

• كل ما كان في كتابه

اوستن
البحر
2018

1 - AF

2 - SLE

3 - Myelofibrosis

4 - Arteriovenous Fistula

الأسئلة

① causes of CRF/ESRD

② Indication of dialysis

③ Replacement therapy of kidney

→ hemodialysis

→ peritoneal dialysis

→ renal transplantation

④ complication of CRF

④ complication of dialysis

- 1- hyperkalemia ECG
- ✓ 2- hemophilia
- ✓ 3- Cushing
- ④ Wegener granulomatosis = small vessel vasculitis
- 5- parkinson

- : Wegener نزل (عنه) ؟!

⑥ Vasculitis Syndrome هل ؟!

- Wegener granulomatosis
- Polyarteritis Nodosa
- Churg - Strauss
- Temporal arteritis.

⑥ Signs / → most common involvement upper Resp. tract

Rhinitis / sinusitis / nasal ulcer → rare

↳ most common Not respond to usual ttt
cough / hemoptysis / dyspnea

→ Kidney involvement (>80%) cause ↓ morbidity & mortality

→ arthritis (60%)

⑥ Investigation / - (C-ANCA) not diagnostic

- biopsy from involvement organ (nasal ^{septum})

⑥ treatment / combination between glucocorticoid & immunosuppressive agent (cyclophosphamide)

اوسكر
ابو
2017

✓ 1 - inferior MI

✓ 2 - Systemic Sclerosis (CREST)
 C: calcinosis. E: esophageal dysfunction.
 R: Raynaud. S: Sclerodactyly. T: Telangiectasia.
 Limited scleroderma.

✓ 3 - ITP

④ Fistula / chronic renal failure complication / indication for hemodialysis

القول الثاني

systemic sclerosis : ~~multiple~~ sclerosis

* clinical picture

① Thickening of skin caused by accumulation of CT

② Raynaud phenomenon

③ GI involvement : esophageal dysmotility
hypomotility of small intestine → bacterial growth
dilatation of large intestine → malabsorption
diverticula

④ pulmonary involvement : pulmonary fibrosis.
restrictive lung dz.
cor pulmonale.

⑤ renal involvement : renal crisis
→ malignant HTN → acute renal failure

* Investigation

: Anticentromere antibody ACA
Anti Scl. 70
ANA

* treatment

No cure for SSC

- for skin manifestation : D-penicillamine

- for Raynaud phenomenon : CCB : Nifedipine

- for hypertension : ACE I

إعطاء indication for hemodialysis :

indication in acute renal failure.

- potassium > 7
- Bicarb < 12
- PH < 7.2
- urea $> 40 \text{ mg\% / day}$ ^{أو} $> 100 \text{ mg\%}$
- cerebral edema / pulmonary edema

indication in chronic renal failure.

- creatinine > 8 in Diabetic patient > 5
- glomerular filtration rate $< 10 \text{ ml/min}$ in Diabetic Patient $< 15 \text{ ml/min}$
- cerebral edema / pulmonary edema
- urea $> 200 \text{ mg\%}$

other indications.

- hyperkalemia
- hypercalcemia
- Drug intoxication
- to correct thermal dysregulation.

إعطاء complication of chronic renal failure :

- [1] CNS : headache - encephalopathy - coma - disequilibrium & - aluminum dementia
Cerebellar ataxia - peripheral neuritis - vertigo - amyloidosis $\rightarrow$ carpal tunnel &
- [2] eye : eyelid edema - chemosis - band keratopathy - conjunctival congestion
transient blindness - hypertensive retinopathy
- [3] Mouth : Kussmaul breathing - urinous odor of breath - coated tongue - red glazed tongue
- [4] Cardiovascular : hypertension - atherosclerosis - pericardial effusion - pericarditis - HF
calcification of valve
- [5] lung : pleural effusion - pulmonary edema - Kussmaul breathing - pleurisy - uremic asthma
- [6] GIT : candidiasis - gastritis - paralytic ileus - malabsorption - dysentery - constipation
pancreatitis - hepatitis ^{بب} dialysis with normal enzyme & bilirubin.
- [7] Kidney : hydronephrosis - polycystic kidney - amyloidosis - pyelonephritis - glomerulonephritis
- [8] ~~RE examination~~ genitalia : gactorrhea - amenorrhea - gynecomastia - infertility
- [9] Skin : edema - pruritis - acidic sweat - papule - earthy look pigmentation
- [10] Muscle : muscle cramps - irritable leg & - muscle atrophy
- [11] Bone : bone weakness - bone cyst - bone fracture [renal osteodystrophy]

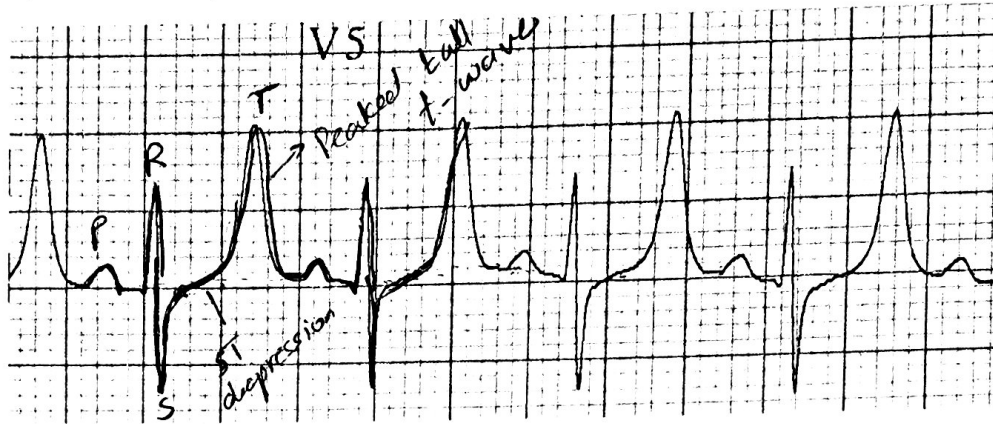
إعطاء complication of hemodialysis :

- [1] Blood : Anemia - bleeding tendency.
- [2] Blood : Anemia - bleeding tendency.
- [3] hypotension - liable for thrombosis $\rightarrow$ ^{ultra} ^{re} ^{parin} $\rightarrow$ liable for bleeding (hemorrhagic pericardial effusion)
- [4] air embolism
- [5] dementia - disequilibrium & - hepatitis - AIDS

6th Year Internal Medicine OSCE Static 2016-2017

Station 1

1- Pt's with T2DM and CRF, now he will go to hemodialysis before he went, his ECG:



1- What are the abnormality? Peak T-wave tall

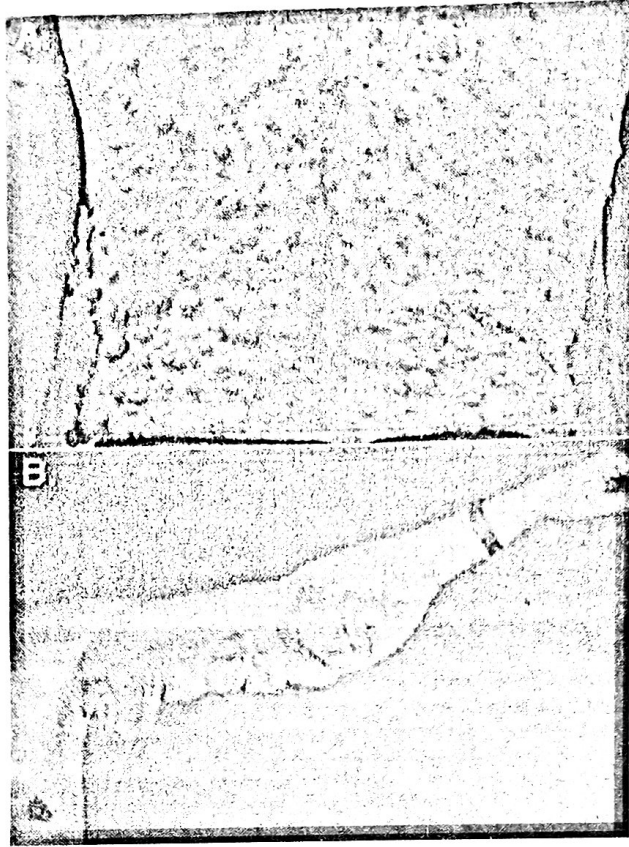
2- DX?

3- Other Changes may be seen in another?

4- What is the mechanism?

5- TTT

Station 2



12/1/11

1- DX?

2- Mode of inheritance, chromosome?

3- Criteria for DX?

4- Another Feature?

5- Type of this disease and the difference?

28

Station 3

DKA

1- Calculate S. Osm? $2 \times \text{Na} + \frac{\text{BUN}}{2.8} + \frac{\text{glucose}}{18}$

2- Dx?

3- Criteria for DX?

1- RBS > 250	4- ketonemia	المعيار
2- PH < 7.3	ليس له قيمة	criteria
3- $\text{HCO}_3^- < 15$	ketonuria	high anion gap

4- Factor that lead this disease?

- 1- presentation of INDDM
- 2- missing dose
- 3- infection (pneumonia - UTI)
- 4- stress
- 5- major surgery

5- TTT?

- 1- Fluid (4-6L)
- 2- insulin (slab dose & maintenance)
- 3- potassium
- 4- Sodium bicarb
- 5- antibiotic

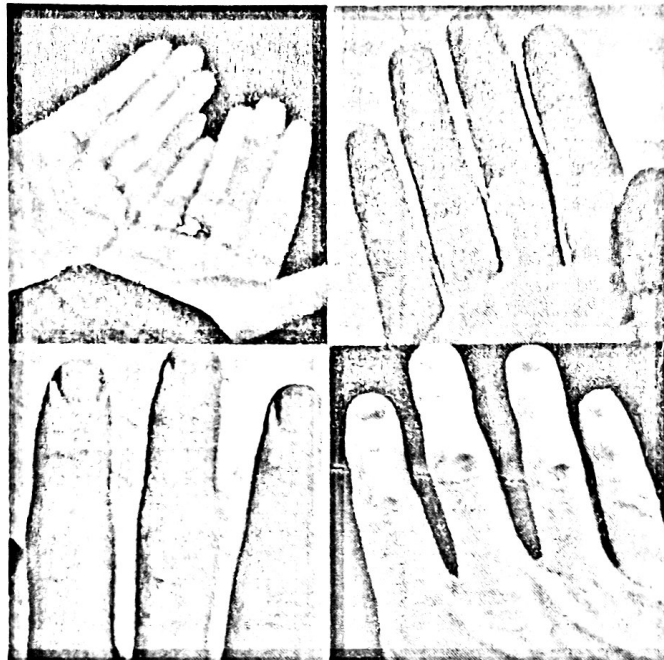
(*) كيف أعرف إنه الرشف قد تم

- 1- relief symptoms .
- 2- tolerate to oral intake .
- 3- anion gap become normal .

Def: recurrent spasm of vs of toes or fingers,
In response to cold or stress.

Station 4

Raynaud's vs ischaemia
- Vasculitis
- Thrombo
Reversible
In irreversible



Spasms

- ear.
- nose.
- lower extremities.

سكتة مفاجئة
Sudden attack

1- DX? Raynaud's Phenomenon

2- Sequelae of the color of the disease?

- ① White → Vasoconstriction.
- ② Blue → cyanosis.
- ③ Red → Rapid B.L. flow (Reversible) change

3- Cause of each color?

1 White → Blue → Red
V.C. cyanosis. Rapid B.L. Flow.

4- Types and causes?

Primary ① = Raynaud's Dz. Characterized by Vasospasm w/o other illness without an associated underlying dz.

Secondary ② → Vasospasm Associated with other dz.
L most commonly ① Scleroderma.

5- TTT?

* → CCB → (nifedipine). ② MCTD.

⇒ How dif. ① & ② By **Nail Fold** capillary scopy.

① → Normal capillaries in normal loop pattern.

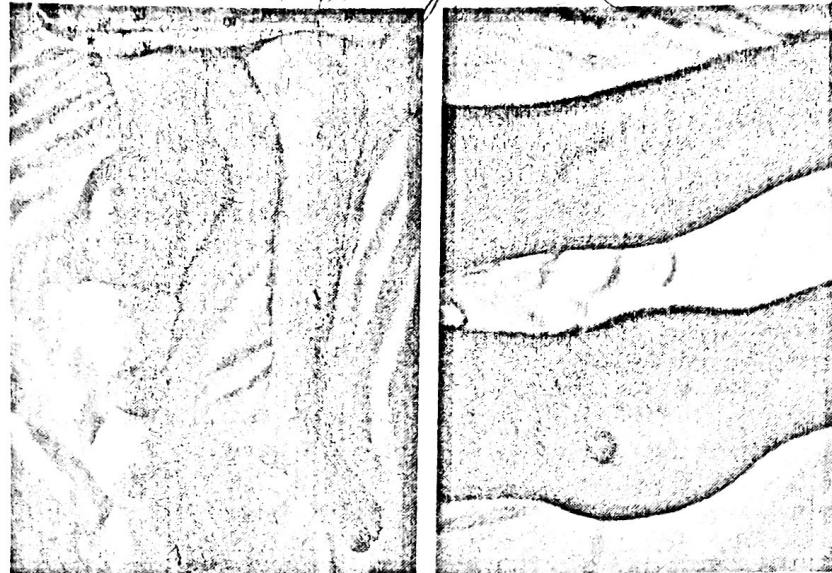
② → abnormal pattern, dz

لوسعة الـ ٥ الـ ١٠
Coagulation not Pk

Station 5

- I (A) x linked. - VIII
II (B) x-linked → IX
III (C) → AR = L II

Acute
complication
of hemophilia



- [عوامل أخرى]
① Deep Bleed.
② Ectymoses
③ Joint Bleed
④ Hem Hematoma.

How diff. type
A, B vs Factor
only by level

confirm
by Assay
or
vial

1- DX? He

2- Mode of inheritance? X-linked recessive ~ so common in M/F

3- Types of this disease?

①	A	→	Factor VIII def.	X-link	80-85%
②	B	→	Factor IX def.	X-link	10-15%
③	C	→	Factor XI def.	AR	very rare

4- Classify manifestations according to level of VIII ~ Severity of hemophilia ~

level	① mild	→	< 1%	bleeding after severe trauma, surgery → [Adulthood]
VIII	② moderate	→	1-5%	Bleeding after mild trauma - during Adulthood
V	③ severe	→	5-25%	Spontaneous severe bleeding - deep tissue bleeding

5- Complication from therapy?

- Formation of inhibitors (Antibodies) → Bethesda inhibits Assay
- Viral infections
- B, C, A, HIV
- Peritonitis

Yours,
Mohammed Awni Taha

[C/P] But complication → ① Soft Tissue Bleeding < Joints.
② Intracranial Bleeding.

Complication
مضاعفات

Acute → muscle Joint Bleed
Long term → Joint and nerve damage

6th Year Internal Medicine OSCE Dynamic 2016-2017

1. Cardiology:

- AS with Dr. Alaa Naim
- PS with Dr. Moh'd Habib

2. Respiratory:

- Asthma with Dr. Waleed Daowd
- Pl. Effusion with Dr. Mahmoud Al-Khozondar

3. Neurology:

- MS with Dr. Shawqi
- CVA with Dr. Sabra

4. Examination:

- DVT with Dr. Aiman
- Abd. (Ascites) with Dr. Hasan Khalaf

5. History:

Gout with Dr. Moh'd Zaqout or Dr. Rami Al-Abadla

**Yours,
Mohammed Awni Taha**

OSCE Internal Medicine 2016

I- Static

الاجبة

1- ECG with ST-Elevation in Lead V₁, V₂, V₃. (Ant-septal).

- What is abnormal finding in this ECG?
- What is your Dx? *Ant. wall MI septal.*
- What is the Artery involved? *LAD.*
- New Murmur was heard along the pericardium, what is the new event? *MR* (Acute complication)
- Complications *Early → OR RVI - HB - MR*

2- Pic of Hemarthrosis and Hematoma

Hemophilia.

- What is in the pic?
- What is your Dx? *P. HTN*
- What is the Mode of Inheritance?
- What are the Complications?
- Classification of severity

hemato

2017

types of hemophilia

- (A) classic 80-85% (↓ factor VIII)
- (B) christmas 10-15% (↓ factor IX)
- (C) absence or ↓ Factor very rare (XI) autosomal recessive.

3- Pic of Portal HTN Gastropathy?

- What is your Dx? *P. HTN*
- Define Liver Cirrhosis?
- What is the etiology of Liver Cirrhosis?
- What are the Complications of Liver Cirrhosis?
- What is the Optimal TTT of Liver Cirrhosis? *transplantation.*

Liver

4- Pic of Dermatomyositis (Heliotrope rash and Gottron's sign) (قديمة) 2009

- What is in the pic?
- What is your Dx?
- How to confirm your Dx?
- Treatment

Complications

- (1) Portal HTN (variceal)
- (2) liver insufficiency [synthetic encephalopathy - Jaundice]
- (3) HCC

II- Dynamic

1- DVT Examination

2- Hx of DKA

3- Asthma Examination

4- Aortic Stenosis Examination Ejection systolic murmur.

Written By: Maisara Al-Rayyes

Liver cirrhosis: inflammation of cells followed by fibrosis and nodular formations that ultimately result in loss of liver architecture

Etiology →

- (1) inf. viral Hep. chronic liver hep.
- (2) metabolic: Wilson's hemochromatosis, NASH, nail.
- (3) Prolonged cholestasis: PBC - PSC
- (4) Autoimmune Hepatitis
- (5) Drugs, toxin
- (6) Alcohol, stem).

- ① Type of ECG changes.
- ② V's involved.
- ③ Dx: Acute — wall MI

④ Risk Factors = modifiable

1. HTN
2. Dyslipidemia
3. DM.
4. Smoking
5. obesity.
6. life style

non modifiable.

1. ~~Sex~~ Age
2. Family Hx.
3. sex

⑤ What are ACS involve?

1. Unstable Angina
2. STMI
3. Non STMI
4. sudden cardiac death.

Treatment of Acute MI

① MONA

morphine
oxygen
Nitroglycerin
Aspirin (ASA)

② BA

- BB
- ACEI

⑥ What complications:

Inf. wall MI | ~~RR~~ MR (2°)

① Rt. ventricular infarction
(Hypovolemic shock).

- ② Complete Heart Block.
 - ③ Papillary muscle Rupture.
 - ④ Acute mitral Regurge.
- mechanical complications.

ألي

- ① Rupture of Papillary muscle
- ② = = IVS
- ③ = = Free wall
- ④ Pseudo Aneurysm.

Congested neck v.
muffled heart sounds.
hypotension

Ant. أنواع

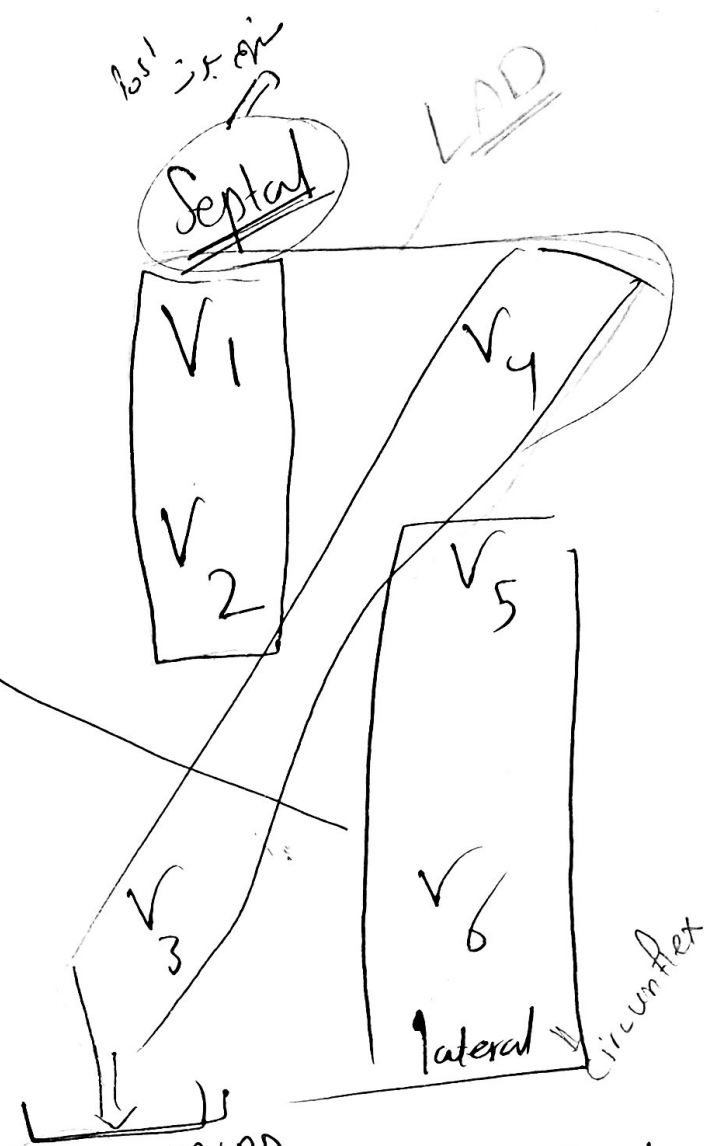
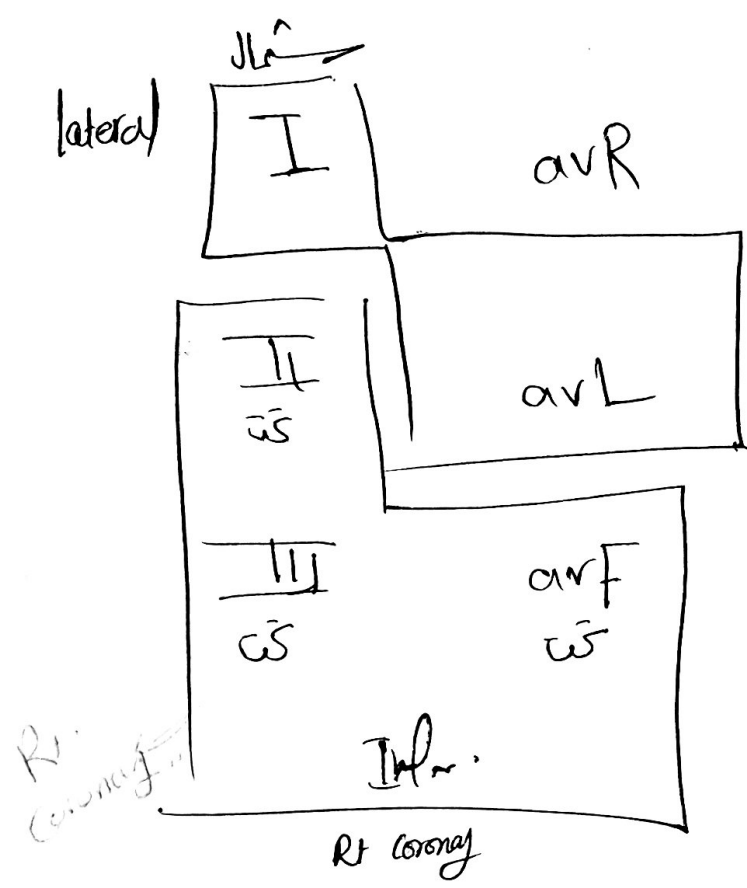
Early

- ① Arrhythmia
- ② Early Pericarditis.
- ③ LVF, Heart block.
- ④ Thromboembolic complications.

Late.

- ① Post MI angina.
- ② Late Pericarditis
- ③ CHF
- ④ LV aneurysm
- ⑤ Frozen Chest shoulder comp. of tx.

- V_1, V_2, V_3 ont
- $V_1, V_5, I, aVL \Rightarrow$ Ant. wall. } $\Rightarrow$ Ant. Ant.
- aVF, II, III (inf)



MI and Arteries

- A { ① Inf. wall MI : II, III, aVF $\rightarrow$ Rt. Coronary.
- B { ② Septal MI : V_1, V_2
- { ③ Ant MI : V_3, V_4
- { ④ Ant. Septal : $V_1 \rightarrow V_4$ $\rightarrow$ LAD
- C { ⑤ Post (tall R V_1-V_2) $\rightarrow$ Lt. circumflex + Rt. coronary
- { Lat. MI $\rightarrow$ aVL, V_5, V_6
- { Ant. lat. $\rightarrow V_4 \rightarrow aVL$ (Left main stem) $\rightarrow$ Lt. circumflex



*

الأمراض

1. portal hypertension gastropathy.

2. liver cirrhosis = consequence of chronic liver disease.

results from necrosis of hepatocytes followed by fibrous tissue deposition and formation of regenerating nodules with loss of hepatic architecture.

3. alcohol

chronic hepatitis B & C

Drug induced liver disease : methotrexate / α -methyl dopa / Amiodarone

other causes : NSAID - ~~B~~ Primary biliary cirrhosis - Secondary biliary hematochromatosis - primary Sclerosing Cholangitis
 α -1 antitrypsin deficiency - Wilson dz
 autoimmune hepatitis - Sarcoidosis (granulomatous dz)
 venous outflow obstruction : Budd Chari & cardiac cirrhosis - cryptogenic

4. ① portal HTN

↓
 PSE
 varices
 ascites
 SBP
 HRS

② hepatocellular failure

↓
 Jaundice
 coagulopathy

③ HCC

5. liver transplantation

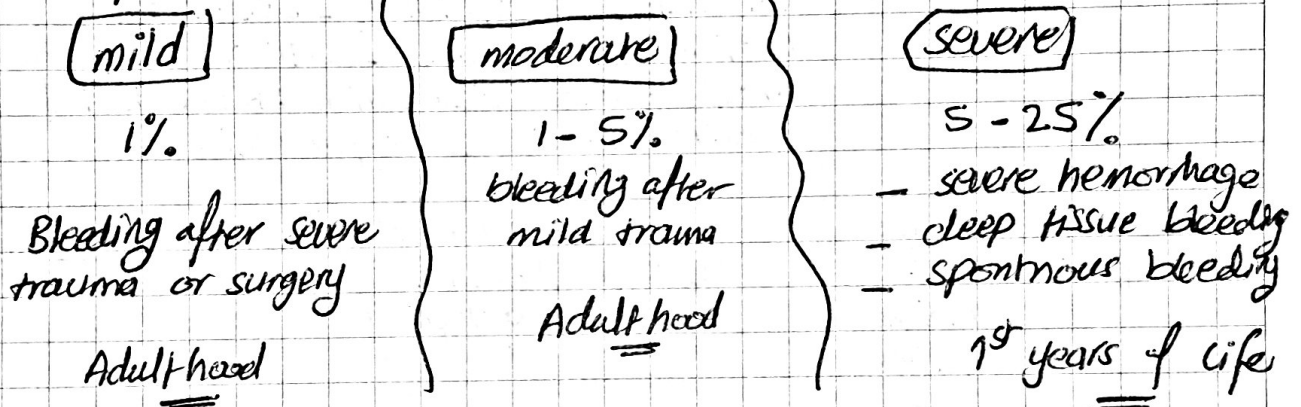
①. ~~muscle~~ hematoma - hemarthrosis (joint bleeding)

②. hemophilia

③. X-Linked recessive

④. Complication : - spontaneous bleeding
~~platelet disorder~~
 - soft tissue bleeding ← muscle
 - GI bleeding ← joint
 - urinary bleeding
 - Intracranial bleeding
 - Bruising.

⑤. Classification :



⊛ treatment : mild → desmopressin (Not work in type B) سبرين
 Severe → replacement of specific factor

⊛ types of hemophilia : A = classic 80 - 85% (↓ factor VIII)
 B = christmas 10 - 15% (↓ factor IX)

autosomal recessive سبب وراثي ⊛ C = absence or ↓ Factor XI very Rare

⊛ Complication of therapy : - Formation of inhibitors (antibody)
 - viral infection : A, B, C, HIV, Parovirus
 - Bethesda inhibitor Assay.

Swot

Swot
2016

Internal medicine OSCE 2016

Static

1. Myotonic dystrophy (picture of patient's face) ✕

- What is name for this face? Hatchet face
- What is the Dx?
- What are the complications and other manifestations?
- How to Dx?

2. ECG (pericarditis) concave ST↑

- What are the ECG findings shown here? PR depression.
- What is the Dx?
- What are other ECG findings not shown here in this ECG?
- ttt? NSAID

→ hypocalcemic patient.

3. Picture of trousseau sign (patient suffering from Hypothyroidism and Addison's)

What is the sign shown here?

How to elicit this sign? ⇒ a blood pressure cuff is placed around the arm and inflated to a pressure greater than systolic pressure and held in place for 3 mint.

What are the laboratory findings?

→ hypocalcemia
low albumin

4. ABG

Calculate Anion Gap

What type of ABG abnormality is this?

What are the causes of this type of ABG findings?

What are the causes of normal anion Gap?

5. ITP Case. (4th year 2015) Idiopathic Thrombocytopenic Purpura.

- ① PR depression
- ② concave ST elevation
- ③ diffuse changes in all leads
- ④ late dynamic changes. *

hypoparathyroidism

pericarditis

chvostek sign

- CNS irritability
- cataract - tingling
- QT ↑ HTN

* Pericarditis on ECG

- ① PR depression pathognomonic.
- ② ST elevation ~ concave
- ③ Diffuse changes...
- ④ dynamic complication Late..

Pericarditis

Early

Chest pain
When laying ↑
Relief → sitting forward.
Friction Rub

Tx ⇒ NSAIDs

Late..

Dressler's.

~~ECG~~ ✓
Fever
pleurisy.
Effusion

Tx: NSAID
Steroids..

ECG
① ST ↑ concave
② PR depression.

Tx: NSAIDs.

Atrial Fibrillation

2015
1/1

اندوكراسي و الكيندي

اوسكي
2015
1/1

2015
COPD, Pleural Effusion
1/1

Station 1 - Cushing.



1- Describe ?

2- how would differ between syndrome and disease?

3- diagnosis? → Cushing syndrome..

4- Investigation? → ① 24 hour urine Free cortisol.

② high dose overnight dexamethasone suppression test
level of ACTH

5- Treatment ?

→ surgery
→ medical.

③ other CT * MRI to exclude pituitary adenoma.
④

①. Gynecomastia

- Trunkal Obesity.

- moon Face..

- stria cutaneous striae

- intercapular buffalo hump.

② Syndrome Vs disease..

↓ Cushing

[many causes.
that lead to
Adrenal hyperplasia

① Cushing dz.

② pituitary-hypothalamic
adenoma.

③ nonendocrine dz..
tumors produce
(CTR) or (ACTH).

④ Adrenal carcinoma.
adenoma.

nodular hyperplasia etc

The cause ↓
↑ level of cortisol
is due to microadenoma
in Pituitary gland
~ excess release ↓
ACTH → stimulate
Adrenal gland..

* unresectable adrenal tumors
are treated with:
Ketoconazole or metoprolol

Station 2

صورة ECG - Inf. MI

"غير عن الي بالملفات القديمة كلها .. كانت جديدة"

1-Describe the findings in this ECG.

✓✓

2-إيش أول أشي بتطلبه للمريض- right sided lead ECG

3-What is the diagnosis ? Inf. MI

4-What is the vessel involved in this patient. ? Right coronary A.

5- Mention complications may occur in this patient.

- Thyrotoxicosis
- SIADH → SIADH due to malignancy...
other causes
manage

2014..

Trousseau sign < Hypothyroid
Addison.] - sign
causes.
Dx.
lab.

مصاب بالسكر ⇒ DKA

⑤ Complication :

LAD

anr.

early complication

- Arrhythmia
- early pericarditis
- left ventricular failure
- heart block.
- Thromboembolic complication

Late complication

- Post MI angina
- late pericarditis
- CHF
- LV aneurysm
- Frozen chest - shoulder
- complication of ttt.

mechanical complication

- rupture of free wall → cardiac tamponade
- rupture interventricular septum
- rupture of papillary muscle → MR
- Pseudo aneurysm.

اجابة السؤال

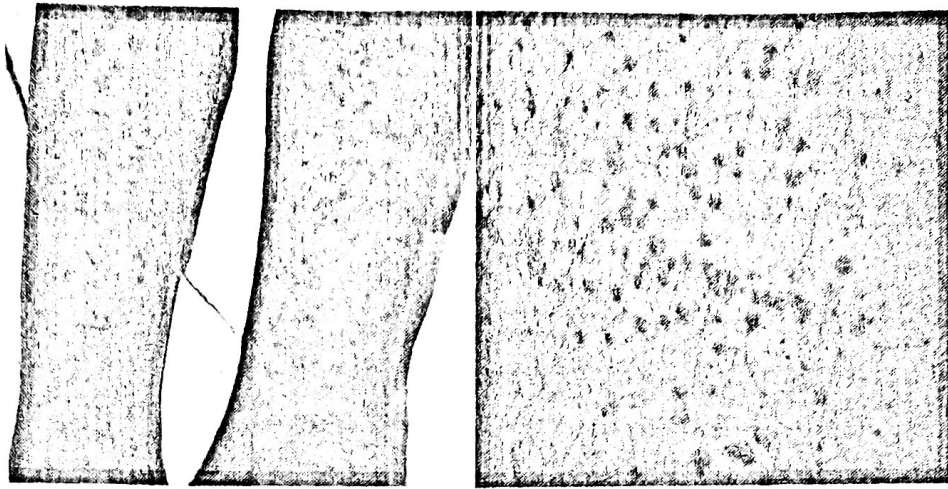
- Rt ventricle infarction ; hypodynamic shock
- complete heart block
- papillary muscle rupture → MR

سؤال في امريكي / اجابة 2017

ما الفرق بين type 1, type 2

	① Acute / Benign	② chronic /
	Children	adult
	F:M 1:1	F:M 3:1
Onset	! Abrupt	
Platelet	< 20 000	< 50 000
	2-6 w	Long term
Spontaneous remission	Common	rare
TTT	Steroid IV. Ig	splenoectomy
	preceded by viral infection	Pregnancy → exacerbation associated with systemic dz is autoimmune hemolytic a.
		[EVAN \$]

Station 3



1- Describe and how this disease differ from other?

Splenomegaly is absent, Megakartocyte in PBS with defective budding, normal PBS & creatinine

2- Diagnosis and type of this disease?

3- Investigation?

4- Plan of Treatment .

- ① petechia : $< 3\text{ml}$ doesn't blanch, not raised. | No splenomegaly | platelet may reach zero.
- ② ITP : ~~Immune~~ Idiopathic thrombocytopenic purpura.
- ③ Investigation : - Bleeding time $\uparrow$
platelet count $\downarrow$
normal clotting time / PT / PTT
specific : IgG
Bone marrow fill with megakaryocyte
normal spleen on examination.
- ④ - avoid trauma / injury / unnecessary surgeries
- Prednisone initial therapy
- splenectomy
- IV Immaglobulin or RhoGAM
- *) مع المرض قد
IgG يدمر splen $\uparrow$
توجد في ال platelet
فترفع macrophage $\uparrow$ معها
Immuno destruction
of platelet

diagnostic criteria of SLE 4/11 :

- | | | |
|---------------------|--|-----|
| 1- Malar rash | 6- Serositis (pleuritis/pericarditis) | SLE |
| 2- Discoid rash | 7- renal involvement | |
| 3- photosensitivity | 8- Neurological disorder (seizure / psychosis) | |
| 4- oral ulcer | 9- hemolytic disorder (hemolytic A. / leukopenia / Thrombocytopenia) | |
| 5- arthritis | 10- Immunological disorder (anti ds DNA - anti sm -) | |

Station 4



1- Describe and what is the sign?

2- Diagnosis? SLE

3- Pt's was dyspneic (wrote in the case of question)

.. What is the causes? Serositis and pleural effusion

4- How you will confirm your diagnosis?
 → ANA
 → Anti ds DNA
 → anti smith / anti Ro

5- Complication ?

① Butterfly rash
Jaccouds deformity

② SLE

③ Serositis / pleural effusion
 $\wedge$
 pleuritis pericarditis

④ ANA

Complement level $C_3, C_4 \downarrow$ in active lupus

elevated level of ds-DNA antibody in active lupus

anti Smith / anti Ro

⑤ eye : conjunctivitis , retinal vasculitis
 heart : pericarditis (Libman Sack endocarditis)

Kidney : lupus nephritis $\rightsquigarrow$ HTN

GIT : mesenteric occlusion

Lung : shrinking lung \$

Neuro : stroke , cerebral infarction , psychosis

Blood : autoimmune thrombocytopenia / hemolytic A.

Pregnancy : repeated abortion

→ consequence ANA $\rightarrow$ $\oplus$ $\rightarrow$ Anti DNA $\rightarrow$ $\oplus$ $\checkmark$ $\therefore$ SLE

$\ominus$ $\rightarrow$ anti Ro / la $\oplus$ $\checkmark$ $\therefore$ SLE

اوسكي
الاسئلة
2015

1- Multiple Myeloma

2- Thyrotoxicosis

3- Psoriatic arthritis

4- SIADH : due to major abdominal & Thoracic trauma (recent RTA)

5- ECG : ant. MI

②

skull X-ray :
Bone Pain no swelling

لآلمة في العظام / لا تورم

الأسئلة ②

Multiple Myeloma

MM = abnormality of plasma cell.

غير معروف
البين

Causing overproduction replacing the bone marrow
as well as & overproduction of functionless immunoglobulin.

clinical picture : - most common manifestation : Bone Pain (Back, Rib)

- infections : pneumococcus, hemophilus.

- Renal failure / anemia

- hypercalcemia symptoms : polyuria, polydipsia

- altered mental status / weakness / fatigue / pallor

- hyperviscosity & symptoms : Blurry vision, confusion
mucosal bleeding.

④ Investigation : most common finding

→ [normochromic, normocytic anemia]

- low anion gap

→ protein electrophoresis = ↑ monoclonal Ig ✓

①
أسئلة
skull X-ray

{ X-ray : punched out lytic lesion, pathologic fracture

CT, MRI

Bone marrow Biopsy

[Bence Jones protein] ←

Treatment : - younger patient : autologous bone marrow transplantation
- older patient : combination of melphalan + prednisone

③ How does this dz cause renal failure ?!

1- accumulation of light chain forming casts in distal tubule → renal obstruction
2- Myeloma light chain are directly toxic on proximal renal tubule.
3- elevated calcium level

4- Dehydration

2013

اوسكي
سادسة
2013

Static OSCE exam >> 6th yr 2013

1. Case of jaundice, itching and hepatosplenomegaly صورة

PBC

- Sign .. Xanthelasma
- What is the Dx? Primary biliary cirrhosis
- How to diagnose? (autoantibodies,, US,, etc)
- Drug used to treat? .. ursodeoxycholic acid

2. ECG with a history of recurrent thrombosis

- What is the Dx? AF
- What are the causes of this case?
- What are the complications?
- How to prevent further thrombus? Warfarin

DVT

Recurrent temporary
Blindness
Amaurosis Fugax

3. X ray skull with a history of anemia and bone pain and lab results

- What do you see .. lytic lesions
- Diagnosis .. Multiple Myeloma
- How does this disease cause renal failure?
- How to confirm the diagnosis?

Most common
symptom

Multiple myeloma.

4. Two pictures of buccal pigmentation with a history of orthostatic hypotension

Addison

- What do you see and what is the mechanism causing it?
Pigmentation << increase ACTH secretion along with melanin stimulating hormone which is secreted by the same cells that secrete ACTH.

- What is the diagnosis? Addison dz.

- Define orthostatic hypotension?

- How to diagnose this disease? Serum cortisol, ACTH stimulating test, plasma ACTH, serum aldosterone
→ definitive diagnosis = cosyntropin / ACTH stimulation test.

5. Two pictures of Mouth " tightening " and hands " white and red fingers "

Systemic
sclerosis.

- What do you see ? mouth tightening ,, palmar erythema

- Dx ? systemic sclerosis

- Investigations to do ? ACA ,, anti Scl 70 ,, ANA

- Causes of death? Renal crisis ,, lung fibrosis

Raynauds

With my best wishes



تكرروا كمان بسادسة 2014 case SLE + herpes zoster as neuro case +

2015
acromegaly

2016
3013
mm.

Orthostatic hypotension :

→ decrease in systolic BP of 20 mmHg
or decrease diastolic " " 10 mmHg
or increase in heart rate of > 10
within 3 min when compared from sitting
or supine position.
↓
of standing

management of addison :

- glucocorticoid
- mineralocorticoid
- Sodium chloride replacement



Static

- ✓ A case of salicylate poisoning ABG's and electrolytes, with calculation of anion gap "a tough one with no damn clinical clue on the paper",
- ✓ A case of SIADH on electrolytes and urine electrolytes panel the dx is SIADH due to lung malignancy, what r other causes of and how to manage,
- ✓ A picture of podagra, not clear though but the clue in the few words above the picture talking about the patient having acute pain and being on thiazide diuretics, questions r what is this sign,
What r typical x ray manifestations,
What is the diagnosis, what is the management,

Chest x ray of cardiomegally and pulmonary edema,
What is the dx, the causes and the management,
- ✓ ECG with inferior MI , what is the abnormalities, the diagnosis, the artery being affected,
- A picture with a short history telling u this patient has jaundice and vitilligo, the diagnosis is pernicious anemia the questions are: what is the diagnosis? Why the patient is jaundiced, what r the investigations, the management.
- ✓ A static asking u about the symptoms and sign of thyroid disorders without specifying hypo or hyper, stupid station but manageable.

Aug
14
2013

AL-AZHAR UNIVERSITY

FACULTY OF MEDICINE

STATIC OSCE EXAMINATION FOR 4TH YEAR 2013

INTERNAL MEDICINE

Name of the Student :

Station 1 : ECG

Station 2 : CBC

Station 3 : Multiple myeloma

Station 4 : HSP

Total Marks :

AF

AB

A

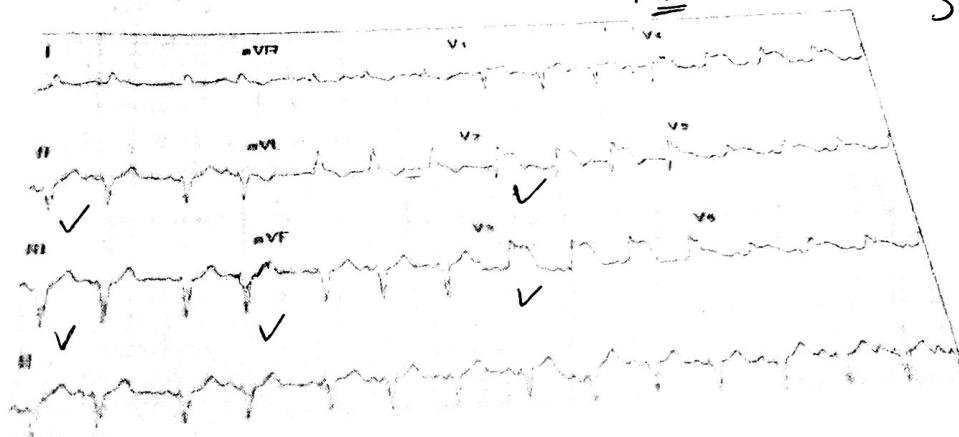
Station-1

A 55-year-old male with strong family history of Coronary Artery Disease present to Emergency Department with severe chest pain. The following ECG was recorded and admitted to CCU without any complications. 4 days after admission he developed severe pulmonary oedema and on auscultation there was a pan systolic murmur over his precordium.

MR

ST ↑ ⇒ inf. lead.

Ant.



- Q.1- What is the abnormal findings in this ECG?
- Q.2- What is the diagnosis of this ECG? *Inf. wall MI*
- Q.3 - What is the artery responsible for this ECG findings?

- Q.4 - What are the mechanical complication leading to the new pulmonary oedema?
- Q.5 - What are the risk factors for CAD?

Rupture of papillary muscle

non. / 1 mod. libl

muscle MR
1st. wall
anem

* multiple
 myeloma
 myelofibrosis.
 plasma cell.

Station - 2

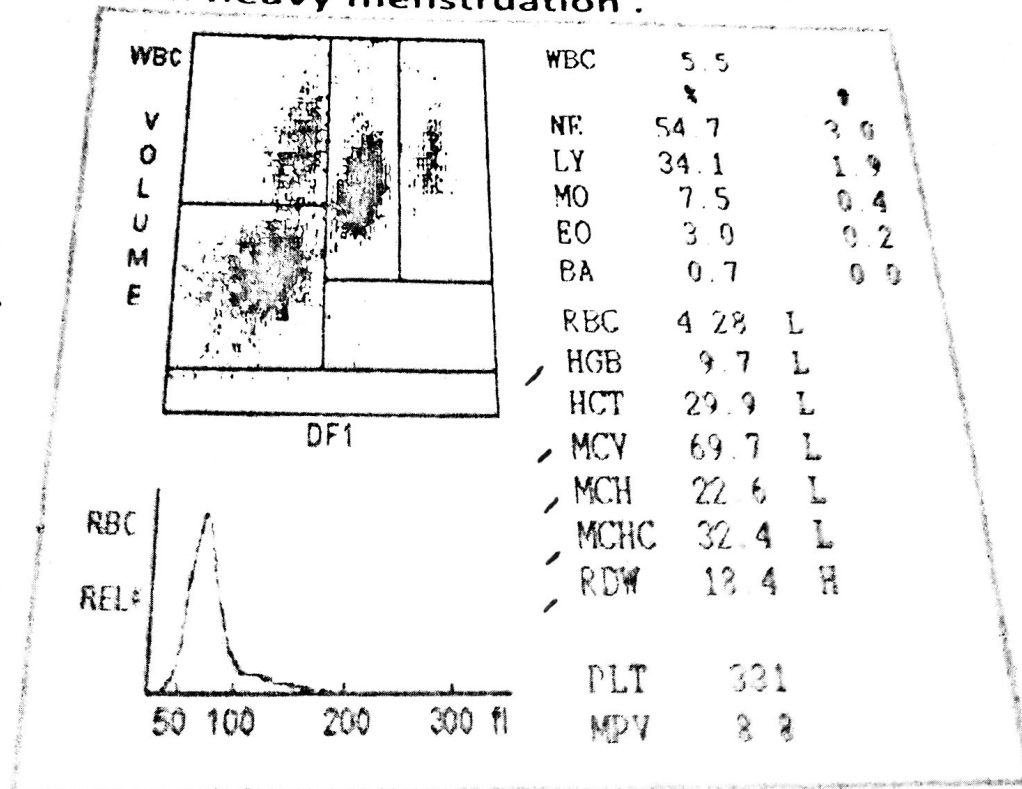
A 55 - year - old male present with abnormal facial appearance for more than 1 year.
 The patient have profuse sweating .
 Patient noticed his ring become tight and he have to change his shoes frequently .
 Below a picture of his face and Skull X Ray .



- Q.1 - What are the signs in the face and skull ?
- Q.2 - What is the diagnosis of this patient ?
- Q.3 - Mention (4) clinical features of this disease
- Q.4 - How you will investigate this patient ?
- Q.5 - How you will treat this patient ?

Station-3

This is a CBC record of a 20-year-old female with heavy menstruation.



Q.1- What is your interpretation of this CBC?

Q.2- What is the most likely diagnosis of this female and the cause?

Q.3 – What is the differential diagnosis?

Q.4 – How you will investigate this female?

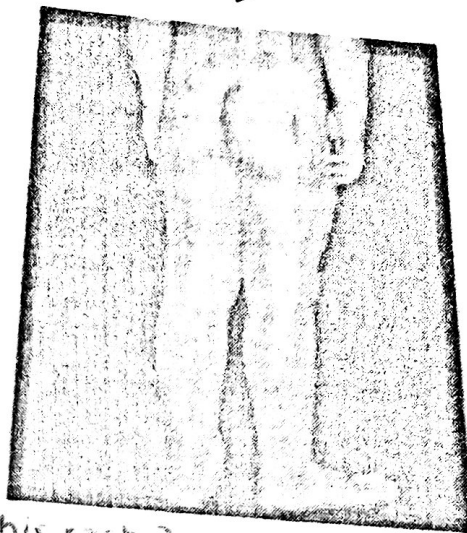
Q.5 – How you will treat this female & the duration of treatment?

A 15-year-old boy complaining of skin rash over his lower limbs of 2 days duration, associated with joint pain and abdominal pain. Two weeks back he had upper respiratory tract infection. CBC: Normal Coagulation profile & Platelet

HSP

Criteria 2/4

- 1- Palpable Purpura
- 2- Age of onset < 20
- 3- Bowel Angina
- 4- WBC PMN on afferides periphery



Preceded by
- URTI

- Strep.
- vaccination

Q.1 - What is this rash?

Q.2 - What is the most likely diagnosis of this boy?

Q.3 - What are the other manifestation of this disease?

Q.4 - What other investigations you will request?

Q.5 - How you will manage this patient & What is the outcome of this patient?

- 1- Palpable rash on extensor surfaces muscles (muscles gravity) purpura
- 2- HSP: most common cause of nonthrombocytopenic purpura

3- Classic tetrad

① 100% Palpable Purpura, 100%

② Arthritis - Arthralgia 75% → no articular damage
↳ effusion: serous / Knee

③ Abdominal Pain 50%
④ Renal Dz 20-50% (Mesangial Prol. w/ IgA)

⑤ GIT Bleeding (30%) → melena may be the presenting symp

⑥ Heart → myocardium

⑦ Lung → pulmonary hemorrhage

mcc
• intussusception
• Obst → ileocolic

* Investigations:

① CBC → normal PH. and c, anemia (git)

② immunoassay (IgA, IgM)

③ Renal Bx - mesangial deposition IgA

④ ANCA -ve ⑤ U/S

⑥ XIII ↓ ⑦ chest X-ray

⑧ ASO titer

good
Children

Adult
Bad

Rash
on lower limbs
Bum
1-10
RR

① ST elevation lead II, III, aVF

② inf. wall MI

③ Rt coronary A.

④ rupture of papillary muscle cause: pulmonary edema / mitral regurg

⑤

↓ modifiable

Risk factors

Non modifiable

HTN / Diabetes / dyslipidemia
smoking / obesity / lack physical activity

Age
family history
gender

pancytopenic
murmur

السؤال الثاني

① Hb ↓ MCV ↓ MCH ↓ MCHC ↓ RDW ↑

② iron deficiency anemia [microcytic hypochromic anemia]
Cause : Blood loss [menorrhagia].

③ DDX :
- iron deficiency anemia
- Thalassemia
- anemia of chronic dz
- sideroblastic anemia
- lead poisoning
→ microcytic hypochromic anemia.

④ Investigation : Serum Iron
Ferritin / TIBC / bone marrow stain.

⑤ treatment : ferrous sulfate tablet : continued until Hb/Ht have normalized and additional 2-3 months to restore iron store.

(lower jaw)

- ① mandibular overgrowth lead to **prognathism**
 maxillary widening
 teeth spacing and malocclusion
 Brow ridge and forehead protrusion
 generalized expansion of skull at the fontanelle

② Acromegaly

- ③ enlargement hand & feet & nose & lip (soft tissue swelling)
 - voice become deeper
 - increased sweating
 - Obstructive sleep apnea
 - Internal organ are enlarged : heart, lung, spleen, liver, kidney
 - Carpal tunnel &
 - menstrual problem
 - Cardiac anomalies & HTN.
 - metabolic change & Diabetes

④ investigation : ① IGF-1

- ② measuring of GH after 100gm glucose given orally
- ③ then **CT**, **MRI** to localized tumors.

⑤ treatment : - Primary H+ is Surgery : Stabilise or ↓ tumor size
 (Trans sphenoidal surgery.)

- Somatostatin analogues → DOC
- Dopamine agonist → if surgery not curative

① purpuric - skin lesions

② Henoch - Schonlein purpura (HSP)

③ - 100% palpable purpura.
 - arthritis - arthralgia
 - abd. pain - GIT bleeding ∴ GIT vasculitis
 - renal dz : IgA nephropathy & hematuria.
 - heart : myocarditis
 - lung : pul. hemorrhage

IgA
mediated
vasculitis.

④ - Serum Immunoglobulin (IgA) - Bleeding time ↑
 ANA / RF ⊖ - normal PF, PTT, Platelet count
 Factor XIII reduced - RBC casts in urine.

CBC : leukocytosis / thrombocytosis

ESR ↑ ~~Platelet Count (low)~~.

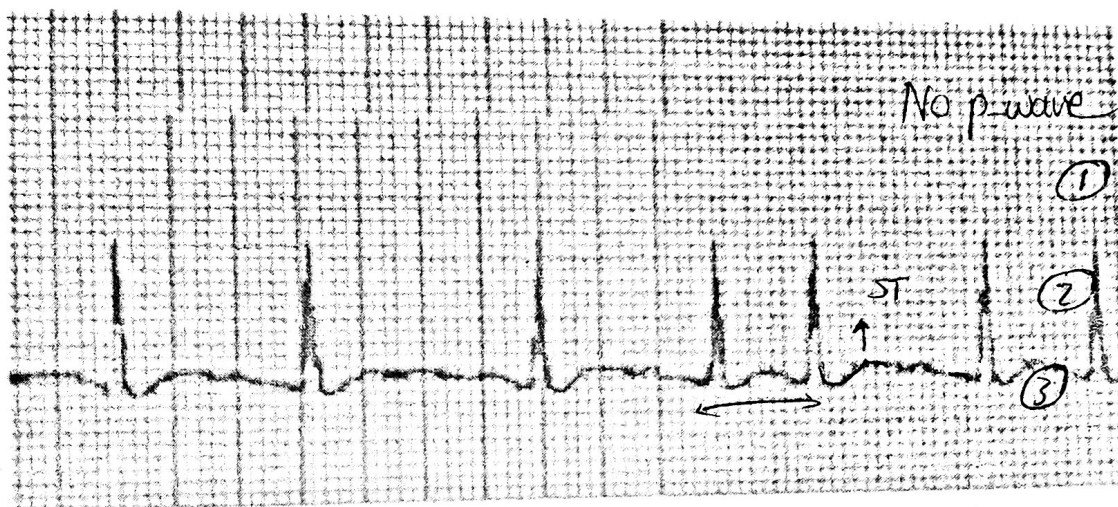
- treatment : Ⓢ NSAID for arthralgia

Ⓢ Steroids for renal / CNS / Intrapulmonary hemorrhage & GIT

AF

This is a patient of 50-year-old presents to the cardiology clinic with history of shortness of breath and difficulty in sleeping flat and the patient awaked frequently from sleep. which improved when the patient stand. On examination patient is looking dyspneic and orthopnic. Pulse is irregular. Heart examination revealed accentuated first heart sound and mid diastolic murmur on left 5th intercostals space.

*irregular
*MS stenosis



① Absent P-wave
② irregular
③ small fibrillatory f-wave

- Q.1 - What is the diagnosis of this ECG? Atrial Fibrillation.
Q.2- What is the causes of this ECG finding?
Q.3- What is the complication for this ECG diagnosis?
Q.4- What is the most likely diagnosis of this patient cardiac lesion?
Q.5 - How can you prevent the most serious complication?

(AF) — Tx ⇒
 unstable → DC shock.
 stable → Rythm and rate control.
 prevent complication : Anticoagulant + Warfarin.

Rate.
BB - CCB - Digoxin
Amiodarone
Flecainide Rythm

30osis -

- HTIV atherosclerosis..
- mitral stenosis (valvular).
- thyrotoxicosis

AF
SLE
Psoriatic Arthritis.
myelo Fibrosis.
IDA-celiac.
MI
IDA-celiac
ABCr : Respiratory Failure
HSP.

Palestinian Medical School
 4th Year Final Clinical Examination (2012) - INTERNAL MEDICINE

Station No.
 Examiner Answer Sheet

Q1. Atrial Fibrillation (2M)

Dx on ECG

idiopathic

all cardiac disease

Q2. Mitral Valve Disease (2M)

. Ischaemic Heart Disease & ACS ✓

extra cardiac

. Cardiomyopathy ✓

Myocarditis

① Thyrotoxicosis

. Hypertension ✓

valvular H. dz

② pneumonia

. Thyrotoxicosis

③ alcoholism

. Alcoholism

④

Q3. Embolic Stroke. (2M)

. - Intestinal ischemia.

. - Limb ischemia

causes of AF

Heart dz.

other ^{my} physiologically

① HTN

② valvular heart disease

③ Ischemic heart dz

④ CMP

⑤ myocarditis

⑥ post Cardiac surgery.

⑦ pericarditis.

⑧ Hyper thyroidism..

Q4. Mitral Stenosis + Atrial Fibrillation. (2M)

Q5. Anti coagulation with warfarine. (2M)

HR control % digoxin / BB blocker / CCB

ttt of AF

anticoagulant (oral) or warfarin

Noax

This is 30-year-old lady present to the clinic with darkening of her exposed parts of the skin after exposure to the sun.

History from this patient reveals that she had a long history of hands joint pain and recurrent aphthous ulcers in her mouth.

She was informed that she is anaemic.

On examination there is abnormal face and hands.

Q.1 - What is the cause of her skin darkening?

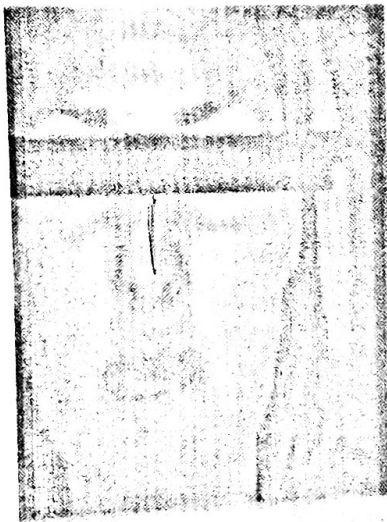
Q.2 - What is the sign in her face & the differential diagnosis & how you differentiate?

Q.3 - What is the name of her joint deformity and how you can prove it?

Q.4 - What is the most likely cause of her anaemia?

Q.5 - What is your initial diagnostic test?

Q.6 - What is the final diagnosis?



SLE ⊛ Diagnostic criteria : need $\frac{4}{11}$ to diagnose

- 1- Malar rash
- 2- Discoid rash
- 3- photo sensitivity
- 4- oral ulcer

5- arthritis

6- Serositis (pleuritis / pericarditis)

7- renal involvement

8- neurologic disorder (seizure / psychosis)

9- hematologic disorder (hemolytic anemia / leukopenia / thrombocytopenia)

10- Immunologic disorder
(anti dsDNA
anti-SM)

Palestinian Medical School

AL - AZHAR UNIVERSITY

4th Year Final Clinical Examination (2012) - INTERNAL MEDICINE

Station No.

Examiner Answer Sheet

Q1. Photosensitivity (1M)

Q.2- Facial butter fly rash (1)

Mitral rash (Not cross the nasal bridge) (1M)

Q.3- Jaccouds deformity which corrected by the patient or physician (2M)

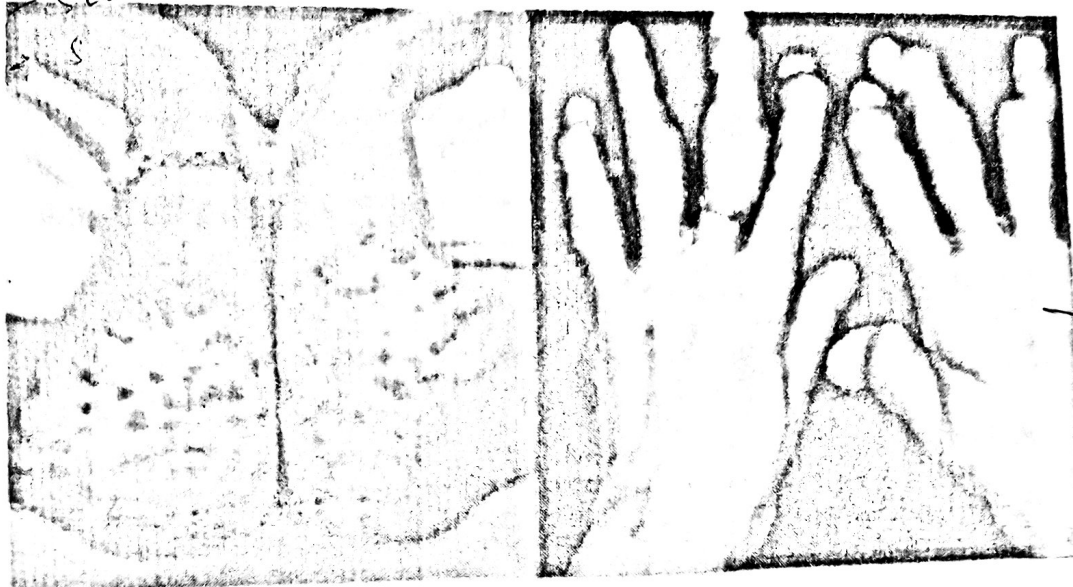
Q.4- Haemolytic Anaemia . (1 1/2M)

Q.5- ANF (1 1/2M) ∴ anti nuclear factor .

Q.6 - SLE (2 M) ∴

Psoriatic
Arthritis.

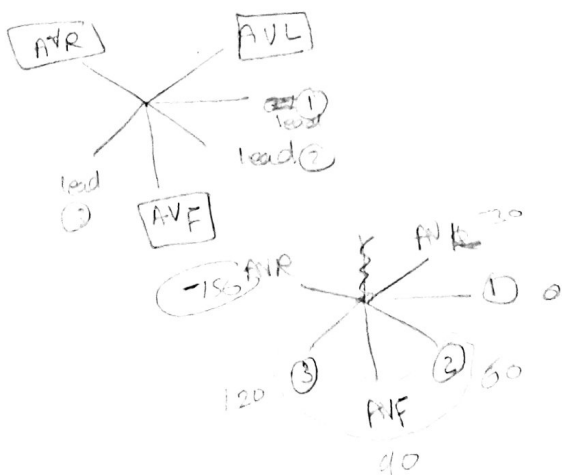
-gout
-OA
-CLE



sausage
shaped
digit

This is a 50 - year - old male presents with skin rash over back of elbow and knees followed by hands joint pain for long time . This is followed by hands deformity .

- Q.1- What is the skin rash over elbows ? *psoriatic plaque*
- Q.2- What pattern of joint deformity for this patient ? *arthritis metatarsal*
- Q.3 - What are the possible abnormal findings you can see in the *nails*
- Q.4- What is the most likely diagnosis of this patient ?
- Q.5- What are other patterns of presentation of this disease ?
- Q.6 - What is the best treatment of this patient ?



⊛ other seronegative arthropathy :

- 1- Ankylosing Spondylitis
- 2- Reactive arthritis
- 3- psoriatic arthritis
- 4- enteropathic Arthropathy

Medical School
4th Year Final Clinical Examination (2012) - INTERNAL MEDICINE
AL - AZHAR UNIVERSITY
Station No.
Examiner Answer Sheet

Q.1- Psoriatic plaques

(1M)

Q.2- Arthritis mutilans

(2M)

Q.3 ① Nail pitting

② Onycholysis انفصال الظفر

(2M) Total (½ for each)

③ Transverse ridges

④ Hyperkeratosis

⑤ Yellow nails

Q.4- Psoriatic Arthritis

(2M)

Q.5- Symmetric polyarthritis

(2 M) Total (½ for each)

- Asymmetric polyarthritis or Oligoarthritis

- Spondylitis

- Distal Interphalangeal joint disease

Q.6- Methotrexate

(1 M) Total (½ for each)

- TNF @ blocker (Infliximab)

- PUVA

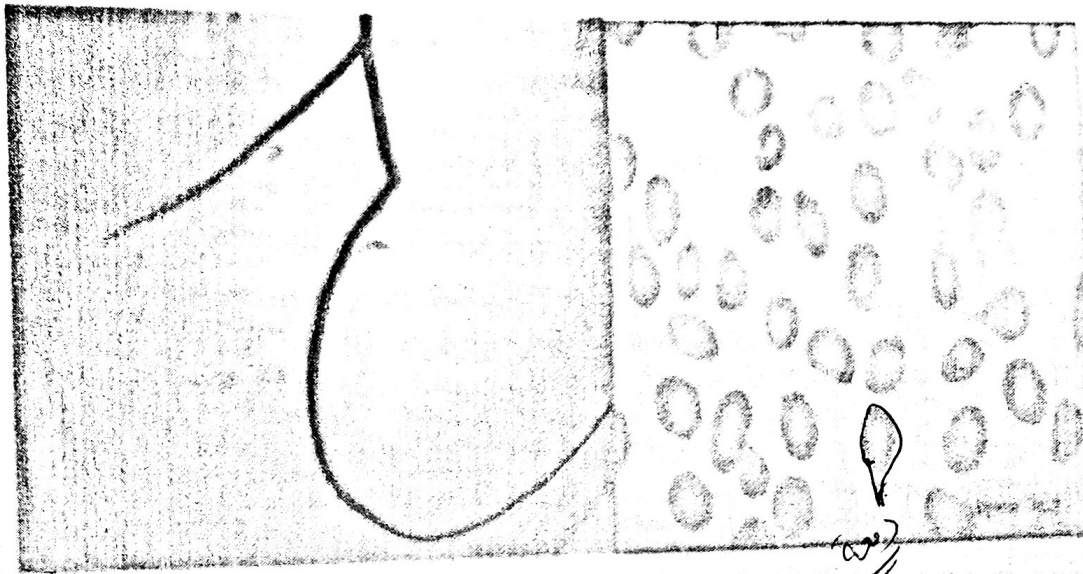
- Intra articular steroid Injection

- NSAIDs

Myelofibrosis

This is a 60 - year - old male present to the clinic with prolonged history of fatigue and dragging abdominal pain mainly in the left hyochondrium area associated with anorexia , night sweats , joint pain and low grade fever. CBC : Hb : 8 gm / dl & Pancytopenia

Clinical examination & Peripheral blood smear show finding in these two pictures .



tear drop cells.

- Q 1- What is the findings in his abdomen ?
- Q 2- What is the abnormal finding in his peripheral blood smear ?
- Q 3- What is the most likely diagnosis ?
- Q 4 - What is the bases for diagnosis ?
- Q 5 - What is the treatment for this patient ?

Palestinian Medical School
4th Year Final Clinical Examination (2012) - INTERNAL MEDICINE
Station No.
Examiner Answer Sheet

AL - AZHAR UNIVERSITY

Giant
Pit

Q.1 - Huge splenomegaly & Hepatomegaly . (2M)

Q.2 - Tear drop cells . (2M)

Q.3 - Myelofibrosis . (2 M)

Q.4 - ① Huge splenomegaly (2M) - 1/2 for each)

Why you dx

② Anaemia & Pancytopenia Platocytosis

③ Bone marrow aspiration (Dry tap)

④ Bone marrow trephine (Fibrosis)

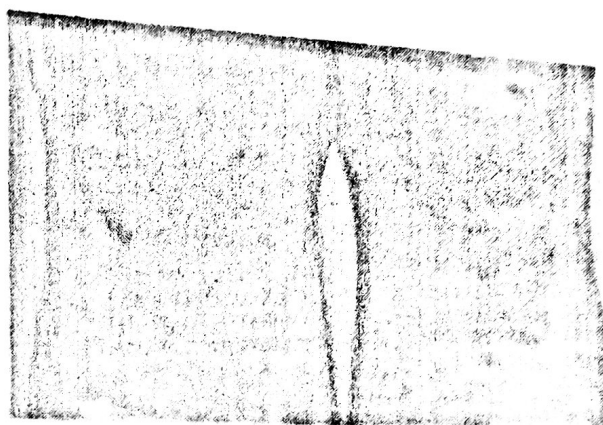
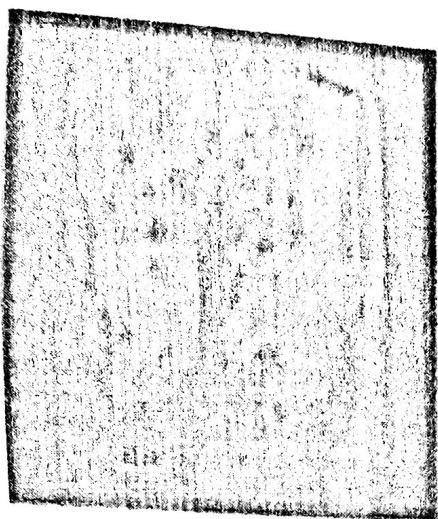
5. Absence of Philadelphia chromosome . ⇒

in
CML
no myelofib.
↓

Q.5 - Periodic blood transfusion (2 M) - 1/2 for each] .

- Erythropoietin & Oxymetholone
- Splenectomy .
- Allogenic peripheral stem cell transplantation
- Bone marrow transplantation
- Hydroxyurea

Celiac dz



This is a young patient present with chronic diarrhoea, abdominal bloating with recurrent mouth ulcers. He had intense & severe itching associated with weight loss. On examination the patient looking pale with skin rash over the knees & buttocks.

- Q.1 - What is the cause of his itching and the skin rash?
- Q.2 - Why the patient looking pale?
- Q.3 - What is the most likely diagnosis?
- Q.4 - How to prove your diagnosis?
- Q.5 - What is the character of his diarrhoea?
- Q.6 - What are the complications of this disease?

characteristic of celiac dz:

- wt loss
 - hypercalcaemia
 - easy bruising
 - elongation of prothrombin time
 - chronic diarrhoea
 - dermatitis herpetiformis.
- absorption of fat soluble vitamins.

most accurate test is small bowel biopsy.

Diagnosis:

1. IgA endomysial antibody
2. IgA tissue transglutaminase antibody
3. IgG " "
4. IgA deamidated gliadin peptide
5. IgG " "

treatment: gluten free diet (no wheat - oats - rye - barley)

celiac

Palestinian Medical School
 4th Year Final Clinical Examination (2012) - INTERNAL MEDICINE
 Station No.
 AL - AZHAR UNIVERSITY

Examiner Answer Sheet

itching

Q.1 - Dermatitis herpetiformis . (2 M) = vesicular rash on the extensor surface of the body.

Pale

Q.2 - Anemia (Dimorphic anaemia / Iron & Folate deficiency)
 (2M) - 1 for each

micro macro

weight

Q.3- Coeliac Disease . (1 1/2M)

prone

Q.4- CBC / Serum calcium / Serum Albumin / S.Ferritin & folate)
 - Positive serology tests (IgA Antiendomysial / TTG
 - Endoscopy with duodenal biopsy) (1 1/2) 1/2 mark for each

celiac

Character

Q.5 - Steatorrhea , oily , explosive . (1M) greasy/oily/floating/fatty foul smelling

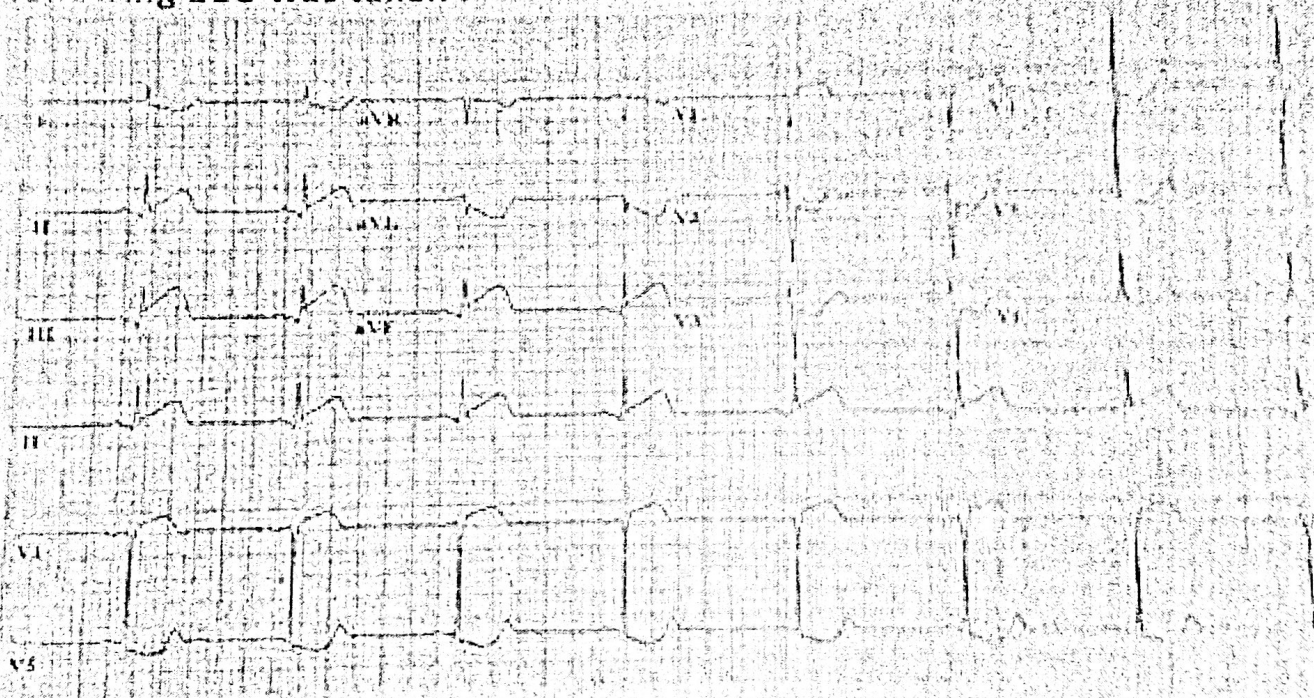
Q.6 Fat soluble vitamin deficiency (ADEK) (2 M) 1/2 for each

- Enteropathy Associated T cell Lymphoma
- Esophageal & Oropharyngeal Squamous carcinoma = adenocarcinoma
- Non Hodgkins Lymphoma
- Association with other Autoimmune Diseases
- Hepatic steatosis and raised ALT & AST)
- Dental enamel hypoplasia & growth retardation
- CNS (Ataxia , P.Neuropathy)
- Osteopenia & Arthralgia
- Infertility
- D. Herpetiformis
- Ulcerative Jejunoileitis
- Hyposplenism

Aug
2012

Palestinian Medical School AL-AZAHAR UNIVERSITY
6th Year Final Clinical Examination (2012) - INTERNAL MEDICINE
Station No.

A 67 - year - old male patient presents to emergency Department with crushing central chest pain of two hours duration. No past history of similar attacks. The patient is heavy smoker, diabetic and hypertensive on anti - hypertensive drugs. His brother died at age of 45 years due to myocardial infarction. On examination the patient is distressed, in severe pain. Blood pressure 80/50 mm/Hg, Pulse rate is regular and with normal rate. JVP ; normal. Chest examination revealed normal lung fields. The following ECG was taken.



Ans
ml

- Q.1-Describe the abnormal findings in this ECG.
- Q.2- What coronary artery feeds this area of the heart .
- Q.3 – What are the risk factors in this patient leads to this disease.
- Q.4-What is the most likely complete diagnosis for this patient?
- Q.5 – What is the definitive treatment for this patient

2012

Q1. ST Elevations in Leads II, III, aVF (2 M)

Q.2- Right Coronary Artery (1 M)

Q.3- Age, sex, smoking, obesity, DM, HTN, Family history (2 M)

Q.4- Acute inferior Wall Myocardial Infarction (2 M)

- Most Likely Right Ventricular Infarction (1 M)

Q.5- IV Fluids, Analgesia, Oxygen. (1 M)
PCI (1 M)

③ Risk Factors

Modifiable	Non-modifiable
HTN	Age
Diabetes	Family history
Dyslipidaemia	Gender
Smoking	
Obesity	
Lack physical activity	

⑤ treatment

① MONA
- morphine
- oxygen
- Nitroglycerine
- Aspirin
+ BB + ACE I

② reperfusion therapy
- PCI
- Thrombolytic
[streptokinase].

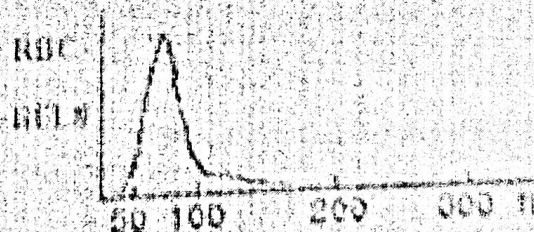
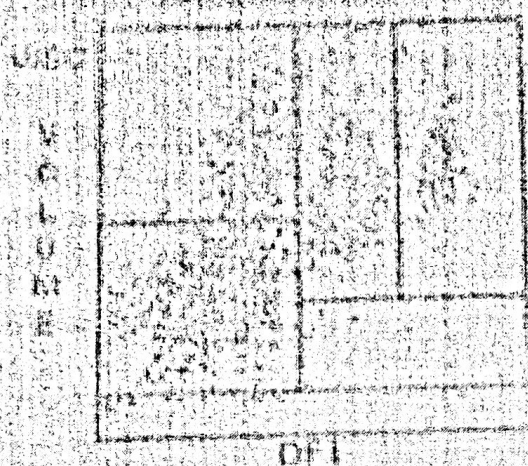
obesity
+ family history
obesity
lack exercise
PCI

Question 10: A 60-year-old female patient

presented with the following symptoms:

1. Fatigue and weakness
2. Pale skin
3. Headache
4. Dizziness
5. Shortness of breath
6. Rapid heart rate
7. Loss of appetite
8. Weight loss
9. Irritability
10. Difficulty concentrating

The patient's physical examination revealed the following findings:
1. Pale conjunctivae
2. Tachycardia
3. Normal lung sounds
4. Normal bowel sounds
5. Normal reflexes
6. Normal neurological examination
7. Normal laboratory tests (CBC, ESR, TSH, etc.)
8. Normal chest X-ray
9. Normal echocardiogram
10. Normal CT scan of the head



WBC	5.5	
NE	4	
SE	54.7	1.1
LY	34.1	1.0
MO	7.5	0.1
EO	3.8	0.0
BA	0.7	0.0
RBC	4.28	1
HGB	9.7	1
HCT	29.4	1
MCV	69.7	1
MCH	22.6	1
MCHC	32.4	1
RDW	16.4	1
PLT	331	
MPV	8.8	

Q.1 What is the abnormal finding in this patient CBC?

Q.2 What is the cause of her skin rash?

Q.3 What is type of anaemia this patient has & What is the differential diagnosis?

Q.4 What is the investigations needed in this patient?

Q.5 What is the most likely causes of anaemia in this patient & What is the most likely final diagnosis?

Q.1. $\downarrow$ Hb $\downarrow$ MCV $\downarrow$ MCH, MCHC : Low / RDW $\uparrow$ Hct (2M)
microcytic *hypochromic* *poikilocytosis*

Q.2. Dermatitis herpetiformis (1 M)

Q.3. Microcytic Hypochromic Anaemia (1M)

- ① - Iron Deficiency Anaemia. (1/2 M)
- ② - Anaemia of chronic disease (1/2 M)
- ③ - Thalassemia (1/2 M)
- ④ - Sideroblastic anaemia / Lead poisoning (1/2 M)

Q.4. (2M) Serum iron, Ferritin, TIBC, Bone Marrow Stain

- * Stool Lipid Assay
- * Serum IgA Endomysial Antibodies
- * OGD with Duodenal Biopsy $\rightarrow$ best for celiac

Q.5 - (2 M) Iron Deficiency Anaemia due to Coeliac Disease :

- ✓ Iron Malabsorption
- ✓ Menorrhagia

السؤال الثاني من الأسئلة

a 20 year old lady presents to our P. department with abdominal bloating and chronic diarrhea since childhood has excessive menstrual bleeding associated with clots on examination she looks pale, No lymphadenopathy. There is intense skin rash over extensor aspect of her knee the following CBC is attached:

Palestinian Medical School
6th Year Final Clinical Examination (2012) - INTERNAL MEDICINE
Station No. _____

AL-AZHAR UNIVERSITY

A 55-year-old woman with a long-standing history of dry cough and increasingly short of breath on exertion. On examination, she looks dyspnoeic. There is finger clubbing. Chest examination: There are bilateral fine end inspiratory (Velcro) Crackles at the lung bases. Her room air arterial blood gas (ABG) are the following:

pH 7.36

PCO₂ 35

PO₂ 55

HCO₃ : 25

Q.1- What is the explanation of her ABG? *Resp. alkaline II*

Q.2- What is the expected Pulmonary Function Test Pattern for this patient? *Restrictive Pattern*

Q.3- What is the finding in this pattern of Pulmonary Function Test?

Q.4- What is the most likely causes of this pattern? *Interst. LD*
vascular L.R.

Q.5 - What is the most likely diagnosis of this patient?

Idiopathic pulmonary fibrosis

FEV1 / FVC (normal) > 80%

→ FEV1 & FVC ↓

→ TLC & RV ↓

Q1. Respiratory Failure Type I (1 1/2 M)

Q.2- Restrictive Pattern (1 1/2 M)

Fing at Job

Q.3- TLC & RV : Decreased (1M) ↓

COPD

- FEV1 & FVC : Decreased (1 M)

- FEV1\FVC : Normal or More than 80 % (1M)

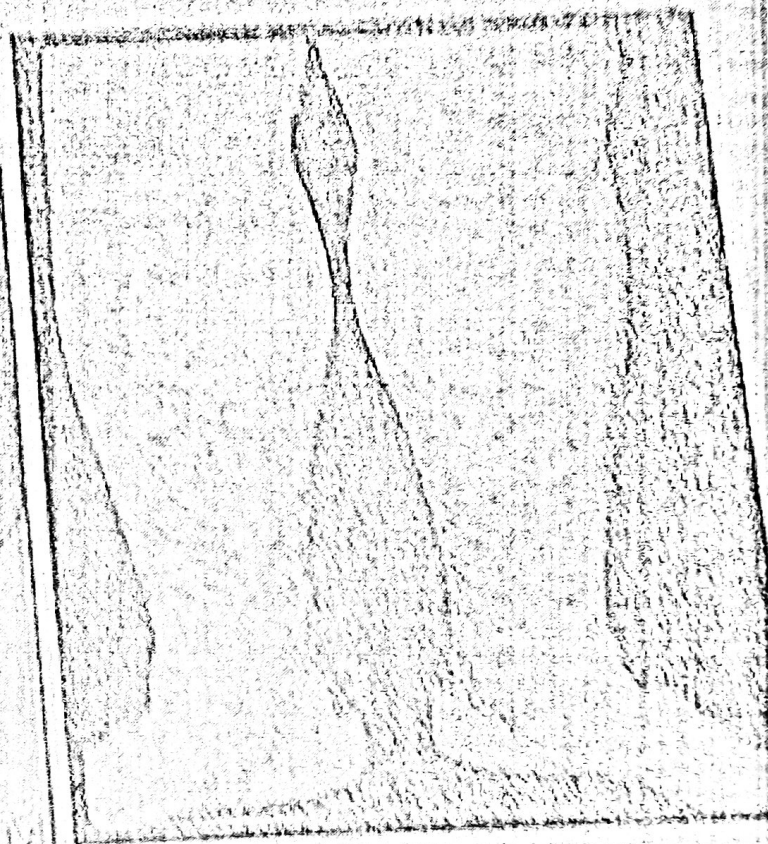
Q.4- Interstitial Lung Diseases (1M)

- Pulmonary Vascular Diseases (1 M)

Q.5- Idiopathic Pulmonary Fibrosis (2 M)

12/6/12
clubbing
*
ECOPD

A 17-year-old male presents to out patients department with new onset of skin rashes involved the buttocks, extensor surfaces of the knees and elbows & legs. This skin rashes not blanched with pressure. Full blood examination, including platelets are normal.



- Q.1 – What is the abnormal finding in this patient thigh & legs ?
- Q.2- What is the differential diagnosis of this type of rash ?
- Q.3- What is the most likely diagnosis of this patient ?
- Q.4- What are other presentation can be elicited in this patient?
- Q.5- What is the treatment of this patient ?

Q.1-Purpuric skin lesions (1M)

B.D

L PH = all

Q.2- Thromocytopenia (1M)

Q.3 - Henoch-Schonlein Purpura (2 M) = vascular purpura *

① Q.4 IgA mediated vasculitis (1 M)

② GIT Vasculitis with Abdominal pain & GIT haemorrhage (1M)

③ IgA Nephropathy with haematuria (1 M) —

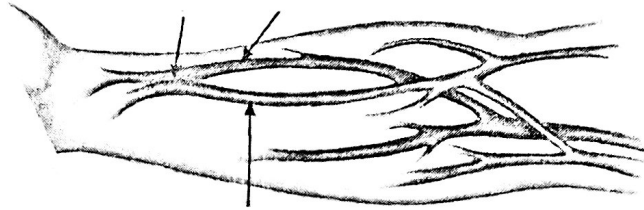
④ Arthropathy (1M)

Q.5- NSAIDS for arthralgia (1 M)

- Steroid for renal, CNS, Intra pulmonary haemorrhage and
GIT Bleedings (1M)

اوسكي
الوعه
2011

1



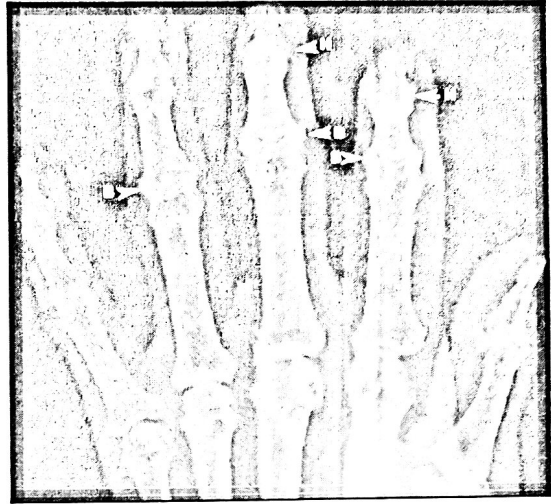
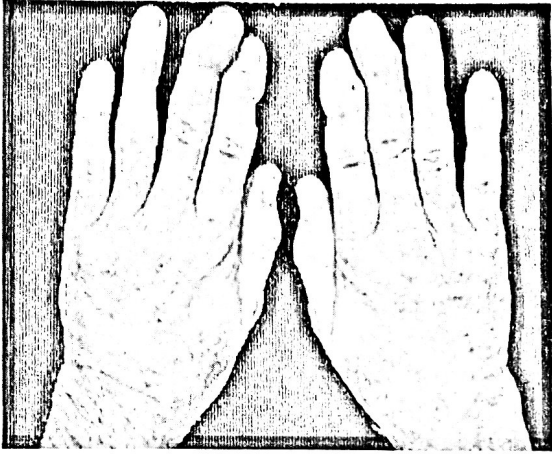
1-diagnosis: AV fistula

2- causes:.....

3-test used to diagnose: !!!!!

4-

5-



The station was a little confusing: typical story of RA but images of OA and and asked about Bouchard's and Heberden's nodules !!! >>

osteophyte → DIP = Heberden's nodule
 → PIP = Bouchard's nodule.

most joint affected. : knee ↗ base of thumb.



It found in 6th 2009

.....

They were just 4 stations I don't remember the 4th 55555555

About the dynamics

1-communication about GERD

2-Asthma with dr.Zaqout

3-flouconazole liver toxicity with dr.Qishawi

4-AS with alaa naim

.....

On the other groups I remember that there was AF with Ashraf Shath!!!

اوسى
2009
اسه

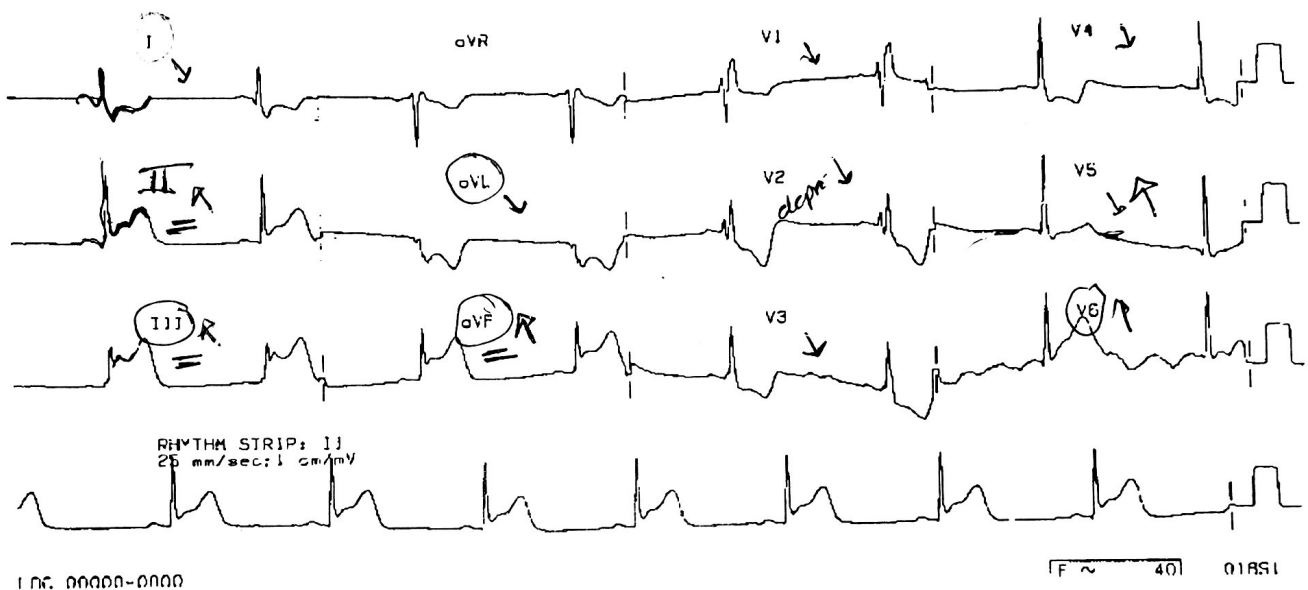
Palestinian Medical School

AL - AZHAR UNIVERSITY

6th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Station No. *Inf. MI*

A 55 year old man with 4 hours crushing chest pain. The following ECG was taken.



Q.1-Describe the findings in this ECG. *ST elevation in II, III, aVF*
ST ↓ in I

Q.2-What is the vessel involved in this patient. ? *Rt. coronary.*

Q.3-What is the final diagnosis ? *Acute Inf. wall MI*

Q.4- Mention 3 sinister complications may occur in this patient.

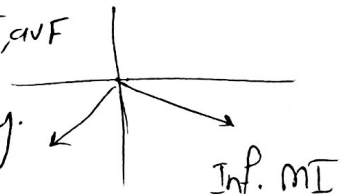
Inf MI complications ?

①-Transient Complete Heart Block. *→*

②- Acute valvular dz *MR* ✓

③- Rt. Ventricular infarction *→*

Rt. coronary *مخالفو*



Palestinian Medical School

AL - AZHAR UNIVERSITY

6th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Examiner Answer Sheet

Station No.

Q.1- ST Elevation in the inferior leads II , III a VF (2)

Reciprocal ST depression in the Anterior leads (.1)

Q.2- Right coronary artery (2)

Q.3-Acute inferior wall myocardial infarction (4)

Q.4-Transient complete heart block (2)

- Acute mitral regurgitation (2)

- Right ventricular infarction (2)

Acute
Chronic
Late.
Post MI angina
Arrhythmia
Early pericarditis

- ① ST elevation in → lead. II, III, aVF (inf lead).
- ② Reciprocal changes ST depression → in ant. leads..
- ③ Rt. Coronary artery.
Acute inf wall MI.

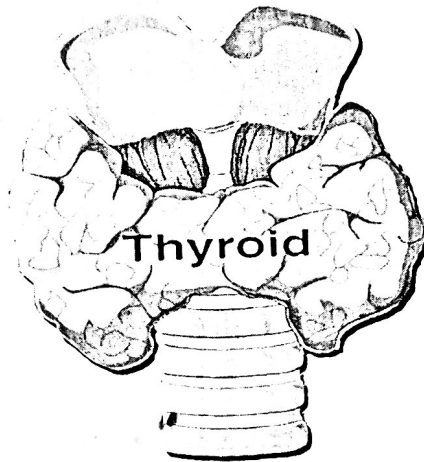
Hypo thy

Station No.

Hyper

1 - general:

- Hair Loss.
- Excessive sweating.
- Tach



inspection

Q.1-When examining a patient with thyroid disease (Hyper / Hypothyroidism) list 5 possible signs you might observe from a general inspection ?

Q.2- When examining the same patients [hand], list 3 things you might observe

Q.3-When examining the same patients [eye], what might you be looking for ?

special. Q.4-Suggest (3) other signs you may detect when examining a patient with thyroid disease.

palpation

Q5.-When palpating the thyroid gland what would ask the patient to do to help confirm your diagnosis ? *drink water*

6th Year Final Clinical Examination (2009) - INTERNAL MEDICINE
Examiner Answer Sheet

Station No.

inspection

Q.1-Thinning/ Dry / Brittle hair.

(5)

Any Thyroid dz.

① -Loss of outer third of eyebrows

② -Facial myxoedema

③ -Agitation

④ -Lid retraction

⑤ -Weight changes (↑ = loss x ↓ = gain)

hand

Q.2-Palmer erythema (↑)

① -Sweaty / dry palms

② -Tremors

③ -Thyroid acropachy

④ -Onycholysis

(3) *Hyper*

Hypo

Tired
sleepy.

↑ wt.

hoarse voice..

myalgia.

↓ Cognitive fun..

↑ Coarse brittle hair

eye

Q.3-Lid lag ①

② -Exophthalmus

③ -Proptosis

④ -Ophthalmoplegia

(3)

examination

Q.4 ① Proximal myopathy ↓

(3)

② - Slow / brisk reflexes ↓

③ -Pre-tibial myxoedema ↑

④ - Tachy / bradycardia ↑↓

Q.5- Swallow water

(1)

- Gout -

Palestinian Medical School

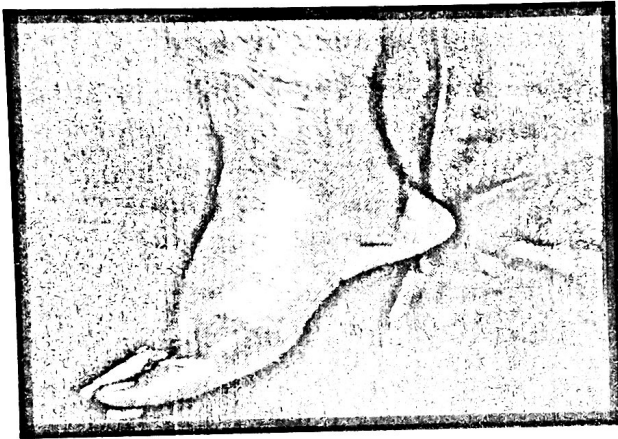
AL - AZHAR UNIVERSITY

6th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Station No.

A 55- year old male known case of hypertension and congestive heart failure .The patient on ACE-Inhibitor ,Furosemide and Hydrochlorthiazide .The patient awaked after midnight with severe pain in his right foot and he noticed something abnormal in his foot. The following photo was taken.

early morning
attack



⊗ mostly affect big toe
become swollen - redness - hotness .

Q.1- What is this sign called & What is the most likely diagnosis?

Q.2 How can you confirm your diagnosis ?

Q.3-What is the characteristic X -ray findings ?

Q.4-What is the most likely precipitating factors in this patient ?

Q.5- How you will manage this patient ?

Palestinian Medical School

AL - AZHAR UNIVERSITY

6th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Examiner Answer Sheet

Station No.

mono Sodium

Q.1 - Podagra / Acute gouty arthritis (4)

Q.2 - Aspirating and inspecting synovial fluid demonstrating MSU crystals. (2)

Polarised microscopy with negative Birefringent crystals (2) *needle sh*

* Q.3 - Soft tissue calcification and punched out juxta-articular erosions around the interphalangeal joints (2)

X-Ray.

Q.4 - Diuretics (Furosimide & Thiazide) (2) $\uparrow$ Ca⁺⁺ causing hypercalcaemia.

①

Q.5 - Oral Colchicine for acute stage (3) *rarely used but still available*

② Change the thiazide

③ Allopurinole for chronic case

Diagnosis:

- Aspiration & inspecting of synovial fluid

MSU crystal

- crystal: needle shape

- polarised microscopy:
③ Birefringent.

- WBC 5000 - 50000

treatment: ① stop & change Thiazide.

- ✓ - NSAID
- ✓ - steroid (in renal impairment).
- ✓ - colchicine
- Allopurinole / probenecid

Soft tissue calcification
↓
punched out
Juxta articular
Erosion
in?

✓ Colchicine
✓ Allopurinole

SIADH

Palestinian Medical School

AL - AZHAR UNIVERSITY

6th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Station No.

A 78 - year - old lady presented with dyspnoea and lethargy ,. She has never smoked cigarettes. Chest X -ray showed a prominent left hilum.

$\downarrow$ Na 110 mmol / l 135 - 145
 $\uparrow$ K 3.5 mmol / l 3 - 5
 $\downarrow \uparrow$ Urea 10 mg / dl (4.0 mmol / l) 20 - 25 mg.
 $\downarrow$ Creatinine 0.7 mg / dl (80 μ mol / l) 0.7 mg $\rightarrow$?? 0.8 - 1.2
 $\uparrow \uparrow$ Glucose 162 mg / dl (9.0 mmol / l)
 Urine osmolality 625 mosmol / kg 65 - 110
 Urine Na 35 mmol / l 500 - 800

10 mg $\times$ $\rightarrow$ mmol

Na 135 - 145

CBC , Thyroid function test , Calcium and cholesterol levels were normal. CT Chest showed a 5 cm irregular mass in the left hilum that had extended into the adjacent lung tissues.

Q.1- Calculate the plasma osmolality.

Q.2- Why there is a low serum sodium ?.....

Q.3- What is the complete diagnosis ?.....

Q.4- What is the other aetologies for this disorder ?.....

Q.5- What is the immediate management for this patient ?.....

SIADH
 $\uparrow$ ADH
 $\downarrow$ Na⁺ < 130
 Posm < 270
 Urine Na > 20
 Suppress RAAS..
 $\downarrow$ BUN, $\downarrow$ creat.
 $\downarrow$ uric acid.
 $\downarrow$ albumin
 no edema

Plasma Osmolality = mosm / L.

$$\begin{aligned}
 &= 2[\text{Na}] + \frac{\text{glucose}}{18} + \frac{\text{BUN}}{2.8} \\
 &= \frac{2 \times 110}{2 \times 110} + \frac{162}{18} + \frac{4}{2.8} \\
 &= 220 + 0.5 + \frac{10}{7} \\
 &2*
 \end{aligned}$$

K⁺ $\downarrow$ Na⁺ water retention
 Retention Na⁺ / 1.5 L
 Edema. no Na⁺ loss
 no edema. Na⁺ loss
 w/o edema w. edema

Palestinian Medical School

AL - AZHAR UNIVERSITY

6th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Examiner Answer Sheet

Station No.

Q.1. 240 mosmol / kg (3)

∴ ← (ملي) ←

Q.2- Syndrome of Inappropriate ADH secretion (SIADH) (2)

Q.3- SIAD due to left brochogenic carcinoma (4) paraneoplastic cancer

Q.4- Pneumonia , Pulmonary TB ,. (4) ↑ Ca⁺⁺

- Drugs (Carbamezapine , Thiazide , Cyclophosphamide)

- CNS (Head injury , SAH , Meningitis)

dp - Lymphoma , Acute intermittent porphyria

Truma

نوع

SCC
small

Q.5- Water restriction (2)

② Demclo cycline - chronic

② Dem clo cycline (when water restriction difficult)

Causes of SIADH

سبب
2005
①
SIADH
Truma

① Malignancy ← SCC.
Pancreatic carcinoma.
ectopic ADH secretion.

② Non malignant

→ pulmonary dz (TB - pneumonia)
lung Abscess
CNS (head injury , encephalitis , cerebral vascular Accident)
Drugs → chlorpromide

①: osmolality

$$2 \times \text{Na} + \frac{\text{mg/dL glucose}}{18} + \frac{\text{mg/dL urea}}{2.8}$$
$$(2 \times 110) + \left(\frac{162}{18} \right) + \left(\frac{10}{2.8} \right) =$$
$$220 + 9 + 3.57 = \boxed{232.5} \text{ mOsm/Kg}$$

normal osmolality
(280 - 295)

∴ low osmolality

④: etiology:

- CNS dz = infection / stroke / tumor / trauma / vasculitis / pain
- pulmonary dz = pneumonia / TB / PE / asthma
- Neoplastic dz = lung cancer / pancreatic cancer
duodenal cancer / Thymus cancer.

⑤ in mild cases: restrict fluid

in severe cases: hypertonic saline / Dem. col. cycline

in cases Not able to correct underlying dz:

Conivaptan / tolvaptan / demeclocycline
{ these medication inhibit }
{ ADH in Kidney and cause }
Diuresis

Palestinian Medical School

AL - AZHAR UNIVERSITY

6th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Station No.

A 49 - year - old lady known to have vitiligo and early menopause presents to OPD complaining of walking difficulties for several weeks. She also complains of annoying parasthesia of the toes and finger tips which has been worse at night. The following photoes and CBC was obtained :



CBC :Hb 8.2Gm /dl , WBC 3800 , Platlet 106.000

MCV :120 ,MCH :36 , MCHC :39

S.Bilirubin (T:3mg /dl -Direct 0.8 mg / dl)

S.Albumin & AST ,ALT : NORMAL.

Q.1-Why the face is lemon like ?

Q.2- What is the most likely diagnosis& the complication in this patient

Q.3- How you will investigate this patient ?

Q.4- What is the differential diagnosis of this type of anaemia ?

Q.5.-What is the treatment and what is the duration of treatment ?

Clinical manifestation :

- Parasthesia
- abnormality in gait & fine coordination
- overt psychoses
- Increase in deep tendon reflex
- Impairment of vibratory & position sense
- Ataxic & spastic gait.

Oral manifestation :

- Pale or Icteric mucosa
- Erythematous macules + irregular border
- Atrophic tongue / fissure changes .
- Lingual parasthesia
- Burning or itching .

Palestinian Medical School

AL - AZHAR UNIVERSITY

6th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Examiner Answer Sheet

Station No.

Q.1-Combination of anaemic pallor and jaundice due to mild haemolysis. (3)

Q.2- Pernicious anaemia / SCDC. (3) : *subacute combined degeneration of spinal cord.*

Q.3- Peripheral smear (macrocytes , ovalocytes , hypersegmented polymorphs

(2)

➔ Serum B.12 LEVEL (1)

➔ Anti-bodies screen (1)

➔ Bone marrow examination (2)

➔ Schilling test. (2)

① Peripheral smear — macrocyte hypersegm

② B₁₂ level

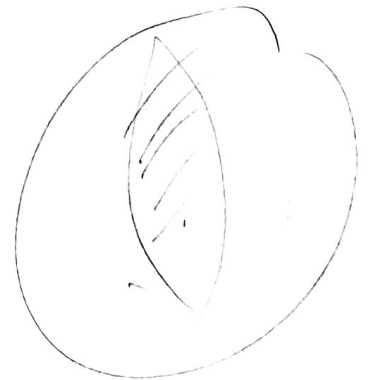
③ BM exam

④ Schilling test

Q.4-FOLATE DEFECIENCY (2)

Q.5- Intramuscular B.12 Injection I.M every 1-3 months & for life (4)

على كل 1-3 اشهر
حقن B₁₂ في العضل
↓
للمدة 3-12 شهرا



Palestinian Medical School AL – AZHAR UNIVERSITY
6th Year Final Clinical Examination (2009) - INTERNAL MEDICINE
Examiner Answer Sheet

Station No.

normal anion gap [10-18]

Q.1- Calculate the Anion Gap ? 34/ 40 (3)

18-16

Q.2- What is the metabolic abnormality ? High Anion Gap Acidosis

Lactic Acidosis (3)

Q.3- What is the most likely cause in this patient ? Salicylate poisoning (2)

why ?

Q.4 Mention 2 causes of Normal Anion Gap Acidosis ? (2)

① Renal Tubular Acidosis /

③ *uretero sigmoido stomy.*

② Diarrhoea

③ Ureteral Diversion / Pancreatic fistula / Hyperalimentation / Saline resuscitation / Acetazolamide

A 25- year- old lady admitted from emergency department in unconscious state . She is tachycadic , tachpnoeic , but haemodynamically stable. The following results are obtained :

Na 144 mmol/l $135 - 145$

K 6 mmol/l ↑ 144 -

Urea 40 mg/dl urea .

✕ Chloride 101 mmol/l

→ Glucose 77 mg/dl

Arterial Blood Gases (ABG on room air)

→ pH 6.96

- Po₂ 106 mmHg (14.1 kPa)

- Pco₂ 14 mmHg (1.9 kPa)

- Bicarbonate 9 mmol /l

Q.1- Calculate the Anion Gap ? $(144 + 6) - (101 + 9) = 150 - 110 = 40$

Q.2- What is the metabolic abnormality ?

Q.3- What is the most likely cause in this patient ?

Q.4 Mention 4 causes of Normal Anion Gap Acidosis ?

Palestinian Medical School AL - AZHAR UNIVERSITY
6th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Student Answer Sheet

Station No.

28

Student Name :

This 50- year – old man with a history of increasing dyspnoea became acutely breathless and was admitted as an emergency .The chest X –ray on the radiology box was taken .

Q.1- What three important abnormal features are present on this CXR

- 1- Enlarged heart size cardiomegaly
- 2- Kerley B line
- 3- pulmonary venous congestion

Q.2- What underlying condition is indicated by these features ?

..... pulmonary edema HF

Q.3- Give the three likely diagnosis , which could cause this condition

- 1- IHD
- 2- HF HTN
- 3- valvular heart Dz

Q.4- Give two treatments that you would give immediately MUNA

- 1- O₂
- 2- Nitrate / Dpiate / Diuresis ...

Palestinian Medical School

AL - AZHAR UNIVERSITY

6th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Examiner Answer Sheet

Station No.

Q.1- What three important abnormal features are present on this CXR

- ① Enlarged heart size (1)
- ② Kerley (B) lines (1) HF
- ③ Pulmonary venous congestion (1)

Q.2- What underlying condition is indicated by these features ?

Heart failure or pulmonary oedema (2)

[1]

[2]

قلبي / رئوي

العضلة ✓
الشرايين ✓
الرئة ✓

Q.3- Give the three likely diagnosis , which could cause this condition

- ① Myocardial infarction / IHD / Cardiomyopathic heart disease (3)
- ② Hypertension
- ③ Valvular heart disease

① HTN
② cardiomyopathy
HF
③ valvular

Q.4- Give two treatments that you would give immediately (2)

- ① Oxygen/ Propped up position
- ② Opiate /Nitrate / Diuretics

O₂ .
analgesics
diuretics .

2009

Inf. * متوقع حينا

2009

2009
الاجبة
① كارتو

2009

Palestinian Medical School

AL - AZHAR UNIVERSITY

4th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

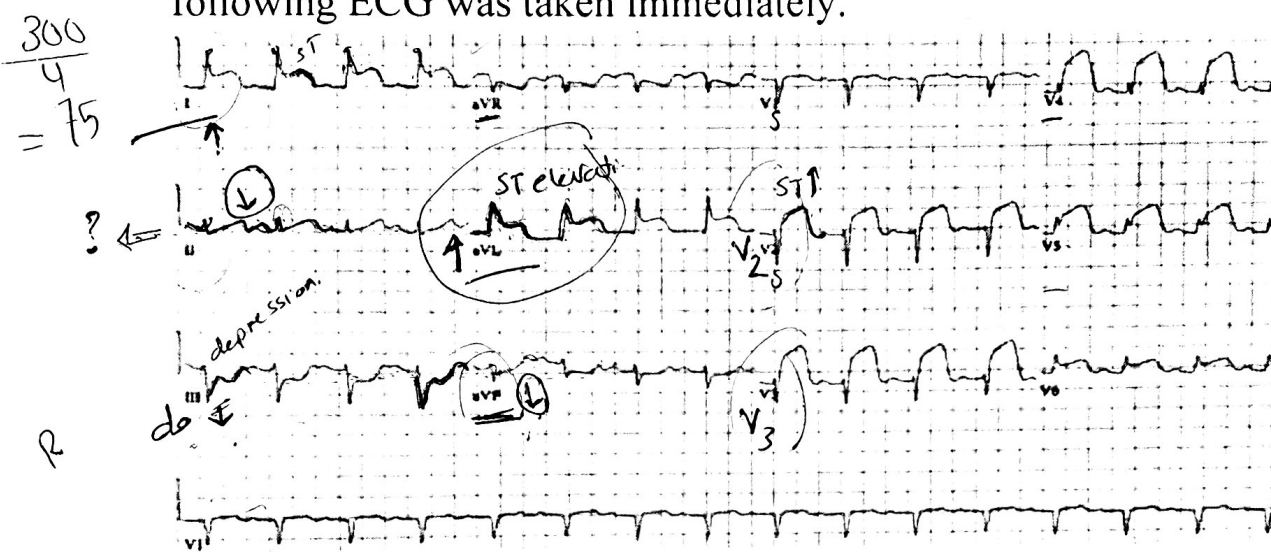
Station No. 1

Rt. sided
ECG

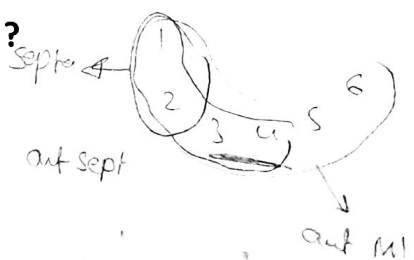
MONA
PCI - SK
Rt. V.
infarction.

أصعب شئ في العلاج
لو سألنا في
Reception
at 1. l.

✓ A 45 - year - old male presented to Emergency Department with chest tightness for four hours duration associated with profuse sweating. The following ECG was taken immediately.



- Q.1- What is the abnormal findings in this ECG ? ST elevation $V_1 \rightarrow V_6$
I - aVL
- Q.2- What is the diagnosis ?
- Q.3- What is coronary vessel responsible for this ECG finding ?
- Q.4- Mention four important risk factors for this patient :
- Q.5- What is the immediate treatment for this patient ?



- 1- ST elevation in lead $V_1 \rightarrow V_6$ (ant.).
 aVL aVF
- 2- ST depression. aVF - III - II

Dx. Acute Ant. wall MI

Coronary. LAD art.

modifiable $\rightarrow$ Obesity - dyslipidemia.
non modifiable $\rightarrow$

- ✓ Ant. wall MI
- Lactic Acidosis
- Derridomyocytis
- PBC
- IDA.
- COPD. Resp. Failure
- DKA
- ✓ Inf. MI
- Thyroid.
- SIADH
- gout
- pernicious Anemia
- Cardio.

Answer Sheet

Station No. 1

Q.1- What is the abnormal findings in this ECG ?

1. ST Elevation in anterior leads (I, Avl, V1 up to V6) (2)

2. Reciprocal changes ST depression in inferior leads (II, III and Avf) (2)

Q.2- What is the diagnosis ? Acute Anterior Wall Myocardial Infarction (4)

Q.3- What is coronary vessel responsible for this ECG finding ? LAD artery (2)

Q.4- Mention four important risk factors for this patient : *left ant. descending a.*

Hypertension, Dyslipidaemia, DM, Smoking, Family History, Age, Lack of physical activity. Obesity. (4)

Q.5- What is the immediate treatment for this patient ?

1- MONA + BAH (Morphine, Oxygen, Nitroglycerine, Aspirin, Beta Blocker, ACE Inhibitor) (3)

2- Reperfusion therapy (PCI, Thrombolytics (tPA, Streptokinase) (3)

Risk factors:

① Modifiable : ② non modifiable

- ① HTN
- ② Dyslipidemia
- ③ DM
- ④ Age
- ⑤ family Hx
- ⑥ Gender

- ④ Smoking
- ⑤ Lack of physical activity
- ⑥ Obesity

immediate Tx

First Aid.

① MONA + BAH

- Morphine
- Oxygen if $SpO_2 < 90\%$
- Nitroglycerine
- Aspirin 300 mg Chewable
- BB
- ACEI

② Reperfusion Therapy

- ① PCI
- ② Thrombolytics "streptokinase"

②

Lactic Acidosis.

Palestinian Medical School

AL - AZHAR UNIVERSITY

4th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Station No.

A 32-year-old lady brought to Emergency Department in unconscious state after repeated generalised tonic-clonic seizures. ABC

Resuscitation was done and immediate Arterial Blood Gases

(ABG) was obtained and the results as follow:

✓ Na: 145 mmol/l

✓ K: 5 mmol/l

✓ Chloride 90 mmol/l

✓ Bicarbonate 10 mmol/l

Po₂: 90 mmHg

✓ Pco₂: 40 mmHg

$$Na = 142 \quad (35 - 145)$$

$$K = 3 - 5$$

$$Cl = 96 - 106$$

$$-HCO_3 = 22 - 28$$

$$pH = 7.35 - 7.45$$

$$-PO_2 = 75 - 100$$

$$-PCO_2 = 35 - 45$$

Q.1 Calculate the Anion Gap?

Q.2- What is the type of this Acid Base Disturbance?

Q.3-Mention 4 causes of this Acid Base Disturbance.

Q.4 - What is the final diagnosis?

Q.5-What is the treatment of this diagnosis?

$$\begin{aligned} \text{Anion gap} &= [Na^+] - [HCO_3^- + Cl^-] \\ &= 145 - [10 + 90] \\ &= 145 - 100 \\ &= 45 \end{aligned}$$

$$\begin{aligned} [Na^+ + K^+] - [HCO_3^- + Cl^-] \\ (145 + 5) - (10 + 90) \\ 150 - 100 = 50 \end{aligned}$$

High metabolic Acidosis.

causes of Acidosis

metabolic
low anion gap
- Myeloma
- low albumin level
- lithium

metabolic
Normal anion gap
- Diarrhea
- Renal tubular acidosis
- ureterosigmoidostomy

metabolic
Increased anion gap
LA MUD PEI
- Lactate (sepsis, ischemia)
- Aspirin
- Methanol
- uremia
- DKA
- Paraldehyde
- propylene glycol
- INH / isopropyl alcohol
- ethylene glycol.

Respiratory
hyperventilation to any cause

Normal anion gap (10-18)

Palestinian Medical School

AL - AZHAR UNIVERSITY

4th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Answer Sheet

Station No.

Q.1 Calculate the Anion Gap ? 45 / 50 (4)

Q.2- What is the type of this Acid Base Disturbance ?

High Anion Gap Metabolic Acidosis. (4)

Q.3-Mention 4 causes of this type of Acid Base Disturbance.(6)

1-Lactic Acidosis

2- Diabetic Keto- Acidosis

3-Uraemia / Paraldehyde

4-Methanol Ethylene glycole /Salicylate /Starvation.

Q.4 - What is the final diagnosis ? Lactic acidosis (Type A)(3)

secondary to epilepsy.(1)

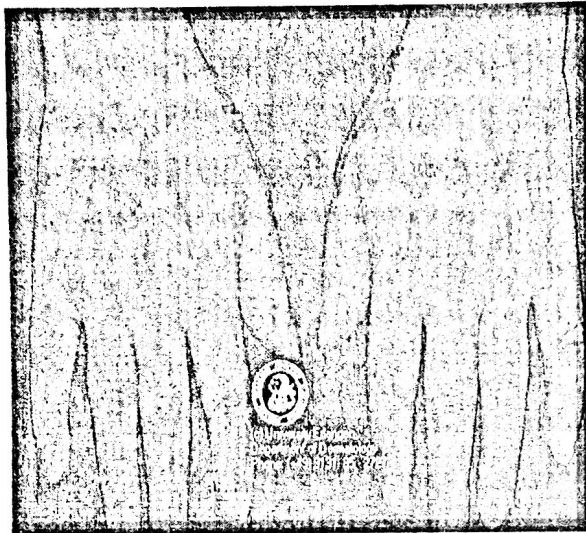
Q.5-What is the treatment of this diagnosis ?

I.V sodium Bicarbonate (2)

① pH ↑ metabolic
② CO₂ ↓ normal-compensated
③ HCO₃ ↓ metabolic
④ anion gap (+) - (-) = 10-18 normal.
⑤ determine cause of M. Acidosis (+)

Station No. 3

A 50 – year – old male presented to out patient department with difficulty in standing after prologed sitting on chair and difficulty in ^{Trunk.} rising his hands above the shoulder .In system review he noticed increase difficluty in passing stool and constipatin. The following photos ^{git} was taken for the patient hands & face. ^{dismotility.}



Q.1- What is the signs on the hands& the face called and which of them pathognomonic and characteristic ?

Q.2- What is the most likely diagnosis ?

Q.3- How you will investigate this patient to confirm the diagnosis ?

Q.4-How you will treat this patient ?

Q.5-What serious pathology can be associated with this diseases
deep investigation to Rule it out need ?

Palestinian Medical School

AL - AZHAR UNIVERSITY

4th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Answer Sheet

Station No. 3

Q.1- Gottrons sign (3) (Pathognomonic)(1) : scaly lesions seen over knuckle
Heliotrpe rash(3) (characteristic)(1) : purple lilac discoloration of the face, eyelid
2 sun exposed areas.

Q.2- Dermatomyositis.(4) : it is an inflammatory myopathy

Q.3-1- Blood Tests (CPK , AST , Aldolase , ESR , ANA , Anti -JO- , Anti
RNP)(1)

2- EMG.(1) : electromyography . show myopathic potential
(short duration/low amplitude)

3- Muscle Biopsy (1)

↓
autoantibody
support the
possibility of autoimmune
origin of the dz

Q.4- Steroid , Methotrexate , I.V Immunoglobulin.(2)

* Q.5- Internal Malignancy.(3)

مريض

Scleroderma / RA / Sjogren + Primary Biliary cirrhosis.

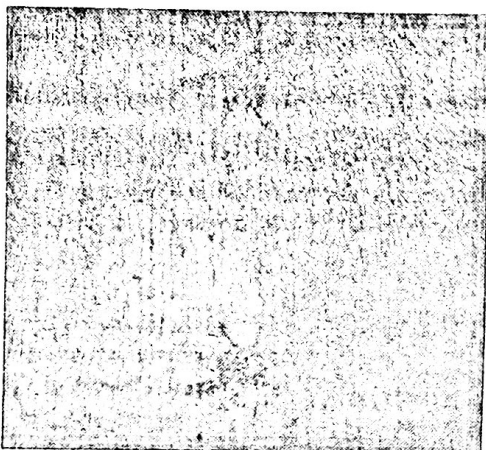
Palestinian Medical School

AL - AZHAR UNIVERSITY

4th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Station No. 11

A 50 -year - old lady non alcoholic presented to out patient department complaining of intense itching, ^{PBC} fatigue, ^{most common symptom in PBC} joint pain, anorexia ^{PBS} and abnormal things appears around her eyes and over her anterior upper chest. CBC , electrolytes & serum aminotransferrases are normal and high serum Alkaline Phosphatase and high serum protein. The following photos was obtained for her face and chest . ^{most common abnormality}



xanthelasma ..

© 2003 Elsevier Limited. All rights reserved.

- Q.1- What are the signs in these photos ? ^{spider}
- Q.2- What is the most likely diagnosis ?
- Q.3- How you will confirm your diagnosis (Investigations)
- Q.4- What is the complications of this disease ?
- Q.5- What is the only drug proved to alter the prognosis ?

antimitochondrial
antibody ⊖

primary sclerosing cholangitis

PBC

⊖

IBD ⚡
ulcerative colitis

Palestinian Medical School

AL - AZHAR UNIVERSITY
4th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Answer Sheet

Station No. 11

Q.1- Spider naevi (3) , Xanthelesmat (3).

Q.2- Primary Biliary Cirrhosis. (4)

- Dx
- Q.3- Ultrasound Abdomen (2) → most specific test .
- (2) - Anti-Mitochondrial Antibodies (2) / Antigen M2 (5) Total IgM ↑
- (3) - Liver Biopsy (2) (+) (6) GGTP ↑
- (4) - IgM

Q.4- Portal hypertension complications and decompensated liver disease. (2)

(1) (2)

Q.5- Ursodioxycholic Acid (2)

زاد
- Ursodioxycholic Acid liver cirrhosis -

ursodioxycholic acid

Palestinian Medical School

AL – AZHAR UNIVERSITY

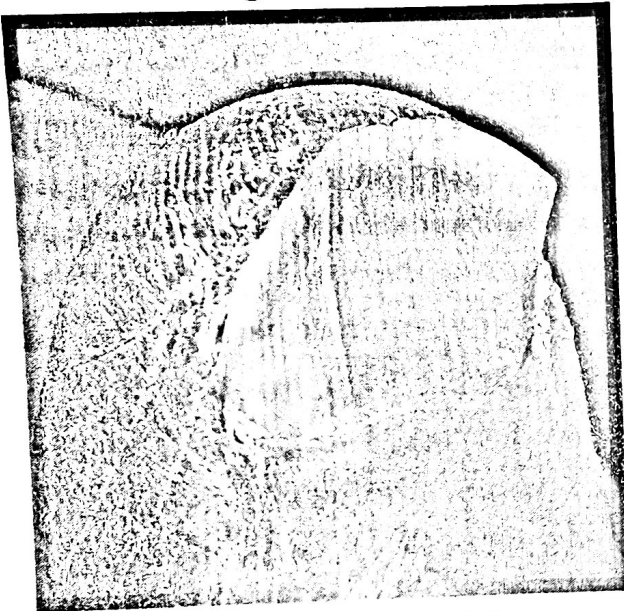
4th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Station No. 12

A 29 – year – old lady presented to out patient department complaining of easy fatigability and progressive dyspnoea on effort .Patient had history of menorrhagia. No history of joint pain .No family history of anaemia,not on any medications.

Physical examination of the patient show pallor , no lymphadenopathy and abnormal finding of her nails and face.

The following CBC was obtained and show the following :



Hb : 8 gm / dl , HTC : 24

MCV : 56

MCH : 22 MCHC : 30

Platlet count : 450.000

Q.1- What is this sign called ?

Q.2- What type of anaemia this patient have ?

Q.3- What is the differential diagnosis of this type of anaemia Q.4-

What is the most likely diagnosis for this patient ?

Q.5- Mention 4 general aetiological causes for this type of anaemia.

Finding is

Palestinian Medical School

AL – AZHAR UNIVERSITY

4th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Strudent Answer Sheet

Station No. 12

Q.1- Koilonychia (3)

Q.2- Microcytic Hypochromic Anaemia (4)

Q.3-

1- Iron Deficiency Anaemia (1)

2- Anaemia of Chronic Disease. (1)

3- Thalassaemia (1)

4- Lead Poisoning (1)

5- Sideroblastic Anaemia (1)

Q.4- Iron Deficiency Anaemia. (4)

Q.5-

1- Increase blood loss (Menorrhagia , chronic GIT loss) (1) ^{most common cause}

2- Increased requirement (Pregnancy , Lactation , child growth) (1)

3- Poor dietary intake (1)

4- Malabsorption (1) ^{celiac}

$\frac{1}{2} \text{ } \frac{1}{2}$

80%

Des. 7.80%

FVC

TLC & RV

FVC

↑

↓

↑

↓

↑

↓

↑

↓

Palestinian Medical School
4th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

AL – AZHAR UNIVERSITY

9) - INTERNAL MEDICINE

Station No.

Q.2- Respiratory failure Type II. (3)

1- Chest wall diseases (Kyphoscoliosis , Flail chest) (2)

Syndrome , Nuromuscular Dystrophy) (2)

4- Respiratory drive (Narcotic over dose) (2)

Q.5 Acute infective exacerbation (3)

Q.5 Acute infective exacerbation (3)

[illegible]

Palestinian Medical School

AL - AZHAR UNIVERSITY
4th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

Station No.

A 20 - year - old , Diabetic , thin built , non married university student , with recurrent hospital admission with Diabetic Keto - Acidosis due to insulin non compliance. Recently admitted to the hospital after an episode of sore throat , fever and repeated vomiting .

Examination on admission : Looking ill , rapid breathing , dehydrated with very dry tongue and throat congestion. Temperature : 39 , Pulse rate : 110 , Respiratory rate 35 / minute, B.P 90/60. Blood Sugar : 450 mg / dl. Urine ketones by dipstick : negative. ABG : Severe Metabolic Acidosis

Diagnosis of metabolic acidosis was considered and treatment with I.V fluids and Insulin, On follow up urine turned to be Positive for Ketone Bodies.

Q.1- What is the criteria for diagnosing Diabetes Mellitus?

Q.2- What are the main Two types of Diabetes Mellitus ?

Q.3- What is the most Two causes that lead to DKA ?

Q.4- What are the Acute & Chronic complications of D.M ?

Q.5 - What is the most important aspect of management to prevent recurrent attacks of DKA in such patient ?

Patient Education

Answer Sheet

Station No.

Q.1

- Symptomatic patient :

fasting
postprandial
glucose

FPG > 126 mg/dl or RBS > 200mg/dl (Single reading) (2)

- A Symptomatic patient : FPG > 126 mg/dl or RBS > 200mg/dl (2 readings)(2)

IDDM

NIDDM

Hb A_{1c} > 6.5% [diagnostic]

Q.2- Type 1 DM & Type 2 DM (3)

Q.3- Omitting insuline therapy(2)

- Infection(2)

1- non compliance.
2- infection.

3- emotional stress

4- excessive alcohol ingestion

Q.4- Acute Complications (3)

① - DKA

② - NKHS non ketotic hyperosmolarity [HONK]

③ - Hypoglycaemia, snow flower cataract , Diabetic Amyotrophy.

- Chronic Complications (3)

① - Diabetic Retinopathy

② - Diabetic Nephropathy

③ - Diabetic Neuropathy

(Low immunity , Infections , Diabetic foot & PVD , Risk for MI & STROKE)

Q.5- Patient Education (3)

Acute

① DKA ✓

② Non ketotic hyperosmolar
Coma NKHC. ✓

③ Hypoglycemia. ✓

Chronic

① macro

↳ coronary art
DZ IHD → MI

↳ cerebral ischemia
↳ stroke

↳ PVD → claudication
DF.

② micro vasculo

↳ Retinopathy.
↳ Nephropathy

↳ peripheral neuropathy

↳ Foot dz.
(ulcer
infection)

Station No. 7

A 20 - year - old , Diabetic , thin built , single university student , with recurrent hospital admission with Diabetic Keto - Acidosis (DKA) due to insulin non compliance. Recently admitted to the hospital after an episode of sore throat , fever and repeated vomiting .

Examination on admission : Looking ill , rapid breathing , dehydrated with very dry tongue and throat congestion. Temperature : 39 , Pulse rate : 110 , Respiratory rate 35 / minute, B.P 90/60. Blood Sugar : 450 mg / dl. Urine ketones by dipstick : negative. ABG : Severe Metabolic Acidosis

Diagnosis of metabolic acidosis was considered and treatment with I.V fluids and Insulin, On follow up urine turned to be Positive for Ketone Bodies.

Q.1 - What are the most Two causes that lead to DKA ? *omit drug * infection.*

Q.2 - Which ketone bodies appear in the blood and urine in DKA ?

Q.3 - Why on admission the urine ketone was negative and subsequently become Positive ?

Q.4 - What is the most important aspect of management to prevent recurrent attacks of DKA in such patient ? *Dipstick only detected Acetone, which later appeared*

Q.5 - What is the criteria for diagnosing Diabetes Mellitus in general?

- ① Acetone
- ② Acetoacetic Acid.
- ③ Hydroxybutyric Acid.

Palestinian Medical School
4th Year Final Clinical Examination (2009) - INTERNAL MEDICINE

AL - AZHAR UNIVERSITY

Answer Sheet

Station No.

Q.1

- Symptomatic patient :

FPG > 126 mg/dl or RBS > 200mg/dl (Single reading) (2)

- A Symptomatic patient : FPG > 126 mg/dl or RBS > 200mg/dl (2 readings)(2)

} + 11.1 A1c
= > 6.5 %

Q.2- Type 1 DM & Type 2 DM (4)

OGTT > 200mg
= 11.1 mmol

Q.3- Omitting insuline therapy(2)

- Infection(2)

Q.4- Beta - hydroxy butric acid(1)

- Aceto Acidic Acid(1)

-Acetone(1)1

Q.5- Dipstix detect only acetone which appear late.(2)

Q.6- Patient Education (3)

Criteria
Fasting 126
RBS 200

Symptomatic

Asymptomatic pt.

* Random Bld. sugar (RBS)
> 200 mg/dL OR > 11.1 mmol/L
* OR Fasting Bld sugar (FBS)
> 126 mg/dL or > 7 mmol
~ One Reading

* FBS > 126 mg/dL
OR RBS > 200
(Two Reading)

* محاضرة 4 *

* 14/2/2019 *

* RNA ^{موجود} single strand

* بعض أنواع RNA يمكن نلاحظ فيها ss حالة دائمة

لكن RNA دائماً يوجد ss

mRNA :- يحمل رسالة وراثية التي تستخدم في

الخلاصة أمينية وتوضع الصفة الوراثية أو الـ DNA

tRNA :- يحمل الحفاز الأميني بناءً على التوافق بين كودون

mRNA وكودون الـ tRNA ، مثل الكاف في الأمينية

في السلسلة الببتيدية إلى ربح نشأ

rRNA :- وهو RNA الموجود في تركيب الرايبوسومات

snRNA :- لا يتكون mRNA في transcription

في يقطع من لنواة يكون RNA في شكل exons, introns

exons :- هي المناطق المشفرة في تحتوي على الكودونات

Exons, introns, exons

AUGG CAGCC AGG UUGCC AG

هذه النيوكليوتيدات لا تمثل رسالة وراثية أي لا تمثل

الخلاصة أمينية في السلسلة الببتيدية ، هي مناطق غير

مشفرة non coding sequence

* في Eu : تتكون الكروموسومات متفرعة و linear

* Genome :

* أي Genome لأي كائن حي نه بكله عن المحتوى الجيني تبعاً ^{الكائن} بمعنى كل الصفات الوراثية.

* لا يمتد هي الصفات الوراثية في جملها، أي أن كل فرد هو هاتو ال Genome المو.

* في 2 different regions :- Genome :-

① nuclear genome ، موجود في النواة ، وهي الكروموسومات الموجودة في النواة .

② الصفات الوراثية الموجودة في cytoplasm : " genome "

مثل ، طياتو كنبيا ، proplast في نباتات
له محتوي own DNA محتوي

it's own DNA

Genome عبارة عن nuclear ، طياتو كنبيا و

proplast genome .

* الجولات من حدة 27 *
* 27 *
* 27 *

جیسے رقم 2

جیسے رقم 2

اللہ موجود ہے *operon* و *promoter* واحد
 * في خلايا *prokaryotic* + ~~و في خلايا *eukaryotic*~~
 والى *Euk.* ← كل جين له *promoter* واحد
 ویکڈیٹا موجود ہے *promoter* واحد

met ← *met* ^{حفظ آمینو} اولیٰ ^{تترجم} بتترجم

یعنی *DNA* ← ^{انتقال} لنویسہ ان ^{ملا} *mRNA* سنتسخ ل
 و تترجم ل ^{حقاً} بدھا ^{تقطیع} *mRNA* یونیٹ یونیٹ
 في الخلية

المستونين م كان موجود

Stop codone م في *stop codone* في *DNA*
 و لہ موجودہ في *mRNA* ...
 یعنی ^{ملا} *mRNA* لنویسہ ان *mRNA* ^{مٹلوا}
stop codone

met ← لیستہ برسرہ ^{حفظ آمینو} ہاں ل
 عبارت عہ ^{نیویسہ ان} *mRNA* سنتسخوا ل
 و *mRNA* ^{سنتسخ} ^{حفظ آمینو}

* خيط طفيف للسجاد
لشبح السجاد

(15)

كيف genetic material في الخلية - compaction
euk. compaction ← في عدة مستويات من الـ

المستوى Level compaction
هو مستوى nucleosome هو جسم نووي
مكون من 8 بروتينات الهستون و 2 جزيئات DNA

octameric structure nucleosome
↓ ↓
8 جزيئات قطع

* مكون من 8 وحدات بناء من

2H2A 2A ← مستوى 2

2H2B 2B ← مستوى 2

2H3 3 ← مستوى 2

2H4 4 ← مستوى 2

4 أزواج من (الهستون)

* الـ 8 يتجمعوا مع بعض كقوة وأجاسير DNA
لتتكون حولها كقوة nucleosome
لشبح nucleosome؟ لتتكون في جزيء نووي DNA
وجزيء نووي " هو الهستون "

* nucleosome ← يتكون من 146 base pair DNA

146 base pair

المرحلة المتضخمة أكثر كثرة على هو الكروموسوم الموجود
 مرحلة *metaphase* كروموسوم
 أكثر انضغاط لأنو يكون *transcriptionally in active*

* الكروموسوم يتكامل الوظيفة بعد م تكون الخلية
 بمرحلة انقسام يكون له شكل وظيفي، الشغل الوظيفي
 للقيام بوظائفه هو وظيفة الكروموسوم لحمل الجينات التي
 فيه تنتسخ لـ mRNA و mRNA يُترجم لأحماض
 أمينية وأحصل مع بروتينات تقوم بالوظيفة.
 هذا الكروموسوم يكون ظاهر على شكل أو يكون
 منقسم للكروموسوم
 مناطق مضغوطة وهي مناطق لا تنتسخ ولا تُترجم
 heterochromatine تكون مضغوطة

... tightly
 * أكثر مناطق بصير فيها *compaction* هي
centromere و *telomere*
 هات هيك هادي المناطق *heterochromatine*

- ① tightly compact
- ② transcriptionally inactive

م بصير فيها عليه نسخ مضغوطة كثير وهي أصل
 لا وظيفة لها. و هيك هيك م بصير فيها
 عليه نسخ. و شكلها هذا الضخم لا نسخ
 و لأنها تتابع عن بعضها البعض.

* لما أحكى عن جين من جاك عن Genetic
Genomics !!
Function ← NA وهذا DNA → N.A
RNA

* ثم هو فروع علم الوراثة؟! (كيف يمكن دراسته؟)
الوراثة يمكن دراستها في 4 مستويات

Mendelian Genetics & Classical Genetics ← Level 1 *
أو Transmission

* الوراثة التقليدية أو الوراثة طنانية
والتي تتعلق كيفية انتقال الصفات
للب العيون صادة وتنتج * مرض مرتبط بالجنس * تعدد باطل وهذا

Cytogenetic ← Level 2 *
Cell يعني

the study of Chromosome

and the carrier types !!

* عزل الكروموسومات في الخلية ونسوفه عدد
الكروموسومات زائد ولا ناقص عدد الكروموسومات
بترتيب T 8 فن الجينات لما بدى أدلة
جين بدراسة Transmission لكن لما جاك مع مجموعة
كروموسومات وهادي الكروموسومات موجودة بالخلية
بدى أشتوف كروموسوم 22 في زيادة فيه؟

الانحصر الطفل
يعنى يعمل عزل DNA تبعو

لكن 'triglyceride' مَ طلع من ~~الخلايا~~ الخلية هو طلع من ~~الخلايا~~ الخلية
 طبعاً في جينات تُنسخ ولا تُترجم ؟؟
 الخلية في بيوطي Libosomal RNA مَ بتترجم بلع
 تُنسخ RNA .

الخلية في بيوطي tRNA تُنسخ ولا تُترجم

~ ~ ~ snRNA ~ ~ ~

~ ~ ~ scRNA ~ ~ ~

* الخلية الذي تُنسخ وتُترجم بيوطي AA و عمل
 عن بروتين .

* في الخلية عندي مَ يقارب 25,000 جين
 في أحياء متشابهة في الجينات .

لأنهم يتعطل نفس البروتين عن الإنزيم
 في ربح يقوم بنفس الوظيفة .

* يعني الإنزيم في برو عمل 'triglyceride' هو
 نفس الإنزيم في ربح عمل ~ ~ ~ عند الفأر .
 من هذه وظائف مشتركة لهذه
 الجينات .

May

الفرق بين genetics و gene

* ^{functionally and physically} unit of heredity *
هي وحدة الوراثة وظيفياً و فيزيائياً

الحمض نوويي DNA هو
المسافة بين النيوكليوتيدات
رقم 17
رقم 25000
رقم 17000

* Functionally
هذا الحمض في بروتين
الترقيم أو بروتين

* م في جين بروتين كروموسوم ولا lipid
م في جين م بروتين بروتين
للا الجينات التي تُترجم بروتين AA's
أعلى بروتين

* وهذا البروتين قد يكون مسؤولاً عن تكوين أو
تثبيت الروابط الإسترية م بين glycerol و F.A
عنه أعلى triglyceride

(23)

Level 4 ← population genetics

* الدراسة العشوائية - دراسة الصفات الوراثية في

مجتمع معين .
يعني المجتمع الفلسطيني م في عيونه نرى عيونه
اليابانيين أو المصريين .

مثلا لو فلسطيني تزوج حسنة و جاها ع فلسطين
من جانب حسنة جيه جديدة للبلد .

* كيف يتغير هذا الجين في البلد ؟

صغير هذا الجين يتغير في المجتمع ولا
أب رضيع هذا الجين فلسطيني (يعني ظاهر
في المجتمع) .

* يعني ظهور صفات جديدة .

* طوبى للصفات الوراثية *

* قبل 1948 كانت لوراثة إيتانية و Cytogenetics
هي السائدة ولعبت دوراً في الوراثة الجزيئية
والعشوائية هي السائدة .

فأخبرهم تحت الميكروسكوب وأعمل *Caryotyping*

physical appearance the *Chromosome*

* مجموعة كروموسومات وكيف صورنا
تظهر تحت الميكروسكوب هذا اسم
Caryotyping يعني في هادي الحالة م بدرج
transmission ولا بد في حالي هادي لصفة
نقل بواحدة حرة وهكذا ، بل بدرج
Caryotyping

molecular genetics ← *Level 3* *

* الدراسة الجزيئية
* هو تقنيات لصفة الوراثة
وتقنيات التفاعل مع *genetics material* DNA
يعني أجيب جسمي وأقصو *Genome human*
وأزخ هذا الجسم في البكتريا ، لبكتريا
نتج هذا البروتين

* *recombinant* لإعادة ترتيب DNA

* قطع قطعنا DNA من هنا

بـ DNA من هنا في *recombinant*
DNA

• مرحلة الانقسام → الفلية في بكتيريا → فاضية تقول
• أوتقمة . ولكن بعد مرحلة الانقسام عليه تقول
• أوتقمة .
• الحالة يكون الكوم .

في هذه الحالة يكون الكروموسوم في أعلى درجة
الـ compaction ويكون لك كروموسوم heterochromatin.

Genetics علم الوراثة
transmission انتقال الوراثة

transmission is also

Genetics يَظُنَّ تعريفها
وحدات الوراثة أو مكونات الوراثة أو الأشياء
المسؤولة عن التوريث كما

Genetics :- كيف تتوحد الجينات في الشكل
!!! الوظيفي !!

لَوْ أَنَّ الْيُوسُفَ مِنْ كَيْفٍ effect تَبَعُوهُ لَمْ يَخُذُوا
الْجِبَّةَ بِعِطْفِئِهِ لَازِمٌ وَهَذَا الْإِتْرَافُ مَسْئُولٌ عَنْ رَبِّهِ
عَادَةً كُنَّا مَعَ قَادَةِ كُنَّا وَهَذَا ^{الْجِبَّةُ} بِمَنْشُورٍ
pathway وَهِيَ عَادَةٌ أَخَذَ هَذَا الْفَرْقَ تَبَعُ
الْعَيْنِ

الـ radial loops و topoisomerase لها دور مع بعضها أكثر

9 * المناطق التي يصير فيها نسخ وترجمة نسخ
euChromatinic "مناطق" كروماتينية حرة
region

← تتؤدي إلى نسخ وترجمة ولها طراز أخص
أمينية وتركيب بروتينات ذات وظيفة

EuChromatine صفاتها :-

- ① less condense. أقل الكثافة يعني أنها فاكهة عن بعضها لكن
- ② euChromatine region. أقل compact
- ③ transcriptionally active.

نشطة يعني لو أجاز ~~polymerase~~ RNA وكمياتها، لا تزال
رج يفتح strands في بعضهم وعمل نسخ لهذه المنطقة
ويطلع mRNA يخرج، ليس بلامرر بتسكوا، الرابو سوان
تتبع ويعلمين أمانه أمينية و بروتين ...

* radial loop → Less compact.

* الكروموسوم في بنواة بكون مع شكل ألياف كروماتينية
طالبت أ الخلية في الانقسام يصير تقصر وتلفظ أكثر
وبظفر مع شكل كروموسوم. والي هو الكروموسوم والي metaphase
* في الوضع الطبيعي للخلية لكي تم فيها انقسام يعني
لا تقدر بدور انقسام الخلية



30 nm

في مستوى الجسيمات

* Level 3

النشاط في مستوى 30 nm fiber مع nuclear matrix
 الكروموسومات موجودة في النواة، والنواة تحتوي
 matrix وتحتوي على بروتينات وهذه البروتينات
 تجمع ال 30 nm fibers وتقل compaction أكثر

* كل مرة في compaction يقل كاس loop

30 nm like Loop وبعد ^{super}coiling
 compaction وتقل compaction
 loop ال كاس ال كاس

... HRS ...

200 انتظام مناطق التيلومير انتقلت
 201 من و من يأخذ به من كناضه
 ايا حية المافرة من أفقد الخلية Euk. عقيمة
 م وظائفها الأساسية من حالت الخلية
 من حيث طول منطقة التيلومير بعد عمر
 (خلية)

سبب سبباته وتعالى أعطى منطقة التيلومير
 mechanism ترجع المتقدمة عنه من موت

مناطق Repetitive ← يعني هنا ضمير جسيم
 له غير متفرقة من يتوسط أى جسيم
 المتأخر إلى هنا جسيم هي على يتكرر

nongenic sequence من يتوسط أى وظيفه
 highly satellite ← هي repetitive لك هي
 repetitive متكرر GCCG و هذا

مناطق متكرر

← يعني sequence GCCCG

تكرر أى مرة مرة من يتوسط جسيم
 و هي satellite

* ~~الكروموسومات~~
 * في منطقة الشروع كساب : والو مواقع :
 * sub meta, meta الخ
 * منطقة DNA وأنها مكتظة
 (highly compacted)

* في كروموسومات ال - كلب : في منطقة
 منطقة الأطراف هي المنطقة التي تحدد عمر
 الخلية

* طالت تنقسم الخلية دائماً لآ انتقال للخلوة منطقة
 الطرف للكروموسوم ^{استقامته} تنقسم DNA
 * كل م' الخلية تنقسم تنقسم منطقة
~~تقصير~~

telomeric
 هي منطقة
 من أطراف
 الكروموسوم
 حيث
 يحدث
 تقصير

→ هي منطقة
 ارتداد بـ /

ف لو انقسمت الخلية مرة ثانية تبصر تقصير
 عند الملاحظة ، كما نرى ولما تنقسم من ، كما نرى
 الميزة في تفقد الخلية ^{eukar} وظائف أساسية في توت
 الخلية . من لو طول منطقة telomer "التلومير"
 200 ألف
 من بعد 200 انتقال منطقة التلومير تقبل

ال في نيوكليوتيد لو شغوا رح يعطوا meth

لن في stopcodone هاه stopcodone هاه
مرتبه اا سب الرسمة والي بالانزيم
لانف لازم يكون للجيم اشارة بنطية
واشارة خايه وهذا الجيم لولو كودود
وكودود خايه

* الجيم والي في pro :- origin

الجيم والي في pro Functional regions + operon
نفسو ومناطق توقف الجيم
مناطق 3' 5' جين

في كودودوا البكتريا في intergenic regions

non transcript مناطق DNA ولا يلا

نسخوا رز هادي من (operon) هذه مناطق
3' جين لا نسخ ل mRNA لا ترجم ولا نسخ

* في فلاي pro لازم يكون في منطقة اسمها

Origin of Replication "وام"

الكودودوا عدد اعلو عليه تضاعف

لازم منطقة ابراعضا

E. coli ← حوالي 6 آلاف جين

* الجينات تأتي في شكلها وظيفي وتقطع جزيئات موجودة
في شكل أوع منطقة أو في شكل متكامل يسمى operon

هو الوحدة الوظيفية للجين في خلايا
prokaryotic وحيوي كـ، كالمادة المعقولة
الحيوية

* المنطقة الفرعية من جينها mRNA
* operon يحتوي على منطقة promoter
← المثلث المعقولة

* كما يكون عندي DNA ويبدأ أعلاه transcription
في بدو ييجي ونزعم النسخ عنده النسخ DNA
ويطوي mRNA

ونزعم RNA polymerase لانهم يعرفون على منطقة
promoter

لهم موجودة قبل كبر يعرفون عليها وينسخ الجين عن
يطوي mRNA

* وكلهم موجودين في operon

أولاً DNA البكتيريا أولاً واستر على شكل loop domain وبعد supercoiling

* أحياناً ننخفض مساحة DNA تقريباً ألف مرة من حجمها الحقيقي مما يعطي قدر من الخلية DNA effect within

في كروموسوم الخلية البكتيرية ← مهم جداً لفهم الخلية في كروموسوم DNA prokaryotic cells

أولاً الخلية أحياناً عارضة لما نوهو single circular DNA وفي كماله في بعض الخلايا البكتيرية بلازميد

* يعطي قدرة البكتيريا على مقاومة المضادات الحيوية موجودة في البلازميد

ويُعتبر البلازميد extra DNA يعني أنه منفصل موجود في الخلية البكتيرية منفصل موجود في الخلية الشم

* 12 خنثوي ؟؟ خنثوي 4.6 مليون base pair Haemophilus influenza خنثوي على

حفظ

* لو جيت دائرة ع شكل سلكة :- لفتها اول لفتح
شكل 8 ولفيتو كمان مرة - وهذا
تصغير لها اذا الحجم
من هادا ال supercoiling محتاج انزيم توبو isomerase
فطالبت DNA بالانزيم وبي افكو ؟ محتاج توبو
isomerase . زي م لفتو ب توبو isomerase وبيكو
ب توبو isomerase وليش افكو ؟ عشان ^{أعمل} transcription
replication

* DNA عايد بي عمل replication لازم يتباعدها ال
2 strand عن بعض . ولها ملفوفات في كيف دباعدهم
عن بعض بفكهم
فهادي supercoiling وظيفتها في supercoiling
الانزيمات التوبو isomerase
وفي relax

* topoisomerase اسم ثاني وهو gyrase
له بنية supercoiling
كل domain واحد بلفهم يعني حل
loop

الاسلاك لما ب supercoiling او ب loop domain



* ~~Exercises~~ 8 -

* الملاحظة في الكروموسومات : بأنه حجم المادة الوراثية أكبر من حجم الخلية

* الخلية مثلاً *E. coli* = ال Genome بها 4.6 mega base

لو حللنا النيوكليوتيد بشكل مستقيم " خطي " 6 رز
نلاحظ بأنه مثلاً أكثر من مليون مرة من طول الخلية
البكتيرية .

يعني DNA الخلية البكتيرية لو أخذناه بشكل
خطي في معنا حوالي ألف مرة من حجم الخلية البكتيرية
لو في ضيق ، استلزم أن ضيق DNA وخصائه
بشكل خطي هو 2 meter في الخلية الواحدة
طولها أطول من كل شيء في نفسه .

ن كيف genetic material ← enclose within
the nucleus

!!??
بشكلها

لو جئنا مع بكتيريا ^{cell} packed within nucleus

ولو مع Eukaryoti nucleus

والكل لانه super volume ضيق

عملوا التجارب

وانتجوا ^{32}P و ^{35}S في غذاء جراثيم بكتيرية مع

Mawrice بل كتحليلهم جزء في DNA ووروزا لينة
تتاركووا في ذلك في ملاحظاتهم البلورية فيها
DNA بـ 3.4

ولكن ووزا لينة في أوقات الجاثرة لأنهم لم
تلقوا في كبريت و لكن تكبروا لها
عملوا جراثيم جاثرة الثوريكانه بالها

الكروموسومات :-

الخلايا pro و euk في مختلف في طبيعة كروماتيد

الوراثة

في pro DNA single circular لكن في Euk. في
في كروموسومات مختلفة

الكروموسوم عبارة عن بروتين + DNA
سواء في pro أو euk

لكن ما هي هذه البروتينات ؟

RNA : DNA linear و circular

الكروموسومات في البكتيريا عبارة عن single circular DNA
موجود في منطقة - nucleoid

S.n.RNA يقوم بوظيفة إنزيمية

* إنزيم له طبيعة nuclear نووي

* معظم الإنزيمات بروتينية لكن في الحنف عندي إنزيمات

له طبيعة نووية - تعالينا الفهم لك في اه

* مباحث enzyme قبل introns

إنزيمات نووية
له طبيعة نووية
S.n.RNA

* ليش S.n.RNA موجود في spliceosome

* تعرف على المنطقة على حدة

فما و على حدة تنقطع فيها في رفع introns بها عنده

مشاهدة مع RNA

وهذا S.n.RNA يحتوي على منطقة مكملة لأطراف ال

introns

* S.no.RNA - لنووي في لنوطين مسؤول عن ال modification

* S.C RNA *

موجود في السيتوبلازم

S.nRNA, tRNA, rRNA
أي تعدد بلا تباين علم
يقوم S.noRNA

لذلك mRNA قبل تم ربطه من النواة إلى استوبلازم

الإنتران introns
Capping: mRNA على الطرف 5' Cap ربح 3' tail
التيققة 7'

tailing

splicing

Remove of introns

تم نسخ mRNA ربطه برا، لنواة إلى استوبلازم
وهو يحتوي introns
وعليه يها إلى؟؟

splicosome هو عبارة عن بروتين +

small n.RNA
nuclear

وظيفته إزالة ال introns

هنا مناطق غير مشفرة لا تقرأ في
صيغة لتكوين أحماض أمينية، موجودة بين
introns حيث تم صنع الترجمة التلقائية لـ mRNA
من هذه حمات لـ mRNA فـ mRNA
لذلك قبل تم ربطه mRNA من النواة لا يتم نقل
في مراحل للمحاسب