

Patho Unité Neoplasia Cheat Sheet

by damn via cheatography.com/195477/cs/41606/

Nomenclature

Sacromas Mesenchymal tumors

Carcinomas Epithelial tumors

Epidemiology (Acquired predisposing conditions)

- 1. Chronic inflammation
- 2. Immunodeficiency states
- 3. Precursor lesion

Clinical aspects

- 1. Local effects of tumor encroachment of tissues/ organs
- 2a. Functional activity e.g. Hormone synthesis
- 2b. Paraneoplastic syndromes -> Ectopic hormone secretion
- 3. Bleeding & infections when tumor ulcerates thru adjacent surface
- 4. Rupture/infarction
- 5. Cachexia (Weakness, e.g. weight loss)

Molecular basis of cancer

Nonlethal genetic damage

Hallmark: Genetic alteration

Cancer genes (Target of genetic damage)

- 1. Oncogene (Mutated gene)
- Mutation from proto-oncogenes
- 2. Tumor suppressor genes
- Prevent uncontrolled growth
- 3. Apoptosis-regulating gene
- Overexpressed in cancer cell-> Protect against apoptosis
- 4. Regulate interactions between tumor and host cells
- Change recognition of tumor by host immune system

2. Insensitivity to Tumor suppressor signals

Retinoblastoma Gene

- Active hypophosphorylated state: Halts cell cycle
- Inactive hyperphosphorylated state
- -Heterozygosity: X Affect cell function
- Both to be inactivated to affect function

p53

- 1. Cell cycle arrest
- 2. DNA repair

2. Insensitivity to Tumor suppressor signals (cont)

3. Apoptosis

4. Evasion of cell death

- Overexpression of BCL-2 protein -> Long life

8. Evasion of immune surveillance

Host defence against tumor -- Tumor immune

Tumor antigens	Antitumor effectors
- Overexpressed cellular proteins, Oncogenic viral products, Differentiation antigens	- CD8+
- Oncogenic viral products	- NK lymphocytes
- Differentiation antigens	- Macrop- hages
	- Humoral immunity
Immune evasion	
- Immunosuppression	- Selective outgrowth
- Antigen masking	- X MHC expression

- X Costimulation

10. Tumor-promoting inflammation

- Interaction between inflammatory cell& tumor
- 1. Proliferation-promoting factor release
- 2. Growth suppressor removal
- 3. Cell death resistance

Apoptosis of CD8+

- 4. Angiogenesis
- 5. Invasion & Metastasis
- 6. Immune evasion

Benign Malignant differentiation

Benign Malignant

- Differentiation & anaplasia
- 1. Well differentiated
- 1. **Well to undifferentiated** (Anaplasia: Functional& structural differentiation loss)
- Dysplasia (Disordered growth)



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Benign Malignant differentiation (cont)

- Carcinoma in situ (Non-invasive malignant tumor)

Rate of growth

- Correlates w./ level of differentiation
- 2. Progressive & slow 2. Erratic (Unpredictable)

Local invasion

3. **No**, expansion w./ clear 4. **Yes**, infilt boundaries

4. **Yes**, infiltrate & destroy

Metastasis

(1)Seeding of body cavities (2) Lymphatic spread (3) Hematogenous spread

4. Absent

4. Frequently present

1. Self-sufficiency in growth signals

Proto-oncogenes

-Normal genes, promote proliferation

Oncogenes

-Mutant version, function anonymously w./o

growth-promoting signals

Oncoproteins

-Proteins encoded

Self-sufficient in:

1. Growth factors &

3. Transcription factors

receptors

2. Signal transd-

4. Cyclins & CDKs

uction proteins

3. Altered cellular metabolism

Warbug effect

- Aerobic situation: Distinct form of cellular metabolism
- High levels of glucose uptake
- Increased conversion of glucose to lactose via glycoyitic pathway

5. Limitless replicative potential: Telomerase

- Telomerase shorten with each cell division
- Cancer cell have enzyme that regenerate telomerase

6. Sustained angiogenesis

- Controlled by balance between angiogenesis promoter (VEGF) and inhibitors (bFGF)

7. Invasion & Metastasis

- Invasion of extracellular matrix
- a. Loosening of intracellular junctions
- b. Degradation
- c. Attatchment
- d. Migration
- Embolus: Evade WBC killing

9. Genetic instability

- Both copies of DNA repair proteins are lost
- 1. Hereditary Nonpolyposis Cancer Syndrome
- BRCA-1 & BRCA-2 (80% familial breast cancer, not sporadic-associated)

Carcinogenic Agents

1. Chemical Carcinogenesis

Initiation

- Carcinogen exposure -> permanent DNA

Promotion

- Promoter induce tumor in initiated cell (Nontumorigenic)
- Promoting agent enhance proliferation & results in cancer
- 2. Radiation Carcinogenesis
- UV rays (UVB, 280-320nm)
- Ionizing radiation (X-ray, gamma ray, particles)
- 3. Oncogenic DNA viruses
- 1. Papillomaviruses (HPV)
- 2. Epstein-Barr virus (EBV)
- 3. Hep B virus (HBV)
- 4. Kaposi sacroma herpes virus (KSHV)



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