Joash
EMPIRE

Pathology Revision Papers.

1 SEPTEMBER 2015 CLASS GENERAL PATHOLOGY

SECTION A MULTIPLE CHOICE QUESTIONS

1) When cell in exposed to injury, the possible outcome include the following except
a) The cell may adapt to the situation
b) The cell may acquire a reversible injury
c) The cell may obtain irreversible injury
d) The cell may undergo chemical adaptation

2) Cell atrophy can be due to
a) Under nutrition
b) injury
c) Infection
d) Toxicity

3) Metaplasia is
a) Replacement of an organ
b) Cellular differentiation
c) Epithelial adaptation
d) Replacement of differentiated tissue with another differentiated tissue

4) Accumulation of fats in hepatocytes is not caused by the following
a) Decrease use of fat by cells
b) Overproduction of fats in cells
c) Increased uptake of fats into parenchymal cells
d) Inflammation of liver

5) Liquefactive necrosis is characterized by
a) Tissue regeneration
b) Tissue proliferation
c) Digestion of tissues
d) Cell necrosis

6) Metastatic calcification is caused by
a) Hypocalcemia

b) Hypercalcemia
c) Reduced absorption of calcium
d) Poor mineralization

7) In what stage of cell division do genetic material double
a) Telophase
b) Anaphase
c) Interphase
d) Prophase

8) Prognosis is
a) General disease progression
b) Expected outcome of the disease
c) Pathological changes
d) Disease complications

9) Mutation occurs when
a) Change in environmental condition
b) Faulty DNA copying
c) DNA sequencing.
d) Hyperplasia arises

10)Cytopathology techniques can used to
a) early screening for early diagnosis of cancer
b) gastrointestinal analysis
c) Metabolic diagnostics
d) Blood electrolytes analysis

10) Outcome of a disease doesn't include the following
a) Resolution.
b) Progression of the disease
c) Death
d) Anaphylaxis

12) Apoptosis is
a) Programmed cell death in a cluster of other cell.
b) Death of a cluster of cells
c) Calcification of tissue
d) Adaptation to cell injury

13) The following is a step during vascular response in inflammation
a) Vasoconstriction
b) Transudate formation
c) necrosis
d) Ischaemia

14) Cellular response in inflammation doesn't include the following
a) Transmigration of leucocytes
b) Secretion of chemical mediators
c) Rolling
d) Exudate formation

15) Prostaglandin secretion during inflammation is done by
a) Leucocytes
b) Platelets
c) Endothelium
d) Lymph nodes

16) Cytokines role include
a) Vascular leakage
b) Tissue distraction
c) Leucocyte activation
d) Vasoconstriction

17) In serous inflammation there is
a) outpouring of thin fluid generated from serum and the mesothelial cell secretion
b) Exudation of large molecules like fibrinogen
c) Necrotic tissue debris.
d) edema fluid.

18) Catarrhal inflammation
a) Mainly affect mucous membrane
b) Affects subcutaneous tissue
c) is associated with excessive pus generation
d) Usually caused by protozoals

19) The following is not beneficial attribute of acute inflammation
a) Toxins dilution
b) Protective antibodies
c) Improves cellular nutrition
d) Immunity depletion

20) The primary cells in chronic inflammation are
a) B lymphocytes
b) T. lymphocytes
c) Natural killer cells
d) monocytes and macrophages

SECTION B: TRUE FALSE QUESTIONS

1) The following occurs during cellular response during inflammation
a) Transmigration of leucocytes
b) Chemotaxis
c) Phegocytosis
d) Exudation
e) apoptosis

2) The following are diseases causing physical agents
a) Ionizing radiation
b) Heat
c) Very low temperature
d) Sulphuric acid
e) worms

3) Outcome of a disease include
a) Incubation period
b) Latency
c) Resolution
d) Death
e) necrosis

4) Possible outcomes of cell injury include
a) Resolution
b) Adaptation to the situation.
c) Acquire reversible injury
d) Cell death
e) under nutrition

5) Cellular adaptation to injury include.
a) Under nutrition
b) Atrophy
c) denervation
d) hyperplasia

e) metaplasia

6) Examples of metaplasia include
a) Epithelial metaplasia
b) Interstitial metaplasia
c) squamous
d) Osseous metaplasia
e) Epidermal metaphasia

7) Mechanism of necrosis occurrence include
a) hypoxia
b) free radical induced cell injury
c) Cell membrane damage
d) Increased intracellular calcium
e) Influx of proteins

8) Cell necrosis can be
a) gangrenous
b) Caseous
c) Malignant
d) benign
e) Liquefactive

9) Events of acute inflammation include
a) Vascular response
b) Cellular response
c) Healing by fibrosis
d) keloid formation
e) Liquefaction

10) What is the importance of chemotaxis during inflammation
a) Aids movements of leucocytes to the site
b) Opsonization

c) Compliment system activation
d) Phagocytosis
e) Endocytosis

11) Killing or degradation of bacteria during phagocytosis can be due to

a) oxygen dependent mechanisms
b) carbondioxide dependent mechanism
c) myeloperoxidase dependent mechanism
d) cell membrane synthesis inhibition
e) metabolic interference

12)chemical mediators of inflammation include

a) serotonins
b) interferons
c) cytokines
d) hydroperoxidases
e) amonium chlorjde

13)Exudate has

a) clear translucent or pale yellow colour
b) only few mesothelial cells
c) Abundant white blood cell and red blood cell
d) No bacteia
e) No proteins

14) Course of fibrinous inflammation include

a) Abscess formation
b) resolution by fibrinolysis
c) Scar formation
d) fibrous strand formation
e) Edema formation

15) Harmful effect of inflammation include
a) Inappropriate response
b) Tissue destruction
c) swelling
d) Tissue proliferation
e) cell mutation

16) Causes of chronic inflammation are
a) persistent infection
b) progression from acute
c) autoimmunity
d) excessive heat
e) acute infections

17) Healing of injured tissues can be through
a) Regeneration
b) repair
c) tissue differentiation
d) wound contraction
e) immunosuppression

18) Factors influencing wound healing include

a) Vascular supply
b) chemical factors
c) race
d) metabolic status
e) kidney function

19) Complications of wound healing include
a) infection
b) deficient scar formation
c) abscess formation
d) tissue necrosis
e) Amylation

20) stages of fracture healing include
a) hematoma formation
b) scar formation
c) callus formation
d) demolition
e) granulation tissue formation

D/cm/1600/020

D/cm/1600/020

G. Pathology

EXAM NO:D/CM/JULY-FQE/014/..... KMTC/QP-08/EAB

SECTION B: TRUE-FALSE – 30% SCORE ...../150 SCORE ...10.../30

01	T	F
a	X	✓
b	X	✓
c		
d	X	
e		

02	T	F
a	X	✓
b	X	✓
c	X	✓
d		X ✓
e	X	

03	T	F
a		
b	X	
c		X ✓
d	X	✓
e	X	

04	T	F
a		
b	X	✓
c	X	✓
d	X	✓
e	X	

05	T	F
a		
b		
c		
d		
e		

06	T	F
a		X ✓
b		X ✓
c	X	✓
d	X	✓
e		X ✓

07	T	F
a	X	✓
b		
c	X	✓
d	X	✓
e	X	

08	T	F
a		
b		
c		
d		
e		

09	T	F
a	X	✓
b	X	✓
c	X	
d		
e		

10	T	F
a		
b		
c		
d		
e		

11	T	F
a		
b		
c		
d		
e		

12	T	F
a		
b		
c		
d		
e		

13	T	F
a	X	
b		X ✓
c	X	✓
d		X ✓
e		X ✓

14	T	F
a	X	
b		
c	X	✓
d		
e		

15	T	F
a		X
b	X	✓
c	X	✓
d		X ✓
e		X ✓

16	T	F
a	X	✓
b	X	✓
c	X	✓
d		
e	X	

17	T	F
a	X	✓
b	X	✓
c	X	
d	X	
e		

18	T	F
a	X	✓
b		✓
c		X ✓
d	X	
e		X ✓

19	T	F
a	X	✓
b	X	✓
c	X	✓
d	X	✓
e		

20	T	F
a	X	✓
b	X	
c	X	✓
d	X	✓
e	X	✓

21	T	F
a		
b	47	
c	18	
d		
e	34	

22	T	F
a		
b		
c		
d		
e		

23	T	F
a		
b		
c		
d		
e		

24	T	F
a		
b		
c		
d		
e		

25	T	F
a		
b		
c		
d		
e		

26	T	F
a		
b		
c		
d		
e		

27	T	F
a		
b		
c		
d		
e		

28	T	F
a		
b		
c		
d		
e		

29	T	F
a		
b		
c		
d		
e		

30	T	F
a		
b		
c		
d		
e		

EXAM NO:D/CM/JULY-FQE/014/.....

KMTC/QP-08/EAB

KENYA MEDICAL TRAINING COLLEGE

Faculty of Clinical Sciences

Department of Clinical Medicine

2014/2015 Academic Year

Final Qualifying Examinations (July 2015)

MODULE:..... Date:.....

TOTAL SCORE...31.../60

SECTION A: MULTIPLE CHOICE QUESTIONS (MCQS) - 30% SCORE...21.../30

Q1	
a	
b	
c	*
d	X

Q2	
a	X
b	
c	
d	

Q3	
a	
b	
c	
d	X

Q4	
a	
b	
c	
d	X

Q5	
a	
b	X
c	
d	

Q6	
a	
b	
c	X
d	

Q7	
a	
b	X
c	
d	

Q8	
a	
b	X
c	
d	

Q9	
a	
b	X
c	
d	

Q10	
a	X
b	
c	
d	

Q11	
a	X
b	
c	
d	

Q12	
a	X
b	
c	
d	

Q13	
a	X
b	
c	
d	

Q14	
a	
b	
c	
d	X

Q15	
a	
b	
c	X
d	X

Q16	
a	
b	
c	X
d	

Q17	
a	X
b	
c	
d	

Q18	
a	X
b	
c	
d	

Q19	
a	
b	
c	
d	X

Q20	
a	
b	
c	X
d	X

Q21	
a	
b	
c	
d	

Q22	
a	
b	
c	
d	

Q23	
a	
b	
c	
d	

Q24	
a	
b	
c	
d	

Q25	
a	
b	
c	
d	

Q26	
a	
b	
c	

Q27	
a	
b	
c	

Q28	
a	
b	
c	

Q29	
a	
b	
c	

Q30	
a	
b	
c	

September 2015 class
General Pathology

CAT

1. Describe any 5 diagnostic techniques used in pathology (5mks)
2. Describe any 5 environmental causes of diseases (5mks)
3. Explain any 4 cell adaption to injury (8mks)
4. Describe the two main ways in which cell die (4mks)
5. Describe the stages of cell division (normal cell division) (8mks)
6. Describe any five causes of inflammation (5mks)
7. Explain 5 cardinal features of inflammation (5mks)

D/CM/16/0/020

17/40

1) i) Image - scanning, X-rays

ii) Immunohisto chemistry

iii) ~~Biological factor - virus and bacterial~~ Cytopathogenetic

iv) Autophy

v)

2) i) Physical Agents - Trauma, Radiation

ii) Biological factor - ~~viruses and bacteria~~ chemicals increase in drugs.

iii) Infection and infestation - virus and bacteria

iv) Nutrition ~~defience~~ deficiences and excesses - Lack of some nutrients in ~~diet~~ diet.

v) Immunological factor - gene disorders.

3) i) Reversible cells - when treated they become normal.

ii) Irreversible cells - cannot be treated they are dead cells.

iii) Hiperplasia - increase in size of cell.

iv)

4) a) Apoptosis

b) ~~Necrosis~~ ~~Crosis~~ Crosis -

5) In

stage 1) Interphase - Preparation for the cell division takes place.

stage 2) prophase -

stage 3) Metaphase - chromosomes moves away from the centre of the cell and divide itself into two.

(4)

stage 4) Anaphase - start formation of daughter cells.

stage 5 Telophase - Complete formation of daughter cells.

stage 6) Cytokinesis - cell division is completely done.

6) i) Physical agents - chemical, Allergic to trauma, Radiation's

ii) Genetic disorder - inherited Genes

(3) iii) Immune disorders - Chromosome disorder.

iv) Infections and infestation - Virus and bacteria

v) Biological factors - chemicals exposure

7

No 9) i) Redness - due to extreme high temperature.

(3) ii) Heat - due to flow of the blood to regulate body temperature.

iii) Swelling - due to accumulation of plasma fluid.

iv) Pain - due to stretching and destruction of muscles.

v) ~~Loss~~ Loss function - due to dead cells.

3

PATHOLOGY
Class: Sept. 2015 End of Semester 2

INSTRUCTIONS:
Examination policy applies.

1. The following is not a factor in wound healing.
   a) Infection of the wound.
   b) Vascular supply.
   c) Environmental temperature
   d) Movements.

2. The following is stage 1 of fracture healing.
   a) Demolition.
   b) Hematoma formation
   c) Formation of granulation tissue.
   d) Inflammation.

3. Demolition in fracture healing involves
   a) Removal of detached bone fragment.
   b) Removal of clots.
   c) Remodeling of the broken bone.
   d) Removal of the inflammatory exudates.

4. Oncotic oedema is mainly determined by;
   a) Plasma protein levels.
   b) Electrolytes
   c) Tissue drainage
   d) Inflammatory processes.

5. The following is not a type of localized oedema.
   a) Malnutrition related.
   b) Pulmonary oedema
   c) Brain oedema
   d) Lymphatic oedema.

6. The following enzyme is key in edema formation due to congestive cardiac failure.
   a) Angiotension II
   b) Cortisol
   c) Adrenal cortizosteroid.
   d) Adrenaline.

7. Hemorrhage involves.
   a) Excessive blood accumulation to tissues.
   b) Intravascular blood congestion
   c) Stasis e.g. blood in extreme vessel.
   d) Extravasations of blood due to vessel opening.

8. Homeostasis does not depend on
   a) Platelets.
   b) Vascular wall integrity.
   c) Coagulation pathway.

d) Blood volume.

9. Thrombosis can be due to;
   a) Smooth blood flow
   b) Blood hypocoagulability.
   c) Endothelial damage.
   d) Vessels wall permeability.

10. Hypercoagulability risks include;
   a) Increased mobility
   b) Chest infections.
   c) Use of anticoagulants
   d) Tissue injuries.

11. venous thrombi
   a) Has firm attachment.
   b) Arise from endothelial injury site.
   c) Grows in retrograde fashion.
   d) Has loose attachment.

12. Fat embolism is common in
   a) Tissue injury
   b) Obstetric procedures.
   c) Chest nerve injury
   d) Cardiovascular diseases.

13. Infarcts are caused predominantly by
   a) Hypotension.
   b) Infections.
   c) Thrombo – embolic events.
   d) Vasospasms.

14. Red infarcts occurs in;
   a) Organs with multiple arterial supply.
   b) Solid organs.
   c) Single arterial supply.
   d) Uncongested tissues.

15. In shock
   a) There are inadequate nutrients.
   b) There is inadequate tissue perfusion.
   c) There is sufficient oxygen supply
   d) There is wide irreversible tissue damage

16. In cardiogenic shock there is;
   a) Depressed vascular contraction.
   b) Depressed peripheral venous resistance.
   c) There is reduced venous return.
   d) There is depressed cardiac performance.

17. DNA roles include;
a) Genetic information transfer.
b) Regulates body system function.
c) Key in carbohydrates metabolism.
d) Lipid synthesis.

18. Mutations are ;
a) Changes in alleles transfer.
b) Necleotide sequence changes.
c) Cellular changes during differentiation.
d) Cell death during inflammation.

19. Genome mutations are;
a) Chromosomal rearrangement.
b) Nucleotides' miscopying.
c) Chromosomal misaggregation.
d) Nonesense during copying.

20. In silent mutations;
a) Genetic code is redundant.
b) There are obvious significant amino acid sequences changes.
c) These are clear phenotypic manifestation.
d) There are deletions of some codons of bases.

SECTION B : TIME FALSE

Note: In this part; you gain one mark for every correct choice and loose one mark for every incorrect choice.

1. Major categories of genetic diseases include;
   a) Mendelian disorders.
   b) Chromosomal disorders.
   c) Multi-factorial disorders.
   d) Single gene disease.
   e) Hematologic disorder.

2. In type 1 hypersensitivity reaction:
   a) It occurs immediately and rapidly.
   b) It is mediated by immunoglobulin E.
   c) It is characterized by initial and late phase.
   d) Mast Cells and basophils are main cells involved.
   e) Occurs mainly in children.

3. Compliment system play key role in
   a) Type 1 hypersensitivity reaction.
   b) Type III hypersensitivity reaction.
   c) Has no role in any form of hypersensitivity reaction.
   d) Consists mainly of plasma proteins.
   e) Are produced by the liver.

4. In type III hypersensitivity immune reaction: immune complex deposits depends on;
   a) Size of immune complex.
   b) Functional status of mononuclear phagocytic system.
   c) Metabolic status of individuals.
   d) Site of deposit
   e) Cardiovascular disorders.

5. Classes of immune complexes mediated diseases include;
   a) Systemic immune complex
   b) Aritus reaction.
   c) Chronic forms.
   d) Ag – Ab complex.
   e) Endogenous type.

6. In immunologic tolerance;
   a) There is failure of general immune response to all antigens.
   b) Failure to mount immune response to specific antigen.
   c) Is dependent on T – helper cells.
   d) Is mediated by B cells.
   e) It has a negative effect on general immune system.

7. In Central tolerance
   a) Is when T and B lymphocytes of exogenous antigens are deleted.

b) There is a apoptosis of T cells that bears self antigen receptors.
c) TjClonal deletion of immature T cells with self antigen receptors.
d) Clonal anergy
e) Results in generalized immune reaction.

8. Mechanisms of auto immune diseases includes
a) Metabolic factors.
b) Peripheral tolerance failure.
c) Molecular mimicry
d) Polyclonal B- lymphocytic activations.
e) Due to exogenous antigens triggering the immune reaction.

9. Auto – immune diseases can be;
a) Organ specific
b) Organ non specific
c) Microbial type
d) Metabolic type
e) Cardiovascular type

10. Types of hemorrhage include;
a) Petechial
b) Purpural
c) Hemotoma
d) White infacts
e) Echymosis

11. Cell injury can be due to ;
a) Metabolic imbalance
b) Genetic abnormality
c) Physical agents
d) Chemicals
e) Biological agents

12. Response to cell injury include;
a) Recovery
b) Death
e) Adaptation
d) Proliferation
e) Differentiation

13. Causes of cellular atrophy include
a) Disease
b) De-nervation
c) Old age
f) Increased endocrine stimulation.
g) Over nutrition.

14. Reversible cellular changes iclude
a) Osseous metaplasia
b) Fatty changes

c) Pigments accumulation
d) Apoptosis
e) Necrosis

15. Tissue hypoxia can be due to;
a) Anaemia
b) Ischaemia
c) Pulmonary diseases
d) Hypertension
e) Electrolyte imbalance.

16. Phagocytosis during inflammation enhances
a) Recognition and attached
b) Complement system activities.
c) Engulfment
d) Lysosomal activity
e) Raised body temperature.

17. Causes of chronic inflammation include.
a) Persistent infections.
b) Prolonged exposure to non biodegradable mild toxic agents
c) Precedes acute inflammation
d) Auto- immunity.
e) metabolic disorders

18.Beneficial effects of inflammation include
a) Dilution of toxins
b) Nutritional benefits
c) Improves tissue structural integrity
d) Plasma mediator system mediation
e) protective antibodies

19.Edema formation is determine by

a) hydrostatic pressure
b) electrolyte balance
c) infections
d) neurologic disorders
e) Vascular permeability

20. The following are pathological diagnostic techniques

a) cytology
b) histopathology
c) computed tomography imaging
d) Magnetic resonance imaging
e) ultra sound

D/cm/160101.020

AM NO:D/CM/JULY-FQE/014/..... KMTC/QP-08/EA8

KENYA MEDICAL TRAINING COLLEGE

Faculty of Clinical Sciences — Pathology 4th Year Exam — Department of Clinical Medicine

16/2015 Academic Year — Final Qualifying Examinations (July 2016)

SCHEDULE:..... Date: 11/07/2016

TOTAL SCORE: 35 /60

SECTION A: MULTIPLE CHOICE QUESTIONS (MCQS) – 30% SCORE: 18 /30

Q1	
a	✗
b	
c	
d	

Q2	
a	
b	✗
c	
d	

Q3	
a	
b	
c	✗
d	

Q4	
a	✗
b	
c	
d	

Q5	
a	✗
b	
c	
d	

Q6	
a	✗
b	
c	
d	

Q7	
a	
b	✗
c	
d	

Q8	
a	
b	
c	✗
d	

Q9	
a	
b	
c	✗
d	

Q10	
a	
b	
c	
d	✗

Q11	
a	✗
b	
c	
d	

Q12	
a	
b	✗
c	
d	

Q13	
a	
b	
c	
d	✗

Q14	
a	✗
b	
c	
d	

Q15	
a	
b	✗
c	
d	

Q16	
a	
b	
c	
d	✗

Q17	
a	✗
b	
c	
d	

Q18	
a	
b	✗
c	
d	

Q19	
a	
b	✗
c	
d	

Q20	
a	
b	
c	
d	✗

Q21	
a	
b	
c	
d	

Q22	
a	
b	
c	
d	

Q23	
a	
b	
c	
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Q24	
a	
b	
c	
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Q25	
a	
b	
c	
d	

Q26	
a	
b	
c	

Q27	
a	
b	
c	

Q28	
a	
b	
c	

Q29	
a	
b	
c	

Q30	
a	
b	
c	

D/cm/dc0101020

EXAM NO:D/CM/JULY-FQE/014/..... KMTC/QP-08/EAB

SECTION B: TRUE-FALSE – 30% SCORE ...../150 SCORE ...17.../30

01	T	F
a	X	
b	X	
c	X	
d		X
e	X	

02	T	F
a	X	
b	X	
c	X	
d	X	
e	X	

03	T	F
a		X
b	X	
c		X
d	X	
e	X	

04	T	F
a	X	
b		X
c	X	
d		X
e		X

05	T	F
a	X	
b		X
c		X
d	X	
e		X

06	T	F
a	X	
b	X	
c		X
d		X
e	X	

07	T	F
a	X	
b	X	
c		X
d		X
e		X

08	T	F
a	X	
b	X	
c	X	
d	X	
e	X	

09	T	F
a	X	
b	X	
c		X
d		X
e		X

10	T	F
a		X
b	X	
c	X	
d		X
e		X

11	T	F
a		X
b	X	
c	X	
d	X	
e	X	

12	T	F
a	X	
b	X	
c	X	
d		X
e		X

13	T	F
a	X	
b	X	
c	X	
d		X
e	X	X

14	T	F
a	X	
b		X
c		X
d		X
e		X

15	T	F
a		X
b	X	
c	X	
d	X	
e	X	X

16	T	F
a	X	
b		X
c	X	
d	X	
e		X

17	T	F
a	X	
b	X	
c	X	
d	X	
e		X

18	T	F
a	X	
b	X	
c		X
d	X	
e	X	

19	T	F
a	X	
b	X	
c	X	
d		X
e	X	

20	T	F
a	X	
b	X	
c	X	
d		X
e	X	

21	T	F
a		
b	76	
c	20	
d		
e	96	

22	T	F
a		
b		
c		
d		
e		

23	T	F
a		
b		
c		
d		
e		

24	T	F
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25	T	F
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27	T	F
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28	T	F
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29	T	F
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30	T	F
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KENYA MEDICAL TRAINING COLLEGE-MOMBASA

DEPARTMENT OF CLINICAL MEDICINE

GEN: PATHOLOGY SECOND SEMESTER CAT

SEPT.2015 CLASS

ANSWER ALL QUESTIONS

1. Explain wound healing under the following
   a) Healing by first intention **(2 Marks)**
   b) Healing by second intention **(2 Marks)**

2. Explain any 3 local and 3 systemic factors affecting wound healing **(6 Marks)**

3. State and explain in 5 complication of wound healing **(5 Marks)**

4. Briefly discuss the differences between venous and arterial thromboses **(4 Marks)**

5. Explain the process / pathogenesis of Oncotic and non Oncotic edema as well as their causes **(5 Marks)**

6. Briefly explain
   a) Infarction
   b) Red infarcts
   c) White infarcts and the organs in which they commonly occur **(6 Marks)**

D/cm/1160101020

Pathology

18/40

Quiz(2) Local.

i) Size of the wound - small wound takes short period to heal while wounds take a long period to heal.

3 ii) Location of wound - wounds located at the joints tends to take long to heal compared to wound located in area which is not a joint.

iii) Degree of damage → small degree of damage tissue heal faster comp big degree damaged tissue.

Systemic Factor: vascular supply

① Heart failure - patients with heart failure their wounds take a long pe heal due to insufficient blood supply.

2 ② Malnutrition - patients with aneamia take along time to heal due to insuffi blood in the body.

③

③ i) Infection → wounds provide a portal entry of bacteria hence causing the wound.

ii) Incitional hernia → Wounds located in places like abdomen may bu patient cough or vomit hence increasing the period of healing

3 iii) Diseases → Diseases lower the body immune hence affect the proces

iv) Dehesion relapse

~~v) Muscles contraction.~~

Scanned by CamScanner

4)

Venous thrombosis	Arterial thrombosis
i) Loosely attached	Firmly attached
ii) Growth is toward the flow of blood	Growth is against the flow of blood
iii) Rises from the site of ischemia	Rises from the site of pulmonary arteries

Aortic aneurysm

endothelial cells

1) - Healing by First intension - This is healing by replacement of non cellular tissue by similar type of tissues.

- Healing by second intension - This is healing by scar formation.

3

6)

a) infarction - death tissue due to decrease in blood supply or drainage
→ Affect heart

(b) Red infarct → death tissue due to haemorrhage
affect ~~to~~ ~~former~~ → Affect ~~limbs~~. heart

c) White infarct → death tissue due to decrease in blood supply. ~~in tissue~~
or → affect ~~former~~ ~~limbs~~ brain

Diploma 024

September 2015 class
General Pathology

CAT

1. Describe any 5 diagnostic techniques used in pathology (5mks)
2. Describe any 5 environmental causes of diseases (5mks)
3. Explain any 4 cell adaption to injury (8mks)
4. Describe the two main ways in which cell die (4mks)
5. Describe the stages of cell division (normal cell division) (8mks)
6. Describe any five causes of inflammation (5mks)
7. Explain 5 cardinal features of inflammation (5mks)

D/cm/12010/210

KENYA MEDICAL TRAINING COLLEGE-MOMBASA

DEPARTMENT OF CLINICAL MEDICINE

GENERAL PATHOLOGY FIRST SEMESTER CAT

SEPT 2016 CLASS **9/12/2019**

TIME: 9.00AM-11.00AM

INSTRUCTIONS: ANSWER ALL QUESTIONS

1. State and explain any 6 diagnostic techniques used in pathology. **(6 mks).**
2. Describe any 6 environmental causes of diseases **(6 mks).**
3. State and explain any 4 cellular adaptations to injury **(8 mks).**

32/40

1 Diagonistic techniques in pathology

a) Histopathology

Study of tissues in the organism to identify Cause of the desease. Tissue to be studyed is called Biopsy ~~Biopsy~~ ~~is~~. Fixation is done to biopsy so as to be stained and viewed in a microscope fixation also help to prevent autolysis

b) Cytopathology

Study of cells to identify cause of the desease methods in which cells are removed from the body include fine needle intrusion, exfoliative and abrasive Cytopathology

c) Clinical genetics

Study of ~~wake~~ inhaited chromosomal disodas in germ cells and acquired chromosomal disoders in Somatic cells by use of molecular techniques such as Suther blot

d) Immunohistochemistry technique

Study of antigens in the body which could have been the cause of the desease

e) Autotrophy autopsy

Study done on a dead body to identify the cause of death

f) Microbiological technique

Investigation of body fluids to identify microscopic organism in the fluids due to infection

D/cm/17010/210

2) Environmental causes of diseases

a) Psychogenic factors

Psychological factors like stress can lead to occurrence of a dessase

b) Infestation and infection

Infestation is ertenal while infection is internal this is due to viruses, parasites and even fungi

c) Physical factors

Physical factors are like variation of temperature which cause dessases how

d) Chemical factors

Chemical substances such as drugs can cause dessase

e) Immunological factors

Related to immune system of the body can be immunodeficiency hypersensitive

f) Nutritional deficiency and excess

lack of food can lead to malnutrition while excess amounts of food can lead to obesity

3 Cellular adaptations to injury

a) Hypotrophy

This is increased in size of a cell due to injury there is increased protein synthesis and increase in size and number of organelle This leads to increase in size of cell and also increase in size of the organ.

b) Hyperplasia

This is increase in number of cells due to an injury. This will also lead to increase in size of an organ. This explains swelling when muscle are stretched

c) Metaplasia

This is replacement of a differentiated tissue by another differentiated tissue. examples are squamous metaplasia and osseous metaplasia

Squamous metaplasia is where by differentiated tissue is replaced by squamous type of differentiated tissue osseous metaplasia is whereby connective tissue is replaced by a bone

Autotrophy Atrophy

This is decrease in size of size due to the degradation of organelle old age, Denervation reduced endocrine stimulation, Disuse and undernutrition are ~~cause of~~ may lead to autotrophy Characterised by presence of of vacuoles filled with degraded organeles

KENYA MEDICAL TRAINING COLLEGE-MOMBASA CAMPUS

DEPARTMENT OF CLINICAL MEDICINE

END OF SEMESTER ONE EXAMINATION

GENERAL PATHOLOGY PATHOLOGY

SEPT 2016 CLASS

SECTION A: MCQ CHOSE THE MOST CORRECT ANSWER

1. In pathology, aspects of disease studied includes;
   a) Progression.
   b) Morphological changes.
   c) Diagnostic techniques.
   d) Cell death.
2. Etiology of a disease refers to;
   a) Pathogenesis of a disease.
   b) Idiopathic
   c) Cause
   d) Morphological changes.
3. The following is a class of etiological factors:
   a) Cytopathologic.
   b) histopathologic
   c) Genetic
   d) Morphologic
4. Pathogenesis refers to;
   a) Disease cause.
   b) Morphological changes
   c) Mechanism by which disease agent generates pathological changes.
   d) Structural alteration
5. Physical agents causing diseases include;
   a) Trauma
   b) Expired drugs.
   c) Cyanide
   d) Metazoa
6. The following is a form of cellular adaptation to injury
   a) Necrosis
   b) Hyperplasia

c) Pigmentation.
d) Mutation.

7. Hyperplasia is
   a) Increase in cell number ✓
   b) Increase in cell size
   c) Replacement of specialized cell by another
   d) Cellular differentiation
8. Cellular atrophy may be due to;
   a) Increased workload.
   b) Degeneration.
   c) Regeneration.
   d) Reduced endocrine stimulation. ✓
9. The following is not a form of pigment accumulation in the cells.
   a) Trigycerides accumulation. ✓
   b) Bilirubin
   c) Melanin
   d) Hemosiderin.
10. In coagulative necrosis there is;
   a) Fatty acid release.
   b) Acute hemorrhage.
   c) Digestion of mines.
   d) Sudden interruption of blood supply. ✓
11. Apoptosis
   a) Is followed by inflammation.
   b) There is death of cluster of cells.
   c) Death of single cells with cluster of cells. ✓
   d) It is not a physiologic process.
12. In meiosis
   a) Occurs only in female.
   b) Occurs in both male and female. ✓
   c) Results into 2 daughters.
   d) Only occurs in plants.
13. During metaphase
   a) Nucleus reappears.
   b) Chromosomes disappear
   c) Chromosomes arrange at the equate r of cell ✓
   d) Spindle fibers disappears
14. In prophase
   a) The nucleolus disappear. ✓
   b) Nuclear membrane reforms.

c) Chromosomes separates at centromere
d) Cytokinesis occurs.

15. The following is not a role of cell membrane
a) Receptor site
b) Transportation
c) Structural integrity.
d) Protein synthesis ✓

16. The following do not cause all injury.
a) Metabolic imbalance.
b) Genetic abnormalities
c) Toxins
d) Amino acids. ✓

17. Inflammation is
a) Cellular adaptation to injury.
b) Morphological changes in the cell.
c) Localized vascularised tissue response to external stimuli. ✓
d) Pathogenesis of a disease.

18. Readness during acute inflammation is due to
a)disease causing agent
b) Increased blood supply and stasis ✓
c) Chemical mediators
d)white blood cells

19) Chemo taxis is
a) Exudation of plasma contents
b) Engulfment and digestion
c) Attraction of leukocytes to the inflammatory site ✓
d) Blood flow to the site

20) Complement system role in inflammation include
a)Identification of the disease agent ✓
b) Activation of macrophages
c) Stimulate T lymphocyte secretion
d) Blood stasis

SECTION B: TRUE AND FALSE QUESTIONS-RESPOND TO ALL ANSWERS NEGATIVE MARKING IS APPLIED.

1. Physical agents causing inflammation include;

a) Fungi F
b) Autoimmunity F
c) Trauma T
d) Toxins F
e) Drugs. F

2. The following constitutes cardinol signs of inflammation.
   a) Hemorrhage F
   b) Clotting F
   c) Swelling T
   d) Pain T
   e) Discharge F
3. Vascular response in acute inflammation is characterized by;
   a) Vasodilation T
   b) Vasoconstriction. T
   c) Blood stasis T
   d) Paring and rolling of white blood cells. F
   e) Chemotaxis F
4. Chemotactic factors include;
   a) Fibrins.
   b) Vitamin K.
   c) Complement system components. T
   d) Leukotrines.
   e) Cytokines.
5. Phagocytosis during acute inflammation involves;
   a) Endothelia attachment and escape.
   b) Engulfment. T
   c) Recognition and attached. T
   d) Vasoconstriction F
   e) Edema formation
6. During anaphase
   a) Diploid sets of daughter chromosomes separate.
   b) Spindle fibres push daughter chromosomes opposite direction.
   c) Cytokinesis occurs.
   d) Nucleolus reappears.
   e) Nuclear membrane form.
7. Malignant / neoplasm cells / tissues.
   a) Are well differentiate
   b) Have rapid growth.
   c) Are poorly differentiated.
   d) Invasive / infiltrates on tissues.

e) Mortal, cells dies by apoptosis.

8. Cell necrosis can occur due to
   a) Hypoxia T
   b) Free radical induced cell injury. T
   c) Cell membrane damage. T
   d) Apoptosis. F
   e) Reduced intracellular calcium level. T
9. In liquifactive necrosis
   a) Occurs due to ischemic injury.
   b) There is suppuration and pus formation.
   c) There is fat accumulation.
   d) Formation of calcified tissues.
10. Cellular response in acute inflammation entails.
   a) Vasodilatation of arterioles. F
   b) Oozing of protein rich exudates. F
   c) Transmigration of leukocytes. T
   d) Phagocytosis T
   e) Swelling. F
11. Key factors in killing and degradation during phagocytosis include;
   a) Lysozymes. T
   b) Protein components / defenses.
   c) Leukotines.
   d) Lactoferins.
   e) Interleukins.
12. Chemical mediators in acute inflammation include.
   a) Cytokines.
   b) Serotonins.
   c) Prostaglandines.
   d) Nitric oxide.
   e) Fibrins.
13. Exudates is
   a) Clear and translucent. F
   b) Colored and turbid. T
   c) Doesn't coagulate
   d) Has abundant WBC & RPC
   e) Occurs in inflammatory conditions.
14. Serous inflammation
   a) Is enacted in severe injuries.
   b) Characterized by fibrinous exudates.
   c) Causes out flowing of thin fluids in adjacent spaces.

d) Resolves without reactions.
e) Always leads to chronic inflammation.

15. In suppuration inflammation, the purulent discharge produced consist of;
    a) Pus cells. T
    b) Transudate fluid. F
    c) Necrotic tissue debris.
    d) Edema fluid
    e) Blood vessels.
16. Beneficial effects of acute inflammation include;
    a) Wound healing. T
    b) Antibodies provision. T
    c) Key in protein synthesis.
    d) Fibrin formation. T
    e) Toxin dilution. T
17. Harmful effects of inflammation include;
    a) Tissue damage. T
    b) Swelling and disability. T
    c) Immune promotion. F
    d) Cell nutrition deficiency.
    e) Sinus formation.
18. Chronic inflammation causes include
    a) Persistent infections. T
    b) Acute inflammation.
    c) Auto – immunity. T
d) Micro – organisms.
e) No biodegradable with partial toxicity.
19. Key or primary cells in chronic inflammation are
    a) T helper cells. F
    b) Natural killer cells. F
    c) Monocytes. T
    d) Macrophages T
    e) Plasma cells. F
20. Systemic effects of inflammation
    a) Fever T
    b) Leukocytosis T
    c) Sight loss
    d) Leucopenia T
    e) Behavioural and response.

KMTC/QP-08/TIS

KENYA MEDICAL TRAINING COLLEGE

Faculty of Clinical Sciences
Final Qualifying Examinations

Department of clinical medicine
2013/2014 academic year

Exam Number: [illegible]

Course Name: General Pathology

MCQ

Score: 30 /300

Score: 44 /60

1	T	F
a		
b	X	
c		
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2	T	F
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3	T	F
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c	X	
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4	T	F
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c	X	
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5	T	F
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6	T	F
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7	T	F
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8	T	F
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9	T	F
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10	T	F
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11	T	F
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12	T	F
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13	T	F
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14	T	F
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16	T	F
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17	T	F
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18	T	F
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19	T	F
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KMTC/QP-08/T15

KENYA MEDICAL TRAINING COLLEGE

Faculty of Clinical Sciences
Final Qualifying Examinations

Department of clinical medicine
2013/2014 academic year

Exam Number: 01GM/13010/310

TRUE / FALSE

Course Name: General pathology

Score: 14 /300

Score: _____ /60

1	T	F
a		✗
b		✗
c	✗	
d		✗
e		✗

2	T	F
a		✗
b		✗
c	✗	
d	✗	
e		✗

3	T	F
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b	✗	
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d		✗
e		✗

4	T	F
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5	T	F
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b	✗	
c	✗	
d		✗
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6	T	F
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7	T	F
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8	T	F
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19	T	F
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d	✗	✗
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20	T	F
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21	T	F
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C

22	T	F
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b	✗	
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e	✗	

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29	T	F
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30	T	F
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KENYA MEDICAL TRAINING COLLEGE-MOMBASA CAMPUS

DEPARTMENT OF CLINICAL MEDICINE

SECOND SEMESTER CATS

GENERAL PATHOLOGY

SEPT 2016 CLASS

9/5/2017

ANSWER ALL QUESTIONS

1. What is edema? (1mk)
2. Explain the 5 determinants of edema formation and how each contributes to edema formation (10 mks)
3. Define hemorrhage and explain the 2 main causes (5 mks)
4. Define hemostasis and 3 major components (5 mks)
5. Briefly explain how hemostasis is achieved following rupture blood vessel (4 mks)
6. Describe shock and explain the 3 main categories (5mks)

D/CM/17010/210

21/30

1 Edema

Edema is the accumulation of fluid excessively in the interstitium or body cavities.

2. Determine of edema formation and how each contribute to edema formation

a) Hydrostatic pressure

There are two forms of hydrostatic pressure namely capillary hydrostatic pressure and Interstitial hydrostatic pressure. Capillary hydrostatic pressure allows outflow of fluid from the blood vessel into the interstitium. When it is increased it promote Edema formation. Interstitial hydrostatic pressure allows out flow of fluid from interstitium into blood vessel reducing chance of edema formation

b) Oncotic pressure

There are two forms of oncotic pressure plasma oncotic pressure and interstitial oncotic pressure plasma oncotic pressure is depended on proteins and allows osmosis of fluid from interstitium into the blood vessel. Its decrease which results from decrease of proteins like in malnutrition leads to edema formation. Interstitial oncotic pressure allows osmosis of fluid into the interstitium from the blood vessel which may cause edema

c) Vascular Permeability

Vascular wall is always Semipermeable allowing only small molecules like glucose to cross to interstitium especially in hover large molecular substances like proteins and blood cells. On inflammatory reaction there may be increased vascular permeability leading to influx of fluid into the interstitium. The fluid is rich in proteins & refered to as exudate. In non-inflammatory reaction the fluid is not rich in proteins and is refered to as transudate

d) lymphatic vessels
lymph freely flows in the lymphatic system carrying nutrients
2 When there is blockage in the lymph vessels it causes accumulation of fluid leading to formation of edema

e) Sodium and water retention
Failure of Sodium potasium pump leads to accumulate
2 of Sodium this attracts influx of water due to osmotic imbalance. Influx increase accumulation of fluid leading to edema.

3. Hemorrhage
Hemorrhage is extravasation of blood outside the blood
1 vessels

Main causes of hemorrhage
i) Internal factors
Increased blood pressure causes blood vessels to burst causing internal bleeding which may be fatal

ii) External factors
External factors may like injuries and cuts which cause
2 damage to blood vessels causing out flow of blood

4) Homeostasis
Homeostasis is prevention of blood loss through a blood
1 vessel as well as maintainance of fluid form of the blood

Major component of homeostasis
Major component of homeostasis include endothelial injury, Coagulation
1 pathways and platelets. Endothelial injury is breakage

9

of a blood vessel exposing blood to external environment Platelets are blood cells in the plasma responsible for hemostasis. There are two coagulation pathways namely intrinsic coagulation pathway and extrinsic coagulation pathway. Intrinsic coagulation pathway is activated by factors inside ~~outside~~ a blood vessels while extrinsic coagulation pathway is activated by factors outside a blood vessel

5. How hemostasis is achieved following rupture of blood vessel

Endothelial injury causes breakage of blood vessels exposing platelets with factors which activate the coagulation pathways. Factors are collagen which activate intrinsic coagulation pathway and glass which activate extrinsic coagulation pathway Coagulation factors includes the chrismate factor, Vitamin K Coagulation pathway involves activation of coagulation factors like activation of prothrombin to thrombin. End result is formation of platelet plug, ceasing of bleeding and later formation of a scar

6) Shock

Shock is the failure of Cardiovascular system to maintain adequate cellular perfusion prevent tissue from getting adequate nutrients and oxygen.
Shock normally occurs when there is reduced preload, reduced afterload and reduced myocardial contractility
Shock is categorised according to its causes into hypovolumic Shock, Distributive Shock and myocardial Shock
hypotolumic Shock results in decrease of preload which might be caused by bleeding and burns
Myocardial Shock is caused by factors which affect

Contractility of the heart. They may be external factors suppressing heart action like pneumothorax or internal factors affecting contractility like myocarditis

Distributive shock is a generalised shock caused by reduction of the afterload

1 KENYA MEDICAL TRAINING COLLEGE – MOMBASA

DEPARTMENT OF CLINICAL MEDICINE

PROMOTIONAL EXAM

GEN. PATHOLOGY

SECOND SEMESTER

SEPTEMBER 2016 RCO CLASS

DATE: 27/6/2017

TIME: 9.00AM-11.00AM

1. Candidates are supposed to be sited
2. 15 minutes before the exam starts.
3. Nobody is allowed to carry notes/books/mobile phones during the examination.

4. Answer all questions indicate whether true or false by putting X on the answer sheet provided.

5. Printed questions are from pages 1-10

SECTION A MULTIPLE CHOICE QUESTIONS ONLY ONE ANSWER IS CORRECT

1) During acute inflammation, vascular response is characterized by the following except
a) Vasodilatation
b) Vasoconstriction
c) Stasis
d) Chemo taxis
2) Cell atrophy can be due to
a) Under nutrition
b) injury
c) Infection
d) Toxicity
3) Shock can be of the following class except
a) Hypovolemic shock
b) Cardiogenic shock
c) Distributive shock
d) Vascular shock
4) Accumulation of fats in hepatocytes is not caused by the following
a) Decrease use of fat by cells
b) Overproduction of fats in cells
c) Increased uptake of fats into parenchymal cells
d) Inflammation of liver
5) Liquefactive necrosis is characterized by
a) Tissue regeneration
b) Tissue proliferation
c) Digestion of tissues
d) Cell necrosis
6) Metastatic calcification is caused by
a) Hypocalcemia
b) Hypercalcemia
c) Reduced absorption of calcium
d) Poor mineralization
7) In what stage of cell division do genetic material double
a) Telophase
b) Anaphase
c) Interphase
d) Prophase

8) Prognosis is
a) General disease progression
b) Expected outcome of the disease
c) Pathological changes
d) Disease complications
9) Mutation occurs when
a) Change in environmental condition
b) Faulty DNA copying
c) DNA sequencing.
d) Hyperplasia arises
10) Cytopathology techniques can used to
a) early screening for early diagnosis of cancer
b) gastrointestinal analysis
c) Metabolic diagnostics
d) Blood electrolytes analysis
11) Outcome of a disease doesn't include the following
a) Resolution.
b) Progression of the disease
c) Death
d) Anaphylaxis
12) Apoptosis is
a) Programmed cell death in a cluster of other cell.
b) Death of a cluster of cells
c) Calcification of tissue
d) Adaptation to cell injury
13) The following is a step during vascular response in inflammation
a) Vasoconstriction
b) Transudate formation
c) necrosis
d) Ischaemia
14) Cellular response in inflammation doesn't include the following
a) Transmigration of leucocytes
b) Secretion of chemical mediators
c) Rolling
d) Exudate formation
15) Prostaglandin secretion during inflammation is done by
a) Leucocytes
b) Platelets

c) Endothelium
d) Lymph nodes

16) Cytokines role include
a) Vascular leakage
b) Tissue distraction
c) Leucocyte activation
d) Vasoconstriction

17) In serous inflammation there is
a) outpouring of thin fluid generated from serum and the mesothelial cell secretion
b) Exudation of large molecules like fibrinogen
c) Necrotic tissue debris.
d) edema fluid.

18) Catarrhal inflammation
a) Mainly affect mucous membrane
b) Affects subcutaneous tissue
c) is associated with excessive pus generation
d) Usually caused by protozoals

19) The following is not beneficial attribute of acute inflammation
a) Toxins dilution
b) Protective antibodies
c) Improves cellular nutrition
d) Immunity depletion

20) The primary cells in chronic inflammation are
a) B lymphocytes
b) T. lymphocytes
c) Natural killer cells
d) monocytes and macrophages

SECTION B: TRUE FALSE QUESTIONS NEGATIVE MARKING IS APPLICABLE

1) The following occurs during cellular response during inflammation
a) Transmigration of leucocytes T
b) Chemotaxis T
c) Phegocytosis T
d) Exudation T
e) apoptosis F

2) The following are diseases causing physical agents
a) Ionizing radiation T
b) Heat T
c) Very low temperature T
d) Sulphuric acid F
e) worms F
3) edema formation is determined by
a) Hydrostatic pressure T
b) Oncotic pressure T
c) Vascular permeability T
d) Lymphatic channels function T
e) Sodium and water retention Incubation period T
4) In genome mutations
a) Due to chromosome missegregation. T
b) gain or loss of one or more whole chromosomes. F
c) exemplified by aneuploidy & polyploidy.
d) often incompatible with survival to the situation.
e) Acquire reversible features
5) Gene mutations types include the following
a) Single base pair change (Point Mutation) T
b) Deletions & Insertions T
c) Expansions of repeat sequences T
d) Chromosomal disaggregation F
e) Loss or gain of chromosome F
6) In mendelian disorders
a) affects transcription, mRNA processing, or translation
b) Abnormal protein or decreased protein
c) may affect any type of protein
d) monogenic mendelian disorders
e) Mainly recessive
7) Mechanism of necrosis occurrence include
a) hypoxia T
b) free radical induced cell injury T
c) Cell membrane damage T
d) Increased intracellular calcium T
e) Influx of proteins F

8) Cell necrosis can be
a) gangrenous T
b) Caseous T
c) Malignant F
d) benign F
e) Liquefactive T
9) Events of acute inflammation include
a) Vascular response T
b) Cellular response T
c) Healing by fibrosis F
d) keloid formation F
e) Liquefaction F
10) What is the importance of chemotaxis during inflammation
a) Aids movements of leucocytes to the site F
b) Opsonization
c) Compliment system activation
d) Phagocytosis F
e) Endocytosis
11) Killing or degradation of bacteria during phagocytosis can be due to
a) oxygen dependent mechanisms T
b) carbondioxide dependent mechanism
c) myeloperoxidase dependent mechanism T
d) cell membrane synthesis inhibition
e) metabolic interference

12) In Mendelian autosomal dorminant disorders
a) Caused by Loss of function mutations
b) Caused by Gain of function mutation
c) Phenotypic expression shows variable penetrance
d) Always shows variable penetrance
e) All carriers are affected

13) Homeostasis depends on three general components

a) Vascular wall T
b) Platelets T
c) Coagulation pathways Hydrostatic pressure T
d) Oncotic pressure T
e) Vascular permeability T

14) In type 1 immune reactions

a) It depends on b cells and IgE T
b) It is cell dependent F
c) It is complement depended
d) Mainly responds to microbial agents
e) Usually systemic in nature

15) Thrombosis can be triggered by the following

a) Endothelial injury T
b) Stasis or turbulence of blood flow T
c) Blood hypercoagulability T
d) Inappropriate response F
e) Tissue destruction F

16) Causes of chronic inflammation are

a) persistent infection T
b) progression from acute T
c) autoimmunity
d) excessive heat F
e) acute infections

17) In type ii immune reaction

a) There is complement dependent reaction
b) Antibody dependent cell mediated cytotoxicity
c) Cellular dysfunction
d) Formation of immune complexes
e) Immuno-suppression

18) Factors influencing wound healing include

a) Vascular supply T
b) chemical factors
c) race F
d) metabolic status
e) kidney function

19) Complications of wound healing include

a) infection T
b) deficient scar formation T
c) abscess formation T
d) tissue necrosis
e) Amylation F

20) stages of fracture healing include
   a) hematoma formation
   b) scar formation
   c) callus formation
   d) demolition
   e) granulation tissue formation

KMTC MOMBASA CAMPUS CLINICAL MEDICINE SEMESTER....II..... KMTC/QF 08/TIS

D/CM/17010 2/Rev. MCQ: SUBJECT Clinical Pathology DATE 23/05/2017

29/30 46/50

1	T
a	
b	
c	
d	X
e	

2	T
a	X
b	
c	
d	
e	

3	T
a	
b	
c	
d	X
e	

4	T
a	
b	
c	
d	X
e	

5	T
a	
b	
c	X
d	
e	

6	T
a	
b	X
c	
d	
e	

7	T
a	
b	
c	X
d	
e	

8	T
a	
b	X
c	
d	
e	

9	T
a	
b	X
c	
d	
e	

10	T
a	X
b	
c	
d	
e	

11	T
a	
b	
c	
d	X
e	

12	T
a	X
b	
c	
d	
e	

13	T
a	X
b	
c	
d	
e	

14	T
a	
b	
c	
d	X
e	

15	T
a	X
b	
c	
d	
e	

16	T
a	X
b	
c	
d	
e	

17	T
a	X
b	
c	
d	
e	

18	T
a	X
b	
c	
d	
e	

19	T
a	
b	
c	
d	X
e	

20	T
a	
b	
c	
d	X
e	

21	T
a	
b	
c	
d	
e	

22	T
a	
b	
c	
d	
e	

23	T
a	
b	
c	
d	
e	

24	T
a	
b	
c	
d	
e	

25	T
a	
b	
c	
d	
e	

26	T
a	
b	
c	
d	
e	

26	T
a	
b	
c	
d	
e	

27	T
a	
b	
c	
d	
e	

28	T
a	
b	
c	
d	
e	

30	T
a	
b	
c	
d	
e	

KMTC MOMBASA CAMPUS CLINICAL MEDICINE SEMESTER II KMTC/QP-08/TIS

D/CM/ I Tolo / 319 TRUE/FALSE: SUBJECT Clinical pathology DATE 2/10/19

1	T	F
a	✗	
b	✗	
c	✗	
d		✗
e		✗

2	T	F
a	✗	
b	✗	
c	✗	
d		✗
e		✗

3	T	F
a	✗	
b	✗	
c	✗	
d	✗	
e	✗	

4	T	F
a	✗	✗
b	✗	✗
c		
d		
e		

5	T	F
a	✗	
b	✗	
c	✗	
d		✗
e		✗

6	T	F
a	✗	
b	✗	
c	✗	
d	✗	
e		✗

7	T	F
a	✗	
b	✗	
c	✗	
d	✗	
e		✗

8	T	F
a	✗	
b	✗	
c		✗
d		✗
e	✗	

9	T	F
a	✗	
b	✗	
c		✗
d		✗
e		✗

10	T	F
a		✗
b		✗
c	✗	
d		✗
e		✗

63
7
56

11	T	F
a	✗	
b		✗
c	✗	
d	✗	
e	✗	

12	T	F
a		
b		
c		
d		
e		

13	T	F
a	✗	
b	✗	
c		✗
d		✗
e		✗

14	T	F
a	✗	
b		✗
c		
d		
e		

15	T	F
a	✗	
b	✗	
c	✗	
d		✗
e		✗

16	T	F
a	✗	
b	✗	
c		✗
d		✗
e		✗

17	T	F
a		
b		
c		
d		
e		

18	T	F
a	✗	
b		
c		✗
d		
e		

19	T	F
a	✗	
b	✗	
c	✗	
d		
e		

20	T	F
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c		
d		
e		

21	T	F
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22	T	F
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b		
c		
d		
e		

23	T	F
a		
b		
c		
d		
e		

24	T	F
a		
b		
c	✗	
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e		

25	T	F
a		
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26	T	F
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26	T	F
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27	T	F
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28	T	F
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30	T	F
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