

NEET - SS

LAST MINUTE

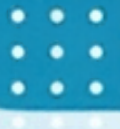
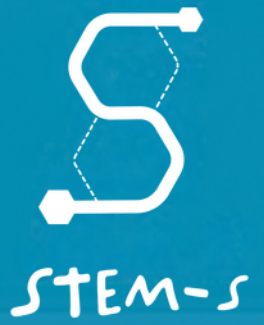
REVISION

NOTES

VERSION 2.0

MEDICAL ONCOLOGY GROUP

PART: 1



Presented By:
STEM-S

Index — Part 1

NEET-SS Last Minute Revision Notes · Medical Oncology · Page numbers as printed on the notes.
*Printed as 7 on the page.

Topic	Page
1. Breast	1-20
Molecular classification; indications for neoadjuvant chemotherapy (NACT)	1
Benefits of NACT; pathological complete response (pCR)	2
Chemotherapy regimens (AC→T, FEC→T, dose-dense); HER2-positive drugs	3
Trastuzumab cardiotoxicity; hormone-receptor-positive (pre- vs postmenopausal)	4
CDK4/6 inhibitors; triple-negative breast cancer (TNBC)	5
Metastatic breast cancer principles; bone-targeted therapy	6
BRCA-mutated breast cancer; PARP inhibitors	7
High-yield breast oncology trials	8*
Breast oncology drugs: endocrine, CDK4/6, HER2, chemotherapy, TNBC, PARP, bone agents	9-12
Drug summary tables by class	13-15
Exam traps: AI in premenopause, tamoxifen myths, trastuzumab, CDK4/6, NACT, metastatic, bone mets, HER2 status	16-19
5-line NEET SS memory traps	20
2. Gastrointestinal & Hepatobiliary	21-42
Oesophagus: histology, treatment by stage	21
Oesophagus: CROSS neoadjuvant CRT; perioperative FLOT	22
Metastatic oesophageal cancer; key points	23
Stomach: histology, molecular markers, treatment by stage, perioperative chemo	24
Older regimens; metastatic first line; targeted & immunotherapy	25
Palliative chemo principles; last-day take-home; oesophagus key trials	26
Oesophagus exam crux; gastric key trials; one-look summary	27
Colon key trials; colon exam crux; rectal key trials	28
Colorectal: polyp risk, FAP vs Lynch syndrome	29
Localized colon cancer treatment; RAS wild-type trap	30
Rectum summary; oesophagus traps (CROSS vs FLOT, SCC vs adeno)	31
Oesophagus & stomach exam traps	32-33
Stomach memory line; gall bladder (BILCAP, ABC-02)	34
Pancreas trials and summary	35
Pancreatic cancer: pathology, location, molecular core (KRAS)	36
Pancreatic cancer: resectable / borderline treatment	37
Pancreatic cancer: metastatic disease	38
Hepatocellular carcinoma (HCC): molecular pearls, aetiology & risk factors	39
HCC: BCLC staging	40
Intermediate HCC: TACE	41
Advanced HCC: treatment decision flow	42
3. Haematological Malignancies	43-79
Acute leukaemias overview: AML, ALL key facts	43
ALL prognosis & treatment; CML hallmark	44

STEM-S

Last Minute ◆ Revision ◆ (LMR)

BREAST CANCER



• Molecular Classification

Subtype	ER/PR	HER2	Ki-67	Key Treatment
Luminal A	+	-	Low (<14%)	Endocrine therapy
Luminal B	+	- / +	High	Endocrine ± Chemo
HER2-enriched	-	+	High	Anti-HER2 + Chemo
Triple Negative (TNBC)	-	-	High	Chemotherapy ± IO

•Exam Pearl

- Luminal A → least aggressive, best prognosis
- TNBC → most aggressive, early relapse

2. Indications for Neoadjuvant Chemotherapy (NACT)

•NACT preferred when:

- Tumor > 2 cm
- Node-positive disease
- HER2-positive
- Triple-negative breast cancer
- Locally advanced breast cancer

•Trastuzumab Cardiotoxicity

- NOT dose-dependent

- Reversible

Unlike anthracyclines (dose-dependent, irreversible)

- If question asks “cumulative dose toxicity” → **Anthracycline, NOT trastuzumab**

•Duration Confusion

Trastuzumab = 1 year

- 2 years gives **NO added benefit** (HERA)

- “Extend trastuzumab to 2 years” → ❓

•Residual Disease After NACT (HER2+)

Do NOT continue trastuzumab

- Must switch to **T-DM1** (KATHERINE)

- Biggest NEET SS trap

3. Neoadjuvant Chemoradiation

- CROSS Regimen

Component	Details
Chemotherapy	Paclitaxel + Carboplatin (weekly)
Radiation	41.4 Gy
Best response	SCC > Adenocarcinoma

- Benefits**

- ↑ R0 resection
- ↑ OS

- Exam Trap**

- CROSS ≠ perioperative chemotherapy
- Preferred for SCC

4. Perioperative Chemotherapy (Adenocarcinoma)

Regimen	Use
FLOT	Distal oesophagus / GE junction adenocarcinoma

FLOT =

- 5-FU
- Leucovorin
- Oxaliplatin
- Docetaxel

BCLC STAGING — THINK IN 3 VARIABLES

BCLC = Tumor burden + Liver function + Performance status

The BCLC system combines tumor status, cancer-related symptoms/performance status and hepatic reserve. Child-Pugh class is used to assess liver dysfunction; ECOG performance status captures functional status.

BCLC	Core features	Default treatment direction
O Very early	Single nodule ≤ 2 cm; Child-Pugh A; ECOG 0	Ablation / resection / transplant according to suitability
A Early	Single or ≤ 3 nodules ≤ 3 cm; Child-Pugh A/B; ECOG 0	Curative local therapy / transplant
B Intermediate	Multinodular; Child-Pugh A/B; ECOG 0	TACE if candidate
C Advanced	Portal invasion and/or extrahepatic spread; Child-Pugh A/B; ECOG 1–2	Systemic therapy
D Terminal	Child-Pugh C; ECOG ≥ 2	Best supportive care

BCLC TRAP

Do not stage HCC by tumor size alone. Liver function + performance status can change the stage and treatment allocation.

EARLY HCC — RESECTION vs TRANSPLANT vs ABLATION

The choice depends on tumor characteristics AND liver reserve/portal hypertension.

Resection: first-line option for noncirrhotic patients with early-stage HCC; also used in selected cirrhotic patients when liver function and portal pressure permit.

Optimal resection candidate: solitary nodule + no portal hypertension $\rightarrow$ 5-year OS ~50–70%.

Suboptimal resection setting: multinodular disease or portal hypertension $\rightarrow$ reported 5-year OS ~35–55%.

Liver transplantation treats BOTH tumor and underlying cirrhosis and is especially valuable when resection is unsuitable because of poor hepatic reserve.

Milan criteria as a key selection framework: 1 nodule < 5 cm OR up to 3 nodules, each < 3 cm, without major vascular invasion/extrahepatic disease.

Ablation: radiofrequency ablation (RFA) is a primary curative technique for small tumors; median survival ~50–60 months.

Milan CRITERIA — RAPID RECALL

1 lesion < 5 cm OR ≤ 3 lesions, each < 3 cm; no major vascular invasion / extrahepatic spread.

Milan CRITERIA — RAPID RECALL

1 lesion < 5 cm
OR ≤ 3 lesions, each < 3 cm; no major vascular invasion / extrahepatic spread.

APL: the emergency subtype

- Defining lesion: PML::RARA, usually t(15;17).
- Clinical danger: severe coagulopathy/DIC with major bleeding risk.
- Do not wait for complete molecular confirmation when APL is strongly suspected: urgent differentiation therapy is required.
- Treatment is based on all-trans retinoic acid (ATRA) ± arsenic trioxide (ATO), with chemotherapy used according to risk/setting.
- Differentiation syndrome is a characteristic treatment complication.

 **APL = ACT FAST: A = ATRA immediately • C = Coagulopathy/DIC • T = t(15;17) • FAST = don't wait for confirmation** — Suspected APL is a hematologic emergency because early death is often related to hemorrhage/DIC.

Treatment framework

- Fit/newly diagnosed patients: intensive induction is commonly cytarabine + anthracycline-based therapy ('7+3') with molecularly directed additions in selected patients.
- FLT3-mutated AML: FLT3-directed therapy may be added to intensive treatment.
- CD33-positive disease: gemtuzumab ozogamicin can be incorporated in selected settings.
- IDH1/IDH2-mutated AML: IDH-directed agents are important targeted options, especially in appropriate non-intensive/relapsed settings.
- Older/unfit patients: lower-intensity azacitidine or decitabine + venetoclax is a major treatment platform.
- After remission, consolidation is guided by disease risk, fitness and MRD; allogeneic hematopoietic cell transplantation is considered particularly for adverse-risk disease and selected intermediate-risk patients.
- Relapsed/refractory AML: salvage therapy, targeted therapy when an actionable mutation is present, and transplant in eligible responders.

 **7+3: 7 days cytarabine + 3 days anthracycline** — Classic intensive AML induction framework; modern regimens may add targeted agents or use alternative approaches based on molecular risk and fitness.

FINAL MEMORY MAP 8;14 Burkitt → MYC → Starry sky → fastest
14;18 Follicular → BCL2 → indolent → transformation to DLBCL
11;14 Mantle → Cyclin D1 → CD5+
11;18 MALT → API2/MALT1 → H. pylori context
2;5 ALCL → ALK → CD30+
HTLV-1 ATLL → flower cells + hypercalcemia

MGUS vs SMOLDERING MYELOMA vs ACTIVE MM

Entity	M-protein / marrow	End-organ damage / MDE
MGUS	Serum M-protein <3 g/dL; clonal BM plasma cells <10%	Absent; no amyloidosis attributable to plasma-cell disorder
Smoldering MM	Serum M-protein ≥3 g/dL and/or urine monoclonal protein ≥500 mg/24 h and/or BM plasma cells 10–60%	No MDE / no myeloma-related organ damage
Active MM	Clonal BM plasma cells ≥10% or plasmacytoma	≥1 CRAB feature attributable to disease OR a myeloma-defining biomarker

MEMORY MGUS = small clone, no damage. SMM = larger clone, still no damage. MM = clone + damage/MDE.

SOLITARY PLASMACYTOMA

Localized clonal plasma-cell tumor in bone or soft tissue without systemic myeloma.

Bone plasmacytoma: solitary lytic bone lesion with clonal plasma cells and otherwise normal marrow/no systemic disease.

Extramedullary plasmacytoma commonly arises in the upper aerodigestive tract, especially nasal/nasopharyngeal mucosa.

Treatment is primarily local radiotherapy; systemic therapy is not routinely required unless progression/systemic disease occurs.

EXAM PEARL Single lesion + clonal plasma cells + no systemic myeloma = solitary plasmacytoma.

POEMS SYNDROME

- POEMS is a plasma-cell disorder characterized by Polyneuropathy, Organomegaly, Endocrinopathy, Monoclonal plasma-cell disorder, Skin changes.
- The monoclonal protein is usually λ -restricted and often associated with a small plasma-cell clone.
- A major pathogenic feature is markedly increased VEGF.
- Other manifestations: sclerotic bone lesions, papilledema, thrombocytosis/polycythemia, edema, pulmonary hypertension, skin hyperpigmentation/hypertrichosis and clubbing.
- Neuropathy is typically a progressive demyelinating sensorimotor polyneuropathy.
- POEMS differs from classic myeloma because the plasma-cell burden can be small and bone lesions are often sclerotic rather than purely lytic.

•VERY HIGH-YIELD EXAM MATCHES (ONE-LINERS)

- Angiogenesis → Bevacizumab
- Immune evasion → Checkpoint inhibitors
- Defective DNA repair → PARP inhibitors
- Cell cycle dysregulation → CDK4/6 inhibitors
- Anti-apoptosis (BCL-2) → Venetoclax

HALLMARKS OF CANCER

Hallmark	Key Pathway / Target	Targeted Drugs (Examples)	Cancer Example / Exam Pearl
Sustaining proliferative signaling	EGFR	Erlotinib, Gefitinib, Osimertinib	EGFR-mutant lung cancer
	HER2	Trastuzumab, Pertuzumab, T-DM1	HER2+ breast cancer
	BCR-ABL	Imatinib, Dasatinib	CML
Evading growth suppressors	CDK4/6-RB pathway	Palbociclib, Ribociclib, Abemaciclib	HR+ breast cancer
Resisting cell death (apoptosis)	BCL-2	Venetoclax	CLL, AML
Enabling replicative immortality	Telomerase	(No routine clinical drug)	Exam point: theoretical target
Inducing angiogenesis	VEGF	Bevacizumab	CRC, lung
	VEGFR TKI	Sunitinib, Sorafenib, Pazopanib	RCC, HCC
Activating invasion & metastasis	MET / ALK	Crizotinib, Alectinib	ALK+ lung cancer
Dysregulating cellular metabolism	mTOR	Everolimus, Temsirolimus	RCC, NET
Evading immune destruction	PD-1	Pembrolizumab, Nivolumab	MSI-H tumors, melanoma
	PD-L1	Atezolizumab, Durvalumab	TNBC, biliary
	CTLA-4	Ipilimumab	Melanoma
Genome instability & mutation	PARP	Olaparib, Talazoparib	BRCA-mutated breast/ovary
Tumor-promoting inflammation	COX / cytokines	(Indirect role)	Conceptual, not exam drug

60-SECOND EXAM TABLE

If the question says...	Think...
Aggressive epithelial ovarian cancer	High-grade serous / Type II / TP53
BRCA ovarian cancer	Homologous recombination defect → PARP inhibitor relevance
Ovarian spread + ascites	Transcoelomic/peritoneal dissemination
Ovarian first-line chemotherapy	Carboplatin + paclitaxel
Young woman + estrogenic symptoms	Granulosa-cell tumor
Virilization + ovarian tumor	Sertoli-Leydig
Ovarian germ-cell tumor + AFP	Yolk sac tumor
Ovarian germ-cell tumor + LDH	Dysgerminoma
Ovarian germ-cell tumor + β -hCG	Choriocarcinoma
Postcoital bleeding	Cervical cancer
HPV oncogenic proteins	E6 → p53; E7 → Rb
Cervical cancer + pelvic wall	Stage III
Endometrial cancer + postmenopausal bleeding	Classic presentation
Endometrial cancer + obesity/unopposed estrogen	Type I/endometrioid pattern
Endometrial serous cancer	Type II / aggressive
Molar pregnancy + no fetus	Complete mole
Molar pregnancy + triploidy/fetal tissue	Partial mole
Persistent β -hCG after mole evacuation	Persistent GTN → evaluate/treat

NEET - SS

LAST MINUTE

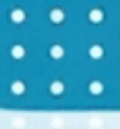
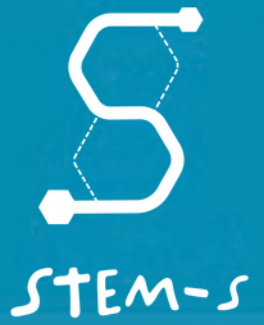
REVISION

NOTES

VERSION 2.0

MEDICAL ONCOLOGY GROUP

PART: 2



Presented By:
STEM-S

First 30 seconds: classify the case

Step	Ask yourself	High-yield answer
1	Small-cell or non-small-cell?	SCLC vs NSCLC
2	If NSCLC → histology?	Adenocarcinoma / squamous / other
3	Advanced NSCLC → actionable driver?	EGFR, ALK, ROS1, BRAF, KRAS, MET, RET, NTRK, HER2 etc.
4	No actionable driver → PD-L1?	PD-L1 level guides immunotherapy strategy

EXAM FRAME In advanced NSCLC, think: HISTOLOGY → MOLECULAR DRIVER → PD-L1 → systemic treatment.

Histology: the practical distinction

NSCLC includes adenocarcinoma, squamous carcinoma and large-cell/other non-small-cell tumors. When morphology is difficult, immunohistochemistry helps establish lineage.

Tumor	Useful markers / clues
Adenocarcinoma	TTF-1 and Napsin-A support pulmonary adenocarcinoma
Squamous	p40/p63 support squamous differentiation
Neuroendocrine / SCLC	Synaptophysin, chromogranin, NSE; diagnosis integrates morphology + IHC

PNEUMONIC Adeno = TTF-1 + Napsin-A; Squamous = p40/p63.

- EGFR mutations occur particularly in nonsmokers/never-smokers and are classically associated with exon 19 deletion and exon 21 L858R.
- Osimertinib is highlighted as an important EGFR TKI and is used in the adjuvant setting for resected EGFR-mutant NSCLC as well as metastatic disease.
- The supplied trial table shows improved outcomes with osimertinib compared with earlier-generation EGFR TKIs in EGFR-mutant NSCLC.

MEMORY HOOK EGFR → think 19 deletion / 21 L858R → TKI, especially osimertinib.

3. UROTHELIAL (BLADDER) CANCER

• Muscle-Invasive Bladder Cancer (MIBC)

Setting	Standard
Neoadjuvant	Cisplatin-based chemotherapy
Surgery	Radical cystectomy
Adjuvant	If no NACT given

• Exam Pearl

Neoadjuvant chemo **improves survival**

• Metastatic Bladder Cancer

Line	Treatment
First-line (fit)	Cisplatin + Gemcitabine
Cisplatin-unfit	Carboplatin-based
Maintenance	Avelumab
Second-line	Pembrolizumab

• Exam Pearl

Avelumab maintenance is practice-changing

• Intravesical Therapy

Drug	Indication
BCG	NMIBC (high-risk)
Mitomycin C	Alternative

• ExamTrap

- BCG is **immunotherapy**, not chemotherapy

1. TUMOR LYSIS SYNDROME (TLS)

- **Causes**

- High-grade lymphomas (Burkitt)
- Acute leukemias
- After chemotherapy (especially hematological)

- **Lab Abnormalities**

- ↑ Uric acid
- ↑ Potassium
- ↑ Phosphate
- ↓ Calcium

- **Management (ORDER MATTERS)**

Step	Treatment
Prevention	Hydration
Hyperuricemia	Rasburicase
Alternative	Allopurinol (prevention only)

- **Rasburicase treats existing uric acid**
- Allopurinol does **NOT** reduce uric acid already formed

PATHOGENESIS — THE BIG EXAM DISTINCTION

Antigen type	Typical antibodies	Response to immunotherapy
Intracellular / onconeural	Hu (ANNA-1), Yo (PCA-1), Ri (ANNA-2), Ma2, CRMP5, amphiphysin	Predominantly cytotoxic T-cell mediated → usually poor neurologic response; tumor treatment is crucial
Cell-surface / synaptic	NMDAR, AMPAR, GABA-BR, LGI1, CASPR2, DPPX, GlyR	Antibody-mediated → generally better response to immunotherapy

EXAM PEARL Intracellular antigen → T-cell mechanism → poorer reversibility. Cell-surface antigen → antibody-mediated → better treatment response.

ANTIBODY-SYNDROME-TUMOR: MUST KNOW

Antibody	High-yield syndrome	Tumor association
Hu / ANNA-1	Encephalomyelitis; sensory neuronopathy; encephalitis; autonomic dysfunction	SCLC
Yo / PCA-1	Rapidly progressive cerebellar degeneration	Breast, ovarian/gynecologic cancer
Ri / ANNA-2	Cerebellar degeneration; opsoclonus-myoclonus; brainstem encephalitis	Breast; gynecologic; SCLC
Ma2 / Ta	Limbic/diencephalic/brainstem encephalitis	Testicular germ-cell tumor
CRMP5 / CV2	Encephalomyelitis; optic neuritis/retinitis; peripheral neuropathy	SCLC; thymoma
Amphiphysin	Stiff-person syndrome; encephalomyelitis	Breast cancer; SCLC
GABA-B receptor	Limbic encephalitis with prominent seizures	SCLC

6. TARGETED THERAPY IN STS

Drug	Indication
Pazopanib	Non-adipocytic STS
Trabectedin	Liposarcoma, LMS
Eribulin	Liposarcoma

- Pazopanib **not effective in liposarcoma**

7. IMMUNOTHERAPY — LIMITED ROLE

- Modest activity in:
 - UPS
 - Synovial sarcoma (selected)
- Immunotherapy is **not standard** in most STS

8. GIST — MUST NOT MISS (VERY COMMON CONFUSION)

- **Biology**
 - Driven by **KIT (CD117)** mutation
 - Responds dramatically to TKIs
- **Treatment**
 - **Imatinib** first-line
 - Sunitinib / Regorafenib in resistance
- **Do NOT use doxorubicin in GIST**

Olaparib Across Cancers: Identifying the Right Patient

1

Olaparib prescribing begins by matching the cancer type, disease setting, biomarker and previous treatment. A BRCA alteration alone is insufficient. Standard dose is 300 mg orally twice daily; duration varies from one year in early breast cancer to two years in first-line ovarian maintenance, or until progression in metastatic settings.

□ WHEN CAN OLAPARIB BE PRESCRIBED?

Cancer and setting	Biomarker and Clinical Eligibility	Practical Prescription
Newly diagnosed advanced ovarian/ fallopian tube/primary peritoneal cancer (SOLO-1)	Germline or somatic BRCA1/2 pathogenic variant; CR or PR after first-line platinum chemotherapy	300 mg twice daily for 2 years. Continue beyond 2 years only if residual disease remains and benefit continues.
Advanced ovarian cancer after chemotherapy containing bevacizumab (PAOLO-1)	HRD-positive (BRCA1/2 alteration and/or qualifying genomic instability); CR or PR after platinum–bevacizumab	Olaparib 300 mg twice daily + bevacizumab. Olaparib for 2 years; bevacizumab ≈ 15 months total.
Platinum-sensitive recurrent ovarian cancer (SOLO-2)	Germline or somatic BRCA1/2 pathogenic variant; CR or PR after platinum rechallenge	Maintenance until progression or unacceptable toxicity. Avoid routine rechallenge after progression on a prior PARP inhibitor.
High-risk HER2-negative early breast cancer (OlympiA)	Germline BRCA1/2 pathogenic variant; completed neoadjuvant/adjuvant chemotherapy and fulfils high-risk criteria	300 mg twice daily for 1 year. Give after radiotherapy; endocrine therapy may continue concurrently.
HER2-negative metastatic breast cancer (OlympiAD)	Germline BRCA1/2 pathogenic variant; previous chemotherapy. HR-positive disease should have received endocrine therapy or be unsuitable for it.	Continue until progression or unacceptable toxicity.
Metastatic pancreatic adenocarcinoma (POLO)	Germline BRCA1/2 pathogenic variant; no progression after ≥16 weeks of first-line platinum chemotherapy	Switch to maintenance olaparib and continue until progression or unacceptable toxicity.
mCRPC after abiraterone or enzalutamide (PROfound)	Qualifying germline or somatic HRR alteration; strongest benefit with BRCA1/2, particularly BRCA2	Olaparib monotherapy until progression. Continue ADT or confirm previous bilateral orchiectomy.
Previously untreated mCRPC (PROpel)	Germline or somatic BRCA1/2 pathogenic variant	Olaparib plus abiraterone and prednisone/prednisolone; continue ADT.

Update / trial	Setting	What changed	Key Points
DESTINY-Breast09	1L HER2+ metastatic	T-DXd-based frontline strategy; T-DXd + pertuzumab showed major PFS improvement vs THP	HER2+ ≠ HER2-low; T-DXd → ILD/pneumonitis
DESTINY-Breast06	HR+/HER2-low or ultralow metastatic	T-DXd expanded activity beyond HER2-positive disease	HER2-low/ultralow is treatment-defining
PATINA	HR+/HER2+ metastatic	Maintenance palbociclib + anti-HER2 therapy + endocrine therapy after induction improved PFS	Maintenance CDK4/6 + HER2 blockade
SERENA-6 / camizestrant	HR+/HER2- metastatic	ctDNA-detected emergent ESR1 mutation during AI + CDK4/6 → switch endocrine backbone to camizestrant + CDK4/6	ESR1 can be detected before radiologic progression
Imlunestrant + abemaciclib	ER+/HER2-, ESR1-mutated advanced/metastatic	FDA approval Sep 2026 after ≥1 endocrine line	ESR1 mutation → oral SERD strategy
ASCENT-04 / KEYNOTE-D19	1L metastatic TNBC, PD-L1 CPS ≥10	Sacituzumab govitecan + pembrolizumab improved outcomes vs pembrolizumab + chemotherapy	TROP2 ADC + ICI; CPS selection
ASCENT-03	1L metastatic TNBC, not candidates for PD-1/PD-L1	Sacituzumab govitecan vs chemotherapy; 2026 approval context	ICI-ineligible TNBC → TROP2 ADC
DESTINY-Breast11	High-risk early HER2+	Neoadjuvant T-DXd strategy produced important pCR/EFS data	Neoadjuvant ADC paradigm
DESTINY-Breast05	HER2+ residual disease after neoadjuvant	T-DXd vs T-DM1	Residual HER2+ disease → ADC escalation

ADC	Target	Major cancers / use	Signature toxicity
Trastuzumab deruxtecan (T-DXd)	HER2	Breast, gastric/GEJ, lung and other HER2-expressing tumors	ILD/pneumonitis
T-DM1	HER2	HER2+ breast	Thrombocytopenia, hepatotoxicity
Sacituzumab govitecan	TROP2	TNBC and other epithelial cancers	Neutropenia, diarrhea
Datopotamab deruxtecan	TROP2	Breast/lung; expanding indications	ILD, stomatitis, ocular toxicity
Mirvetuximab soravtansine	FR α	FR α -high ovarian cancer	Ocular toxicity
Polatuzumab vedotin	CD79b	DLBCL	Neuropathy, cytopenias
Belantamab mafodotin	BCMA	Multiple myeloma	Corneal/ocular toxicity
Pivekimab sunirine	CD123	BPDCN	Myelosuppression / ADC-related toxicity

APL TREATMENT INDUCTION (LOW RISK)^{b,c,d,e}

See references ([APL-7](#)) and [APL-B 1 of 6](#) for details on regimens

Preferred Regimens

ATRA + daily arsenic trioxide^{f,g,1}
(category 1)

or

ATRA + intermittent arsenic trioxide^{f,g,2}
(category 1)

If blood count recovery by day 28 (platelet $>100 \times 10^9/L$, absolute neutrophil count [ANC] $>1 \times 10^9/L$), proceed with consolidation. BM aspirate and biopsy may be considered to document $<5\%$ blasts and no abnormal promyelocytes^{h,i,j}

CONSOLIDATION THERAPY^{i,k}

Continue consolidation as per protocol (see references and [APL-B 1 of 6](#))

[Post-Consolidation Therapy \(APL-5\)](#)

Useful in Certain Circumstances (if arsenic is not available or contraindicated)

ATRA + idarubicin^{f,g,3}
(category 1)

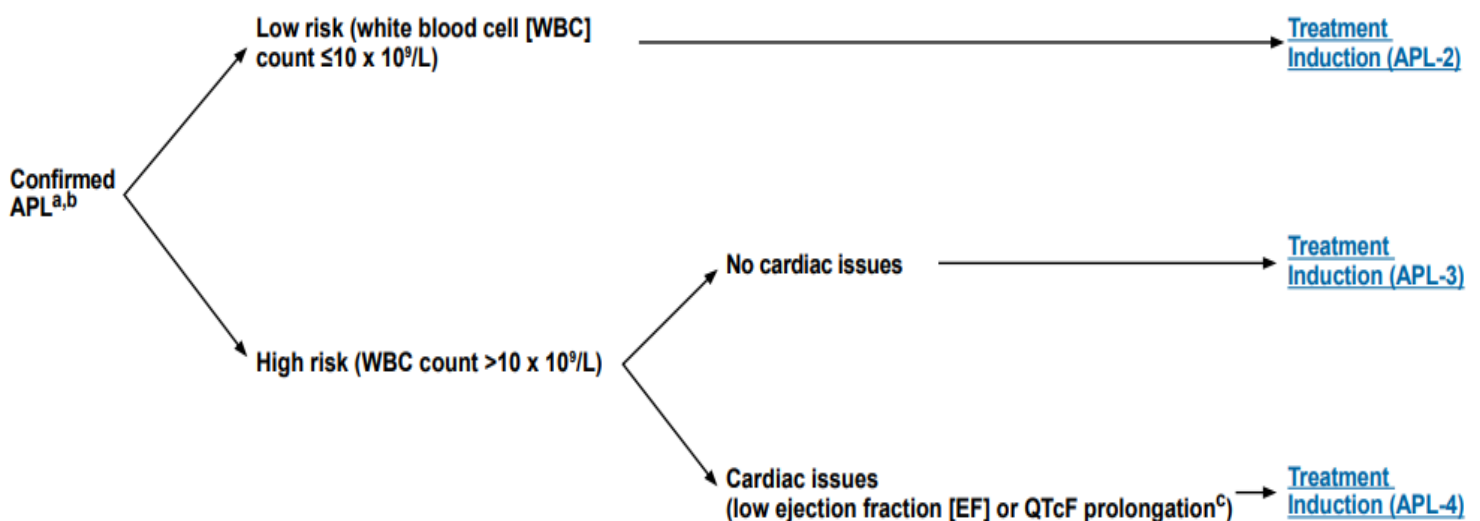
or

ATRA + gemtuzumab ozogamicin^{f,4}

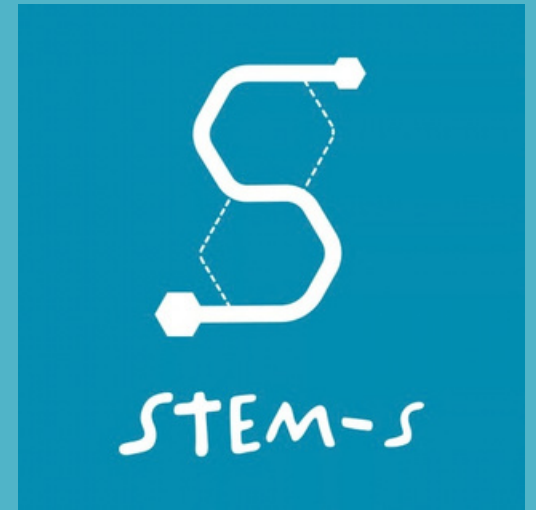
BM aspirate and biopsy days 28–35 to document $<5\%$ blasts and no abnormal promyelocytesⁱ before proceeding with consolidation^k

Continue consolidation as per protocol (see references and [APL-B 1 of 6](#))

APL CLASSIFICATION AND TREATMENT RECOMMENDATIONS



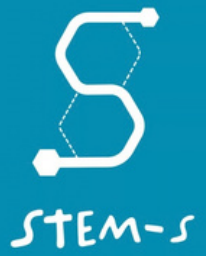
STAGE WISE TREATMENT OF ALL



INI-SS MEDICAL ONCOLOGY NOTES

PRESENTED BY- STEM-S

CANCERS



BREAST CANCER



DIAGNOSIS

WORKUP

PRIMARY TREATMENT

DCIS
Tis,N0,M0,
Encapsulated or
solid papillary
carcinoma (SPC)^a

- History and physical exam
- Diagnostic bilateral mammogram
- Pathology review^b
- Determination of tumor estrogen receptor (ER) status
- Genetic counseling for patients at risk^c of hereditary breast cancer
- Breast MRI^{d,e} as indicated

Breast-conserving surgery^f (BCS) without lymph node surgery^g

Total mastectomy with sentinel lymph node biopsy (SLNB)^{g,h} ± reconstructionⁱ

Whole breast radiation therapy (WBRT) (category 1) with or without boost to tumor bed^{h,j,k,l}
or
Accelerated partial breast irradiation/partial breast radiation (APBI/PBI)^{h,j,k,l}
or
No RT^{h,j,k,l} (category 2B)

Postsurgical Treatment (DCIS-2)

Postsurgical Treatment (DCIS-2)



HEPATOCELLULAR CARCINOMA (HCC) SCREENING^a

Patients at risk for HCC^b:

- Child-Pugh A or B cirrhosis, any etiology
 - Hepatitis B or C^d
 - Alcohol-associated cirrhosis^e
 - Nonalcoholic steatohepatitis
 - Other etiologies^{d,f,g,h}
- Child-Pugh C cirrhosis,^c transplant candidate
- Without cirrhosis
 - Hepatitis B^{c,i}

Ultrasound (US)^{a,j}
+
Alpha fetoprotein (AFP)

AFP positive^k
or
US nodule(s) ≥ 10 mm

Additional
workup
([HCC-2](#))

US nodule(s) < 10 mm → Repeat US + AFP in 3–6 mo

US negative^l → Repeat US + AFP in 6 mo



CLINICAL PRESENTATION

RISK ASSESSMENT^b

Incidental finding of nodule suspicious for lung cancer

- Multidisciplinary evaluation^a
- Smoking cessation counseling

Patient factors

- Age
- Smoking history
- Previous cancer history
- Family history
- Occupational exposures
- Other lung disease (chronic obstructive pulmonary disease [COPD], pulmonary fibrosis)
- Exposure to infectious agents (eg, endemic areas of fungal infections, tuberculosis) or risk factors or history suggestive of infection (eg, immune suppression, aspiration, infectious respiratory symptoms)

Radiologic factors^{c,d}

- Size, shape, and density of the pulmonary nodule
- Associated parenchymal abnormalities (eg, scarring or suspicion of inflammatory changes)
- Fluorodeoxyglucose (FDG) avidity on FDG-PET/CT imaging

Solid nodules
[Follow-up \(DIAG-2\)](#)

Subsolid nodules
[Follow-up \(DIAG-3\)](#)

Lung nodules in asymptomatic patients at high-risk detected during lung cancer screening with LDCT

[NCCN Guidelines for Lung Cancer Screening](#)



**American Joint Committee on Cancer (AJCC)
TNM Staging System For Prostate Cancer (8th ed., 2017)**

Table 1. Definitions for T, N, M

Clinical T (cT)

T	Primary Tumor
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
T1	Clinically inapparent tumor that is not palpable
T1a	Tumor incidental histologic finding in 5% or less of tissue resected
T1b	Tumor incidental histologic finding in more than 5% of tissue resected
T1c	Tumor identified by needle biopsy found in one or both sides, but not palpable
T2	Tumor is palpable and confined within prostate
T2a	Tumor involves one-half of one side or less
T2b	Tumor involves more than one-half of one side but not both sides
T2c	Tumor involves both sides
T3	Extraprostatic tumor that is not fixed or does not invade adjacent structures
T3a	Extraprostatic extension (unilateral or bilateral)
T3b	Tumor invades seminal vesicle(s)
T4	Tumor is fixed or invades adjacent structures other than seminal vesicles such as external sphincter, rectum, bladder, levator muscles, and/or pelvic wall.

Pathological T (pT)

T	Primary Tumor
T2	Organ confined
T3	Extraprostatic extension
T3a	Extraprostatic extension (unilateral or bilateral) or microscopic invasion of bladder neck
T3b	Tumor invades seminal vesicle(s)
T4	Tumor is fixed or invades adjacent structures other than seminal vesicles such as external sphincter, rectum, bladder, levator muscles, and/or pelvic wall

Note: There is no pathological T1 classification.

Note: Positive surgical margin should be indicated by an R1 descriptor, indicating residual microscopic disease.

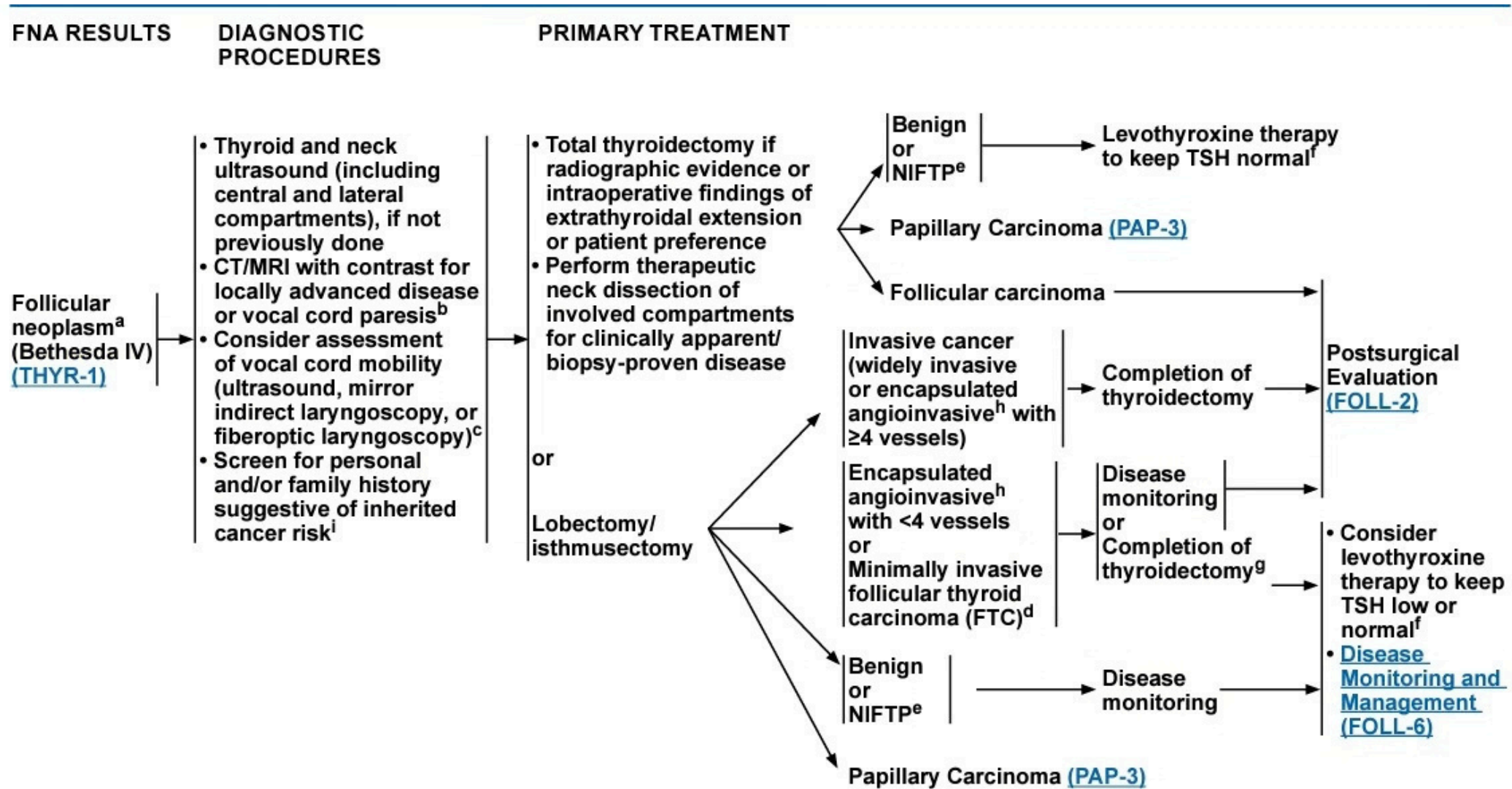
N Regional Lymph Nodes

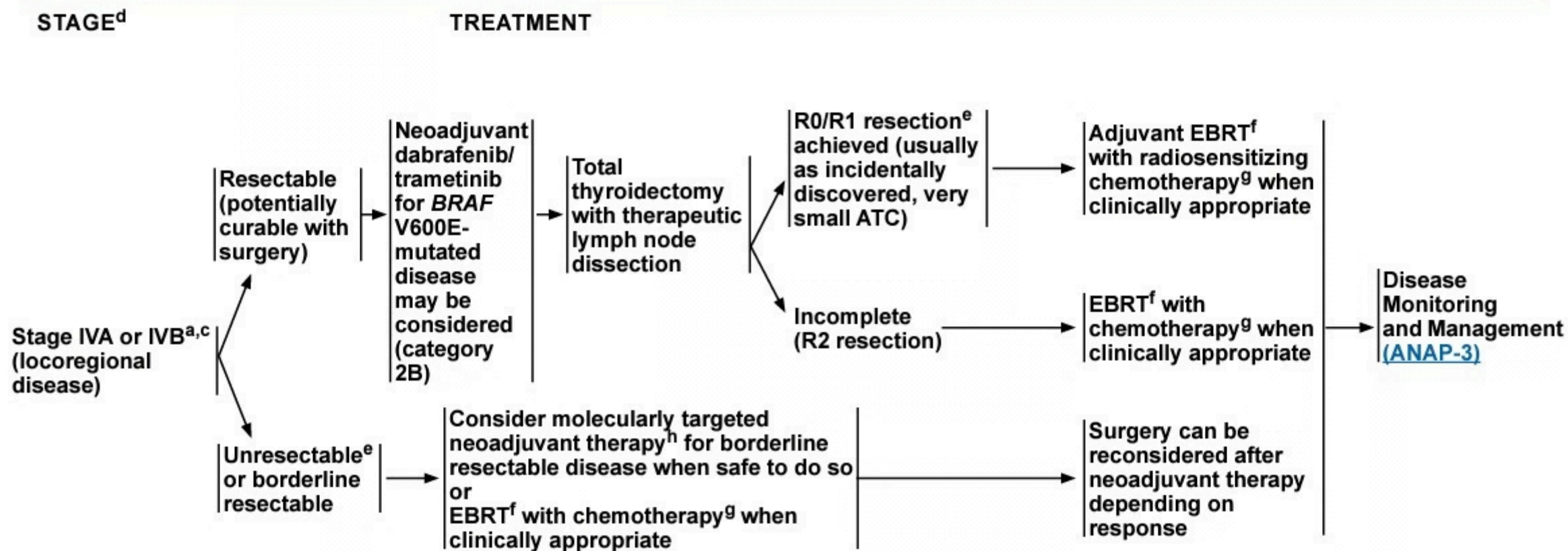
NX	Regional lymph nodes cannot be assessed
N0	No positive regional nodes
N1	Metastases in regional node(s)

M Distant Metastasis

M0	No distant metastasis
M1	Distant metastasis
M1a	Nonregional lymph node(s)
M1b	Bone(s)
M1c	Other site(s) with or without bone disease

Note: When more than one site of metastasis is present, the most advanced category is used. M1c is most advanced.





Study Smart for NEET SS

Medical Group

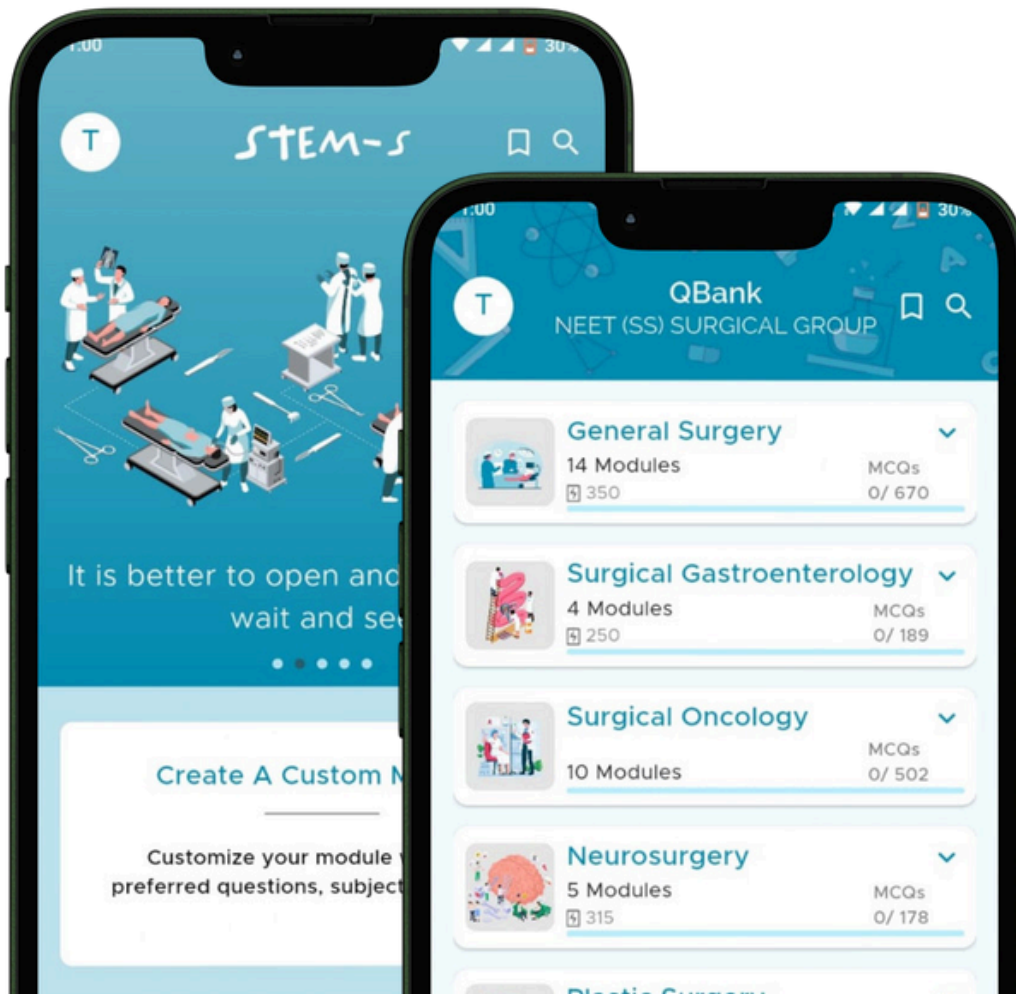
Surgical Group

Pediatric Group

Medical Oncology
Group

Critical Care Medicine
Group

Subscribe Now



Get STEM-S on

