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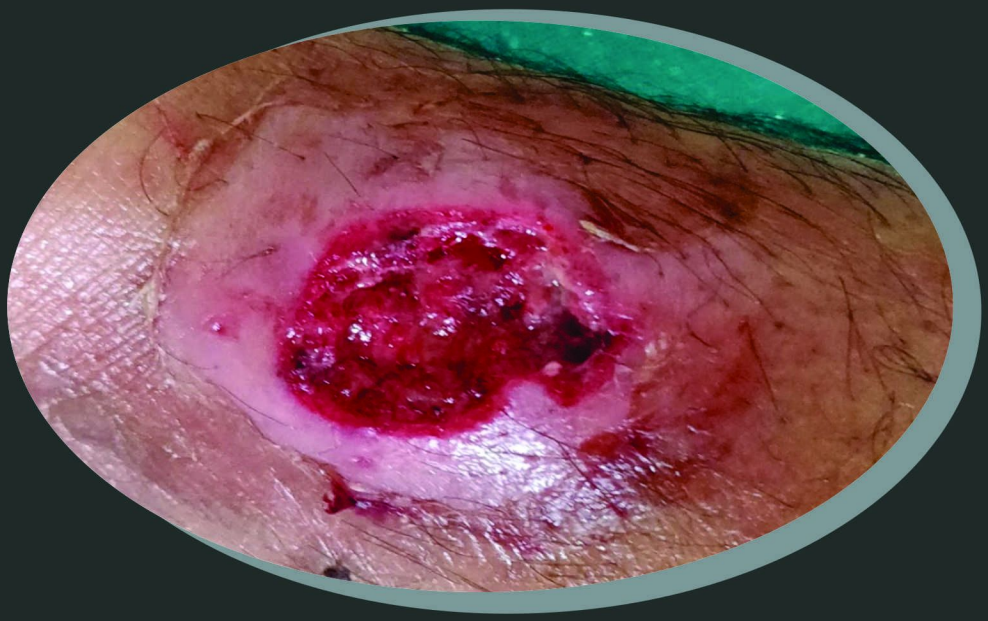
A QUICK REVIEW BOOK ON GENERAL PATHOLOGY PART-I



JV'n Dr. Bharat Sharma

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CHAPTER -1

INFLAMMATION

Inflammation is an immune response of the body against any injury. It defends the body against any injury by eliminating it from the body. In case it is unable to eliminate the harmful agent it also stops it from spreading.

Causative agents of inflammation are:

1. Infectious agents- Bacteria
2. Physical agent- Trauma
3. Chemicals- Corrosive Poisons
4. Foreign Bodies in the Body
5. Immunological agents- Antigen antibody reaction

General Features of inflammation

Almost every human shows the general features of the inflammation. The Roman writer Celsus in 1st century A.D. named the famous 4 cardinal signs of inflammation as:

i) rubor (redness);

ii) tumor (swelling);

iii) calor (heat); and

iv) dolor (pain).

fifth sign

vi) functiolaesa (loss of function) was later added by Virchow.

The function of inflammation is to eliminate the initial cause of cell injury, clear out necrotic cells and tissues damaged from the original insult and the inflammatory process, and initiate tissue repair. Almost every time it is helpful for the body to remove harmful agents to the body but in that process inflammation some time produce harmful effect on the body too.

Significance of the Inflammation

1. It defends the body against any injury
2. Limit the spread of harmful agent
3. Clear out necrotic cells
4. Further process in repair and healing of the affected part of the body.

Types of Inflammation

In general consideration the inflammation is divided into two basic types

1. **Acute inflammation**
2. **Chronic inflammation**

But some text books of pathology also give one more type of inflammation which is **subacute inflammation** which occurs between acute and chronic inflammation.

Acute Inflammation

- Acute inflammation is as name suggested short in duration and quickly resolving in its nature. Mostly it does not last more than 2-3 weeks.
- Recovery and healing occurs as soon as resolution of the acute inflammation is happened in the body.
- It is the first reaction of the body against any causative agents like trauma, chemical injury, bacterial infection etc.

- Main cells of inflammation are Neutrophils.
- Cardinal features of inflammation is present.

Chronic Inflammation

- Chronic inflammation is as name suggested occurs in long duration and its nature of recovery is very slow. It can last for months to years.
- Recurrent acute infection can lead to the chronic inflammation.
- It can also occur when any foreign body present in the body.
- Main cells of inflammation are Lymphocytes and Macrophages.
- Healing occur by fibrosis.
- Cardinal features of inflammation is absent.

Stimuli for Acute Inflammation

1. Infectious agents- Bacteria, virus fungus
2. Physical agent- Trauma, heat, cold
3. Chemicals- Corrosive Poisons
4. Foreign Bodies like splinter
5. Immunological agents- Antigen antibody reaction, hypersensitivity reaction
6. Ischemic Injury to organ leading to necrosis etc.

Pathogenesis of Acute Inflammation

Pathogenesis of the acute inflammation can be studies in two events:

1. Vascular Event
2. Cellular Event

1. Vascular Event: Vascular event of the inflammation can be further studies in the following headings

a. Haemodynamic changes in the affected part

b. Change in the permeability in the affected part

After injury to the body the following steps occur in the body:

- Firstly, transient vasoconstriction occurs
- Then persistent progressive vasodilatation occurs
- Then changes occur in hydrostatic and in oncotic pressures
- Which causes blood flow to become slow
- And then leucocyte margination occurs

After that leucocytes are emigrated from vascular space to the extra vascular space. For leucocytes emigration there is changes in the permeability of the vessels occur which causes formation of gaps in the vessel. These permeability changes occur by the following methods:

i. Contraction of endothelial cells

ii. Retraction or mild endothelial damage

iii. Direct injury to endothelial cells

iv. Leucocyte-mediated endothelial injury

v. Leakiness in neovascularisation

vi. Increased transcytosis

After that cellular changes start to occur in the body which is also the next event of the acute inflammation.

2. Cellular Event of acute inflammation

It consists of two important processes:

A. Exudation of Leucocytes

B. Phagocytosis

The above processes are described below

A. Exudation of Leucocytes: Escape of the leucocyte from microvasculature to the injured tissue space is the most important cellular event.

   The changes leading to the margination of leucocytes to microvasculature is as follow:

   I. Changes in the formed element of the blood
   II. Rolling and adhesion
   III. Emigration
   IV. Chemotaxis

B. Phagocytosis

Engulfment of solid particles by cell is known as phagocytosis or cell eating.

The most important cell which perform this phagocytosis function are

a. Polymorphonuclear Neutrophils
b. Monocytes and Macrophages

The phagocytosis involves 3 steps:

i. Recognition and attachment
ii. Engulfment
iii. Killing and degradation

i. Recognition and attachment: is occurs by the receptors which are mannose receptors, scavenger receptors and opsonin receptors.

ii. Engulfment: first there is formation of cytoplasmic pseudopods which engulf the microbes and forms the **phagosome**. Then phagosome act with the lysosome and formation of**phagolysosome**is happened. After that the final process is started which is

iii. Killing and degradation: after the formation of phagolysosome the lysosomal enzymes kills the micro-organism and finally degradation occurs by the hydrolytic enzymes.

Killing occurs in neutrophils and macrophages by following methods

a) Reactive oxygen spices
b) Reactive nitrogen spices
c) By lysosomal granules
d) Neutrophil extracellular traps

Outcomes of Acute Inflammation

Finally, when all the processes of acute inflammation is over following outcomes can be encountered in the host site:

1. Resolution

2. Regeneration

3. Healing

4. Suppuration

5. Chronic inflammation

Chronic Inflammation and Its Outcomes

- Chronic inflammation is as name suggested occurs in long duration and its nature of recovery is very slow. It can last for months to years.
- Recurrent acute infection can lead to the chronic inflammation.
- It can also occur when any foreign body present in the body.

- Main cells of inflammation are Lymphocytes and Macrophages.
- Healing occur by fibrosis.
- Cardinal features of inflammation is absent.

SYSTEMIC EFFECTS OF CHRONIC INFLAMMATION

Chronic inflammation is associated with following systemic features:

1. Fever

2. Anaemia

3. Leucocytosis

4. ESR is elevated

5. Amyloidosis

Outcomes of Chronic Inflammation are

- Resolution
- Healing (regeneration, fibrosis)
- Dystrophic calcification

CHAPTER-2

REPAIR AND HEALING

Healing is a process by which body restores the tissue which has been lost during the process of inflammation. That is why, for both acute and chronic inflammation the outcome of both inflammation **healing** is the common one.

Healing mainly involves two process

1. Regeneration- results in the complete restoration of the original tissue.
2. Repair- results in the restoration of the tissue by the fibrosis process which leads to the formation of the scar.

I. Regeneration

- Regeneration is a process by which healing occurs and complete restoration of the original tissue is done.
- In order to maintain proper structure of tissues, cells are under the constant regulatory control of their cell cycle.
- The cells of the body are divided into 3 types
    a. Labial cells- continually multiplying cells e.g. cells of epidermis.
    b. Stable cells- Once they mature they stop to multiply and become stable e.g. hepatocytes. But whenever there is stimulation to these cells they start to multiply again.
    c. Permanent cells- These cells lose their ability to multiply once maturation of these cells occur e.g. neurons.

- The process of regeneration is almost always occurring in following process:

- Proliferation and Reduction of the Gap by original cells: -Firstly the proliferation of the remaining original cell on the part of the injury occurred so the gap which is formed during the process of injury can be reduced.
- Margination and Differentiation of cells: - now proliferated cells comes closer to reduce the gap and then subsequent differentiation occur in the cell to form the tissue
- By this process complete restoration of the original tissue is done.

II. Repair

- Repair is the process by which restoration of the tissue is done by the fibrosis process which leads to the formation of the scar.
- There are two processes which are involved in the process of repair:
  a. Formation of the granulation tissue
  b. Contraction of the wound
- Three phases are observed in the formation of the granulation tissue., These are
  - Phase of inflammation
  - Phase of clearance and
  - Phase of Ingrowth of Granulation tissue-
    (i) Angiogenesis
    (ii) Fibrogenesis
- After that wound contraction occur so that the tissue defect can be reduced and the wound is slowly-slowly decrease in the size by the activity of the myofibroblast which forms the collegenfibers and cause the wound to contracts in its size.

CHAPTER-3

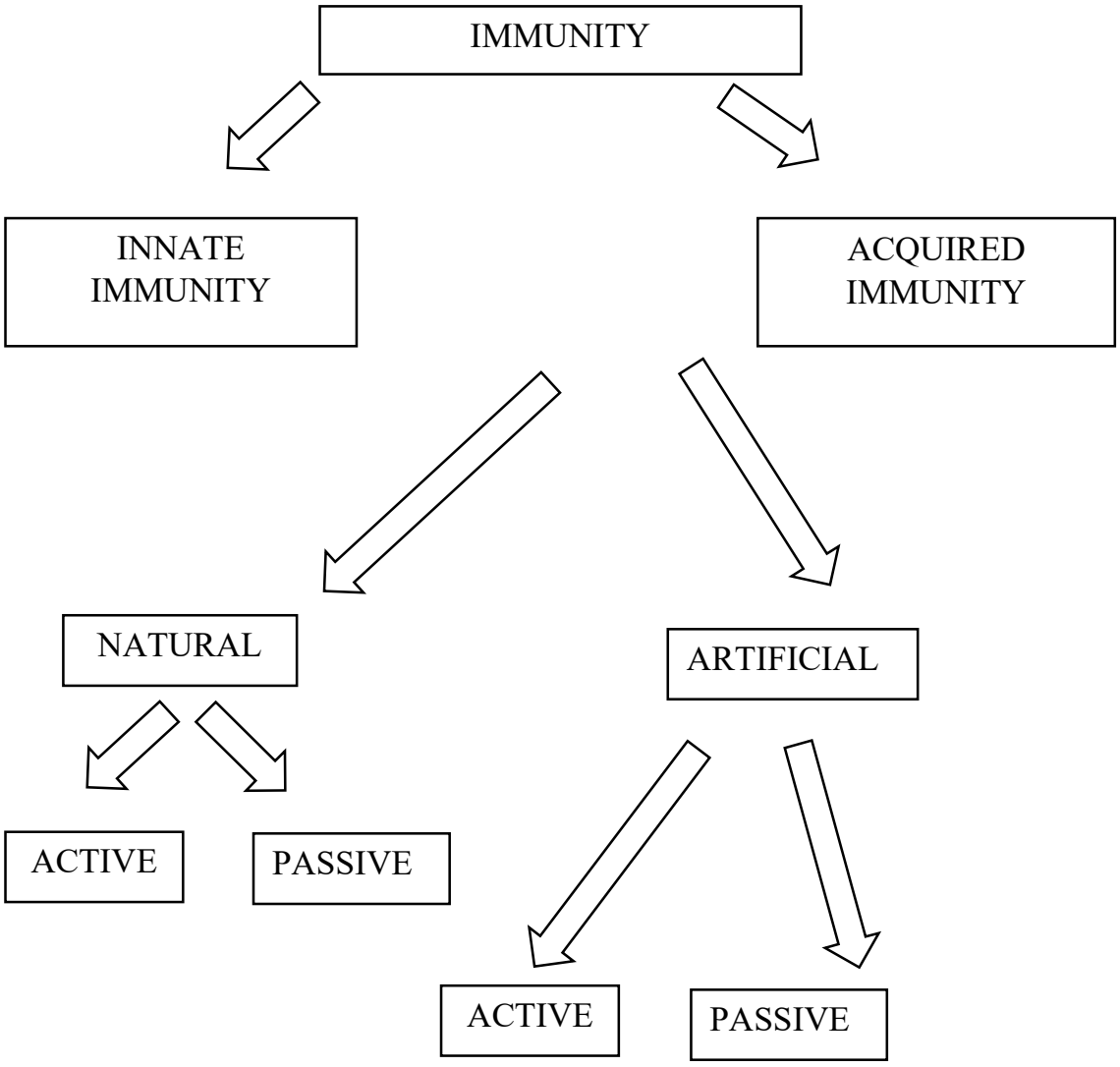
BASICS OF IMMUNITY

- Immunity is the ability of the body to protect against the pathogen or infections.
- Webster dictionary define immunity as a condition of being able to resist a particular disease especially through preventing development of a pathogenic microorganism or by counteracting the effects of its products.
- Immunity is of two types:

  I. Innate immunity

  II. Acquired immunity

- **ANTIGEN** is a toxin or other foreign substance which induces an immune response in the body, especially the production of antibodies.
- **ANTIBODY** is an immunoglobulin, a specialized immune protein, produced because of the introduction of an antigen into the body.
- To function properly, an immune system must detect a wide variety of agents, from viruses to parasitic worms, and distinguish them from the organism's own healthy tissue.



I. Innate immunity

- It is also called natural or native immunity or non-specific immunity.
- Innate immunity is the inborn capacity of the body to resist pathogens.
- If the organisms enter the body, innate immunity eliminates them before the development of any disease.
- It represents the first line of defence against any type of pathogens.

II. Acquired immunity

- Acquired immunity is the resistance developed in the body against any specific foreign body like bacteria, viruses, toxins, vaccines or transplanted tissues.
- This type of immunity is also known as specific immunity.
- It is the most powerful immune mechanism that protects the body from the invading organisms or toxic substances.
- Lymphocytes are responsible for acquired immunity.

There are two main mechanisms of immunity within the adaptive immune system – humoral and cellular.

- Humoral immunity is also called antibody-mediated immunity. With assistance from helper T cells, B cells will differentiate into plasma B cells that can produce antibodies against a specific antigen. The humoral immune system deals with antigens from pathogens that are freely circulating, or outside the infected cells. Antibodies are produces against them and by the process of phagocytosis degradation occurs.
- Cellular immunity occurs inside infected cells and is mediated by T lymphocytes. The pathogen's antigens are expressed on the cell surface or on an antigen-presenting cell. Helper T cells release cytokines that help activated T cells bind to the infected cells' MHC-antigen complex and differentiate the T cell into a cytotoxic T cell. The infected cell then undergoes lysis.

Immune system is made up of

- Organ
- Tissues

ORGANS AND CELLS OF IMMUNE SYSTEM

a) Primary lymphoid organs:

i) Thymus

ii) Bone marrow

b) Secondary lymphoid organs:

i) Lymph nodes

ii) Spleen

iii) MALT Cells (Mucosa-Associated Lymphoid Tissue located in the respiratory tract and GIT) .

Immune system helps in

- Identifying the foreign pathogen
- Defending against pathogen
- Eliminating the pathogen
- Remembering the pathogen for future

All lymphocytes are released in the circulation and are differentiated into two categories.

1. T lymphocytes or T cells, which are responsible for the development of cellular immunity.
2. B lymphocytes or B cells, which are responsible for humoral immunity.

T LYMPHOCYTES

- T lymphocytes are processed in thymus.

- The processing occurs mostly during the period between just before birth and few months after birth.
- Thymus secretes a hormone called thymosin, which plays an important role in immunity. It accelerates the proliferation and activation of lymphocytes in thymus.
- It also increases the activity of lymphocytes in lymphoid tissues.

Types of T Lymphocytes

1. Helper T cells or inducer T cells.

2. Cytotoxic T cells or killer T cells.

3. Suppressor T cells.

4. Memory T cells.

- After the transformation, all the types of T lymphocytes leave the thymus and are stored in lymphoid tissues of lymph nodes, spleen, bone marrow and GI tract.

B LYMPHOCYTES

- B lymphocytes were first discovered in the bursa of Fabricius in birds, hence the name B lymphocytes.
- Bursa of Fabricius is a lymphoid organ situated near the cloaca of birds.
- Bursa is absent in mammals and the processing of B lymphocytes takes place in liver (during fetal life) and bone marrow (after birth).

Types of B Lymphocytes

1. Plasma cells
2. Memory cells

- After transformation, the B lymphocytes are stored in the lymphoid tissues of lymph nodes, spleen, bone marrow and the GI tract.

Normal Immune Response

The physiologic function of the immune system is todefence against infectious microbes. The earliest reaction to microbes is mediated by the innate immunity, which are ready to respond to microbes. These mechanisms include epithelial barriers, NK cells, phagocytes and plasma proteins (e.g., of the complement system). The reaction of innate immunity is almost always inflammation. The defence reactions of adaptive immunity develop slowly, but are more powerful, strong and specialized. Microbes and other foreign antigens are captured by Dendrintic cells and transported to lymph nodes, where the antigens are recognized by naive lymphocytes. Then lymphocytes are activated to proliferate and differentiate into effector and memory cells. Cell-mediated immunity is the reaction of T lymphocytes, designed to combat cell-associated microbes (e.g., phagocytosed microbes and microbes in the cytoplasm of infected cells). Humoral immunity is mediated by antibodies and is effective against extracellular microbes (in the circulation and mucosal lumens). CD4+ helper T cells help B cells to make antibodies, activate macrophages to destroy ingested microbes, stimulate recruitment of leukocytes, and regulate all immune responses to protein antigens. The functions of CD4+ T cells are mediated by secreted proteins called cytokines. CD8+ CTLs kill cells that express antigens in the cytoplasm that are seen as foreign (e.g., virus-infected and tumour cells). Antibodies secreted by plasma cells neutralize microbes and block their infectivity, and promote the phagocytosis and finally destruction of pathogens.

HYPERSENSITIVITY REACTIONS

Hypersensitivity is defined as an exaggerated or inappropriate state of normal immune response with onset of adverse effects on the body,

Hypersensitivity reactions are studied mainly in 4 different types to understand it properly. Depending uponthe duration of its action, on the speed of its development and type of the immune response, these 4 types ofhypersensitivity reactions are:

TYPE I: ANAPHYLACTIC (ATOPIC) REACTION

Type I hypersensitivity occurs when a person comes in a contact with an antigen for which he has been previously sensitized.It is very rapid in nature and anaphylactic immune response is developed due to this reaction.

The reaction appears within 15-30 minutes of exposure toantigen.

ETIOLOGY AND PATHOGENESIS

Type I reaction is mediated by humoralantibodiesof IgE type or regain antibodies in response to antigen. Type Ireaction includes participation by B lymphocytes and plasma cells, mastcells and basophils, neutrophils and eosinophils.

The manifestations of type I reactionmay be variable in severity and intensity. It may manifest as a local irritant(skin, nose, throat, lungs etc.), or sometimes may be severe and life-threateninganaphylaxis.

Examples of Systemic anaphylaxis:

i) Administration of antisera e.g. anti-tetanus serum (ATS).

ii) Administration of drugs e.g. penicillin.

iii) Sting by wasp or bee.

Examples of Local anaphylaxis:

i) Hay fever (seasonal allergic rhinitis) due to pollen sensitisation of

conjunctiva and nasal passages.

ii) Bronchial asthma due to allergy to inhaled allergens like house dust.

iii) Cutaneous anaphylaxis due to contact of antigen with skin characterised

by urticaria, wheal and flare.

TYPE II: ANTIBODY-MEDIATED (CYTOTOXIC) REACTION

Type II or cytotoxic reaction occurs when the body's humoral antibodiesattacks some special types of cell surface antigens and causes destruction of that particular cell.

Type II reaction too appears generally within 15-30 minutesafter exposure to antigen but in myasthenia gravis and thyroiditis it mayappear after longer duration.

ETIOLOGY AND PATHOGENESIS

Type II reactions haveparticipation by complement system, tissue macrophages, platelets, naturalkiller cells, neutrophils and eosinophils while main antibodies are IgG andIgM class. Type II hypersensitivity is tissue-specific and reaction occurs afterantibodies bind to tissue specific antigens, most often on blood cell.

Examples of type II reaction aremainly on blood cells and some other body cells and tissues.

i) Autoimmune haemolytic anaemia

ii) Transfusion reactions

iii) In Graves' disease (primary hyperthyroidism),

iv) In myasthenia gravis,

v) In type 1 diabetes mellitus, islet cell autoantibodies are formed

TYPE III: IMMUNE COMPLEX MEDIATED (ARTHUS) REACTION

Type III reactions occurs when the circulating antigen-antibody complexes deposits on the tissues, which cause the activation of the complement system. Which further activated the inflammatory reaction, resulting in cell injury.

The onset of type III reactiontakes place about 6 hours after exposure to the antigen.

ETIOLOGY AND PATHOGENESIS

Type III reaction is not tissue specificand occurs when antigen-antibody complexes fail to get removed by thebody's immune system. There are 3 types of possible etiologic factorsprecipitating type III reaction:

1. Persistence of low-grade microbial infection

2. Extrinsic environmental antigen

3. Autoimmune process

It may be mentioned here that both type II and type III reactions have
antigen-antibody complex formation but the two can be distinguished—
antigen in type II is tissue specific while in type III it is not so. Moreover, themechanism of cell injury in type II is direct but in type III it is by deposition ofantigen-antibody complex on tissues and subsequent sequence of cell injurytakes place. Type III reaction has participation by IgG and IgM antibodies,neutrophils, mast cells and complement.

Examples of Type III hypersensitivity are

i) Immune complex glomerulonephritis in which the antigen may be

glomerular basement membrane (GBM) or exogenous agents (e.g.

Streptococcal antigen).

ii) Goodpasture syndrome having GBM as antigen.

iii) SLE in which there is nuclear antigen (DNA, RNA) and there is formationof anti-nuclear and anti-DNA autoantibodies.

iv) Rheumatoid arthritis in which there is nuclear antigen.

TYPE IV: DELAYED HYPERSENSITIVITY (T CELL-MEDIATED) REACTION

Type IV or delayed hypersensitivity reaction is tissue injury by T cell-mediatedimmune response without formation of antibodies (contrary to type I, II andIII) but is instead a slow and prolonged response. The reaction occurs about24 hours after exposure to antigen and the effect is prolonged which maylast up to 14 days.

ETIOLOGY AND PATHOGENESIS

Type IV reaction involves role of mastcells and basophils, macrophages and CD8+ T cells.

Examples of Type Iv Reaction

1. Reaction against mycobacterial infection e.g. tuberculin reaction,

granulomatous reaction in tuberculosis, leprosy.

2. Reaction against virally infected cells.

3. Reaction against malignant cells in the body.

4. Reaction against organ transplantation e.g. transplant rejection, graft

versus host reaction.

CHAPTER-4

NEOPLASIA

- The word neoplasia is taken from Ancient Greek.
- Neo ("new") and plasma ("formation", "creation").
- The literary meaning of the neoplasia is new growth,
- Neoplasm is excessive new growth in the tissue which is uncontrolled.

Definition of Neoplasm

According to the different authors neoplasm have many definitions. Some common definitions are given below:

- **Neoplasm:** An abnormal mass of tissue that results when cells divide more than they should or do not die when they should. Neoplasms may be benign (not cancer), or malignant (cancer). Also called tumour.
- **Neoplasm:** A mass of tissue formed as a result of abnormal, excessive, uncoordinated, autonomous and purposeless proliferation of cells even after cessation of stimulus for growth which caused it.
- **Neoplasm:**A neoplasm is a type of abnormal and excessive growth, called neoplasia, of tissue. The growth of a neoplasm is uncoordinated with that of the normal surrounding tissue, and it persists growing abnormally, even if the original trigger is removed.This abnormal growth usually (but not always) forms a mass.When it forms a mass, it may be called a tumour.

- The branch of science dealing with the study of neoplasmsor tumours is called oncology (oncos=tumour, logos=study).
- Neoplasms may be ‘benign’ when they are slow-growingand localised without causing much difficulty to the host.
- Neoplasm can be ‘malignant’ when they proliferate rapidly, spread throughoutthe body and may eventually cause death of the host.
- Thecommon term used for all malignant tumours is cancer.
- Hippocrates (460-370 bc) coined the term karkinos for cancer of the breast. The word ‘cancer’ means crab, thus reflecting the true character of cancer since ‘it sticks to the part stubbornlylike a crab’.

All tumours either Benign or Malignant have two basic components:

1. Parenchyma: had uncontrolled proliferating cells.
2. Supportive Stroma: had the blood vessels and connective tissue which provides nutritional supply to the proliferating cells.

So the tumours have their nomenclature on the basis of the parenchymal component present in them.

- The suffix ‘-oma’ is added to denote benigntumours.
- Carcinoma- Malignant tumours of epithelial origin.
- Sarcomas- Malignant tumours of mesenchymal origin.

Some special categories of the tumours which are mixed together are:

a. Mixed tumours
b. Teratomas
c. Hamartoma
d. Choristoma

CLASSIFICATION OF TUMOURS

Tissue of origin	Benign	Malignant
Squamous epithelium	Squamous cell papilloma	Squamous cell (Epidermoid) carcinoma
Neuroectoderm	Naevus	Melanoma
Adipose tissue	Lipoma	Liposarcoma
Cartilage	Chondroma	Chondrosarcoma
Blood vessels	Haemangioma	Angiosarcoma
Lymph vessels	Lymphangioma	Lymphangiosarcoma
Nerve cells	Ganglioneuroma	Neuroblastoma
Salivary glands	Pleomorphic adenoma (mixed salivary tumour)	Malignant mixed salivary tumour
Hepatocytes	Liver cell adenoma	Hepatoma (Hepatocellular carcinoma)
Bone	Osteoma	Osteosarcoma

CHARACTERISTICS OF TUMOURS

The characteristics of tumours are described under the following headings:

I. Rate of growth

II. Cancer phenotype and stem cells

III. Clinical and gross features

IV. Microscopic features

V. Spread of tumours

   a. Local invasion or direct spread

   b. Metastasis or distant spread.

1. Rate of growth: Neoplastic cells growth is more aggressive in comparison with the normal cells. Rate of Growth of the tumor depends on Growth Factors Secreted by the tumour cells.
   a. Benign Tumour- Grows Slowly
   b. Malignant Cells- Grows Very Aggressively
2. Cancer phenotype and stem cells: In normal condition, in an organ most of the cells are of similar origin and have similar function and the growth and the functions are in control of the body system, but in neoplastic cells they losses all the body control and grow in haphazard manner and they dose not influence by the death signals and escape the death or self-suicide which are some protective features of the normal cells. Due to this imbalance excessive proliferation occurs in the cells and excessive growth occurs. They lose their structural and functional ability, mutations occur in these cells and due to over proliferation and growth they invade the surrounding tissues.
3. Clinical and gross features: Benign tumours are slow growing neoplasm and they are many time asymptomatic and they do not invade the surrounding structures. But occasionally if they are present at a very especial position may causes very sever manifestations e.g. Meningioma. But malignant tumours are the opposite of benign tumours they are more aggressively growing, mostly symptomatic and also invades the surrounding structures and one important feature of the malignant tumour is that

they spread to the distant site (metastasis).Cardinal clinical features of malignant tumours are: anaplasia (the loss of the mature or specialized features of a cell or tissue), invasiveness and metastasis.

4. Microscopic features: Some of the common patterns in tumours
    a. Epithelial tumours generally have Sheets, Acini, Columns or acini.
    b. Mesenchymal tumours have Bundles, Whorls and Separated by the intracellular matrix.

Some important morphological features of the tumours are

i. Differentiation: It is defined as the extent of morphological and functional resemblance of parenchymal tumour cells to corresponding normal cells.

    Differentiation Minimal ⟶ Well- Differentiated Tumour

    Differentiation Poor ⟶ Poorly Differentiated Tumour

ii. Anaplasia: It is lack of differentiation and is a characteristic feature of most malignant tumours. Depending upon the degree of differentiation, the extent of anaplasia is also variable i.e. poorly differentiated malignant tumours have high degree of anaplasia. Some common morphological changes that can be seen due to anaplasia are Loss of polarity of the cell, IncreasedNeucleo-cytoplasmic Ratio, pleomorphism, hyperchromatism, anisonucleosis, functional changes etc.

iii. Inflammatory Reactions: Some tumours show chronic inflammatory reaction, chiefly of lymphocytes, plasma cells and macrophages, and in some instances granulomatous reaction, as a part of the morphologic features of the tumour. This is due to cell-mediated immunologic response by the host in an attempt to destroy the tumour. In some cases, such an immune response improves the prognosis. E.g. seminoma testis, malignant melanoma of the skin etc.

5. Spread of tumours: One of the most important and differentiating feature of the malignant tumour is that they can spread from its origin site to the different sites.

   Some Common Routes via tumours can spread are

   a. Local Invasion or Direct spread to adjoining tissue.

   b. **Metastasis** or Distant Spread is defined as spread of tumour by invasion in such a way that discontinuous secondary tumour mass/masses are formed at the site of lodgement.

      Routes of the metastasis Are

      i. Lymphatic Spread

      ii. Haematogenous spread

      iii. Spread along body cavities and natural passages

DIFFERENCE BETWEEN BENIGN AND MALIGNANT TUMOR

Feature	**Benign**	**Malignant**
Boundaries	Well Differentiated	Poorly Differentiated
Surrounding Tissue	Often compressed	Usually Invade
Size	Small	Large
Pattern	Resembles to the tissue of origin	Often poor resemblance
Basal polarity	Retained	Lost
Pleomorphism	Present	Absent
N:C Ratio	Normal	Increased
Anisonucleosis	Absent	Present
Function	Well Maintained	Lost
Growth	Usually Slow	Rapid
Local invasion	Absent	Present
Metastasis	Absent	Present
Prognosis	Local Complication	Death by local and metastatic complications

GRADING AND STAGING OF CANCER

'Grading' and 'staging' are the important guiding system to a physician for the future prognosis of the neoplasm.

Grading is defined as the gross appearance and microscopic degree of differentiation of the tumour, while staging means extent of spread of the tumour within the patient. Thus, grading is done on pathological basis while staging is on clinical grounds.

GRADING

Cancers may be graded grossly and microscopically. Gross features like exophytic or fungating appearance are indicative of less malignant growth than diffusely infiltrating tumours.

Grading is largely based on 2 important histologic features:

a. the degree of anaplasia,
b. Rate of growth.

Based on these features, cancers are categorised from grade I as the most differentiated, to grade III or IV as the most undifferentiated or anaplastic.

Many systems of grading have been proposed but the one described by Broders for dividing squamous cell carcinoma into 4 grades depending upon the degree of differentiation is followed for other malignant tumours as well.

Broders' grading is as under:

Grade I: Well-differentiated (less than 25% anaplastic cells)

Grade II: Moderately-differentiated (25-50% anaplastic cells)

Grade III: Moderately-differentiated (50-75% anaplastic cells)

Grade IV: Poorly-differentiated or anaplastic (more than 75% anaplastic cells)

STAGING

The extent of spread of cancers can be assessed by 3 ways

1. by clinical examination
2. by investigations
3. by pathologic examination of the tissue removed.

Two important staging systems currently followed are:

A. TNM staging
B. AJC staging.

Currently, clinical staging of tumours does not rest on routine radiography (X-ray, ultrasound) and exploratory surgery but more modern techniques are available by which it is possible to 'stage' a malignant tumour by non-invasive techniques.

TNM staging TNM staging (T for primary tumour, N for regional nodal involvement, and M for distant metastases) was developed by the UICC (Union Internationale Contre Cancer, Geneva). For each of the 3 components namely T, N and M, numbers are added to indicate the extent of involvement, as under:

T0 to T4: In situ lesion to largest and most extensive primary tumour.

N0 to N3: No nodal involvement to widespread lymph node involvement.

M0 to M2: No metastasis to disseminated haematogenous metastases.

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