Cancer
Nursing 101

Introduction to Cancer Unit

Definition of Cancer

1. Cancer - Large group of diseases characterized by uncontrolled growth and spread of abnormal cells. (American Cancer Society)
2. Cancer – A term for diseases in which abnormal cells divide without control. Cancer cells can invade nearby tissues & can spread through the bloodstream & lymphatic system to other parts of the body. (National Cancer Institute)

Associated Terminology

*see handout

History of Cancer

- 3000 BC: Ancient Egypt prescribed pills & pigs ears.
- 500 BC : Hippocrates - first to clarify neoplasms; Carcinoma - tumor that spread out & destroyed host.
  - Galen - described cancer as 'crab-like'
- 1000 : Cancer result of excess of one of 4 bodily humors (fluids) (Humoral theory) : blood, phlegm, yellow bile and specifically black ligle (stool).
- 1500: Pare – surgeon
- 1700 : Discovery of microscope - view abnormal cells; environmental carcinogens
- 1800 : Use of ether (1846)
  - Asepsis proved effective (1867).
  - X-rays discovered-diagnosis of cancer (1895)
  - Radium used to treat cancer (1898) .
- 1900 : National Cancer Institute founded.
  - Chemotherapy on scene in WWII
  - DNA of cell discovered; Viral oncology
  - Smoking connected with lung cancer
- 1913: ACS - 10 MD, 5 lay
- 1972 : Oncogenes discovered (cancer causing genes)
- Early 1980s: Immunology, Interferon (anti-tumor drug)
- Late 1980s: Advances in treatment (chemo – combo & adjuvant therapy; antiemetics; radiation; immunotherapy)
- 1990s: Genetic therapy / Research
- 2000s to present: Vaccines, advances in biologic therapy, continued research

Sociocultural Aspects Related to Cancer

Attitudes

- Patient's Attitude - may delay treatment, education essential.
- Nurses' Attitude – self – evaluation; nonjudgmental

Myths

- Contagious
- Uncontrolled pain
- Hidden Curse

Epidemiology - Statistics

Definitions:

- Incidence - number of newly diagnosed cases of cancer observed within given population within specified time frame. **(*new cases*)**
- Morbidity - total number of cases of a specific disease (cancer) in a specified period of time (usually one year) per unit of population alive. **(*illness rate*)**
- Mortality - total number of deaths from specific disease (cancer). **(*death rate*)**

-Higher in men than women
-Second most common cause of death in US after heart disease
-Leading cause of death in people 40-79 yrs of age

Cancer Facts & Figures

Cancer Etiology

External Factors
    Environmental carcinogens
        Chemical carcinogenesis
        Physical carcinogenesis
        Viral carcinogenesis

Personal Factors
        Immune function
        Advancing age
        Genetics

Etiological Theories

- Immune System Dysfunction: normal immune system fights off ca; neoplastic cell evades its immune-defense system; immunosuppressant's increase risk of ca.

– Factors influencing impaired immune function:
» Malnutrition

» Advancing Age
» Chronic Disease

■ Hormonal Influences: promote or inhibit effects of environment; can allow ca to progress can **also restrain or enhance ca growth**;

■ Carcinogens
– Chemicals: external- occupational

– Diet: certain food additives and contaminants; nitrates and nitrites, artificial sweeteners, increased fat, aflatoxin B fungus (peanuts, corn, rice)

– Radiation: environmental radiation, ultraviolet radiation, ionizing radiation

– Drugs: some depend on host and dosage; cytotoxic agents, immunosuppressant, estrogens, oral contraceptives, steroids

■ Viral Infections: certain viruses have been identified as carcinogenic

■ Demographic Factors
– Age- risk increased with age
– Sex- males > females
– Geographical location- exposure- pollutants
– Culture- behavioral patterns
– Race-increased incidence in African Americans- later dx

■ Psychological Factors: psychological stress increases vulnerability; personality traits- ineffective coping strategies

■ Other Factors
– Multifactorial Theory- combination
– Genetic Inheritance- certain gene inheritance, certain cancers in families, oncogene theory- all cells have potential
– Chronic wounds, trauma- polyps

Pathophysiology: Definitions

1. Oncogenesis - process by which neoplasms are produced: new growths
2. Hyperplasia - abnormal increase in number of cells; increased tissue mass
3. Metaplasia - reversible, benign change of adult cells from one type to another
4. Dysplasia - benign change of cells resulting from chronic irritation - may reverse or progress to cancer
5. Anaplasia - malignant, irreversible change of cells which regress to more primitive level (fetal)
6. Differentiated - cells with recognizable specialized structures and functions (well vs. poorly differentiated)

7. Undifferentiated - cells which have lost their capacity for specialized functions
8. Carcinogenesis - *carcinogen* causes the cell to mutate; mutated cell produces neoplastic cells with an accelerated growth pattern; process by which a normal cell undergoes malignant transformation.

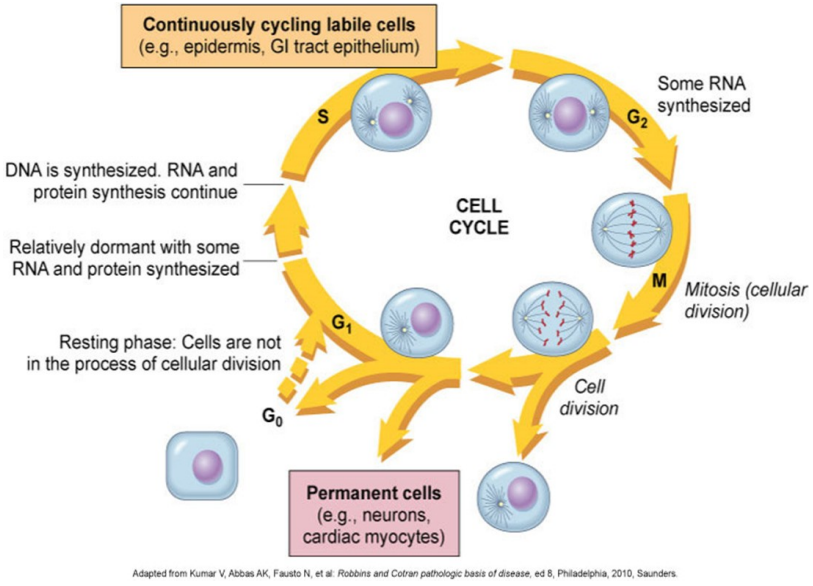
Cell Cycle

- All cells, malignant or normal, progress through 5 phases of cell cycle.
- Stages of Cell Cycle:
  - G0 – Resting Phase – perform all cell functions except proliferation
  - G1 – RNA & protein synthesis
  - S – DNA synthesis
  - G2 – Additional RNA & DNA synthesis
  - M – Mitosis – cell division

* Proliferation – cells waiting in G0 (cell death = cell growth)

Cancer cells cannot remain in G0 so they replicate continuously.

https://www.youtube.com/watch?v=IeUANxFVXKc



Cancer Cells:

- Loss of contact inhibition
- Respond differently to intracellular signals
- Divide indiscriminately

-**Proto-oncogenes-** Normal cellular genes that are important regulators on normal cellular processes; when mutated, they act as **oncogenes** (tumor-inducing)

Normal vs. Cellular Dysfunctions

■ Normal Cell Growth
– Explanation A
» Intracellular mechanism designates when cell growth is needed.
» # cell death = # cell growth

– Explanation B
» Contact Inhibition: growth is controlled by boundaries

■ Cellular Proliferation Dysfunction
- Stem cells > predetermine undifferentiated cells
– Problems with cell growth
– Genetic in origin
– DNA altered

Cancer Cell Proliferation
- Similar to tissue from which they arise
- Divide haphazardly
- DNA substituted/rearranged/mutates
- Misconception: rate of growth is greater
- Indiscriminate & continues growth: Sometimes they produce more than two cells at the time of mitosis = **pyramid effect**
- Loss of contact inhibition
- The time required for a tumor mass to double in size = **doubling time.**

■ Cellular Differentiation Dysfunction
- Normal cells: immaturity (undifferentiated) to maturity (differentiated)
- Cancer cells: genetic lock is unlocked = genetic alterations and mutations; go back to undifferentiated state

Development of Cancer

■ Initiation: 1st stage
- Exposure to agent
- Mutation of genetic structure; inherited or acquired
- Many carcinogens are detoxed, if this fails, cancer alters the DNA – cell will either die or repair

■ Promotion: 2nd stage
- Distinction between 1st and 2nd stage: **activity of promoters is reversible**
- Reversible proliferation
- Some carcinogens are **complete carcinogens** (capable of both initiating and promoting)
- Period of latency (1-40 yrs) – affected cell is hidden; depends on growth rate and environmental factors

- Critical mass- 1 cm = 1 billion cells to be evident; 1 cm= 0.4 inch (palpable) (MRI can detect 0.5 cm tumor)

■ Progression: 3rd & final stage
- Increased growth, invasiveness, metastasis
- Invasion- regionally and metastasize
- Proliferation of cells
- Loss of contact inhibition
- Secretion of lytic enzymes
- Certain affinity for site of metastasis (ie: colon cancer spreads to liver), other cancers unpredictable

Role of Immune System

(T-cells, Natural Killer Cells, Monocytes)

1. Immune response to reject or destroy cancer cells if perceived as non-self.
   a. May be inadequate as cancer cells arise from normal human cells.
2. Some cancer cells have changes on their surface antigens.
   a. Tumor-associated antigens (TAAs)
3. Response to TAAs is **termed immunologic surveillance**.

– Lymphocytes continually check cell surfaces-detect-destroy cells with abnormalities.

– Involves cytotoxic T cell, natural killer cells, macrophages, and B lymphocytes.

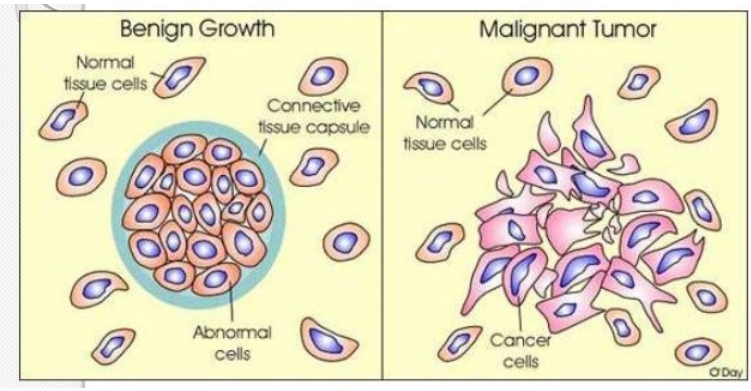
4. Escape mechanisms-CA cells <u>*evade*</u> immune system.
   a. Suppression of factors that stimulate T cells
   b. Weak surface antigens allow CA cells to "sneak through" surveillance
   c. Development of tolerance of immune system
   d. Suppression of immune response to products secreted by CA cells
   e. Induction of suppressor T cells
   f. Blocking antibodies that bind TAAs

- Oncofetal antigens: found on tumor cell surfaces and inside tumor & fetal cells; used as tumor markers; not 100% specific for tumor recurrence
  - Example: PSA (prostate), CA-125 (ovarian), CA-19-9 (pancreatic / gallbladder)

Classifications of Cancer

■ Why classify cancer?
– Form of communication
– Determine effectiveness of treatment (evaluation tool)
– Determine disease progression
– Provide statistical information

- *Site, histological grading, & extent*
- Destroy normal cells, exhaust nutrition and oxygen
- Neoplasm = new growth of tissue
- Cyst- abnormal collection of fluid
- Precancerous lesion- abnormal cell growth
- Tumor & Neoplasm are used synonymously
- Grouped by benign or malignant



■ Benign versus Malignant

– Benign characteristics:

- Latin word bene= good
- Encapsulated
- Localized
- Remains at primary site
- Well differentiated
- Slow growth
- Tends not to recur once removed
- Growth with minimal destruction

– Malignant characteristics:

- Latin word Laius= bad
- Not encapsulated
- Widely infiltrative
- Metastasizes
- Immature cells
- Rapid division
- Tends to recur upon removal
- Destructive cell growth
  - causes death unless treated
  - infection, necrosis, hemorrhage
  - cachexia

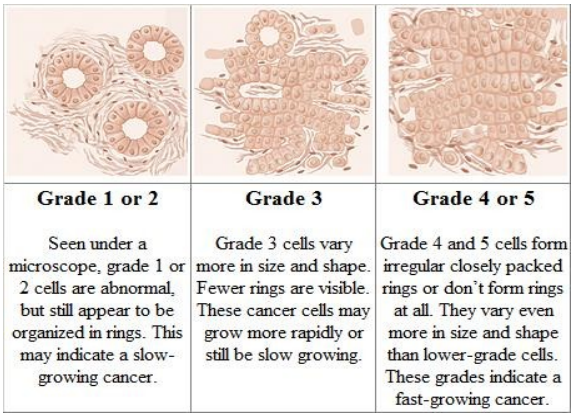
■ Anatomic Site

– Identification by tissue of origin, tissue of anatomic site, & characteristics (benign or malignant)

Site	Benign	Malignant	Example (B vs. M)
- **epithelial cells** -solid tumors - cover internal or external surfaces (skin, lung, GI, uterus)	-oma	- carcinoma	Adenoma vs. Adenocarcinoma
- **non-epithelial tissue** (muscle, bone, fat, connective tissue) - cartilage	- oma - osteoma - fibroma - chondroma	- sarcoma	Osteoma vs. Osteosarcoma
- **nervous tissue** specific for site	- oma (meningioma)	- oma (glioma)	Meningioma vs. Meningeal sarcoma
- **hematopoietic** (lymph tissue	- oma	- lymphoma	Benign Lymphoma vs. Hodgkin's/ non-Hodgkin's
- **hematological** (blood, bone marrow)		- leukemia's	ALL – Acute lymphocytic leukemia

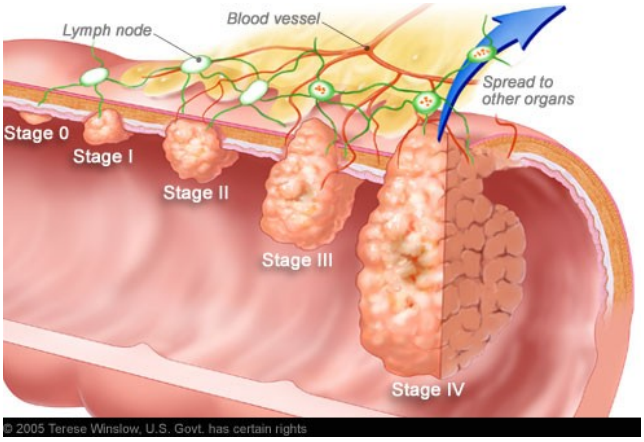
■ <u>Histologic Analysis</u>
– Classified by appearance of tissue / cells
– Biopsy needed to obtain cells

Grade I – well differentiated; differs in size, shape, and organization
Grade II – moderately differentiated; mild dysplasia
Grade III – poorly differentiated; severe dysplasia
Grade IV – undifferentiated; anaplasia
Grade X cannot be assessed



Grade 1 or 2	Grade 3	Grade 4 or 5
Seen under a microscope, grade 1 or 2 cells are abnormal, but still appear to be organized in rings. This may indicate a slow-growing cancer.	Grade 3 cells vary more in size and shape. Fewer rings are visible. These cancer cells may grow more rapidly or still be slow growing.	Grade 4 and 5 cells form irregular closely packed rings or don't form rings at all. They vary even more in size and shape than lower-grade cells. These grades indicate a fast-growing cancer.

■ Extent of Disease
– Staging – describes the extent of the disease; not looking at cell appearance
– Clinical Staging
» Stage 0 – cancer in situ
» Stage I - localized
» Stage II – limited local spread
» Stage III – regional spread, extensive local
» Stage IV – metastasis



■ Extent of Disease
– TNM Classification
» T – tumor size
» N – lymph nodes involved
» M - metastasis

TNM Classification System

Tumor	To Tis T1, T2,T3,T4	No evidence of primary tumor Tumor in situ Ascending degrees of tumor size and involvment
Nodes	No N1a, N2a N1b, N2b, N3b Nx	No abnormal regional nodes Regional nodes-no metastasis Regional lymph nodes-mets suspected; Regional nodes cannot be assessed clinically
Metastasis	Mo M1, M2, M3	No evidence of distant metastasis Ascending degrees of metastatic involvement of host including distant nodes

Metastasis

- Malignant cells detach from parent tissue & migrate; spread of cancer cells;

Difficult to treat, some resistant to chemo or radiation; most frequent sites are lungs, brain, bone, liver

- Direct Extension

  - Local
  - Infiltrates surrounding tissue> hemorrhage, necrosis, ulceration, tissue fixation
  - Not orderly
  - Wide margin excisions with surgery

- Lymphatic Spread

  - Permeation- node to node
  - Embolization - emboli

- Hematogenous Spread

  - Circulatory system
  - Vascular embolism- lung bone liver brain
  - Microemboli – small capillary beds; micrometastasis

- Seeding

  - Along serosal lining spread
  - Planted during surgery

Prevention, Detection and Control

- Nurses have the opportunity / obligation to share information with the community - education!
- Emphasize early cancer detection!

  - Educational programs
    pamphlets, guest speakers, community programs

  - Resources
    Awareness of services

  - Counseling
    Correct misinformation, provide direction, eliminate fears

Seven Warning Signs of Cancer

C - Change in bowel or bladder habits
A - A sore that does not heal
U - Unusual bleeding or discharge
T - Thickening or lump in breast or elsewhere
I - Indigestion or difficulty in swallowing
O - Obvious change in a wart or mole
N - Nagging cough or hoarseness

ACS Recommendations:
(class preparation: write what you've researched and what your classmates share)

- Breast Cancer

- Cervical Cancer

- Colorectal Cancer

- Prostate

Levels of Health Promotion (Prevention)

- Primary Prevention

- General wellness promotion

- Secondary Prevention
- Screening - Early diagnosis & treatment

- Tertiary Prevention
- Assist to highest level of wellness

Education

- Decrease exposure
- Proper nutrition
- Exercise
- Rest
- Regular MD exams
- Reduce Stress
- Allow for leisure time
- Know 7 warning signs
- Self exam
- See MD with any symptoms

Prevention Tips