**NEOPLASIA**

**All tumours have 2 basic components, what are they?**

- Tumour *parenchyma* containing the neoplastic cells
- *Reactive stroma* which is made up of connective tissue, blood vessels and variable cells of the adaptive and innate immune system

The classification of tumours and their biologic behaviour are based on their *parenchymal components*, but their growth are critically based on the stroma.

**Please fill in the blanks in the table below**

| Characteristics | BENIGN | MALIGNANT |
|---|---|---|
| **Differentiation/ anaplasia** | Well differentiated,<br>Structure still typical of tissue of origin | Some lack of differentiation to complete disorder (anaplastic),<br>Atypical structure |
| **Rate of Growth** | Usually progressive and slow<br>May come to standstill or regress<br>Rare mitotic figures and normal | Erratic<br>Maybe slow or rapid<br>Mitotic figures are numerous and abnormal |
| **Local Invasion** | Usually cohesive, expansile, non invasive well demarcated masses | Locally invasive, infiltrating surrounding tissue |
| **Metastasis** | Nil or absent | Frequent, more likely with large undifferentiated primary tumours |

**List the established environmental factors affecting cancer risk:**

- ***Infectious agents*** – 15% of all cancers in the world are either directly or indirectly caused by infectious agents (HPV in cervical, head, neck cancer)
- ***Smoking*** – single most important environmental factor to premature death (implicated in mouth, pharynx, larynx, oesophagus, pancreas, bladder, and lung)
- ***Alcohol consumption*** – alone increases risk of oropharyngeal, larynx, oesophageal carcinomas. Indirectly causes alcoholic cirrhosis which lead to hepatocellular carcinoma. Synergistic with smoking increases upper airways and GIT malignancies
- ***Diet*** – although precise dietary factors remain a matter of debate

- ***Obesity*** – men has 52% and women 62% higher cancer death rates than their slimmer counterparts
- ***Reproductive history*** – long cumulative exposure of estrogen without opposing progesterone
- ***Environmental carcinogens*** – UV, asbestos, arsenic

**Name some antitumour mechanisms**

Can be divided to *cell-mediated* and *humoral*
Cell mediated
- Cytotoxic T lymphocytes
- NK cells
- Macrophages

Humoral
- Activation of the complement cascade
- Induction of antibody dependent cellular cytotoxic (ADCC) cascade by NK cells

**What are Tumour Markers?**

Biochemical indicators of the presence of tumours. They include cell surface antigens, cytoplasmic proteins, enzymes and hormones.

In clinical practice, they refer to the molecule that can be detected in plasma or other body fluids. However, not to be construed as primary modalities of cancer diagnosis, but rather ancillary testing to support the diagnosis.

**Please name examples and state which tumour it is most associated with.**

Remember…markers can be hormones, oncofoetal antigens, isoenzymes, specific proteins, or mucins
hCG – nonseminomatous testicular tumours, trophoblastic tumour
CEA – Ca of colon, pancreas, lung, stomach and breast
alpha fetoprotein – liver, germ cell tumours
PSA – prostate
Immunoglobulins – multiple myelomas, gammopathies
Mucins : CA 125 - ovarian
CA 19-9 – pancreatic
CA 15-3 - breast