PMS COLLEGE OF DENTAL SCIENCE AND RESEARCH
II BDS REGULAR BATCH

REMEDIAL EXAMINATION DECEMBER 2021
GENERAL& DENTAL PHARMACOLOGY AND THERAPEUTICS

02/12/21

Time: 3 hours **Total marks: 70**

Essay **(2x10=20)**

1. Enumerate the drugs used in the treatment of tuberculosis. Explain the mechanism of action, adverse effects and therapeutic uses of Rifampicin. (4+2+2+2=10)
2. Classify the drugs used in bronchial asthma. Mention the mechanism of action and adverse effects of Salbutamol. Outline the treatment for acute severe asthma (status asthmaticus) (4+2+2+2=10)

Short Notes **(4x5=20)**

3. Enumerate the uses and adverse effects of corticosteroids
4. Explain the mechanism of action, adverse effects and therapeutic uses of Amoxycillin. (1+2+2)
5. Classify antifungal agents. Outline the treatment of oral candidiasis. Explain the mechanism of action of the drug used (2+2+1)
6. Classify Anti - Ulcer agents. Discuss the mechanism of action & uses of Pantoprazole (2+1+2)

Answer Briefly **(10x3=30)**

7. Obtundants
8. Insulin analogs
9. Non sedating Antihistamines.
10. Explain super infection. Give two examples
11. Compare and contrast: Domperidone and Metoclopramide
12. Explain the mechanism of action and uses of Acyclovir
13. Mention two chelating agents and its uses
14. Outline the treatment of diabetic ketoacidosis
15. Methotrexate
16. Styptics

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Angela Sasthri

2688

To be fillled by the Candidate

Please (✓)Tick Answered Question Numbers in appropriate boxes

Q.No.	ANSWER	Q.No.	ANSWER	Q.No.	ANSWER	Q.No.	ANSWER	Q.No.	ANSWER	Q.No.	ANSWER
1.		6.		11		16		21		26	
2.		7.		12		17		22		27	
3.		8.		13		18		23		28	
4.		9.		14		19		24		29	
5.		10		15		20		25		30	

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VATTAPPARA, TRIVANDRUM

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2. **Fill this form in capital letters only**
3. ***This form will be Scanned by Computer***
4. **Do not fold the Sheet**
5. **Do not make any stray marks on this form**

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1. **Degree / Diploma** : Write the name of the Degree / Diploma (eg : MBBS,BDS,BAMS,BHMS,BSc. Nursing, B.pharm, MS-Ortho, MD- General Medicine etc.
2. **Exam & Sub. /Paper & Section** : Write the Examination in which student is appeaning (Eg: 1st Year, 1st Phase, Final Year, etc.), Write the subject name (eg. Anatomy - Paper 1 Section A)
3. **Candidate's Name** : Write your name in BLOCK letters
4. **Exam Date** : Enter date of examination **dd/mm/yy** format.
5. Reg. No. Enter the register number(all ninedigits)in the boxes first. Do not leave any boxes empty or do not fill any boxes other than number (0to9) Darken the appropriate ovals neatly with DARK BLUE/BLACK BALLPEN only. The Register Number composition for the course is 99 999 9999 (Eg. 10 001 5483)
6. **Q. P.Code,** Question Paper code is printed on your question paper. For example,Question paper code on anatomy-Paper 1 with Q.P. Code M 1201. In this case, You are required **to fill only the number portion of the Q.P.Code-** i.e, 1201, in the boxes provided for the purpose.
7. **Signature of the Candidate** : Affix your signature within the box.
8. **Please(✓) tick** the answered Question Numbers in appropriate boxes provided on the top of this page

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1. **The answer must be legibly written using BLUE or BLACK INK PEN or BALLPEN.** Any colour pen/pencil can be used only for drawing the figures/diagrams. Underline, if any, must be drawn using RED BALLPEN only.
2. Write answers on both sides of all pages
3. Write the correct number and sub division (if any)of the question on the left hand margin at the beginning of each answer.
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5. **There should not be any other. identification in the answer book.** Any sort of identification will be considered as Malpractice and the registration of the candidate for the paper will be cancelled without any further notice.
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To be filled by Invigilator	To be filled by Custodian
Q P Code	Packet No. / Sl. No. in the Packet
Degree / Diploma : II ND BDS (REGULAR)	
Exam & Subject : PHARMACOLOGY (REMEDIAL)	

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PMS To be filled by Custodian

QP Code | Packet No. | Sl.No. Packed

FOURTH VALUATION
To be filled by Examiner

PART VII

Q. No	MARKS	Q.No	MARKS	Q.No	MARKS	Q.No	MARKS	Q.No	MARKS	Q.No	MARKS
1		6		11		16		21		26	
2		7		12		17		22		27	
3		8		13		18		23		28	
4		9		14		19		24		29	
5		10		15		20		25		30	
SUBTOTALS											

Grand Total: 0 1 2 3 4 5 6 7 8 9 (0 1 2 3 4 5 6 7 8 9)

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Name of the Examiner:

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Serial No. in the Packet: 0 1 2 3 4 5 6 7 8 9

PHARMACOLOGY
REMEDIAL EXAM

42

I. ESSAY

1) The different drugs used in the treatment of tuberculosis can be classified as:

① ⇒ First line drugs (THREE)
- Rifampicin
- Isoniazid
- Pyrazinamide
- Ethambutol.
- Streptomycin.

② ⇒ Second line drugs
- Ethionamide
- Thiacetazone
- Rifabutin
- Rifapentine
- Cycloserine
- Para Aminosalicylic acid.
- Kanamycin
- Capreomycin.
- Amikacin.

③ Tuberculocidal drugs (Kills Mycobacterium tuberculosis)
- Rifampicin
- Isoniazid
- Pyrazinamide
- Streptomycin
- Capreomycin
- Rifabutin. etc.

④ Tuberculostatic drugs (stops the growth of Mycobacterium tuberculosis)
- Ethambutol
- Ethionamide
- Cycloserine.
- Thiacetazone. etc.

RIFAMPICIN

→ Mechanism of Action
- It inhibits the DNA dependent RNA polymerase.
- Thereby inhibits RNA synthesis.
- Tuberculocidal.

DNA —(DNA dep. RNA polymerase ↑ X)→ mRNA

→ Adverse Effects

- Hepatotoxicity
- GIT disturbances – nausea, vomiting, epigastric pain
- Flu like syndrome – fever, chills, body ache
- CNS symptoms – headache, drowsiness.
- Hypersensitivity reactions – rashes, urticaria.
- Staining of secretions – orange-red colour of urine, sweat, saliva, tears.

→ Uses

- Effective for treatment of tuberculosis and atypical mycobacteria.
  Rationale: Kills Mycobacterium tuberculosis & atypical mycobacteria
- Leprosy.
  Rationale: Kills M. leprae.
- Resistant Staphylococcal infections. – acts against all gram positive and gram negative organism.
- Meningococcal infections. – Acts against meningococcus
- Brucellosis – Drug of choice.
- Eradicate H. pylori?

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2). BRONCHIAL ASTHMA

CLASSIFICATION

1) Bronchodilators
2) Corticosteroids / Anti-Inflammatory Drugs.
3) Mast Cell Stabilizers.
4) Leukotriene Antagonists.
5) Anti IgE antibody.

(1) Bronchodilators

(a) Sympathomimetics

⇒ Selective β_2 agonists

Short acting :- Salbutamol
- Terbutaline.

Long acting :- Salmeterol
- Formeterol

⇒ Non-selective β_2 agonists

- Adrenaline
- Isoprenaline
- Ephedrine.

MICROBIOLOGY

REMEDIAL EXAM

26/35

good.

Essay

1.

Mode of transmission :-

- Hepatitis B can be transmitted by 3 modes:

1) parenteral transmission.
2) perinatal transmission.
3) Sexual transmission.

1) Parenteral transmission :- Hepatitis B can be transferred from the blood and other body fluids using of unsterile razors can cause the transmission

2) Perinatal transmission :- Hepatitis B can be trasfred from mother to baby through placental transfusion and breast feeding.

3) Sexual transmission :- Hepatitis B antigen may be found on the body fluids, semen, vagnial secretions and can be transfred through sexual contact.

- Male homosexuals are more prone.
- Hepatitis B can also be transfred from open skin lesion contact.

Pathogenesis

- Hepatitis is an immune mediated response.

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membrane.

- When the viral organism develops before the host immune response develops.
- During the replication, it activates the B & T lymphocytes. Hepatocytes carries viral antigen.
- Hepatitis grows in the hepatocytes and activate the Natural killer cells, macrophages etc.
- Immunodefficient people and infant act as a carrier of hepatitis B.

Clinical manifestations

- Incubation period : 30-180 days.

There are 3 stages.

1) Pre-icteric stage
2) Icteric stage
3) convalescent stage.

1) Pre icteric stage :-

- Incubation period is 6 days - 6 months.
- Gastrointestinal symptoms present.

2) Icteric stage :-

- Jaundice, biliurenia, dark stools are present.

3) Convalscent phase :-

- Nalnise, fatigue are found

- Extrahepatic & Intrahepatic complications are seen.

- Two types of carriers are seen :-

1) Simple carriers :- HBsAg is less. Only large transfer of blood & product can transfer the infecting

2) Super carriers :- HBsAg is in high titre even a small quantity of blood & products can cause hepatitis.

Lab diagnosis

- Hepatitis can be detected by ELISA, PCR

1. Detection of viral markers :-

a) HBsAg

- It is the epidemiological marker of Hepatitis B
- After the infection, jaundice occurs after 2 weeks after infection.

b) HBeAg & HBVDNA :-

- They are the viral markers of :-
  - ~~acute~~ viral ~~replication~~ infectivity
  - Chronic viral infectivity
- They are the markers of multiplying of viral antigens
  - Active replication
  - Chronic.
  - Carriers.

c) HBcAg :-

- They are not detectedble, they are found inside the membrane of HBsAg.

d) Anti HBc ~~Ag~~ IgM :-

- They are the first antibody elevated in the blood.
- Their indication ~~indicate~~ that the organism is infected.

e) Anti HBc IgG :-

- Epidemological marker of Hepatitis B.
- They are positive for infection, chronic stage.

f) Anti HBc :-

- They remain less infectivity, during the stages.
- The person may recovered

Anti HBs Ag :-

- Occurs after the ~~anti~~ HBs Ag has diminished.
- They person is fully recovered.
- They are the markers for Hepatitis B vaccine.

Prophylaxis

1. General preventive measures :-

- Proper health education, use of unsterile objects should be avoided, proper hygiene should be maintained.
-
-

2. Immunisation :-

1) Active immunisation :-

- Recombinant yeast viral Antigen.
- Recombinant vaccine
- Given at 6, 8, 10, 14 weeks along DPT.

2) Passive immunisation

- Human Immunoglobulin (HBIG)

- It occurs when there is close contact of Hepatitis B infection as in dental or surgical infections

Combined immunization :-

HBIG + vaccine is more active.

Short Essay

2. Hypersensitivity is a ~~reacti~~ immune response that cause tissue disease, tissue distruction, organ destructions.

- There are two types of hypersensitivity - Immediate and delayed type hypersensitivity.
- Coombs & gel classified hypersensitivity into:

1) Type I - Anaphylatic
2) Type II - Cytotroopic IgE
3) Type III - Complement.
4) Type IV - Delayed type

Type I, II, III are immediate type
Type IV is Delayed type hypersensitivity

Anaphylaxis

- When a sensitized individual comes in contact with the shocking dose.
- shocking dose can be given in any route.
- Parenteral is more preffered.
- 2-3 weeks is required when a sentisized individual comes in contact with shocking dose.
- Cytotropic IgE antibody are formed against antigen eg binds to the receptor on the mast cells.
- They bind to the Fc portions of the antibody.
- When a shocking dose of the same or immunologically related is given. They bind to the receptors of the mast cells.
- Antigen antibody complex is formed and causes release of chemical mediators
- There are two types of chemical mediators :-

1) Primary mediators :-

a) Histamine :

vasodilation, contraction of smooth muscle

Vasoconstriction, contraction of smooth muscles

2) Secondary mediators :-

a) Platelet aggregating factor :- platelets are released more and aggregate to form haemostatic plug formation.

b) Eosinophil chemotactic factor :-

They are more potent bronchoconstrictors than Histamine

c) Prostaglandin & thromboxane :-

Types of Anaphylaxis :-

- There are two types of Anaphylaxis :-

3/2/21

1) Shulz Dale phenomenon :-

- Ionised tissues from organs are placed in ringer solution
- when erythrocyte is added it causes organ contracts vigously.
- This phenomenon is called Shulz Dale phenomenon

2) Cutaneous Anaphylaxis :-

- When a shocking dose injected intradermally to sensitized individual after 24-72 hrs a

flare and wheal is produced at the site of injection.

3. Cultivation of virus:-

- Virus cannot be grown in culture medium. There are three types of virus inoculation:-
  1) Animal inoculation
  2) Embryonated egg inoculation
  3) Tissue culture

1) Animal inoculation:-

- They are inoculated for:- primary isolation of virus, study oncogenis of virus.
- Mouse is inoculated for arbovirus & coxsackie virus.
- Animal inoculation can be done in any route.
- intraperitoneal, peridontial
- Animal are inoculated and studied for reaction disease and death.
- Animals are sacrificed after the test.
- Pigs, rabbit, mouse are animals that are used for animal inoculation.

2) Embryonated egg inoculation:-

- Hens egg are used to inoculate in

a) Chorioallantoic membrane - Pocks are produced in the membrane. Each virus produced different pocks

b) Allantoic cavity :- Influenza virus is used for vaccine production. chick vaccine.

c) Allantoic sac :- Influenza virus is used for the isolation & inoculation of virus.

d) Yolk sac :-

It is used for the growth, inoculation of virus.

Tissue culture :

There are type :- 1) Tissue culture
2) Organ culture

1. primary cell culture :- a) The freshly prepared organ tissue are taken from humans or animals and subjected

b) They can be grown only for a short period

c) After which they undergo scensure.

2. Diploid cell strain:-

°) They have the same number of Chromosone that of the parent cell.

°) They diploid. in

°) They can be only cultivated upto 50 serves after which they becomee seensces.

3. Continous cell line:-

- They are the first strain.
- They have haploid chromosones.
- they can be also cultivalebe upto a period.

Demonstration of virus

1. Cytopathogenic effect:-

- Most of the organisms undego cytopathogenic morphological changes called as cytopathogenic effects.
- Organism causing cytopathogenic virus is cytopathogi Organism.
- There may be [illegible] of cells [illegible] necrosis & lysis,

- When a haemagglutinating organisms are present addition of erythrocytes can causes agglutination to them organisms & adsorption occurs this is called haemadsorption.

3. Electron microscope :-

- Tissue cells are examined under electronmicroscope.

4. Tumour cells :-

- Tumour cells are formed & piled up to form Tumour emboli

5. Interferon :-

When a non cytopathogenic virus is present & it can be detected by adding a cytopathogenic virus. This is called interfere & non cytopathogenic virus can be detected inside the cell.

7. K

Short Note

5. CMI

- cell mediated immunity it is not a antibody depended reaction.
- It activates the B & T lymphocytes to activate the immune response.
- Role of CMI :-
  - It is used in transplantation & organ transplantation
  - It is effective in delayed type hypersensitivity
  - It is used by the intracellular organisms. facultative anaerobes and both CMI & HMI

Primary response :-

- It occurs when the foreign antigen enters the body for the first time
- Two types of cells are present helper T cells (TH cells) & Cytotoxic T cells. (Tc cells)
- TH cells acts on the antigen with M.H.C class II molecules and kills the microorganism
- Tc cells acts on the antigen with M.H.C class II molecules and kills the microorganism

Secondary response

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if it is from lymphocytes then lymphokines & from macrophages then macrocyte

6.

- Type 4 is the delayed type hypersensity
- It is mediated by T lymphocytes with macrophages lymphocytes.
- It occurs after 24-72 hrs after the infection.
- There are two type of :- 1) Tuberculin type,
  2) Cutaneous skin reaction

a) Tuberculin type :-

- when a shocking dose of tuberculin is injected to the sensitized individual of tuberculin. Injected intradermally. then after 24-72 hrs later the injected site will have wheel & flare reactions.
- The injected place will have more monocytes & lymphocyte.
- Purified protein derivative are present.
- ~~Delayed type tuberculin test~~ can be used to detect tuberculin.
- Several skin test are employed - freig test, lepromin test.

b) Cutaneous skin reaction.

- When skin comes into contact with metals such as Ni and causes reaction to skin, chemicals dies, etc.
- These act as a hapten & enters the skin and reacts with the skin protein to bring out reactions.

7. Kala Azar.

- It is caused by Leishmaniasis.
- It has two life forms :- promastigote & Amastigote.

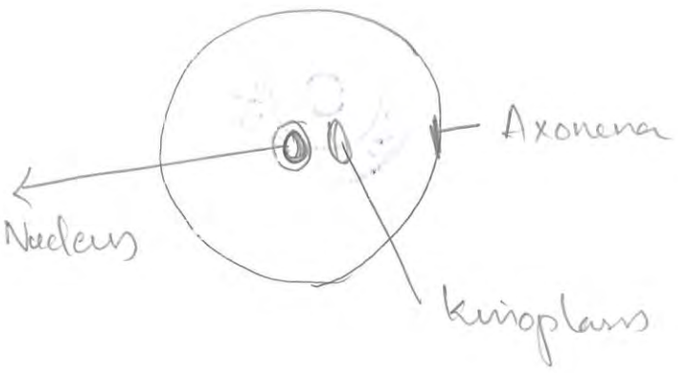
Promastigote occurs in man
Amastigote occurs in sandfly.

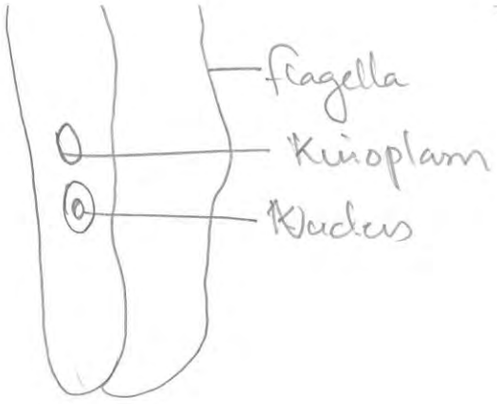
- ~~P~~Amastigote

- It occurs in man. It is aflagellar type.
- Nucleus is located in centre. Kinetoplasm is located near to the nucleus.
- Axonema close to periphery
- oval or rounded shape.

Promastigote.

- It occurs in sandfly after the blood meal
- It is flagellar type; spindle shaped.
- Nucleus is located in ~~front~~ centre. Kinoplasm is located next to nucleus. Axonema is the flagella which is the length of the body.





Clinical features

- Splenomegaly.
- Febrile Aneurysm.
- Anaemia.

Lab diagnosis

- Direct cell culture
  - Biopsy - brain biopsy
  - Culture media - NNN media
  - Direct blood smear
- Indirect cell culture
  - Electronicroscopy - Aldehyde test
  - ELISA
  - RIA

8. Recent viral outbreaks.

a) Severe Acute respiratory distress syndrome (SARS)

- SSRNA
- Occured in 2003

- MOT - close contact, human to human contact; animal.
- Agents - Bats.
- Symptoms
  - fever, chills.
  - diarrhea.
  - Shortness of breath.
  - Acute respiratory problems.
- Prophylaxis
  - No proper treatment protocol
  - No preventive measures.
  - Available antivirals + antibiotics
  - Vaccine under research
  - proper isolation & preventive measures to be taken

b) Novel corona virus.

- 2019 - first occurence in Wuhan china
- SSRNA
- MOT - droplet, close contact, human to human.
- Agents - Bats.

Symptoms

- fever, headache
- body pain
- Diarrhea
- Shortness of breath
- Respiratory problems
- pneumonia.

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- No proper treatment protocol
- Vaccine is under trial
- Available antiviral & antibiotics
- Proper isolation.

c) MERS - Middle east Respiratory syndrone.

- 2012 - occured

MOT - droplets, close contact, human to human

Symptoms

- Acute respiratory problems
- shortness of breath
- fever, chills

Prophylaxis

- No proper treatment protocol
- Vaccine is ~~under trial~~ under research.
- Available antiviral & antibiotic
- proper isolation.

d) Ebola virus :-

- MOT - from water & contaminated food.
when there is a close contact with animals or human infected like blood, body fluids, & dead Ebola patients.
- Symptoms
  - fever
  - chills

- Internal bleeding.
- White blood cells count decreased.
- Seizeures.

Prophylaxis

- No proper treatment protocol
- Vaccine under research
- Available antiviral & antibiotics
- proper isolation.

e) ~~Nosovirus~~ Omicron

- Varient of coronavirus.
- highly infectious.

Symptoms - Nausea, pneumonia
Diarrea
Vomiting
Fever

f) Nipah virus.

Agents - bats.

MOT - close contact.

Symptoms

- fever
- chills
- pneumonia
- Seizures
- coma.

Prophylaxis

- No proper treatment protocol
- Vaccine under research
- Available antiviral & antibiotics
- proper isolation.

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- fever
- chills
- headache
- diarrhea
- Nausea
- Vomiting

4. Vaciparum Malaria

- It has two stages of life cycle ♂ Man & ♀ Anopheles mosquito

Asexual cycle in Man

Sexual cycle in Anopheles mosquito

There are 4 stages :-

Pre erythrocytic stage

Erythrocytic stage

Gametogony

Rest stage

Preerythrocytic stage :-

- Sporozoites present in the hepatocytes and divides and grows into merozoits after 10-12 divisions.
- Merozoitis enters into cell division & converted to merozoites.
- Erythrocytic stage.

Merozits enters and ~~division~~ undergo division.

Gametogony

- Some of them grows into microgametes & macrogametes which are the microgametocytes & macrogametocytes.
- females are macrogametocytes and has a larger round oval them male & bright blue cytoplasm & nucleus.
- Male are microgametocytes & smaller nucleus & light blue cytoplasm.
- Rest stage
- Some of them don't grow into gametes & enters into resting stage. Later after 2 years these get activated & form malaria.

Mosquito cycle

- ~~When the~~ macrogametes - one oocyte is fertilized by the microgametes which are two. fertilization occurs & results in formation of ovum, to oocyte to ookniete transferred in midgut of mosquito
- They are placed in the salivary glands of the mosquito. & released to man after the bite.

1) Thrombosis

53/4+20

> Obstructive nature of circulatory disturbance.

> Process of formation of solid mass from the circulation that is present in the circulation. is called Thrombosis.

> Mass formed is called thrombus.

2

25.3/4

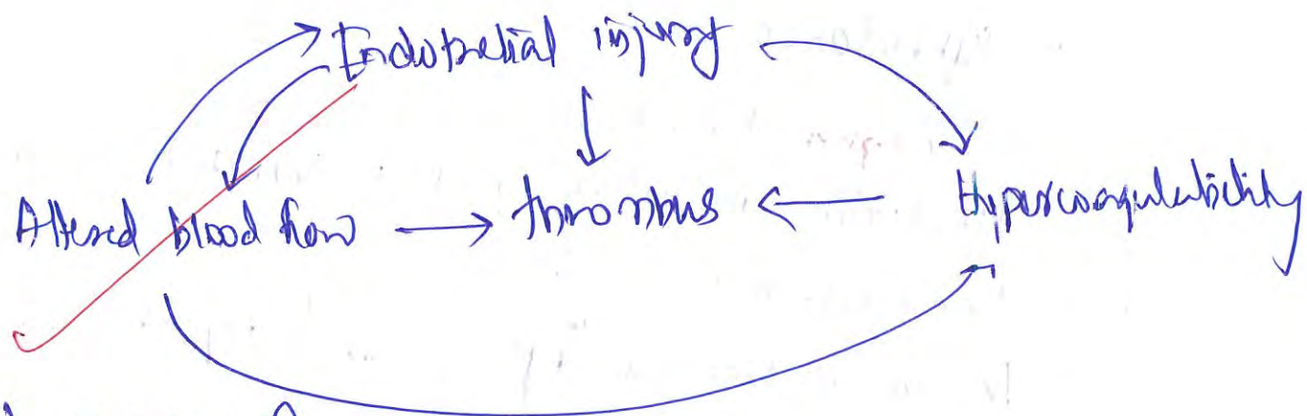
35

Ethiopathology

Events leading to formation of thrombus:

1) Endothelial injury.
2) Altered blood flow.
3) Hypercoagulability.
4) Activation of platelets.
5) Activation of coagulation system.

Virchow's Triad: 3 primary events predisposing to thrombus formation.



(1) Endothelial injury.

> Intact endothelium maintain normal blood flow by following functions:

(a) Subendothelium injury.

Subendothelial extracellular matrix promotes thrombogenesis.

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> Thrombomodulin.
> Tissue plasminogen activator
> Inhibitors of tissue aggregation.

(c) Release prothrombotic factor.
> Thromboplastin
> Von-willebrand factor.
> Inhibit tissue plasminogen activator
> ~~Prog~~ ~~Act~~ Tissue activator.

Factors leading to vascular injury and predisposing to thrombus formation:
⇒ Endocardial injury.
⇒ Ulcerative plaques of atherosclerosis
⇒ Diabetic vasculitis.
⇒ Shock.
⇒ Exogenous chemicals (smoking)
⇒ Endogenous chemicals (hypercholesterolemia).

(2) Altered blood flow.
(a) Turbulance.
> Unequal blood flow.
> Blood cells including platelets migrates to the periphery (close to endothelium.
> Causes endothelial injury and deposits like fibrin and platelets.
> Initiates arterial and cardiac thrombi.

(b) Stasis.
> Slowing of blood flow.
> Allows higher release of O_2 from the blood.
> Inhibits inflow, diffusion of activated clotting factors.
> Initiates venous thrombi.

(3) Hypercoagulability or thrombophilia.

> Group of conditions causing increased risk of developing ~~clot~~ venous thrombi.

> 2 types:

(a) Primary (hereditary)

(b) Acquired (secondary).

(a) Primary factor.

> Deficiency of Antithrombin III.

> Deficiency of protein S.

> Deficiency of protein C.

> Mutation of factor V leiden.

> Increased level of coagulation factor.

> defect in fibrinolytics.

(b) Acquired factors.

(i) Risk factors.

> ~~Protein~~ Advancing age.

> Prolonged bedrest.

> Prolonged immobilisation.

> Obesity

> Smoking.

(ii) Predisposing clinical conditions.

> Heart disease

> Valvular disease (like atherosclerosis).

> Hypercoagulability conditions.

> Shock.

> Tissue damage.

> Late pregnancy.

> Anti cardiolipin antibody

(4) Role of Platelets:

> following endothelium injury, platelet acts an important role in blood haemostasis and as well as thrombus formation

(a) Platelet adhesion

(b) Platelet activation

(c) Platelet aggregation

(5) Role of coagulation system

> Both intrinsic and extrinsic pathway.

Morphology of thrombus:

Gross

> Size and shape depends upon on site of origin.

> Arterial thrombi: White, firm and pale.

> Venous thrombi: Red, soft and gelatinous.

> line of Zahn.

Microscopy:

> line of Zahn in arterial thrombi shows alternate light staining platelet with fibrin mesh layers and dark staining platelet with more red cells.

> venous thrombi: More red cells, lymphocytes and platelet with fibrin mesh.

Fate of thrombi:

(a) Resolution

(b) Organization

(c) Propagation

(d) Thromboembolism

(c) Recanalisation.

> fibrinolytic system dissolves thrombus

> Accentuated by thrombolytic system.

(b) Organisation.

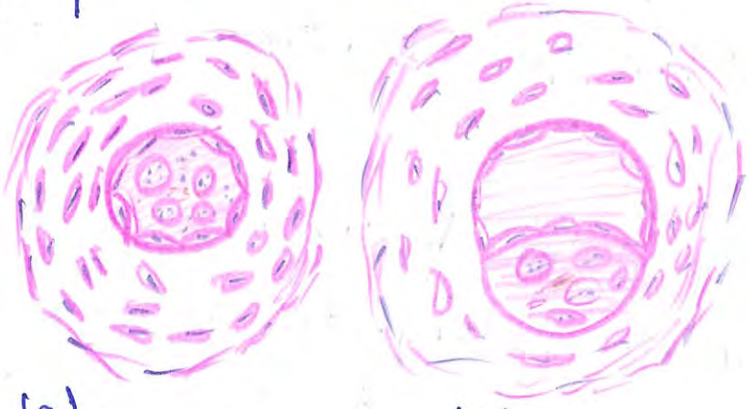
> Phagocytosis of fibrin and cell debris, granulation tissue formation, recanalisation, calcification.

(c) Propagation.

> Increase in size and cause obstruction.

(d) Thromboembolism.

> Detach from the vessel walls and carried to through the bloodstream.



(a) (b)

(c)

(2)

(a) Spread of tumours.

> Cardinal feature of malignant tumours.

2 types: (a) Local invasion. (Direct spread).

(b) Metastasis (Distant spread)

(a) Local invasion.

> Malignant tumours shows infiltration and destruction of adjacent tissues.

> Tumours invade via route of lymphatic

is more resistance to invasion.

eg: giant tumours cells of bone / osteoclastoma

(b) metastasis.

> Metastasis is defined as spread of tumour by local invasion in such a way that secondary discontinous tumour masses are formed at the site of lodgement.

> ~~Commonest~~ Route of metastasis.

(a) Lymphatic spread.

(b) Haemorrhagic spread.

(c) Spread by tissue spaces and natural passages

(a) Lymphatic spread.

> Involvement of lymphnode by malignant cells is more

(i) Lymphatic permeation: lymphatics are readily involved by cancer cells and causes the spread of the tumour cells.

(ii) Lymphatic emboli: Malignant cells detach and form thrombus embolism which is carried to the next draining lymphnodes.

• Regional nodal metastasis: Regional lymphnode draining through invasion by continuity invasion.

eg: Axillary lymphnode

Cervical Lymphnode

• Skip metastasis.

• Retrograde metastasis: Tumour cells spread against the flow of lymph.

(b) Haematogenous spread.

> Tumour cells spread via blood especially veins ~~by~~ because of ~~its~~ its thin wall.

> common site: lungs, liver, bones, kidney, adrenal, brain

> ~~Gross: White to yellowish white, 1-2cm diameter~~

> ~~Cut surface~~:

> Gross: Blood born metastasis of an organ appears as multiple, rounded nodules of varying size and spread along the organs.

> Microscopy: Secondary tumour appears as the structure of primary tumour.

(c) Spread via natural passages and spaces.

I Transcoelomic spread: Metastasis spread along the serosal lining of coelomic and coelomic fluid to varying parts of the body

eg: Carcinoma of stomach spread to ovaries/ ~~cucumber~~ Krukenberg tumours.

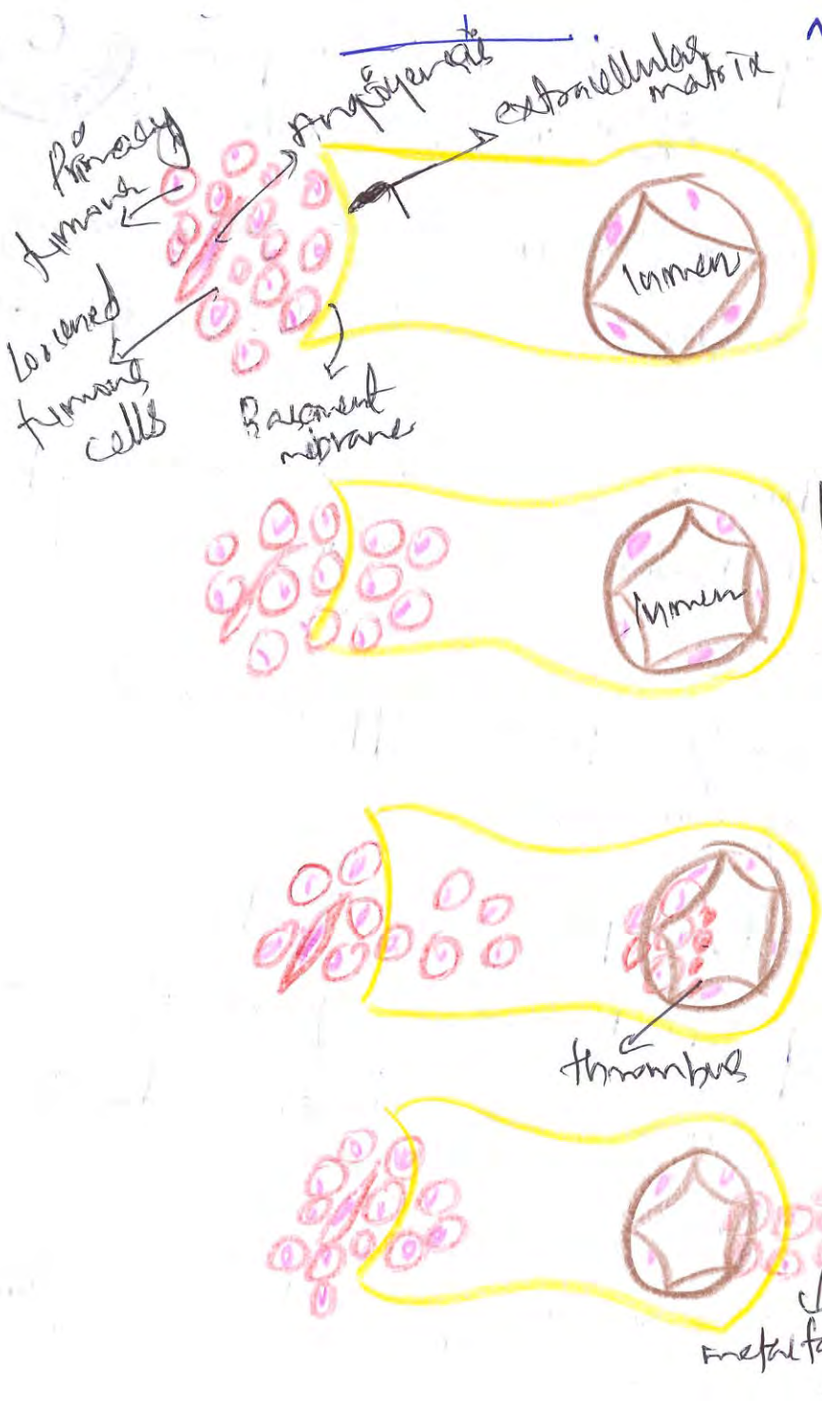
Carcinoma of ~~the~~ bronchus and breast spreads to pleural and peritoneal cavity.

II Spread via epithelial lined surface.

eg: cancer of ovaries and endometrium spread to ~~the~~ fallopian tube

III spread via cerebrospinal fluid eg: brain tumours.

IV Implantation spread of tumour by implantation by surgeon's ~~surgery~~ sutures, needle or very rare



(1) Aggressive clone with angiogenesis, loosening of tumour cells

(2) Invasion into extracellular matrix, destruction of extracellular matrix

(3) Thrombus formation, entry to lumen

(4) Extravasation from lumen, metastasis tumour formation

(3) Atherosclerosis.

> Atherosclerosis is thickening and hardening of large and ~~small~~ medium sized muscular arteries, primarily due to the involvement of tunica intima and is characterised by fibrofatty Plaques or atheromas.

> Commonest and most important arterial disease.

> Common site: aorta, coronary and cerebral arteries

Main clinical manifestations (due to ischemia),

(a) Heart: Angina, MI.

(b) Brain : transient cerebral ischaemic
chronic ischaemic heart disease
(c) Others : peripheral vascular diseases, ---

Etiology.

(A) Major risk factors.
(a) modifiable.
(i) Dyslipidaemia (hypercholesterolemia).
(ii) hypertension.
(iii) Diabetes.
(iv) Smoking.
(b) Constitutional.
(i) Age
(ii) Sex
(iii) Genetic factors.
(iv) Familial and Racial factors.
(B) Emerging risk factors.
(i) Environmental risk factors.
(ii) Obesity.
(iii) hormones.
(iv) Physical inactivity
(v) Stressful life.
(vi) hyperhomocysteinemia.
(vii) homocysteinuria
(viii) Role of alcohol.
(ix) CRP.
(x) Infections.

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(i) Dyslipidaemia.

> Hypercholesterolemia is directly proportionate relationship with atherosclerosis and Ischemic heart diseases.

(a) Atherosclerotic plaque contains Cholesterol and cholesterol esters.

(b) Experimental animals can be induced by atherosclerosis by cholesterol rich diet.

(c) Individuals with hypercholesterolemia can cause with other diseases, which has increased risk of developing atherosclerosis and IHD.

(d) High mortality rate with atherosclerosis

> Classes of lipid: low density lipoprotein (very low density lipoprotein, high density lipoprotein, chylomicrons.

> Lipoproteins are circulated in blood and crosses cell membrane and carried by carrier proteins called apoproteins.

Blood lipid profile:

(i) Normal cholesterol: 140-199 mg/dL, 200-240 mg/dL (borderline), >240 mg/dL (risk of IHD).

(ii) Triglycerides: <150 mg/dL.

(iii) LDL: very dangerous, rich cholesterol level.

(iv) VLDL: rich with triglycerides, but not much as LDL.

(v) HDL: good cholesterol, protect atherosclerosis

> dyslipidemia can be maintained by lowering LDL, HDL and normal cholesterol level by exercise.

ii) Smoking.

> Extend and severity of smoking is danger

iii) Hypertension.

> Risk factor of all clinical manifestation of atherosclerosis

> mechanical injury to cell by increased BP.

(iv) Diabetic mellitus.

> Atherosclerosis is very common and appears in the early stage.

> Type 2DM is due to metabolic syndrome, dyslipidaemia

> Risk of IHD, gangrene foot is high

Constitutional factors.

a) Age: • Atherosclerosis is age related disease
• Appears in the early stage.

b) Sex: More common to men than female

c) Genetic factors: Used to run in a family.

d) Racial factors: More in whites than blacks.

(B) Emerging risk factors.

(i) Environmental risk factors.

> More in developed countries.

ii) Exogenous hormones and endogenous estrogen

> fully developed atherosclerotic lesion.
> fibrous plaque, fibrofatty plaque, atheromas.
> Site: abdominal aorta (most commonly), thoracic aorta, descending ~~thrombi~~ aorta.

> Gross:
- White to yellowish white, 1-2 cm diameter, raised on surface.
- Cut surface: luminal surface appears as firm, white fibrous cap, and central core composed by ~~extract~~ yellow to yellowish white soft plumped structure.

⇒ Microscopy:
> fibrous cap composed of smooth muscle cells, dense connective tissue and extracellular matrix covered by superficially by endothelium
> Cellular area under fibrous cap composed of macrophages, foam cells, lymphocytes, smooth muscle cells
> Deeper central core: composed of necrotic debris, lipids, cholesterol clefts, lipid laden foam cells.
> In older and advanced cells, these become hyaline.

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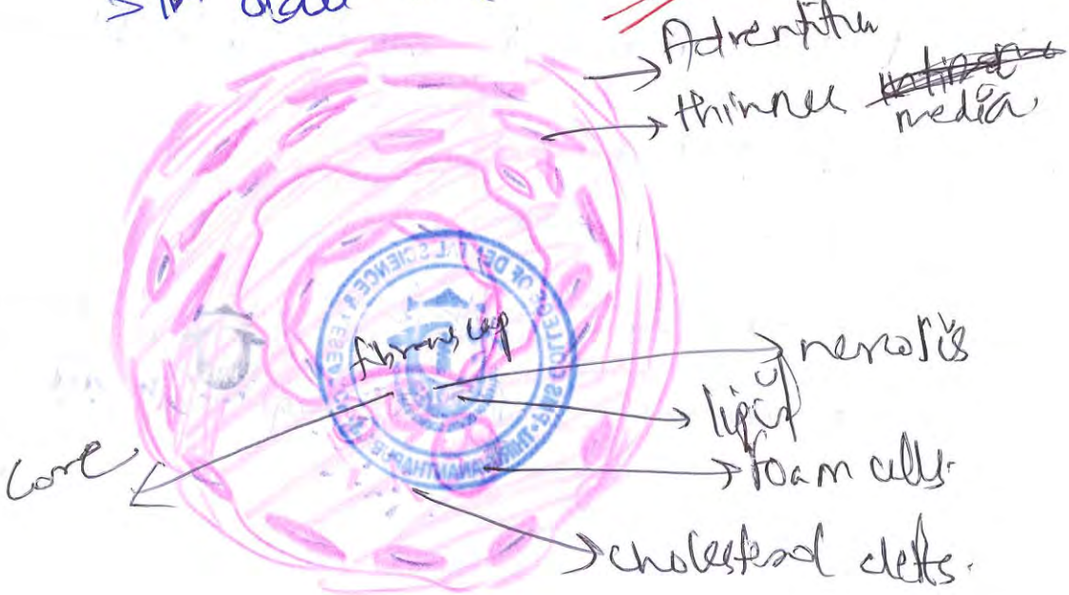


4) Type II HS.

> Type II hypersensitivity reaction or Cytotoxic reaction.

> Appears 15-30 minutes after exposure.

> Humoral Antibody attacks cell surface antigen and ~~ours~~ lysis of target cells.

> ~~Ours~~ occurs against normal cells or antigens and exogenous antigens.

Etiology and Pathogenesis.

> ~~Type~~ Type II HS is tissue specific and Antibody only binds ~~the~~ the Antigen specific tissue.

(a) Antigen on the specific site is attacked and binds to Fc region of Antibody (IgG or IgM) and causes antigen-Antibody complex

(b) Unreacted (unattached Fc region forms link between Antigen and complement.

(c) Activation of classical pathway of complement generating C3b.

(d) The C3b act as opsonin and phagocytes.

(e) The Antigen-antibody not only attacks cells but also destroy MAC in the cells.

Examples:

(a) Cytotoxic Antibodies to blood cells.

> Autoimmune hemolytic anaemia.

> Transfusion reaction.

(b) Cytotoxic Antibodies to tissue components:
> Grave's disease
> Myasthenia gravis.
> Male sterility.
> Type 2 DM
> Hood pasture syndrome.

3

(5) Pleomorphic adenoma (mixed salivary gland tumour)
> Most common tumour of salivary gland.
> Also called mixed salivary tumour because of appearance of ~~mixed~~ mixture of different histological components.
Site: located below and in front of ear, slow growing painless.
. Most commonly seen in middle aged womens.
> Gross: . Pseudocapsulated
. Round, at times multilobulated.
. 2-5 cm diameter
- Bosselated surface.
> Cut surface: Greyish white
variegated.
Soft and mucoid appearance
Admixture.
Cartilaginous (blue) and Glandular

Osseous seen

Microscopy:

> Mixed appearance: Tumour is composed of ductal epithelial cells, myxoid, cartilaginous, connective tissue & types:

(a) Ductal epithelial cells.

(b) Myoepithelial cells.

(a) Ductal epithelial cells is lined by columnar or cuboidal cells.

Composed of ~~sheet~~ acini, ducts, tubules ...

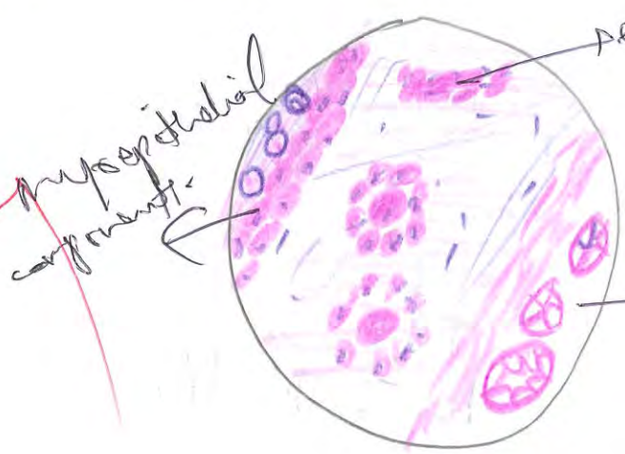
(b) Myoepithelial cells: Composed of sheets, nests, whorls

Prognosis

> Recurrent.

> Compresses the facial nerve

> ~~Invades~~ change to carcinomatous.



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6) Hypertension

> Sustained resting blood pressure above 140/90 mmHg is called hypertension.

Complications of hypertension

(a) Blood vessels ... the CS ... diseases

(d) Eyes : Hypertensive retinopathy.

(e) Nervous System: Stroke.

(a) Hypertensive atherosclerosis.

> Affect arteries.

Includes a) Hyaline arteriosclerosis

b) Hyperplastic arteriosclerosis

c) Necrotising arteriolitis.

(b) Hypertensive Heart diseases.

> manifests as left ventricular hypertrophy.

> Most common heart disease after IHD.

c) Nephrosclerosis

i) Benign:

> kidney is small and contracted.

ii) Malignant:

> Kidney is enlarged.

> Cortex shows flea bitten kidney due to tiny petechial haemorrhages.

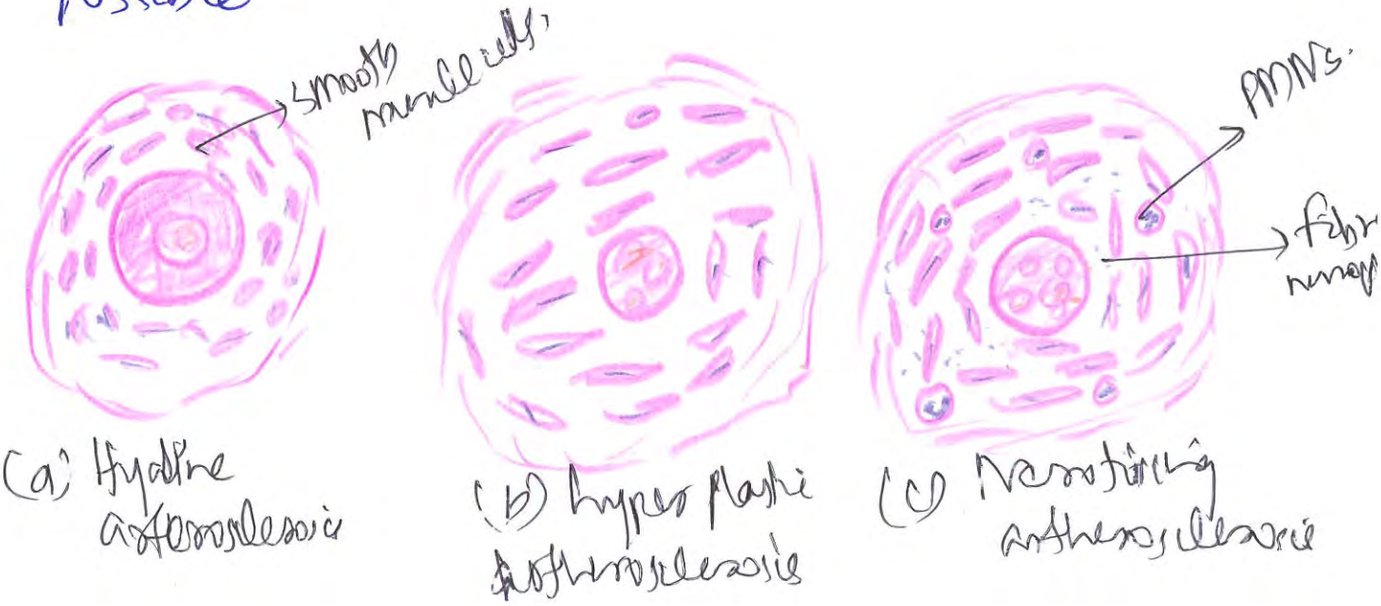
(d) Stroke:

> Intracerebral haemorrhage: Usually of hypertensive origin

> Subarachnoid haemorrhage: Usually of aneurysmal origin

(e) Hypertensive retinopathy.

> Retinal capillary narrowed. So vision not possible.



(a) Hyaline atherosclerosis (b) Hyperplastic atherosclerosis (c) Necrotizing atherosclerosis

(f) Packed cell volume.

> It is the ratio of volume of blood cells to the total blood cells.

> PCV is used for estimation of blood count

> Methods.

a) Wintergen's method.

PCV increases in case of: Anaemia
children.

PCV decreases in case of: pregnancy.

(g) Infective endocarditis.

> It is a very infectious disease

To be fillled by the Candidate

Please (✓)Tick Answered Question Numbers in appropriate boxes

Q.No.	ANSWER	Q.No.	ANSWER	Q.No.	ANSWER	Q.No.	ANSWER	Q.No.	ANSWER	Q.No.	ANSWER
1.		6.		11		16		21		26	
2.		7.		12		17		22		27	
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SUBTOTALS											

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GOLDEN HILLS, VATTAPPARA, TRIVANDRUM

II BDS KUHS Regular Batch (New Scheme)

REMEDIAL EXAMINATION DECEMBER 2021

MICROBIOLOGY

01/12/21

- *Answer all questions*
- *Use diagrams and flowcharts wherever necessary.*

Time: 1½ hours Marks : 35 Essay

1. Explain the mode of transmission ,pathogenesis, clinical manifestations, lab diagnosis and prophylaxis of HBV.

(1x10=10 Marks)

Short Essay

2. Define Hypersensitivity. Type's of Hypersensitivity. Briefly explain Anaphylaxis
3. Cultivation of Virus

(2x5=10 Marks)

Short Notes

1. Falciparum Malaria
2. CMI
3. Delayed type Hypersensitivity
4. Kala Azar
5. Briefly describe recent viral outbreaks

(5x3=15Marks)

GENERAL PATHOLOGY

Time: 1½ hours Max Marks: 35

Essay: (10)

1. Define thrombosis. Describe etiopathogenesis of thrombosis. Mention fate of thrombus. (2+6+2)

Short Essay: (5X2)

2. Describe the modes of spread of malignant tumors with examples. (5)
3. Mention the risk factors of atherosclerosis. Describe the microscopy of atheromatous plaque. (3+2)

Short notes: (3X5)

4. Type II hypersensitivity.
5. Pleomorphic adenoma.
6. Infective endocarditis.
7. Complications of hypertension.
8. Write a note on PCV.

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(7) DESENSITIZING AGENTS.

Desensitizing agents are the substances used in the treatment of hypersensitivity.

(i) Stontium chloride

Stontium chloride react with potassium and forms stontium phosphate.

· It will blocks the dentinal tubules.

(ii) potassium nitrate.

It helps in crystalisation and remineralisation.

(iii) Sodium monofluorophosphate.

It also helps in crystallisation and remineralisation.

(iv) potassium oxalate.

potassium oxalate reacts with the calcium phosphate in hydroxyapetite and forms calcium oxalate which will blocks the dentinal tubules.

Commercially available densensitizing agents.

Eg:- Sensodyne :- 10% Stontium chloride + Sodium monofluoro phosphate.

Thermodent

promise.

(9) VERTICAL BONE DEFECTS.

It is a type of bone defects in which bone loss occurs in obliquely. Also known as angular defects. The base of the defect is apical to the surrounding bone.

Goldmann classified vertical bone defects into

(i) One walled defect
(ii) Two walled defect
(iii) Three walled defect.

(i) Three walled defect

- Only one wall is lost ; 3 walls are intact.
- Has good prognosis.
- Can be managed with regenerative therapy.

(ii) Two walled defect.

- Two walls are lost and other two walls are intact.
- Can be managed with either resective or regenerative therapy

(iii) One walled defect.

- Only one wall is intact
- Poor prognosis.

Circumferential defect:

When the defects are continuous around the tooth surface it is called circumferential defect.

(10) ANTIOXIDANTS.

Antioxidants are substance that will inhibit the oxidative processess by tapping the oxygen species.

It action takes by

(i) by preventing the free radical initiation and progression
(ii) by eliminating the reactive oxygen species.
(iii) by forming chelates with the transition metals.

2. A large amount of reactive oxygen species (ROS) is formed in the body during physical function. These oxygen species are dangerous to integrity of the structural components of cells

- These ROS can damage cells and DNA and can produce several disease
- An increase in ROS can cause periodontal diseases by destructing the MMP's and other structures present in the periodontium.

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(11) RADIUS OF ACTION.

- Page and Schroeder on the basis of Waerhaugs study stated that the bone destruction occurs in 1.5 - 2.5 mm on presence of plaque.
- Beyond 2.5 mm there is no action since it produces angular defects which is not seen as it is narrower and end up in horizontal bone loss.
- Above 2.5 mm bone loss is in Local juvenile periodontitis, and papillon Lavefere disease.

(12) STRUCTURE OF BIOFILM.

- Biofilm is defined as a structural entity in which microbes are embedded in an extracellular matrix.
- Dental plaque consists of over 500 species of microbes.
- Around 150 species of microbes is present in each individual
- 1g of plaque contain around 10^{11} bacteria.
- The intracellular matrix is made of organic and inorganic component
- Organic component consists of polysaccharides, proteins and lipids
- Inorganic component contains Calcium, Sodium, Flouride, potassium, Magnesium

- In supragingival plaque inorganic component is obtained from saliva and in subgingival plaque it is obtained from GCF.

- Dental plaque
  - Supragingival plaque
    - Coronal plaque
    - Marginal plaque
  - Subgingival plaque
    - Tooth associated plaque
    - Unattached plaque
    - Tissue associated plaque.

Supragingival plaque:

Tooth surface :- Gram positive cocci, small rods

Outer surface :- Gram negative rods, filaments, spirochetes.

Tooth associated plaque:

contains gram positive rods and filaments.

Unattached plaque:

Contains gram negative rods, filaments.

Tissue associated plaque:

Contains gram negative anaerobes bacteria.

(13) COMPOSITION OF GCF.

Gingival crevicular fluid is ~~seen~~ in the gingival sulcus that sweeps from the epithelium.

It is an inflammatory exudate.

Composition

→ Cellular elements
- Neutrophils
- Leukocytes
- Epithelial cells.

→ Electrolytes
- Sodium
- potassium
- Calcium

→ Organic substance.
- Carbohydrate
- Lipid
- protein
  - Immunoglobulins
  - Complements.

→ Metbolic and Bacterial products
- urea
- Endotoxin
- Lactic acid
- Antifactive factor
- Cytotoxic factor.

→ Enzyme and enzyme inhibitors

- Alkaline phosphatase
- Acid phosphatase
- Lactic dehydrogenase
- Hyaluronidase
- Collagenase.
- Lysosyme.

(14) LIPPING.

- In the stage of Repair of tissue damage caused by trauma from occlusion, new bony trabeculae is incorporated into the thin alveolar bone. This process is known as buttressing bone formation.
- Two types of buttressing bone formation -
  - Central buttressing bone formation - bone formation with in the jaw
  - peripheral buttressing bone formation - bore formation from lingual and buccal alveolar plates.
- In peripheral buttressing bone formation there is thickening of facial and lingual alveolar bone margins it is called Lipping

15 PREGNANCY GINGIVITIS.

Gingival inflammation occuring in the period of pregnancy.

Etiology

- Due to increase in progesterone and estrogen and their metabolits
- These hormones causes increase in vasculature and there is increased permeability of blood vessels which leads to gingival inflammation.

Course and duration

- It starts in 2nd or 3rd month of pregnancy and will become severe in 8 month
- This will subside spontaneously in the last months of pregnancy or after parturition.

Clinical features

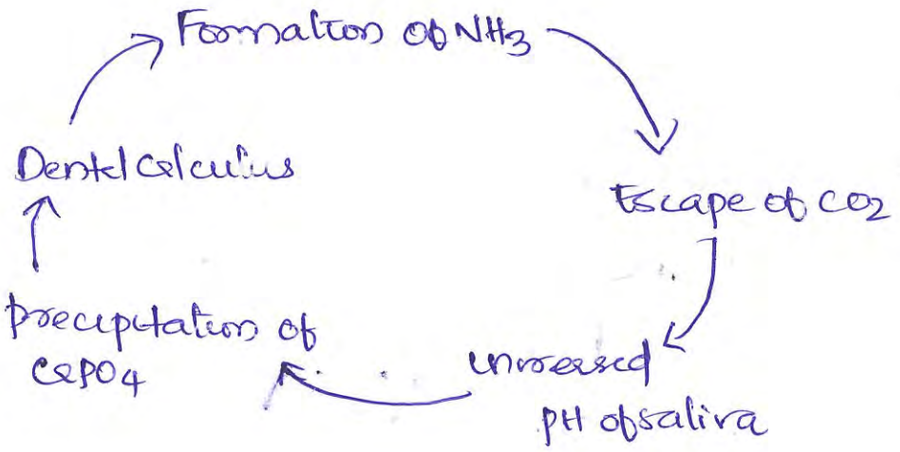
- Severe inflammation of the gingiva
- Gingiva become reddish blue in colour
- Oedematous and rolled out interdental gingiva.
- Spontaneous bleeding.
- Painful pain
- It will get localised to form a mass - pregnancy epulis

Management.

- Preventive regimens are used
- Antibacterial therapy and scaling.

(16) Theories of calculus formation.

(1) Boosta theory



(2) Seeding theory

A matrix of dental plaque - Seeding agent.

↓

formation of small foci of calcifications

↓

enlargement and coalescence of this foci

↓

Dental calculus formation.

(3) Inhibition theory.

Control mechanism - pyrophosphate.

This pyrophosphate is formed from phosphate which reacts with calcium and form dental calculus.

Brushite is transformed to more stable structure like sodium octaphosphate and there by dental calculus is formed.

(5) Enzymatic theory.

The enzymes present in the dental plaque ~~precipitates~~ precipitates ~~Calcium~~ phosphate ions which reacts with calcium to form calcium phosphate - dental calculus is formed.

(8) SLING SUTURES.

- Most common type of suture used in periodontal flap surgery
- Needle is entered from one side and it is encircled around the teeth and the needle is removed through other side
- Two types
  - Simple interceptal
  - Contenuos independant

(1) Aggressive periodontitis

Classification of periodontitis

periodontitis is classified into

1. chronic periodontitis
2. Aggressive periodontitis
   - localised aggressive periodontitis
   - generalised aggressive periodontitis.

Aggressive periodontitis

In aggressive periodontitis there is

(i) Rapid loss of periodonticem and tooth attachment

(ii) Occurs in otherwise healthy individual
(ie there is no other systemic disease that can contribute to periodontal disease)

(iii) positive familial aggregation.

Etiology

- Role of microorganisms

Aggregatibacter actinomycetemcomitans is involved in the aggressive periodontitis

- JP2 strain causes AP.

- Role of immune cells.

It is due to the action of NK cells

- Role of genetics:
  - There is positive familial aggregation
  - Single nucleotide polymorphism.

clinical features

- Aggressive periodontitis usually occurs in young age (less than 35 yrs of age)
- Aggressive periodontitis leads to rapid and severe destruction periodontium leading to loss of teeth.
- It is of 2 types
  - Localised aggressive periodontitis
  - generalised aggressive periodontitis

→ Localised aggressive periodontitis

- Age :- pubertal.
- There is destruction of periodontal structure around incisor and 1st molar.

  There is increased loss of attachment in 2 or more teeth in which one tooth is molar.
- Also known as localised juvenile periodontitis
- strong familial aggregation

Radiographic features.

- severe bone loss in relation to incisor and molars.

→ Generalised aggressive periodontitis

- Age of occurence :- Less than 30 years.
- There is destruction of periodontal structure around 3 or more teeth other than molars and incisors.

- And there is gradual loosening of the teeth.

Radiographic features

- Vertical bone loss is seen
- Classic arc shaped bone loss is seen in generalised AP

Management:

I. Conventional therapy

Conventional therapy include patient education, oral hygiene measures, supra and subgingival scaling and root planning. If it is not subsided by these treatment procedures; surgical therapy is used.

Surgical therapy include:

(i) Regenerative surgery

which include the use of bone graft and regeneration

(ii) Conventional surgery

periodontal flap.

I. Antibiotic therapy

Usage of Tetracycline

- Tetracycline 250 mg four times daily for 1 week.
- Alternatively Doxycycline 100mg can also be used.
- For tetracycline resistant cases:- Amoxycillin + Metronidazole is given.

- This procedure include scaling and root planing (complete removal of plaque and calculus) in 2 appointments with 24hours.
- Brushing the tongue with 1% Chlorhexidine gel for 1minute
- Rinsing the mouth with 0.2% Chlorhexidine solution for 2 minutes
- Irrigating the periodontal pockets with 1% Chlorhexidine solution.

(!!) Host modulation therapy

- periostat 20 mg is used.

If the condition is not improved extraction ~~addition~~ of the loose teeth.

(2) Flap Surgery.

Indications of flap surgery.

- persistence of moderate to deep pocket even after thorough scaling and root planning.
- As a root coverage procedure in recession.
- To increase the width of attached gingiva.
- ~~To place graft (Laterally and GT.~~
- To place graft and in GTR procedure flap is elevated.
- Gingival overgrowth
- Crown lengthening and other restorative procedure.
- To eliminate pocket distal to third molar.

Apically displaced flap.

Indications :-

(i) pocket eradication

(ii) To increase the width of attached gingiva.

Procedure - It can be full thickness flap or partial thickness flap.

Contraindications

- poor oral hygiene status.
- systemic diseases like diabetes mellitis etc.
- Severe bone loss
- High caries activity.

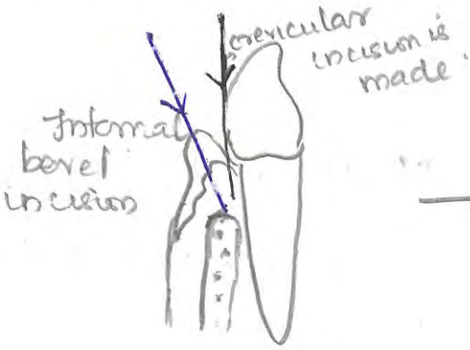
procedure -

- Under local anaesthesia, Intoral bevel incision is made 2mm away from the gingival margin to the crest of alveolar bone.
- Crevicular incision is made;
- flap is reflected
- Interdental incision is given and wedge of tissue is removed.

scaling

- Vertical incision is made as it is displaced from the original position.
- Scaling and complete debridement of plaque and calculus
- Removal of granulation tissue

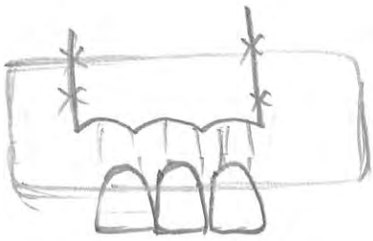
- Find the area is covered with a periodontal pack.



The wedge of tissue is removed after placing an intradental incision

This wedge of tissue contain the granulation tissue & soft tissue wall of pocket

→ vertical incision is given for the apical displacement of flap.



→ Sutures are placed and the area is covered with periodontal pack.

Advantages.

- There is complete eradication of periodontal pocket
- Width of attached gingiva can also be increased.

Disadvantages.

- chance for recession after flap surgery
- Hypersensitively can be increased.
- Not used in aesthetic areas.

(3) Tissue response to TFO.

When the occlusal forces exceed the adaptive capacity of periodontium it leads to trauma from occlusion.

TFO is of 2 types

(i) primary trauma from occlusion.

(ii) secondary trauma from occlusion.

Tissue response to TFO include 3 stages

(i) Injury

(ii) Repair

(iii) Remodelling of periodontium.

(i) Injury.

- When the occlusal forces exceed the adaptive capacity it leads to traumatized occlusion thereby injury to periodontium
- When occlusal force is applied tooth rotates; and the axis of rotation in single rooted teeth is between middle and apical third of root. In multirooted teeth it is located in the middle of interdental bone.
- During optimum force, On pressure side there is compression of PDL and resorption of bone and in tension side there is elongation of PDL and bone apposition

- On severe force on the pressure side the PDL is compressed in large extend, hyalinasation and areas of necrosis is formed and bone resorption occurs.

- On excessive force; on tension side the PDL is elongated and osteoclastic activity is initiated and bone resorption occurs.

- As a result tooth become loose in its socket and can loads its lose of teeth.

- The area where maximum injury occurs is the furcation area.

(ii) Repair

- The periodontium is in constant repair
- The injured periodontium is constantly repaired by the cells present there
- When the deformities cannot keep pace with the repair activity; both get mobile.
- In this stage, as a part of repair, new bony trabecula is incorporated into the thin alveolar bone. This process is known as buttressing bone formation

- Buttressing bone formation is of two types
- central buttressing bone formation - bone formation with in the jaw
- peripheral buttressing bone formation - bone formation from lingual and buccal alveolar plates.

. In peripheral bulbossing bone formation, there is thickening of facial & lingual alveolar bone margins, it is called lipping

(iii) Remodelling

. When the repairing process can't keep pace with the injury remodelling of bone occurs

. There is formation of vertical bone defects without periodontal pocket.

(19) Importance of hand hygiene & barrier technique

. Sterilisation and disinfection is very important in dental practice.

. In an dental office, disease can be easily transmitted from one to another

- from patient to dentist
- from dentist to patient
- from one patient to another
- from dental assistants to patient and vice versa.

Hand hygiene.

. Hand hygiene is important in dental practice

. Hands should be washed thoroughly before and after treating a patient

. Hands should be washed with liquid soaps

and after removal of gloves

- proper handwashing technique should be used.
- Hand should be washed if they were contact in contact with any contaminated surfaces.
- proper gloving technique should be used.
- Gloves should be changed after each patient
- After usage gloves should be disposed safely.
- Only with gloved hand intraoral examination is done; other procedures like impression pouring, x-ray film placement all should be done with gloved hand.
- Hands should be washed if it is visibly soil, contact with any contaminated surface etc.
- Usage of double glove method whenever necessary.

Barrier technique

Personal protection equipment (PPE)

It is used when the procedure include splashing of fluids. It includes

(i) protective clothing
(ii) Gloves
(iii) Masks
(iv) Headcap
(v) Eyewear
(vi) Faceshield.

- PPE is worn in procedures like surgical procedures, restorative procedures, scaling and root planning etc.
- proper wearing of PPE is necessary when the patient is having any blood borne diseases or viral infections like hepatitis, HIV.

(5) Antibiotics and their role in periodontal therapy.

- In periodontal therapy, mechanical therapy is most important
- As an adjunct to ~~pro~~ mechanical therapy, antibiotic therapy is used.
- Antibiotic therapy is not used as an monotherapy.

Common antibiotics used in periodontal therapy.

(i) Penicillins

- Bactericidal
- Broad spectrum of activity with more effective against gram positive beganisms.

Amoxycillin - commonly used antibiotics

Amoxycillin 500 mg thrice daily

Combination of Amoxycillin + clavulanic acid

Augmentin

Augmentin 625 mg twice daily

(iii) clendamycin

active against gram negative organisms.

(iv) Tetracycline

- Bacteriostatic
- Active against gram positive organism.
- Tetracycline is used in addition with scaling and root planning
- If there is chance of periodontal infection
- used in periodontitis

Contraindications

not used in pregnancy period

Adverse drug reactions

- Nausea
- Vomiting
- Abdominal pain
- Stomatitis
- Hypersensitivity reactions.

Dosage

Tetracycline 250 mg four times daily for one week in aggressive periodontitis.

Metronidazole

- Antiamoebic drug
- Active against anaerobic organisms

Mechanism of Action

- Causes the DNA damage → bactericidal.

Uses

- periosonal abscess
- periodontal abscess
- Acute ulcerative necrotizing gingivitis
- Desquamative gingivitis
- Pericoronitis

Other uses

- Brain abscess
- Amoebiasis - Drug of choice
- Giardiasis
- intestine abscess.
- Trichomoniasis

Adverse drug reactions

- Metallic taste
- nausea.
- vomiting
- Disulfiram like reaction with alcohol.

Contraindications

- patients on anticoagulent therapy.
- Alcohol using patients.

Dosage

(A) Desquamative gingivitis :

Desquamative gingivitis is characterized by erythema followed desquamation and ulceration in gingiva.

Classification

- Mild desquamative gingivitis
- Moderate desquamative gingivitis
- Severe desquamative gingivitis.

Disorders associated with desquamative gingivitis

(i) Lichen planus

- Ulcerative lesions
- preceded by ~~vesicle formation~~ papule formation in the gingiva later it rupture and form ulceration
- Stroae of Wickham present

Gingival lesions include

(i) Keratotic lesions

(ii) Ulcerative lesion

(iii) Atrophic lesions.

Management

- Usage of steroids like prednisolone
  Methyl prednisolone.

(II) pemphigoid

(III) pemphigus vulgaris

- vesiculobullous lesion
- Autoantibodies against Dsg 3
- formation of vesicle which rupture and leave crater like ulcerations
- positive Nikolsky's sign
- On direct immunoflourescence → chicken wire appearance

3. Management
- use of steroids.

(IV) Lupus erythematosus
- formation of butterfly shaped rash in the cheek and nasal bridge.

(V) Erythema multiforme
- forms bull's eye lesion

845 J

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Please (✓)Tick Answered Question Numbers in appropriate boxes

Q.No.	ANSWER	Q.No.	ANSWER	Q.No.	ANSWER	Q.No.	ANSWER	Q.No.	ANSWER	Q.No.	ANSWER
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2.	14	7.	1	12	4	17		22		27	
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5	21	10	3	15	8	20		25		30	

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3. **Candidate's Name** : Write your name in BLOCK letters
4. **Exam Date** : Enter date of examination **dd/mm/yy** format.
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PART VII

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PART II

PMS COLLEGE OF DENTAL SCIENCE AND RESEARCH

REMEDIAL EXAMINATION (KUHS) IV BDS PART I REGULAR

ORAL MEDICINE AND RADIOLOGY

MODEL - 01/12/21

3 HOURS MAX MARKS: 70 MARKS

Essays (2X10= 20 marks)

1. Enumerate white lesions of the oral cavity and discuss the clinical features, investigations and management of leukoplakia

2. Describe sialography and its significance in various diseases of salivary glands

Short essays (4X5= 20 marks)

3. Define pain. Discuss the differential diagnosis of chronic unilateral facial pain.

4. Object localization techniques in dental radiology

5. Role of radiography in diagnosis of periodontal disease

6. Discuss the Oral manifestations of HIV and its management

Short Notes (10X3= 30 marks)

7. Myofascial pain dysfunction syndrome

8. Collimation and filtration

9. Schilling test

10. Ludwig's angina

11. Vital tissue staining

12. Role of steroids in oral lesions

13. Importance of medical history

14. Chronic osteomyelitis

15. Topical antifungal drugs

16. Pemphigus vulgaris and its management.

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