

Surgical Oncology

Volume - 1

MARROW
— Super Speciality

Instructions

- The notes must be used in conjunction with the Marrow SS Surgery videos and should not be considered standalone material.

Please note:

- The information in this book has been printed based on the transcript of the Marrow SS Surgery videos.
- The information contained in this book is for educational purposes only. The content provided is not intended to substitute for professional medical advice, diagnosis or treatment.
- This book cannot be sold separately. It has been made available to only select eligible users who have an active subscription to Marrow SS Surgery videos.
- The text, images, slides, and other materials used in this book have been contributed by the faculty, who are subject matter experts. We have merely reproduced them as video transcripts in this book.
- The notes have been consciously designed in a way that is concise and revisable. To ensure this, we have intentionally added only the most relevant modules and images that are needed for you.
- Reasonable care has been taken to ensure the accuracy of the information provided in this book. Neither the faculty nor Marrow takes any responsibility for any liability or damages resulting from applying the information provided in this book.
- This set contains notes of all main videos published in the app until August 2025. You can find notes for any additional videos published after this date within the app under the videos section.

All Rights Reserved

No part of this publication shall be reproduced, copied, transmitted, adapted, modified or stored in any form or by any means, electronic, photocopying, recording or otherwise.

© Marrow

// ME821-PPL

Contents

Volume - 1

Basics of Oncology

1.	Principles of Cancer Staging	1
2.	Etiology of Cancer I	4
3.	Etiology of Cancer II	11
4.	Hallmarks of Cancer	14
5.	Oncogenic Viruses I	20
6.	Oncogenic Viruses II	23

Essentials of Cancer Therapy

7.	Cancer Screening	26
8.	Radiotherapy in Oncology I	32
9.	Radiotherapy in Oncology II	40
10.	Tobacco Use & Cancer Patients	45
11.	Role of Surgery in Cancer Prevention	50
12.	Response Evaluation Criteria in Solid Tumors	58
13.	Oncologic Emergencies	62

Head and Neck Oncology

14.	Salivary Gland Tumor	79
15.	Overview of Head and Neck Cancers	86
16.	Principles of Surgery in Malignancy of The Oral Cavity	100
17.	Oral Cavity and Oropharyngeal Cancers	110
18.	Hypopharyngeal, Nasal and Sinus Cancers	124
19.	AJCC Staging of Oral Cancers (8 th Edition)	131

20.	Neck Dissection	138
21.	Guidelines - Laryngeal Cancer Management	151
22.	Guidelines - Oropharyngeal Cancer Management	158
23.	Guidelines - Nasopharyngeal Cancer Management	165
24.	Human Papilloma Virus in Head & Neck Cancers	170

Thoracic Oncology

25.	Non Small Cell Lung Cancer I	175
26.	Non Small Cell Lung Cancer II	187
27.	Non Small Cell Lung Cancer III	202
28.	Small Cell Lung Cancer	210
29.	NET of Lung	216
30.	Mediastinal Neoplasms	221
31.	Mesothelioma	232

Cancer of the GIT

32.	Esophageal Cancer : Introduction & Evaluation	243
33.	Esophageal Cancer : Management	250
34.	Gastroesophageal Junction Tumors	254
35.	Gastric Cancer I	261
36.	Gastric Cancer II	266
37.	Small Bowel Tumors	272
38.	Colon Cancer - Anatomy	278
39.	Colon Cancer Management	282
40.	Management of Polyps	291
41.	Rectal Cancer	297
42.	Multimodality Management of Rectal Cancers	303
43.	Carcinoma Appendix	310

44.	Management of Appendiceal Tumors	314
45.	Anatomy of Liver, Types of Liver Resections and ALPPS	332
46.	Hepatocellular Carcinoma (HCC) : Clinical Features and Evaluation	340
47.	HCC : Staging and Management	346
48.	Gallbladder Cancer	357
49.	Bile Duct Cancer	367
50.	Pancreatic Cancer	377
51.	Gastrointestinal Stromal Tumors (GIST)	390
52.	GIST : Updates	400

Volume - 2

Genitourinary Cancers

53.	Kidney Cancer	405
54.	Bladder Cancer I	412
55.	Bladder Cancer II	417
56.	Penile Cancer	421
57.	Testicular Cancer I	428
58.	Testicular Cancer II	433

Gynecological Cancers

59.	Cervical Cancer	441
60.	Cervical Intraepithelial Neoplasia	444
61.	Carcinoma Endometrium	453
62.	Lymphadenectomy in CA Endometrium	461
63.	Molecular Classification of Endometrial Carcinoma	467
64.	Ovarian Cancer I	476
65.	Ovarian Cancer II	484

66.	Ovarian Cancer III	493
67.	Role of CRS & HIPEC in CA Ovary	503
68.	Vaginal Carcinoma	512
69.	Vulvar Carcinoma	515
70.	Gestational Trophoblastic Neoplasia (GTN)	519
71.	Human Papilloma Virus Vaccine	524

Endocrine Tumors

72.	Pancreatic Neuroendocrine Tumors (NET) I	531
73.	Pancreatic Neuroendocrine Tumors (NET) II	534
74.	Differentiated Thyroid Cancer I	541
75.	Differentiated Thyroid Cancer II	550
76.	Differentiated Thyroid Cancer III	559
77.	Differentiated Thyroid Cancer IV	572
78.	Medullary Thyroid Cancer	577
79.	Thyroid Carcinoma : Radioiodine Therapy	582
80.	Parathyroid Tumors	588
81.	Adrenal Tumors	594
82.	Pheochromocytoma	603
83.	Men Syndrome I	608
84.	Men Syndrome II	617

Bone, Soft Tissue and Skin

85.	Skin Malignancy : Introduction	622
86.	Malignant Melanoma : Molecular Biology	628
87.	Malignant Melanoma : Subtypes & Staging	633
88.	Malignant Melanoma Evaluation and Management	642
89.	Malignant Melanoma : Adjuvant Therapy	652

90.	Malignant Melanoma : Updates	658
91.	Basal Cell Carcinoma I	659
92.	Basal Cell Carcinoma II	663
93.	Squamous Cell Carcinoma	666
94.	Skin Adnexal Tumor I	671
95.	Skin Adnexal Tumor II	678
96.	Skin malignancy : Image Based Discussion	685
97.	Bone Tumors I	691
98.	Bone Tumors II	698
99.	Soft Tissue Sarcomas I	706
100.	Soft Tissue Sarcomas II	715
101.	Recurrent, Metastatic & Retroperitoneal Soft Tissue Sarcoma	724

Breast and Other Cancers

102.	Breast I	728
103.	Breast II	742
104.	Breast III	756
105.	Breast IV	771
106.	Carcinoma of Unknown Primary	781
107.	Ductal Carcinoma In Situ and Management	791
108.	Oncoplastic Breast Surgery	799

PRINCIPLES OF CANCER STAGING

----- Active space -----

Introduction

00:00:10

Purpose : To know the **extent of the disease**.

TNM Staging :

- most widely used staging system (Anatomical staging system).
- 3 components : TNM.

T category (Primary tumour) :

- invasive : T1/T2/T3/T4.
- Others : TX/T0/Tis.

N category : Regional Lymph nodes (LN).

- N0 : No nodes.
- N1.
- N2.
- N3.

M category : Distant metastasis.

- M0 : No distant metastasis.
- M1 : Distant **metastasis present** (Has sub-categories like a, b, c, d).

Note : It is an evidence based system (upper stage → ↓ Survival).

Eg., Breast cancer :

- T1 : <2 cm. T2 : 2-5 cm. T3 : >5 cm.
- 1.8 cm and 1.9 cm tumours : No difference in survival.
- 1.9 cm and 2.1 cm tumours : Sharp difference in survival.
- Therefore cut-off for upper stage is 2 cm.

Staging groups :

Group	Based on	
cTNM	<ul style="list-style-type: none"> • Clinical/radiological examination • Surgical exploration without resection 	
pTNM	Pathology of resected tumour	
yTNM	Post-Neoadjuvant therapy (NACT)	
	ycTNM	Clinical/radiological examination post-NACT
	ypTNM	Pathology of resected tumour post-NACT
rTNM	Recurrence	
	rcTNM	Clinical/radiological examination of recurrence
	rpTNM	Pathology of resected tumour of recurrence
aTNM	Autopsy (incidental detection)	

----- Active space -----

TNM staging

00:09:40

T Primary tumor	N (Regional nodes)	m (Distant metastasis)
<p>Tx : Cannot be assessed/information not available.</p> <ul style="list-style-type: none"> Eg., Primary tumour operated elsewhere with no records. Extensive tumour where the 1° cannot be identified. <p>T0 : No primary tumour.</p> <p>Tis : in situ.</p> <p>T1-T4 : invasive.</p>	<p>Nx : Cannot assess.</p> <p>N0 : No nodes.</p> <p>N1- N3 : Nodes present</p>	<p>m0 : No distant metastasis.</p> <p>m1 : Distant metastasis.</p>

multiple tumours :

Highest T mentioned

E.g., breast cancer : 3 tumours are present with largest being 6 cm.

- Staging : pT3 (m)/N0/m0 (or) pT3 (3)/N0/m0 where (m) means multiple.
- Actual number of tumours can also be specified like (3).

Synchronous Vs. Metachronous :

- Cut-off is 4 months.
- <4 months is synchronous and >4 months is metachronous.

Unknown primary :

- Evidence of **nodal spread is present**, expected primary site doesn't show up.
- Categorised as T0.
- Example. :
 - Axillary nodes present, no primary seen in breast, clinically → cT0.
 - mastectomy is done and no primary is found → pT0.
 - Staging (As per suspected 1° site) → Ca. Breast, T0/N1/m0, 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 :

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

Isolated tumour cells :

- Cluster of <200 cells.
- Size <0.2 mm.
- Represented as (i+). Eg: pNI (i+).
- It represents in-transit disease & not something that stations & proliferates.
- It is not considered as node positive.
- Except : Isolated cells are also considered node positive.
 - melanoma.
 - merkel cell carcinoma.

----- Active space -----

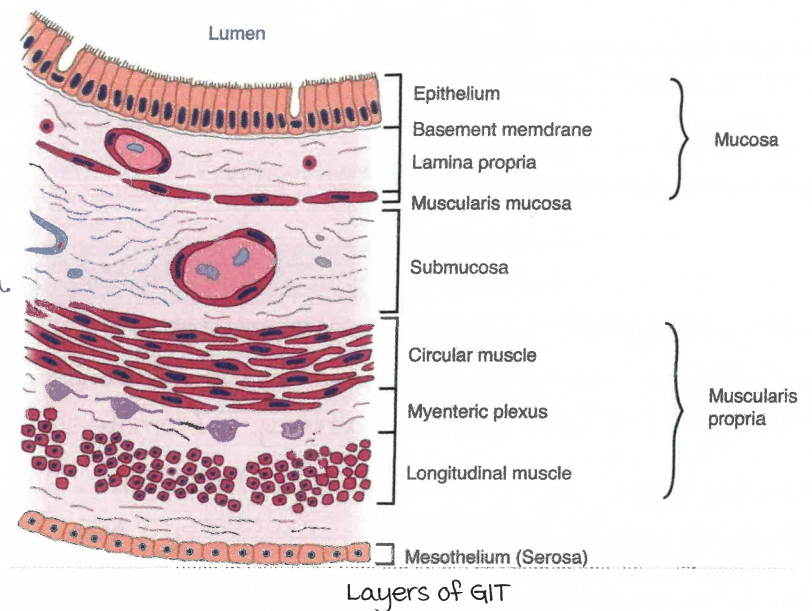
Stage 0 :

In situ and non-invasive cancers.

- Area below the serosa : Subserosa.
- If serosa is absent, it is called adventitia.

Definition :

- Non-invasive : Disease has not crossed basement membrane of epithelium.
- in situ : Disease has not attained spread potential.



In situ :

- Not crossed a boundary to attain spread.
- Boundary can be :
 - Basement membrane : Oral cavity.
 - muscularis mucosa : Colon.
- No potential to spread.
- Nodal/Distant assessment not needed.

Complete pathological response :

- Seen when tumour disappears after NACT.
- Represented as : ycTO/NO/mo.

Non-invasive :

- Represented as Ta.
- Eg: Bladder cancer : pTa/NO/mo.

DCIS :

- Can have nodal spread.
- invasive component maybe missed on pathology.

----- Active space -----

ETIOLOGY OF CANCER I

Introduction

00:00:10

Factors responsible for carcinogenesis :

Inflammation :

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
RET mutations → Non invasive follicular thyroid neoplasm → Promotes tumor development (Promotion of inflammatory pathway)	
E.g. : <ul style="list-style-type: none"> • Aflatoxin & aspergillus causing HCC. • RET mutations. 	E.g. : <ul style="list-style-type: none"> • C/c pancreatitis → Pancreatic carcinoma • H pylori → Stomach cancer • GERD → Esophageal ca • Hepatitis → HCC

Infections causing cancer :

Long standing infections can cause cancer.

----- Active space -----

Cancer	Infection
Bladder cancer	Schistosoma hematobium
Burkitt's lymphoma	EBV
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

Inflammatory Mediators

00:08:48

Cytokines :

- Cytokines have a role in interaction b/w tumor & host immune cells.
- Cytokines are further divided into :
 - Chemokines.
 - Interleukins : IL-1, IL-6, IL-8, IL-17.
 - Interferons : I (α & β), II (γ), III (Δ_1, Δ_2 & Δ_3).
 - Prostaglandins.
 - TNF α .
- TNF : 1^o mediator of inflammation.

NF κ B pathway :

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

----- Active space -----

Inflammation → Cytokines → 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-1 β , IL-8, TNF α & CRP.
- Increase in their levels results in reduced survival (Poor prognosis).
- STAT 6 & STAT 3 high expression showed inverse association of survival in mesothelioma.

Invasion :

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

Angiogenesis :

Pro-angiogenic factors
TNF α
IL-1 β
IL-8

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

Factors for angiogenesis
MIF : endothelial cell activation
TGF β
Angiopoietin-2

Molecular mechanism of carcinogenesis

00:18:05

NF κ B pathway : Protumorigenic.

C/c inflammation → EMT (Epithelial mesenchymal transformation) activation → Increased cell survival by promoting anti-apoptotic proteins → MYC & BCL-XL.

Extracellular matrix remodelling by MMP & VEGF.

STAT 1 & 3 : Persistent STAT 3 → Tumor inflammatory signal + NFκB → Tumor cell survival & angiogenesis

----- Active space -----

Inflammasome :

- Silica & asbestos can trigger inflammasome.
- Activates IL-1β & IL-8 and other mediators (Pro-inflammatory).

Toll like receptors (TLR) :

- Role in :
 - Host defense mechanism.
 - Tissue injury.
- C/c inflammation → C/c TLR pathway activation → Carcinogenesis.

Chemical factors

00:23:09

Scrotal cancer in chimney sweepers : First environmental cancer discovered by Percivall Pott.

Cancer	Chemical factor
Lung	Tobacco, asbestos, nickel
Pleura	Asbestos
Oral cavity	Tobacco, alcohol
Esophagus	Tobacco, alcohol
Gastric	Tobacco
Colon	Tobacco, alcohol
Liver	Aflatoxin, vinyl chloride, tobacco, alcohol
Kidney	Tobacco, trichloroethylene
Bladder	Tobacco, 4-amino biphenyl, 2-naphthylamine, cyclophosphamide, phenacetin
Prostate	Cadmium
Skin	Arsenic, coal tar, PAH, benzopyrenes, cyclosporin A

----- Active space -----

mechanism of chemical carcinogens :

Genotoxic	Non-genotoxic
Directly altering genetic material	Independent of direct insult
mechanism : <ul style="list-style-type: none"> • DNA adducts • Inducing DNA ssb (Single stranded breaks) & dsb (Double stranded breaks) 	MAP (mitogen activated protein) Kinase pathway (or) NFkB pathway They are epigenetic modifiers : <ul style="list-style-type: none"> • Cytotoxic • Receptor mediated (Steroid receptors & tamoxifen)
<ul style="list-style-type: none"> • Direct genotoxic : Cause cancer at site of exposure. E.g : UV induced skin cancer • Indirect genotoxic : Requires metabolic transformation from procarcinogen to carcinogen. E.g : Aflatoxin 	

Both can cause reactive oxygen species DNA damage alter gene expression.

Aristolochic acid :

- From genus of Aristolochia (Plant).
- Used as herbal remedy for weight loss.
- Class I carcinogen.
- Causes A:T to T:A transformation.
- Diseases caused :
 - a. Balkan endemic nephropathy.
 - b. Nephrotoxic : Interstitial fibrosis.
 - c. Upper tract urothelial carcinoma.

PAH (Polycyclic aromatic hydrocarbons) :

- ≥3 fused benzene rings.
- >200 chemicals.

Benzopyrene :

- most studied PAH.
- metabolized by CYP4501A1 & CYP4503A4.
- mechanism of action : DNA adducts.
- excretion : Glutathione pathway.
- Increased lung & skin cancer.
- Found in overcooked food, coal burning and tobacco smoke.

IARC group 1 pharmaceutical carcinogens

00:35:14

----- Active space -----

Drug	Cancer
Azathioprine	Non-hodgkin's lymphoma, SCC of skin, HCC, cholangiocarcinoma.
Cyclophosphamide	Bladder cancer, leukemia
Chlorambucil	Leukemia
Cyclosporine	Leukemia, lymphoma, non-melanomatous skin cancer
Tamoxifen	Endometrial cancer
Estrogen/OCP/HRT	Breast cancer, endometrial cancer

Physical factors

00:37:13

Radiations :

- Ionising radiations : Ionise molecules (Electron is displaced from orbit) by linear energy transfer (LET).
- X rays & γ rays have low LET.
- Particulate matter :
 - Electron, proton, neutron, C ion, α particles.
 - They have high LET.

m/c source of radiation exposure :

- 80% : Radon gas.
- 20% : medical sources.

mechanism of action of ionising radiations :

Direct action	Indirect action
High LET : Direct DNA damage	Low LET
Direct energy transferred to molecule.	Hydrolysis of H_2O releases OH^\cdot radical which causes DNA damage.

Both causes similar lesions in DNA.

1 Gy of ionising radiation :

- 40 dsb.
- 1000 ssb.
- 1000 single base lesions.
- 150 DNA protein crosslinks per cell.

Dsb are critical lesions \rightarrow Cell lethality.

----- Active space -----

Cell response to radiation :

- Base excision repair : For ssb.
- Homologous repair :
 - High fidelity repair.
 - For dsb.
- Non homologous end joining repair :
 - m/c mechanism of repair in ionising radiations.
 - For dsb.
 - Not accurate → Results in mutation.

Theoretical risk models for radiation induced cancer :

Linear, no threshold model :

- most accepted.
- Induction of cancer is directly proportional to dose of radiation even in low dose.

Sublinear/threshold model :

- Below threshold dose, risk is negligible.

Supralinear/stealth model :

- Doses below threshold can trigger activation of DNA damage surveillance & repair mechanism leading to suboptimal activation of cell cycle.

Linear quadratic model :

- Effect of radiation at low doses → Single tract of radiation hitting multiple targets quadratic induction rate.

vulnerable cells :

- most vulnerable :
 - Hematopoietic cell line (Leukemia except CLL) : m/c.
 - Thyroid gland.
- Intermediate : Breast, lung, salivary gland.
- Radioresistant : Skin, bone, GIT.

ETIOLOGY OF CANCER II

----- Active space -----

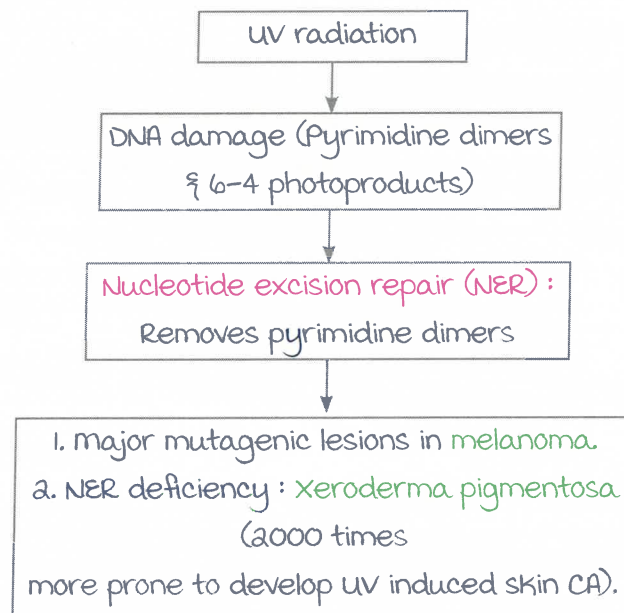
Physical factors leading to carcinogenesis

00:00:08

uv light :

- UV A (320 to 400 nm) : mainly produces ROS → Single strand breaks & base lesions in DNA.
- UV B (290 to 320 nm).
- UV C (240 to 290 nm) :
 - most damaging to DNA.
 - most of the UV C is absorbed by ozone layer.
- UV B & UV C :
 - Forms pyrimidine dimers.
 - And also 6 - 4 photoproducts that consists of covalent ring structures
→ Bending of DNA helix → Interfere in DNA synthesis.

Cellular response to UV radiation :



Asbestos :

- Contributes in causing 5 - 7 % of all lung cancers.
- Mechanism of action : ROS → Single strand breaks + base lesions.
- Asbestos + tobacco : more chance of causing lung CA.
- Tumor suppressor genes p53 & p16INK4A + K-RAS oncogene are associated with lung CA caused by asbestos.

----- Active space -----

- **malignant mesothelioma :**
 - major cause : Asbestos fibres.
 - Associated with **PI6INK4A** & **NF2** gene mutation.

Radiofrequency radiation and microwave radiation :

- Radiofrequency radiation : 3 KHz to 300 MHz.
- microwave radiation : 300 MHz to 300 GHz.
- **Cellphones :**
 - Brain peak specific absorption rate : 4 to 8 W/kg.
 - > 10 yrs of cellphone usage : Increased chance of **glioma and acoustic neuroma**.
 - It is **inconclusive** as large prospective studies have shown no risk.

Electromagnetic fields :

- **Not carcinogenic** (Energy is not high enough to break chemical bonds).

Dietary factors

00:09:33

Dietary fibre :

- All plant polysaccharide & lignin → Resistant to hydrolysis by the digestive enzymes.
- **No association b/w dietary fibre and colorectal cancer.**

	Increased incidence	Decreased incidence
Red meat	Colorectal CA	
Regular milk consumption	Prostate CA	Colorectal CA
Coffee		HCC Endometrial CA Prostate CA
Vit D		Colorectal CA Breast CA Prostate CA
Selenium (acc to SELECT RCT)	No protective effect in prostate CA.	

mechanisms of redmeat being carcinogenic :

- ↑ Anabolic hormones in red meat.
- Polycyclic aromatic hydrocarbons (Cooking at high temperature).
- ↑ Heme in red meat.
- Nitrates → Nitrosamines (In smoked, salted and processed meat).