A NEET SS (SURGERY) PREPARATION COURSE BY MARROW, WITH A TEAM OF SELECTED SUPER-SPECIALITY FACULTY

SURGERY NEET SS

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PRINCIPLES OF CANCER STAGING

Introduction

00:00:10

Purpose : To know the extent of the disease.

TNM Staging :
most widely used staging system.
Anatomical staging system.
3 components :

1. T category : Primary tumor.
   - T x.
   - T 0.
   - T is.
   - T 1. } Invasive
   - T 2. } Invasive
   - T 3. } Invasive
   - T 4. } Invasive
2. N category : Regional lymph nodes (LN).
   - N 0 : No nodes.
   - N 1.
   - N 2.
   - N 3.
3. M category : Distant metastasis.
   - M 0 : No distant metastasis.
   - M 1 : Distant metastasis present.
     Has sub-categories like a, b, c, d.

Evidence based system : Upper stage → ↓ Survival.
Eg.: Breast cancer :
T 1 : <2 cm. T 2 : 2-5 cm. T 3 : >5 cm.
1.8 cm and 1.9 cm tumors : No difference in survival.
1.9 cm and 2.1 cm tumors : Sharp difference in survival.
∴ Cut-off for upper stage is 2 cm.

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2 Surgical Oncology 00

Staging Groups

00:04:10

Group	Based on
cTNM	Clinical examination Radiological examination Surgical exploration without resection
pTNM	Pathology of resected tumor
yTNM : • ycTNM • ypTNM	Post-Neoadjuvant therapy (NACT)
rTNM : • rcTNM • rpTNM	Recurrence
aTNM	Autopsy (incidental detection)

ycTNM : Clinical/radiological examination post-NACT.
ypTNM : Pathology of resected tumor post-NACT.
rcTNM : Clinical/radiological examination of recurrence.
rpTNM : Pathology of resected tumor of recurrence.

TNM Staging

00:09:40

T (Primary tumour) :
Tx : Cannot be assessed/Information not available.
Eg:
- Primary tumor operated elsewhere with no records.
- Extensive tumor where the primary cannot be identified.

T 0 : No primary tumor.
T is : In situ.
T 1–T 4 : Invasive.

N (Regional nodes) :
N x : Cannot be assessed.
N 0 : No nodes
N 1–N 3 : Nodes present.

M (distant metastasis) :

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M 0 : No distant metastasis.

M 1 : Distant metastasis present.

No M x.

Multiple tumors : Highest T mentioned.

Eg.: Breast cancer :

- 3 tumors are present with largest being 6 cm.
- Staging : pT3 (m)/N 0/M 0 (or) pT 3 (3)/N 0/M 0 where (m) means multiple.
- Actual number of tumors can also be specified like (3).

Synchronous Vs. Metachronous :

- Cut-off of appearance of multiple tumors is 4 months from the diagnosis of primary.
- <4 months : Synchronous.
- >4 months : Metachronous.
- Metachronous malignancies are staged separately.
- Synchronous malignancies are staged together.
- Paired organs like lung included in the staging criteria.

Unknown primary :

- Evidence of nodal spread is present.
- Expected primary site does not show up.
- Categorized as T 0.
- Eg.:
  a. Axillary nodes present, no primary seen in breast.
  b. Clinically → cT 0.
  c. Mastectomy is done and no primary is found → pT0.
  d. Staging (as per suspected primary site) → Ca. Breast, T 0/N 1/M 0, Stage II.

Regional nodes :

Sentinel node :

- Represented as (sn).
- If only sentinel node biopsy is done then (sn) can be used.
- If complete dissection is done, then (sn) cannot be used.

FNAC proven nodes :

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- Represented as (f).
- Eg: FNAC proven N1 : pN1 (f).

Isolated tumor cells :

- Cluster of < 200 cells.
- Size < 0.2 mm.
- Represented as (i+). Eg: pN0 (i+).
- It represents in-transit disease and not something that stations and proliferates.
- It is not considered as node +ve.
- Isolated cells are also considered node positive in aggressive malignancies :
  a. melanoma.
  b. merkel cell carcinoma.

Stage 0

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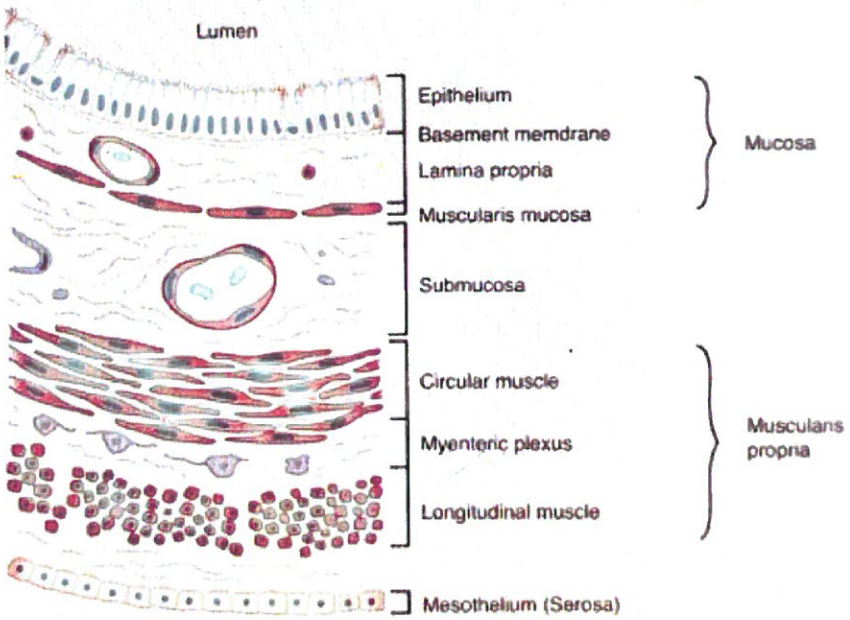
In situ and non-invasive cancers.



Layers of GIT

Area below the serosa : Subserosa.

If serosa is absent, it is called Adventitia.

1. In situ : Not crossed a boundary to attain spread (no

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spreading potential).

- Boundary can be :
  a. Basement membrane : Oral cavity.
  b. Muscularis mucosae : Colon.
- No potential to spread.
- Nodal / distant assessment not needed.

2. Complete Pathological response :
   - Seen when tumor disappears after NACT.
   - T 0/N 0/m 0.
   - Not Stage 0 (stage 0 → in-situ).

3. Non-Invasive : Disease has not crossed basement membrane of epithelium.
   - Very few cancers.
   - Represented as T a.
   - Eg : Bladder cancer : pT a/N 0/m 0.

4. DCIS :
   - Can have nodal spread.
   - Invasive component maybe missed on pathology.

ETIOLOGY OF CANCER I

Etiology & factors responsible for carcinogenesis

00:00:10

Rudolf Virchow proposed that lymphoreticular infiltrate in a tumor originates from chronic inflammation.

Types of inflammation :

Tumor intrinsic	Tumor extrinsic
Cancer initiates and amplifies the inflammatory pathway → Promote survival, growth & invasion.	macroscopic environment of tumor contributes to carcinogenesis.
E.g. : • Aflatoxin & aspergillus (↑ses mutagenesis) causing HCC. • RET mutations → Non invasive follicular thyroid neoplasm → Promotes tumor development (promotion of inflammatory pathway)	E.g. : • Chronic pancreatitis → Pancreatic carcinoma. • H pylori → Stomach cancer. • GERD → Esophageal ca. • Hepatitis → HCC.

Infections causing cancer :

Cancer	Infection
Bladder cancer	Schistosoma haematobium
Burkitt's lymphoma	EBV. HHPV 4
Cervical cancer	HPV
Cholangiocarcinoma	Salmonella typhi, Opisthorchis viverrini, Clonorchis sinensis
Colorectal cancer	JC virus, Streptococcus bovis
Glioma	JC virus

HCC	Hepatitis B, C, D, Schistosoma japonicum, Aflatoxin
Hodgkin's lymphoma	EBV
merkel cell cancer	merkel cell polyoma virus
mesothelioma	SV 40
Adult T cell leukemia/ lymphoma	HTLV I
Prostate cancer	Xenotropic murine leukemia virus
Kaposi's sarcoma	HHV 8

Inflammatory mediators

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The following have a role in interaction b/w tumor & host immune cells (cytokines) :

- Chemokines.
- Interleukins : IL-1, IL-6, IL-8, IL-17.
- Interferons : I (α & β), II (γ), III ($\Delta 1$, $\Delta 2$ & $\Delta 3$).
- Prostaglandins.
- TNFα.

TNF : 1° mediator of inflammation.

NFKB pathway :

- major role in cancer.
- Activator of TNF.
- Initiation & transformation.

mechanism :

Inflammation → Cytokines → Promote release of inflammatory cells → Oxidative damage, DNA mutation → microenvironment in tissue is more conducive to increased cell growth, survival & transformation.

Survival of cell :

- Pro-inflammatory cytokines : IL-1β, IL-8, TNFα & CRP
  ↑sed levels → Reduced survival (poor prognosis).

- STAT 6 & STAT 3 ↑ expression (↑ inflammation) → inverse association of survival in mesothelioma.

Invasion :

- mmP 9 (matrix metalloproteinase 9) :
  a. Gelatinase which degrades type IV collagen.
  b. High expression shows poor prognosis (High chance of tumor invasion).
- HIFα : Increased vascular invasion in HCC → Poor prognosis.
- Cathepsin D : Increased association in inflammatory breast cancer.

Angiogenesis :

Pro-angiogenic factors
TNFα
IL-1β
IL-8

Factors for angiogenesis
MIF : Endothelial cell activation
TGFβ (Head & neck SCC)
Angiopoietin-2

Factors for metastasis
VEGF
FGF 2
PDGF
ICAM-1
VCAM-1
E-selectin
P-selectin
mmP-9

Molecular mechanism of carcinogenesis 00:18:00

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1. NFKB pathway : Pro-tumorogenic.

mechanism :

i. Chronic inflammation → EMT (epithelial mesenchymal transformation) activation → ↑ sed cell survival by promoting anti-apoptotic proteins → MYC & BCL-XL.

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