</th>
</tr>
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<tr>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>1ry Hyperthyroidism</td>
</tr>
<tr>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>2ry Hyperthyroidism</td>
</tr>
<tr>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>1ry Hypothyroidism</td>
</tr>
<tr>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>2ry Hypothyroidism</td>
</tr>
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</table>
Renal function test and eGFR

- CR, UREA (hormonal supplements in adults!!)

### Table 10. Stages of Chronic Kidney Disease

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
<th>GFR (mL/min/1.73 m²)</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Kidney damage with normal or ↑ GFR</td>
<td>≥90</td>
</tr>
<tr>
<td>2</td>
<td>Kidney damage with mild ↓ GFR</td>
<td>60–89</td>
</tr>
<tr>
<td>3</td>
<td>Moderate ↓ GFR</td>
<td>30–59</td>
</tr>
<tr>
<td>4</td>
<td>Severe ↓ GFR</td>
<td>15–29</td>
</tr>
<tr>
<td>5</td>
<td>Kidney failure</td>
<td>&lt;15 (or dialysis)</td>
</tr>
</tbody>
</table>

Chronic kidney disease is defined as either kidney damage or GFR <60 mL/min/1.73 m² for ≥3 months. Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies.
• Microalbuminuria (MA) is defined as a persistent elevation of albumin in the urine of (>20 to <200 microg/min).

• **Other tests section**

• In diabetic patients treat with ACE INH even if the patient is not hypertensive.
Autoimmune Diseases

Routine clinical tests:

- ESR and CRP; ↑↑ in inflammation
- CBC: anemia and ↓ platelet and ↓ WBCs
- routine chemistry panels which may reveal:
  - kidney involvement; ↑↑ BUN & creatinine
  - abnormalities of liver function tests
  - ↑ muscle enzymes (CPK)
Lab Diagnosis of RA:

- **Rheumatoid factor:** -ve in 30% early in illness; can repeat 6 - 12 ms; +ve in numerous other processes (e.g., lupus; scleroderma; Sjögren’s syndrome; neoplastic disease); not an accurate measure of disease progression.
- **Anticyclic citrullinated peptide (Anti-CCP) antibody:** correlate well with disease progression; increases sensitivity when used in combination with RF; more specific than RF.
- **Antinuclear antibody (ANA):** Limited value for screening
- **Complement levels:** Normal or elevated
- **Urinalysis:** hematuria or proteinuria
Lab Diagnosis of SLE:

- Anti-nuclear antibody (ANA)
- Anti-DNA antibody: to determine Abs to the genetic material in the cell
- Anti-Sm antibody: a ribonucleo-protein found in the cell nucleus
- Complement proteins C3 and C4
Vit D def

• Management plan:( vit d 50,000)
• Less than 50 one tab every one week for 8 wks then one tab monthly for 4 mths then repeat test if becomes normal from(75 to 250) preventive dose every 2 mths better to be taken for long life
• Above 50-75 one tab taken monthly for 6 mths then repeat test and if it becomes normal preventive dose better to be taken every 2 mths
Vit B12 def

• Methylcobal injection (b12 injection) better than neurobion because it’s not painful taken E.O.D for 2 wks 6 injection 1gm then tablets taken for 2 to 3 months.
• Stress on diet rich in b12
Roaccutane

• Some tests should be done monthly to follow patient taken roaccutane
• Liver profile, lipid profile (TG), renal profile
• SI: dryness, depression and high lipid and liver profile
Hormonal profile

- (FSH, LH, PROLACTIN AND ESTRAIDOL)
- Should be done 2\textsuperscript{nd} or 3\textsuperscript{rd} day of period look for follicular phase normal FSH higher than LH.
- If LH higher than FSH think in PCO refer for assessment\textbf{(Rotterdam Criteria)}
- High FSH and LH with LOW Estraidol in late thirty females think in premature ovarian failure after repeating the test in 4-6 wks
Stool analysis

- Entameba histolytica treated by flagyl 500mg tds for 5 days
- Round worms (Ascaris) treated by mebendazole (vermox) 100mg bd for 3 days
- Not given below 2yrs
- Thread worms (pin worms) caused by enterobius vermicularis treated by single dose of vermox; the dose may need to be repeated in 2 weeks if infection persists
• Consider treating all household contacts as threadworms are highly transmissible.
Hair fall

• Tests are recommended for hair fall are Ferritin should be more than 70 normal range from (11-250), CBC, Zinc and TSH.

• Normally 100 hairs fall out per day in females and about 40 in males.