

CANCER

- A neoplasm or a new growth consists of a mass of cells which proliferate in an atypical and relentless way, and serve no useful function
- Mechanism is unknown
- Prime event is induction of change in genetic protein by virus or carcinogen
- If activated, these **oncogenes** may induce transformation initiating the first step in the formation of a neoplasm
- Mutant cells are destroyed before they proliferate because of immune surveillance or simple wastage
- Environmental agents in the host are also needed for the “promotion” of tumour growth ie. Hormone-dependent cancers

Benign Vs Malignant Features

	BENIGN	MALIGNANT
CLINICAL FEATURES		
• Growth	Slow	Rapid
• Ulceration	Unusual	Frequent
• Haemorrhage	Unusual	Frequent
• Evidence of metastasis	No	Frequent
GROSS APPEARANCE		
• Tumour edge	Smooth	Irregular
• Cut surface	Bland, Homogeneous	Variegated Due To Haemorrhage And Necrosis
• Secondary spread	No	Adjacent Structures
MICROSCOPICAL FEATURES		
• Resemblance to tissue origin	Good	Often Poor
• Cell size and shape	Uniform	Highly Variable (Pleomorphic)
• Mitotic figures	Few, All Normal	Numerous, Frequently Abnormal
• Local invasion (blood vessels...)	No	Often Present
• Dysplasia in adjacent tissues	No	Sometimes

Classification of tumours

EPITHELIUM	CARCINOMA
Squamous	Squamous carcinoma
Glandular	Adenocarcinoma
Transitional	Transicional cell carcinoma
CONNECTIVE TISSUE	SARCOMA
Fat	Liposarcoma
Muscle	Leiomyosarcoma / Rhabdomyosarcoma
Fibrous tissue	Fibrosarcoma
Cartilage	Chondrosarcoma
Bone	Osteosarcoma
SPECIAL CATEGORIES	
Bone marrow-derived	Leukaemias, lymphomas
CNS	Gliomas
Melanocyte	Melanoma

What causes cancer?

Age: Old age: Colon, lung, prostate
Young age: Leukaemias, testicles

Carcinogens may have cumulative effect

Genetic factors:

Familial polyposis
Retinoblastoma
Breast

Geographical and racial factors:

Stomach cancer in Japanese
Melanomas in Australia

Environmental agents:

Tobacco smoking
Asbestos
β-naphthylamine: transitional cell carcinoma
Vinyl chloride: liver cell carcinoma
Soot: scrotal cancer

Carcinogenic agents:

“ Multistep theory of neoplasia”: cancers do not arise from a single event but that multiple events are involved. Many of the aetiological agents do not actually induce cancer, but causes an alteration in the cell that predisposes it to further changes which eventually lead to cancer

- Chemical carcinogens
- Radiation: Electromagnetic rays
 Particulate radiation
- Viruses: Epstein-Barr
 Human papilloma virus
 Hep B
 Human T cell leukaemia virus 1

Oncogenes

Genes that can transform normal cells into cancer cells.

- Exogenous (viral oncogenes)
- Endogenous: genes that are normally present in the cell but have been altered to produce the oncogene

Oncogenes promoting cell growth:

Normal cell growth is influenced by:

Growth factors

Receptors for growth factors

Transducer molecule

- Increased growth factor production
- Increase in growth factor receptors on cell's surface
- Abnormal growth factor receptors
- Abnormal transducers that will act as if growth factor has bound to the receptor
- Nuclear acting molecules

Mechanisms of spread

- Mechanism of invasion and metastasis are obscure
- Malignant cells secrete a number of factors or "tumour-secretory" products:
 - Angiogenesis factors
 - Proteolytic enzymes
 - Prostaglandins (osteolysis)
- Lymphatic spread: carcinomas
- Venous spread
- Transcoelomic cavities
- Cerebrospinal fluid
- Arteries
- Natural history of tumours is also related to its growth rate

Clinical features

Local effects: Bleed
Discharge of excess mucus
Obstruction, compression
Pressure symptoms
Organ failure
Pain

Systemic effects: Anorexia
Asthenia
Lassitude

- Chronic anaemia or inanition
- Secretion of abnormal proteins and peptides (appropriate and inappropriate hormonal secretion): Cushing's syndrome (ACTH)
- Paraneoplastic syndromes: symptoms in cancer patients that are not readily explained by local or metastatic disease:
 - Hypercalcemia
 - Clubbing of the fingers
 - Skin disorders
 - Peripheral neuropathy
 - Cerebellar degeneration

Factors influencing prognosis

Type of tumour

Metastatic

Local: precancerous changes and in-situ carcinoma

Lesion resembles tumours known to occur in that organ

Histological grade

Crude measure of how much the tumour resembles normal tissue

Mitotic count

Tumour staging

Depends on pathological and clinical information

TNM, Duke's, Ann Arbor's for Hodgkin's

Treatment of cancer

Local excision

Radiotherapy

Chemotherapy

Endocrine-related treatment

Immunotherapy

Palliative treatment