First ever book conceptualized for giving One Touch to Pathology by **Flowcharts • Tables • MCQs • One-Liners**

ONE Touch Pathology

For NEET/NEXT/FMGE/INI-CET

Special Features

- Written and Compiled by Leading Faculty and Subject Expert of Pathology
- Enriched with Latest Updates up to 2022
- Entire theory covered in just **120** pages in Flowcharts, Tables and One-liners format
- **100+** MCQs of Recent Exams covered up to November 2022
- **50+** NEXT Pattern Qs Covered

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ONE Touch Pathology

For NEET/NEXT/FMGE/INI-CET

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Preface

It was always our wish to provide Pathology in the simplest possible form, which is easily understood and student can revise in the shortest time. So, this book "One Touch Pathology" is in line with the name and is our effort to provide concise form of complete Pathology from General Pathology to Systemic Pathology. The entire book is conceptualized with the vision to make it simple in reading and clear in approach.

What are the specialities of this book?

1. **Theory** – a concise form of text (in 120 pages) and most important points to remember for exams are given. The text is in accordance with the recent CBME and NEXT exam curriculum.
2. **High Yield Tables** – most frequently asked points and clinical correlates are tabulated – for easy learning and more visual impact for long-term memory.
3. **Images and Histological Plates** are given along with descriptions. They have been arranged as per the chapters.
4. **Latest Exam Questions** – last 3 years Exam Question papers with solution, explanations and discussions are provided to have an idea about trend of questions and know about the recently asked topics. The answers are given with references from standard resources.
5. **NEXT Pattern Questions** – 50+ NEXT pattern Qs have been added in a topic-wise manner for giving latest trend of approach to the Question Bank.

Do I really need this book?

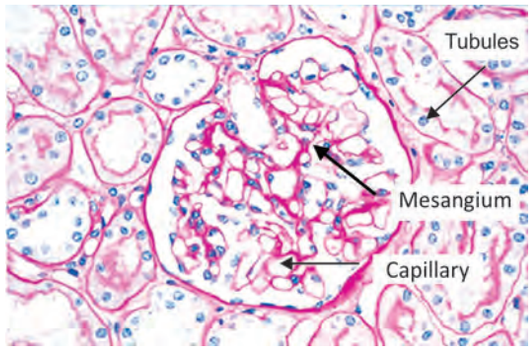
Students who have completed Pathology syllabus from any resource can use this book as revision text. Collection of pearl, One-liners and Important tables are full of valuable information. Students whose exams are round the corner and have not done Pathology, can start preparing from this book. You must remember most of the high yield text, tables and topics from the latest Qs Bank.

We have tried our level best to make this book concise, revision oriented, clutter free and error free, however, any suggestions regarding the improvement of the book will be highly appreciated.

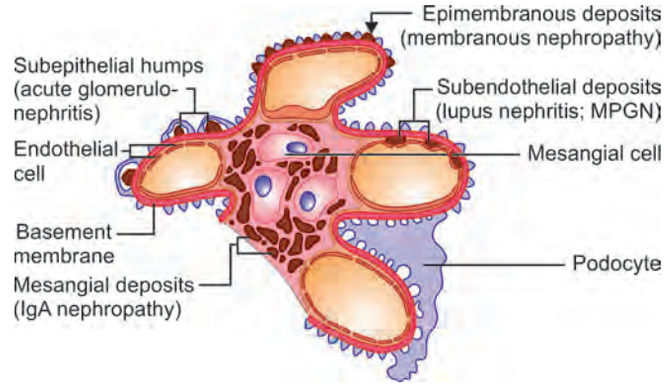
Contents

KIDNEY

GLOMERULUS



Structure of glomerulus



Types of glomerular deposits

PATHOLOGIC RESPONSES OF THE GLOMERULUS TO INJURY

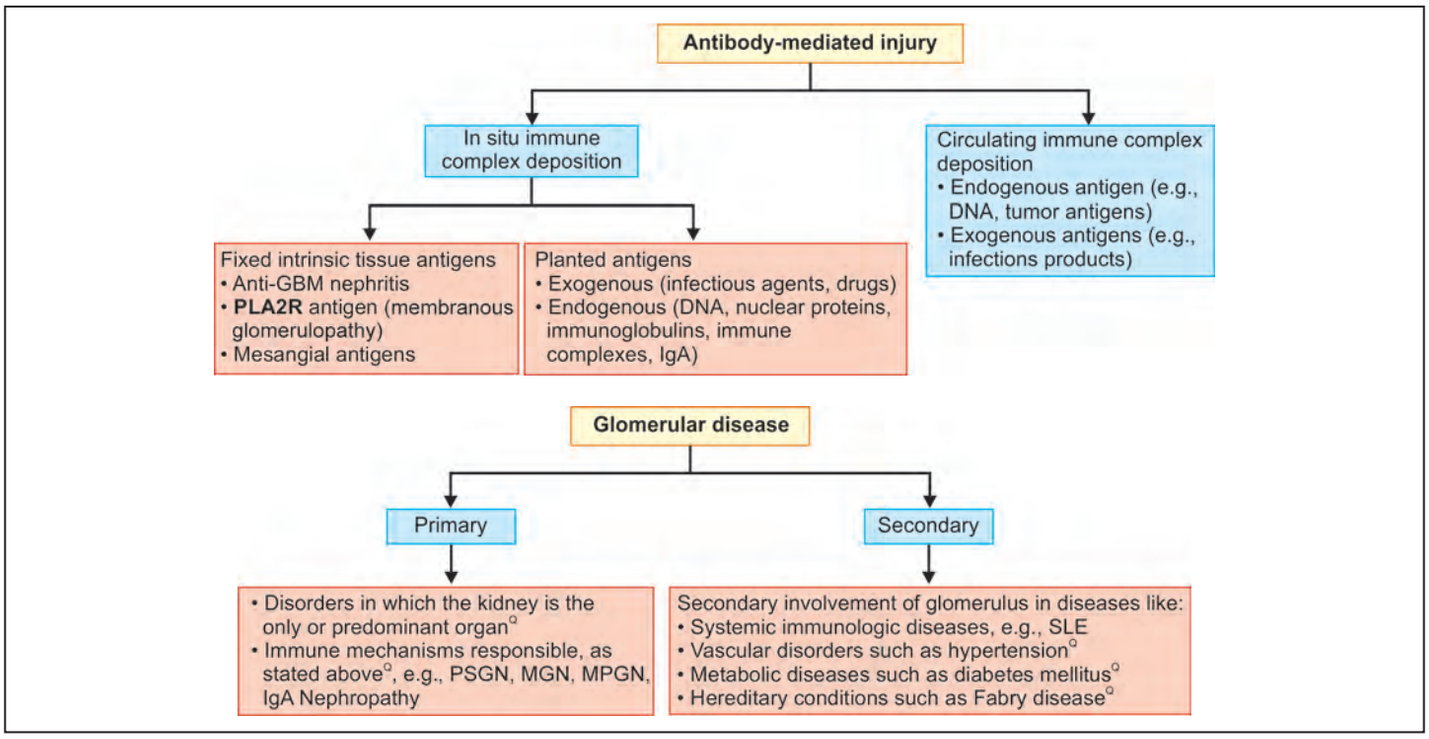
Site of glomerular deposits	Type of glomerulonephritis
Subepithelial deposits	• PSGN • Membranous GN • RPGN • Heymann Nephritis
Subendothelial deposits	• Lupus nephritis • MPGN-I
Membranous deposits	• MPGN II
Mesangial deposits	• IgA nephropathy • HSP

Terminologies used in kidney biopsy	
Terminology	**Description**
Diffuse	Involving **>50%** of the glomeruli in the kidney[Q]
Global	Involving the glomerulus **completely**[Q]
Focal	Involving **<50%** of the glomeruli in the kidney
Segmental	Affecting **a part of each glomerulus**[Q]
Capillary loop Mesangial	Affecting predominantly **capillary or mesangial regions**[Q]

Bowman's capsule- Lined by flattened cells (parietal epithelial cells)

Capillaries- Visceral epithelial cells having foot process (podocytes)
Every capillary has basement membrane lined by endothelial cells

All the capillaries are supported by mesangial matrix



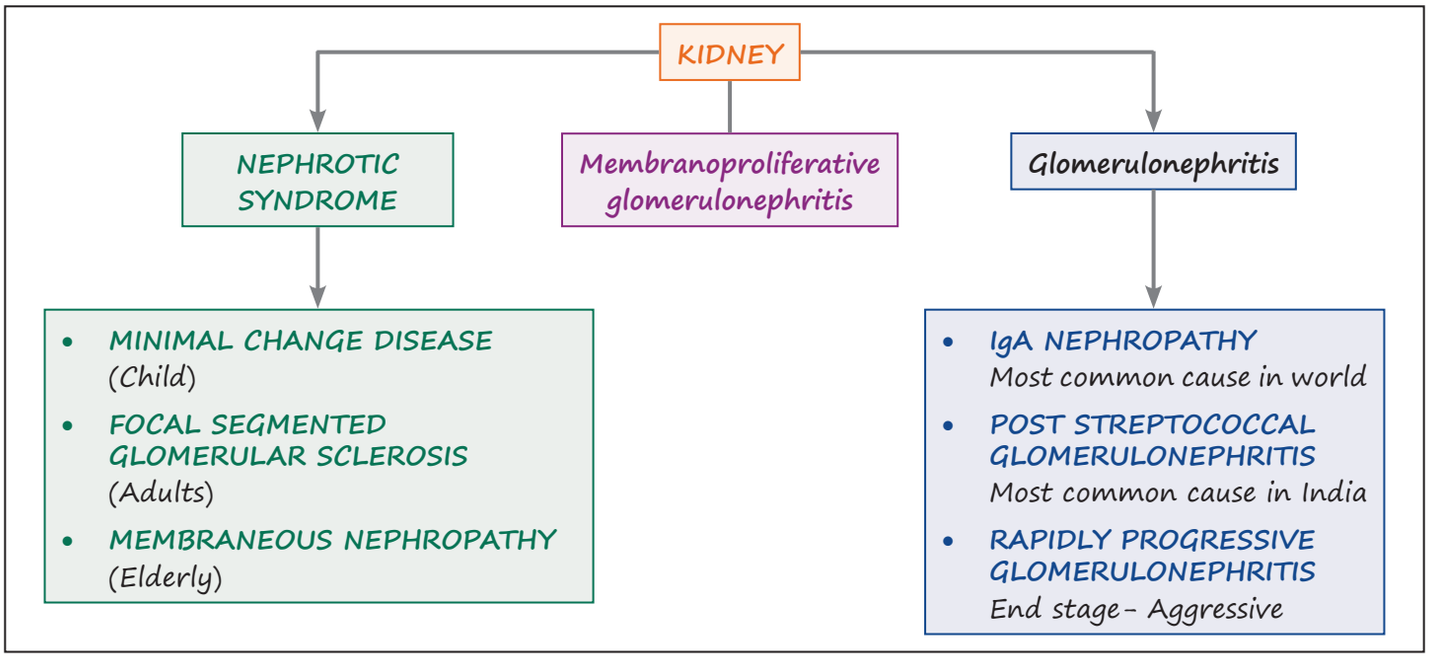
NEPHRON MUTATION

- Most common mutation
- Coded by NPHS-1
- Minimal change like presentation
- Respond to steroids

PODOCIN

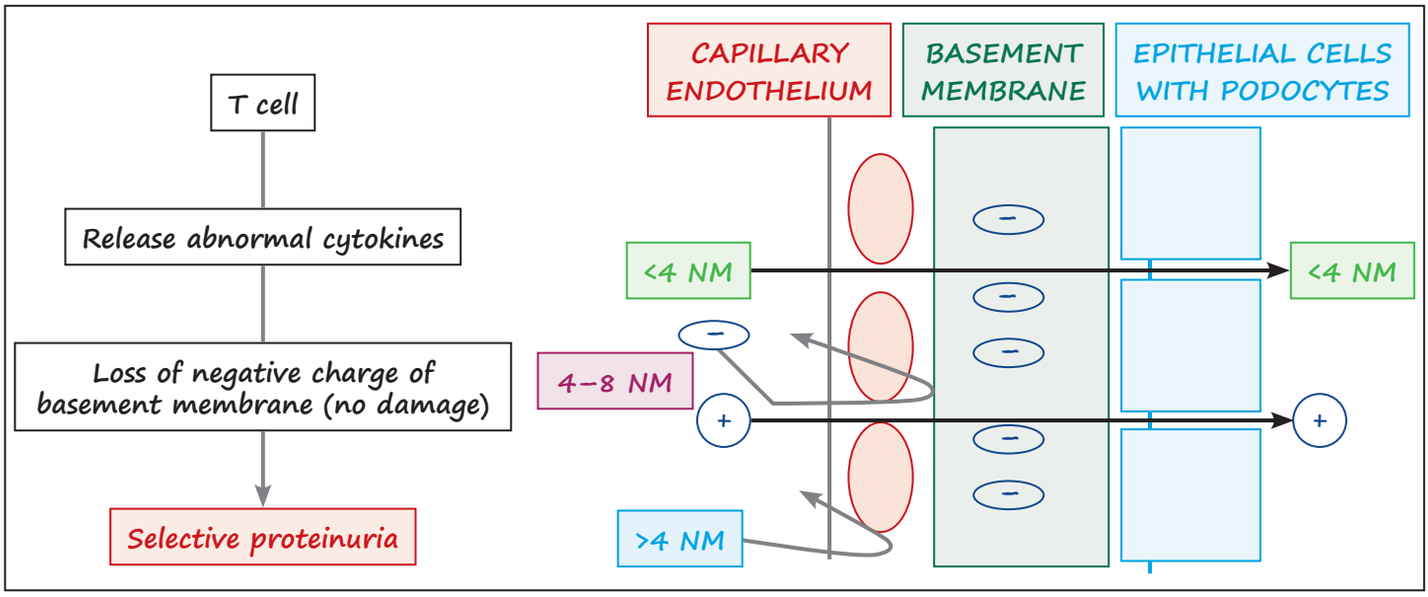
- Coded by NPHS-2
- FSGS like presentation
- Does not respond to steroids

Actin mutation also causes FSGS (α Actinin)



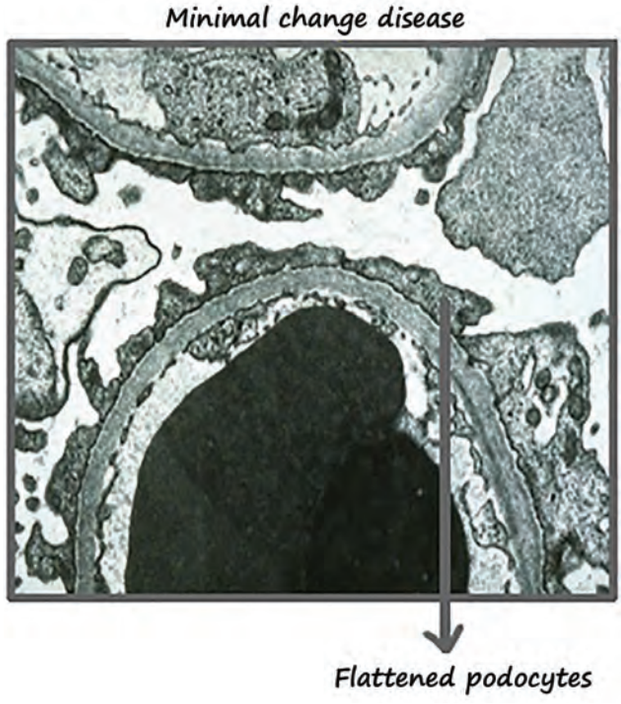
NEPHROTIC SYNDROME

MINIMAL CHANGE DISEASE



Albumin is released as it is negatively charged and smaller in size

- Cytokines also cause flattened podocytes– **Effacement seen in electron microscopy**
- No change in light microscopy and Immunofluorescence



- **Morphology:**
  - **Light microscopy: Glomeruli appear normal**[Q].
  - **Immunofluorescence microscopy: No Ig/ complement deposits.**
  - **Electron microscopy: Diffuse effacement of foot processes of podocytes ("podocytopathy")**[Q].
    - No electron-dense deposits[Q]
  - **Proximal tubules** cells get **laden with lipid** and protein due to tubular reabsorption of lipoproteins: **Lipoid nephrosis**[Q].

FOCAL SEGMENTED GLOMERULAR SCLEROSIS

Due to Podocytes injury

Caused by

- HIV
- Obesity
- Reflex
- Renal ablation
- Sickle cell anemia

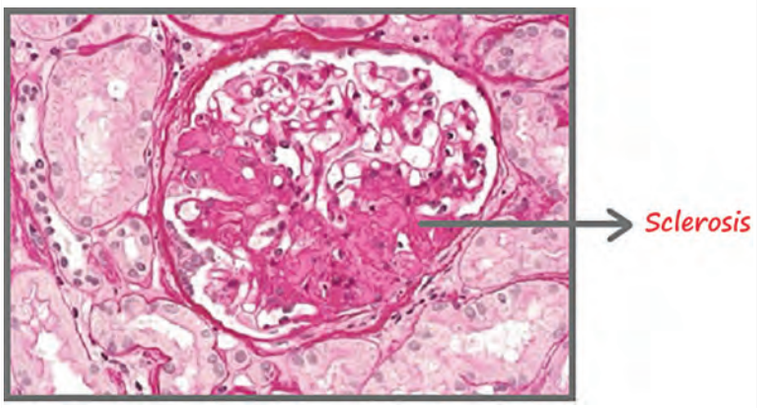
<50% of glomeruli- Focal segmented

Podocytopathies
- Minimal change disease
- FSGS

DAMAGED PODOCYTES → SCLEROSIS

HIVAN

- HIV associated nephropathy
- Collapsing nephropathy
- Proliferation of podocytes causes damage to other structures



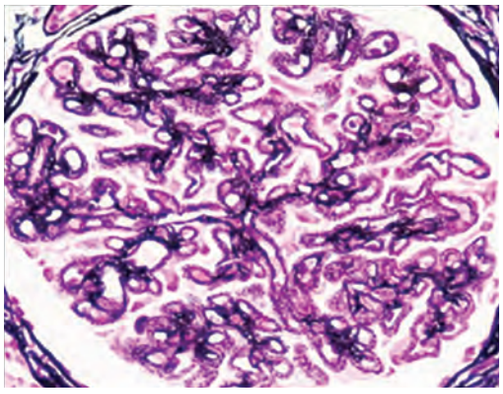
- **Morphology:**
  - **Light microscopy**
    - Collapse of capillary loops in sclerotic areas
    - Deposition of plasma proteins along capillary wall **(hyalinosis)**
  - **Immunofluorescence microscopy**
    - IgM + C3 deposition in sclerotic areas and/or in mesangium.
  - **Electron microscopy**
    - **Diffuse effacement** of foot processes of podocytes.
    - **Focal detachment** of the epithelial cells.
    - **Denudation** of the underlying GBM.

MEMBRANOUS NEPHROPATHY

- Immune complex mediated
- **Antigen - Phospholipid A2**

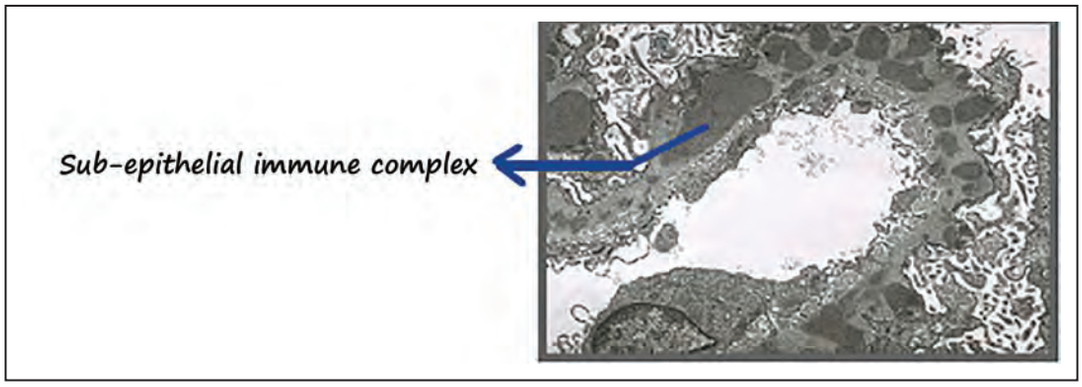
ON LIGHT MICROSCOPY

- Thickened BM
  PAS/Silver stain
- **SPIKES AND DOME PATTERN**



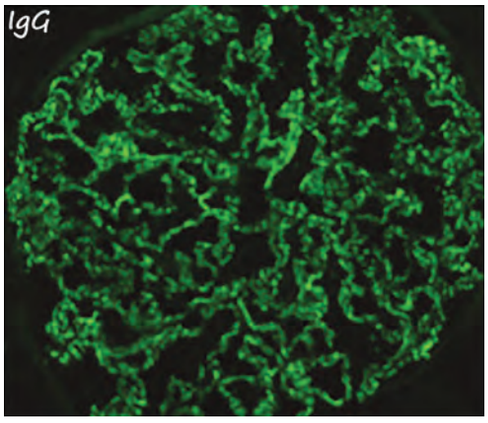
- **Morphology:**
  - **Light microscopy:** Uniform, diffuse thickening of the glomerular capillary wall.Q
  - **Immunofluorescence microscopy: Granular/Lumpy bumpy**Q electron dense immune complexes deposits
  - **Electron microscopy: Granular deposits (Ig + complement)**
    - **Effacement of podocyte** foot processesQ
  - **On Silver methenamine stain:** Prominent **"spikes" and "domes"**Q of silver-staining matrix.

ON ELECTRON MICROSCOPY



IMMUNOFLUORESCENCE

- Fluoro isothicyanate (green color)
- Granular immunofluorescence

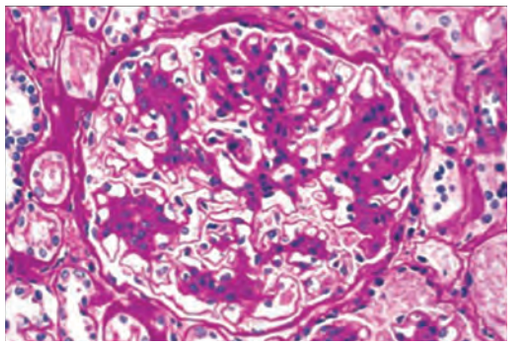


IgA NEPHROPATHY (BERGER'S DISEASE)

- Affects 25–40 years age group
- Due to excess of IgA or not able to metabolise IgA
- Occurs after upper respiratory or lower respiratory infection

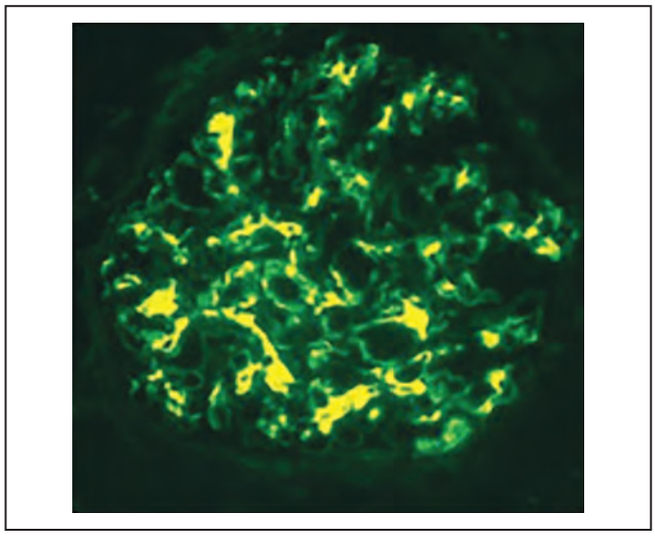
ON LIGHT MICROSCOPY

- Mesangial proliferation is seen



IN IMMUNOFLUORESCENCE

- Granular immunofluorescence (IgA, IgG, C3)
- IgA activates alternative complement pathway (C3)



ON ELECTRON MICROSCOPY

- Deposits increased mesangium

- **Presents with isolated hematuria**
- **Coeliac/liver disease patients have excess of IgA causing predeposition**
- **More prone to get IgA nephropathy**

TREATMENT

- Transplantation
- Reoccurs after transplantation

POST STREPTOCOCCAL GLOMERULONEPHRITIS

- Immune complex mediated
- Caused by B hemolytic streptococci

- **ANTIGEN- STREPTOCOCCAL PROGENITOR EXOTOXIN B - SPE B**

Antigen combines with Antibody

↓

ANTIGEN - ANTIBODY COMPLEX

↓

Deposited in the sub endothelium

↓

Neutrophil flooding in capillary

↓

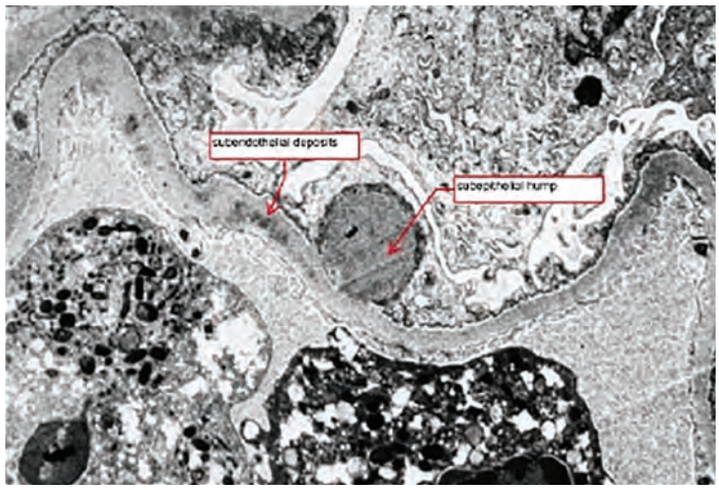
ENDOCAPILLARY PROLIFERATION

↓

Ag- Ab jumps from sub endothelium to subepithelium

↓

LARGE SUBEPITHELIAL HUMP



- Within 6 weeks heals by itself
- Complement deposition (C1, C3, C4)

RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS

Hallmark: BASEMENT MEMBRANE DAMAGE

Basement membrane damage

↓

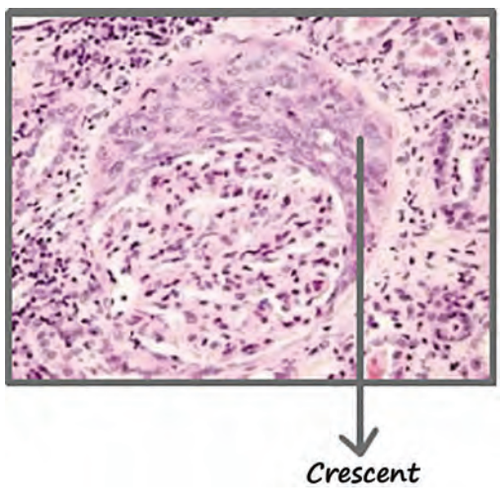
Coagulation protein leaks into bowman's capsule
Fibrinogen mainly

↓

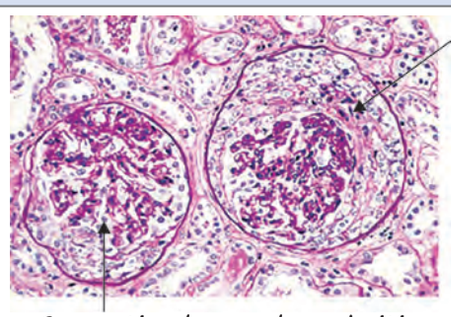
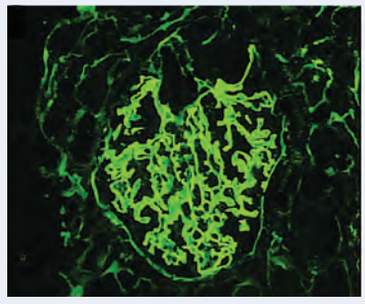
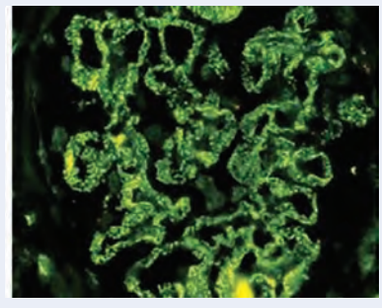
Parietal cells starts proliferating to eat up fibrogen

↓

CRESCENT



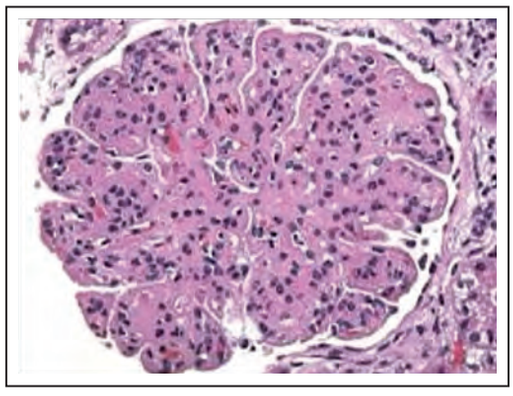
CRESCENT - Fibrinogen, inflammatory cells, parietal cells
Very big crescent can interact visceral epithelial cells

Entity	Type I (20%)	Type II (25%)	Type III (55%)
Mechanism	Anti-GBM antibody	Immune complex	Pauci-immune, c-ANCA/p-ANCA mediated
Etiology	Renal limited **Goodpasture syndrome**Q (Serum antibodies against **alpha 3 NC1 domain of collagen – IV**)	• **Postinfectious** ▪ Poststreptococcal glomerulonephritisQ ▪ Bacterial endocarditisQ • **Noninfectious** ▪ SLEQ, HSPQ ▪ Mixed cryoglobulinemiaQ • **Primary renal disease** ▪ MPGNQ ▪ IgA nephropathyQ	**ANCA-associated** • Idiopathic • **Granulomatosis with polyangiitis (Wegener granulomatosis)**Q • Microscopic polyangiitisQ • Hypersensitivity vasculitisQ
Grossly	Kidneys are enlarged and pale, often with **petechial hemorrhages** on the cortical surfaces. **(FLEA-BITTEN KIDNEY)**Q		
Light m/e	• **Glomeruli: Crescents** are HallmarkQ. • Focal and segmental necrosisQ, endothelial and mesangial proliferationQ. • **Pauci-immune: Segmental glomerular necrosis** is a feature characteristicQ.  Cresentic glomerulonephritis Crescents—formed by • Proliferation of **parietal cells**Q • Infiltration by **WBCs**Q • **Fibrin strands.**Q **Crescents** obliterate the urinary space and compress the glomerular tuft, hence **More the number of crescents → poorer the prognosis**Q		
Immunofluorescence m/e	**Linear GBM fluorescence**Q  **Type I RPGN (linear)**	**Granular immune deposits**Q  **Type II RPGN (granular)**	**No** deposition of immune reactantsQ **No deposits seen** **(Pauci-immune)**
Electron m/e	**Ruptures in the GBM**Q may be present, Type II shows immune complex deposits.		

DIFFERENTIATE BETWEEN TYPES OF RPGN

LIGHT MICROSCOPY	All crescents		
ELECTRON MICROSCOPY	All will show basement membrane damage		
IMMUNOFLUORESCENCE	I Linear	II Granular	III No IM

MEMBRANOPROLIFERATIVE GLOMERULONEPHRITIS



SUBENDOTHELIAL DEPOSITION

↓

Mesangial cells proliferation

↓

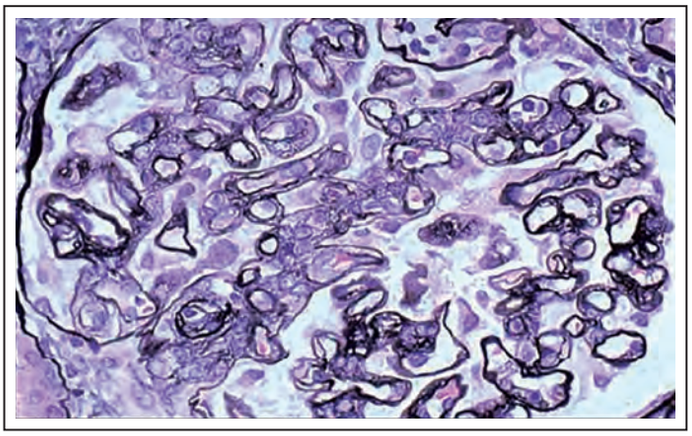
Lobular accentuation

↓

Splitting of BM

ON SILVER STAIN

- Basement membrane is broken
  Tram – Track appearance



MPGN TYPE 2

- **Known as** C3 GLOMERULONEPHRITIS
  DENSE DEPOSIT DISEASE
  COMPLEMENTOPATHIES
- C3 convertase- breakdown C3 to C3a and C3b

C3 → **C3a + C3b**

↑

C3 convertase

- C3 convertase is then broken down by Factor H and Factor I ⊖ **C3 NeF**

C3Ne F blocks the action of Factor H and I
Which causes more breakdown of C3 to C3a and C 3b
C3 will be decreased

- Deposition of dense material in laminate densa of BM (ribbon like)

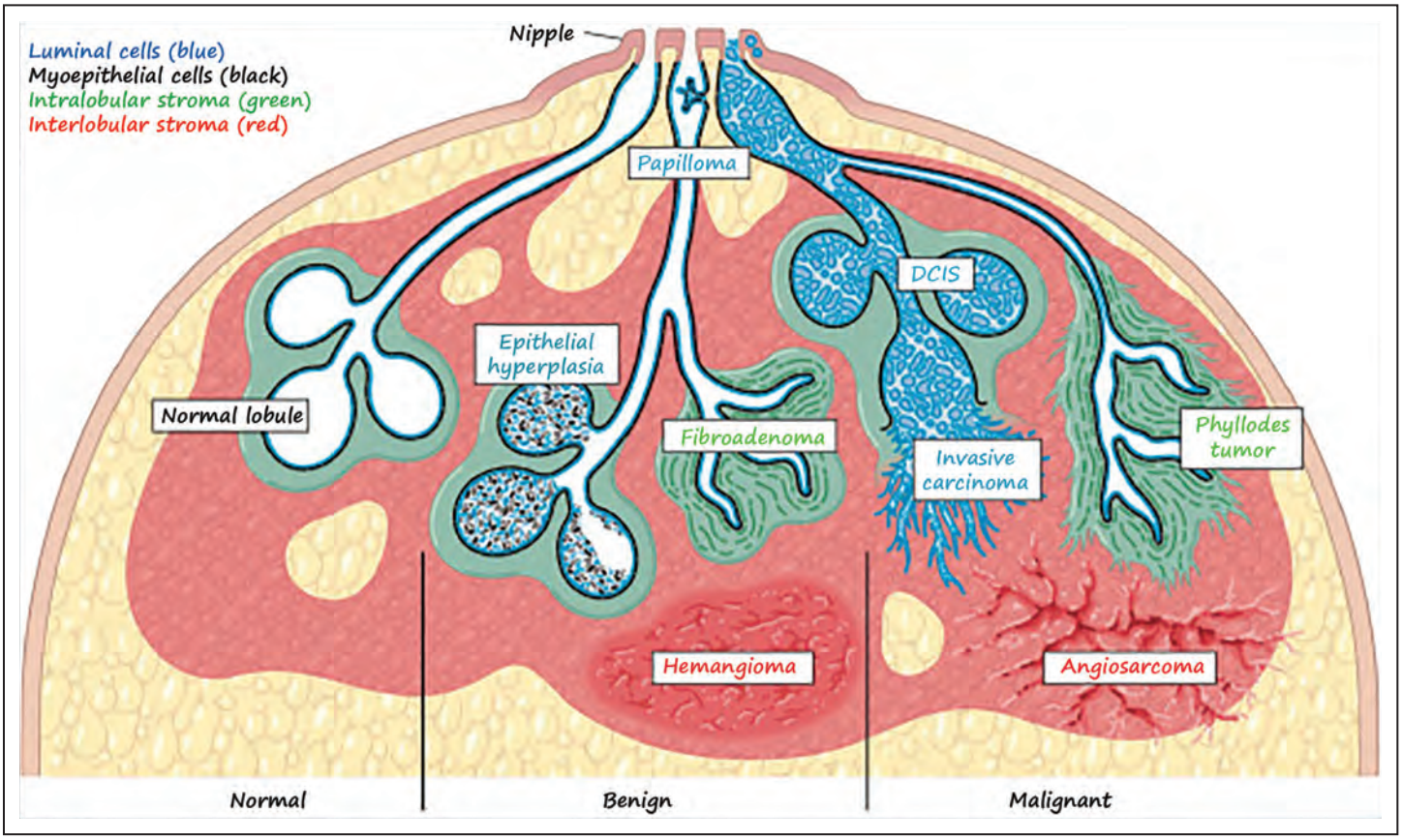
SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

ISN/RPS (2003) classification of lupus nephritis	
Class I	Minimal mesangial LN
Class II	Mesangial proliferative LN
Class III	Focal LN (<50% of glomeruli) III (A): active lesions III (A/C): active and chronic lesions III (C): chronic lesions
Class IV	Diffuse LN (50% of glomeruli) Diffuse segmental (IV-S) or global (IV-G) LN IV (A): active lesions IV (A/C): active and chronic lesions IV (C): chronic lesions
Class V	Membranous LN
Class VI	Advanced sclerosing LN (90% globally sclerosed glomeruli without residual activity)
Adapted from weening jj, D'Agati VD, Schwartz MM	

BREAST

STRUCTURE OF BREAST

- **Breast include:**
  - Two major structures (ducts and lobules),
  - Two types of epithelial cells (luminal and myoepithelial),
  - Two types of stroma (interlobular and intralobular).
- Ducts and lobule are lined by inner luminal cells and outer myoepithelial cells.
- Each lobule is made up of acini and surrounded by intralobular stroma and interlobular stroma.



Proliferation of luminal and myoepithelial cells → Hyperplasia —Seen in→ Pregnancy and puberty

Hypertrophy → Lactating

Loss of Myoepithelial cells → **Malignancy**

Only luminal cells proliferate → Ductal carcinoma insitu
Myoepithelial cells are intact

If at any point, MEC disappear → Invasive carcinoma

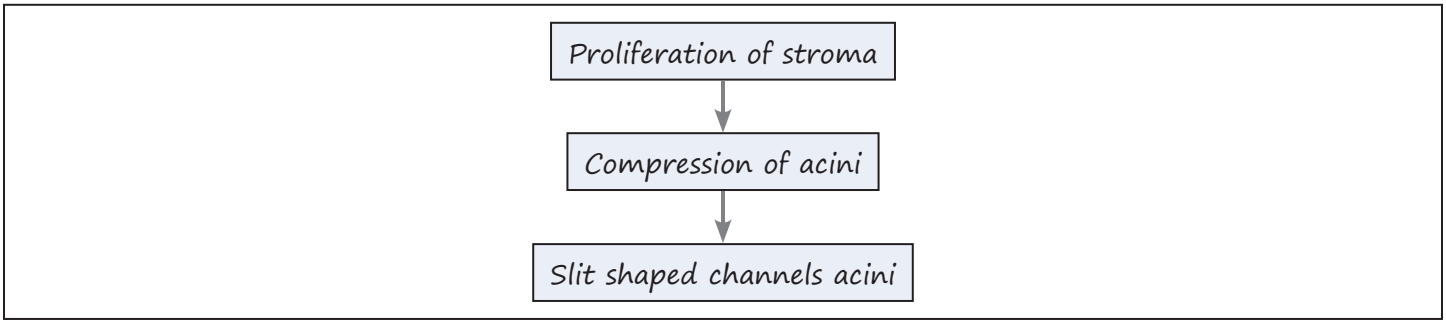
STAINS OF MEC

- P63
- Calponin
- S100
- Smooth muscle actin

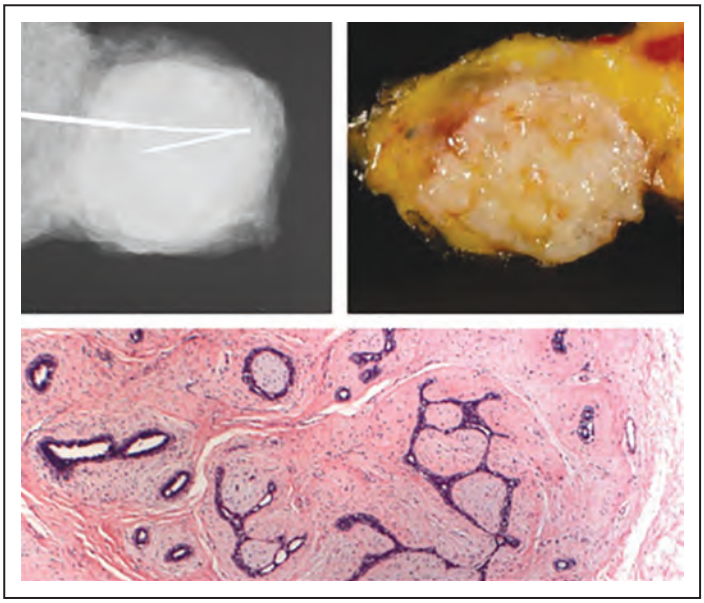
TUMORS OF THE BREAST

FIBROADENOMA

- Most common benign tumor of the female breast.
- Two-thirds of fibroadenomas harbor driver mutations in MED12.
- Arises from intralobular stroma
- Always benign, limited to intralobular stroma



- Tumors are well-circumscribed, rubbery, grayish white nodules that bulge above the surrounding tissue and often contain slit-like spaces lined by epithelium
- FNAC:
  - Intact duct
  - Proliferation of stroma
  - Spindle shaped ducts
  - Antler horn pattern



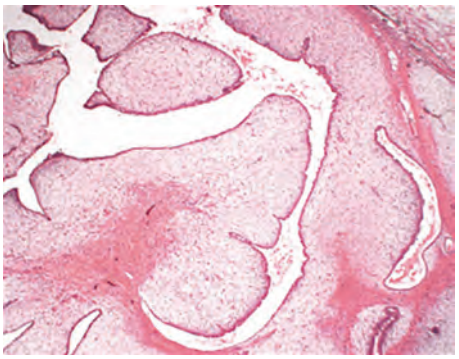
Proliferation of stroma and spindle shaped ducts

Antler horn pattern seen in FNAC

PHYLLODES TUMOR

Arises from intralobular stroma but pushes into interlobular stroma in a finger/plant like manner.

Intralobular form ⟶ Benign
Interlobular form ⟶ Malignant



Compared to a fibroadenoma, there is increased stromal cellularity and overgrowth, giving rise to the typical leaf-like architecture.

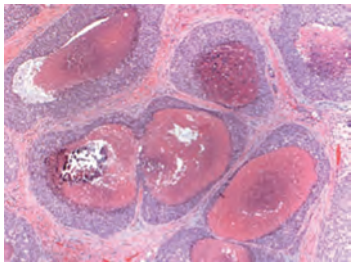
INTERLOBULAR STROMA TUMORS

- Hemangioma ⟶ Benign
- Angiosarcoma ⟶ Malignant
  Skin, liver, breast

DUCTAL CARCINOMA IN SITU

- Clonal proliferation of epithelial cells limited to ducts and lobules by the basement membrane.
- Quickly change into malignancy

Acini with blue color cells at periphery and necrosis at center

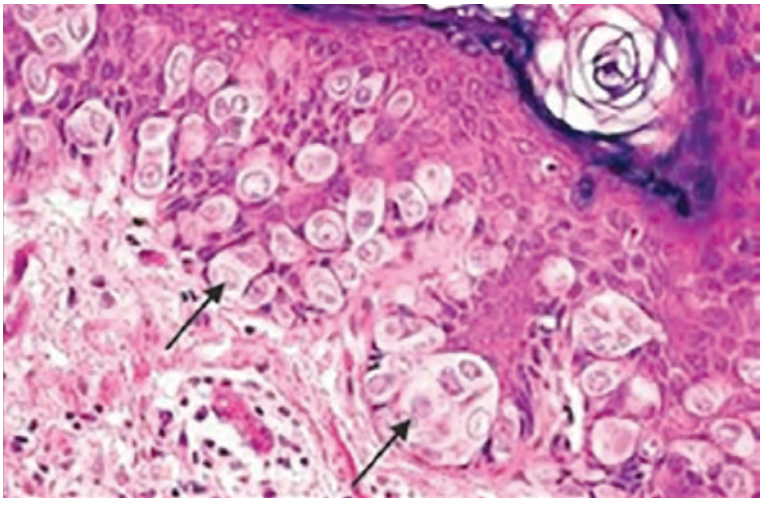


Comedo type DCIS

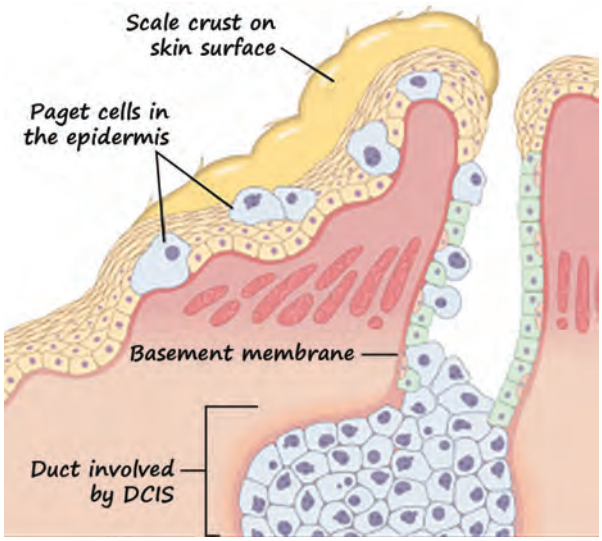
PAGET'S DISEASE OF BREAST

- Retrograde movement of epithelial cells
- Always have DCIS/invasive carcinoma in breast

Can also occur in vulva ⟶ No underlying malignancy

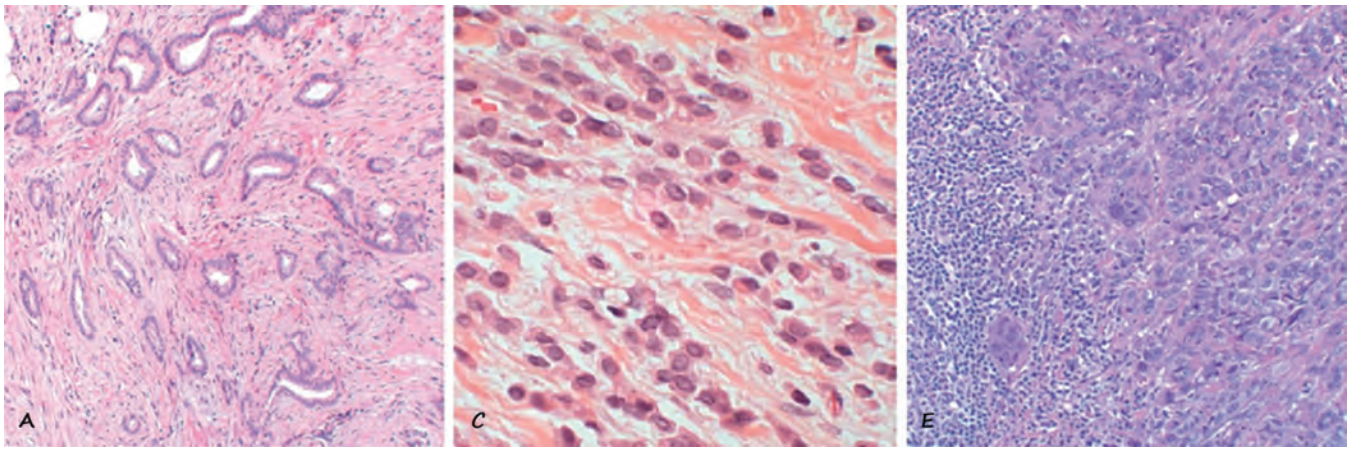


Paget's disease of vulva



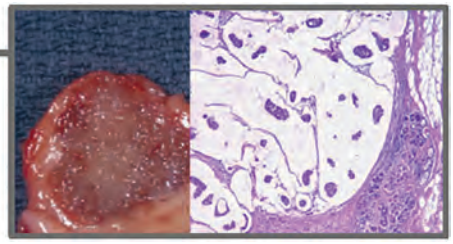
MEDULLARY CARCINOMA OF BREAST

- Mimic fibroadenoma
- Grow as sheets of lymphocytic and plasma infiltrate
- Associated with triple negative mutation



Adenocarcinoma | Lobular carcinoma | Medullary carcinoma

MUCINOUS (COLLOID) CARCINOMA



Seen in menopausal age group

Cancers having good prognosis:
Tubular, Colloid and lobular

RISK FACTORS FOR DEVELOPING BREAST CANCER

Risk factors	Relative risk[a]
Female gender Increasing age Germline mutations of high penetrance Strong family history (>1 first-degree relative, young age, multiple cancers) Personal history of breast cancer High breast density	>4.0
Germline mutations of moderate penetrance High-dose radiation to chest at young age Family history (1 first-degree relative)	2.1–4.0
Early menarche (age <12 years) Late menopause (age >55 years) Late first pregnancy (age >35 years) Nulliparity Absence of breastfeeding Exogenous hormone therapy Postmenopausal obesity Physical inactivity High alcohol consumption	1.1–2.0

CDH-1

- Codes for E, which is a belt desmosome.
  Belt Desmosome: contact inibitation; protects against metastasis

FOR METASTASIS TO DEVELOP THE FIRST MOLECULE TO BE LOST IS E CADHERIN

MUTATION

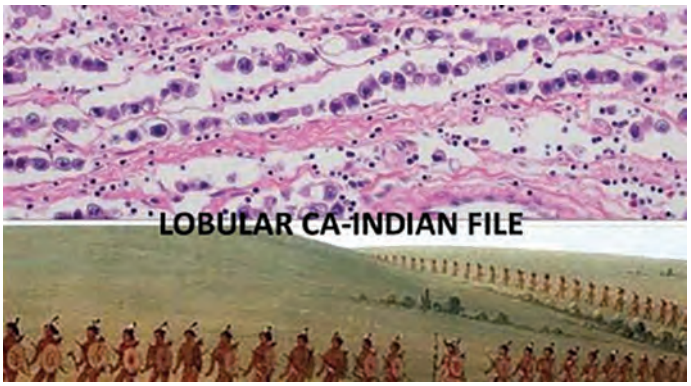
DRIVER MUTATION: First mutation that causes the cancer

PASSENGER MUTATION: Multiple mutation helps in metastasis

E cadherin is a passenger mutation.
It is a driver mutation only for Gastric CA, Lobular caricinoma of breast

LOBULAR CARCINOMA OF BREAST

- Single cells infiltrate the breast
- Indian file pattern
- Bilateral breast cancer
- Contains targetoid mucin



PTEN

- On chromosome 10
- 2nd guardian of genome
- Cowden syndrome

Features

- Hyperkeratosis of hands and foot
- Lot of skin appendage all tumor
- Cancers of breast, endometrium, thyroid

High Yield Facts

- Most common mutation seen in luminal B- **BRCA 2**
- Most common mutation seen in Triple negative - **BRCA 1**
- Triple negative are usually **Medullary CA breast**
- Radiation induced breast cancer- **CHEK 2**
- Mutation associated with Thyroid cancer- **PTEN**

Gene (syndrome)	% of single gene cancers[a]	Risk of breast cancer to age 70[b]	Other cancers	Comments
High Penetrance Germline Mutations (>4-fold increased risk; 3%–7% of breast cancers)				
BRCA 1 (familial breast and ovarian cancer)	~55%	~40%–90%, females; 1%, males	Ovarian (~20% 40%), fallopian tube, pancreas, prostate, others	Majority of cancer's are TNBC
BRCA 2 (familial breast and ovarian cancer)	~35%	~30%–60%, females; 6%, males	Ovarian (~10%–20%), pancreas, prostate, others	Majority of cancers are ER positive. Biallelic mutations cause a form of Fanconi anemia.
TP 53 (Li-Fraumeni)	<1%	~50%–60%, females; <1%, males	Sarcoma, leukemia, brain tumors, others	Majority of cancers are ER and HER2 positive
PTEN (Cowden)	<1%	~20%–80%, females; <1%, males	Thyroid, endometrium, others	Also associated with benign tumors
STKII (Peutz-Jeghers)	<1%	~40%–60%, females	Ovarian, colon, pancreas, others	Also associated with benign colon polyps
CDHI (hereditary diffuse gastric cancer)	<1%	~50%, females	Gastric signet ring cell carcinoma, colon	Majority of cancers are lobular in type
PALPB2 (hereditary breast cancer)	<1%	~30%–60%, females; <1%, males	Pancreas, prostate	Biallelic mutations cause a form of Fanconi anemia
Moderate Penetrance Germline Mutations (2- to 4-fold increased risk 5% to 10% of breast cancers)				
ATM (ataxia-telangiectasia)	~5%	~15%–30%, females		Biallelic mutations cause ataxia-telangiectasia
CHEK2 (hereditary breast cancer)	~5%	~15%–30%, females	Prostate, thyroid, colon, kidney	Majority of cancers are ER positive

MOLECULAR PROFILING

ER-positive, HER-2-negative (also termed "luminal; 60–70%)
Luminal A – Low proliferation (40%–55%)-Most common form of invasive breast cancer
Luminal B – High proliferation (40%–55%)-Most common type of carcinoma associated with BRCA2
HER2-positive (20% of cancers)-2nd M.C. subtype of invasive breast cancer >50% tumors with germline TP53 mutations (Li-Fraumeni syndrome)
ER-negative, HER2-negative Tumors ("basal-like" triple negative Ca; 15%)-The majority of carcinomas arising in women with BRCA1 mutations

IMMUNO HISTOCHEMISTRY

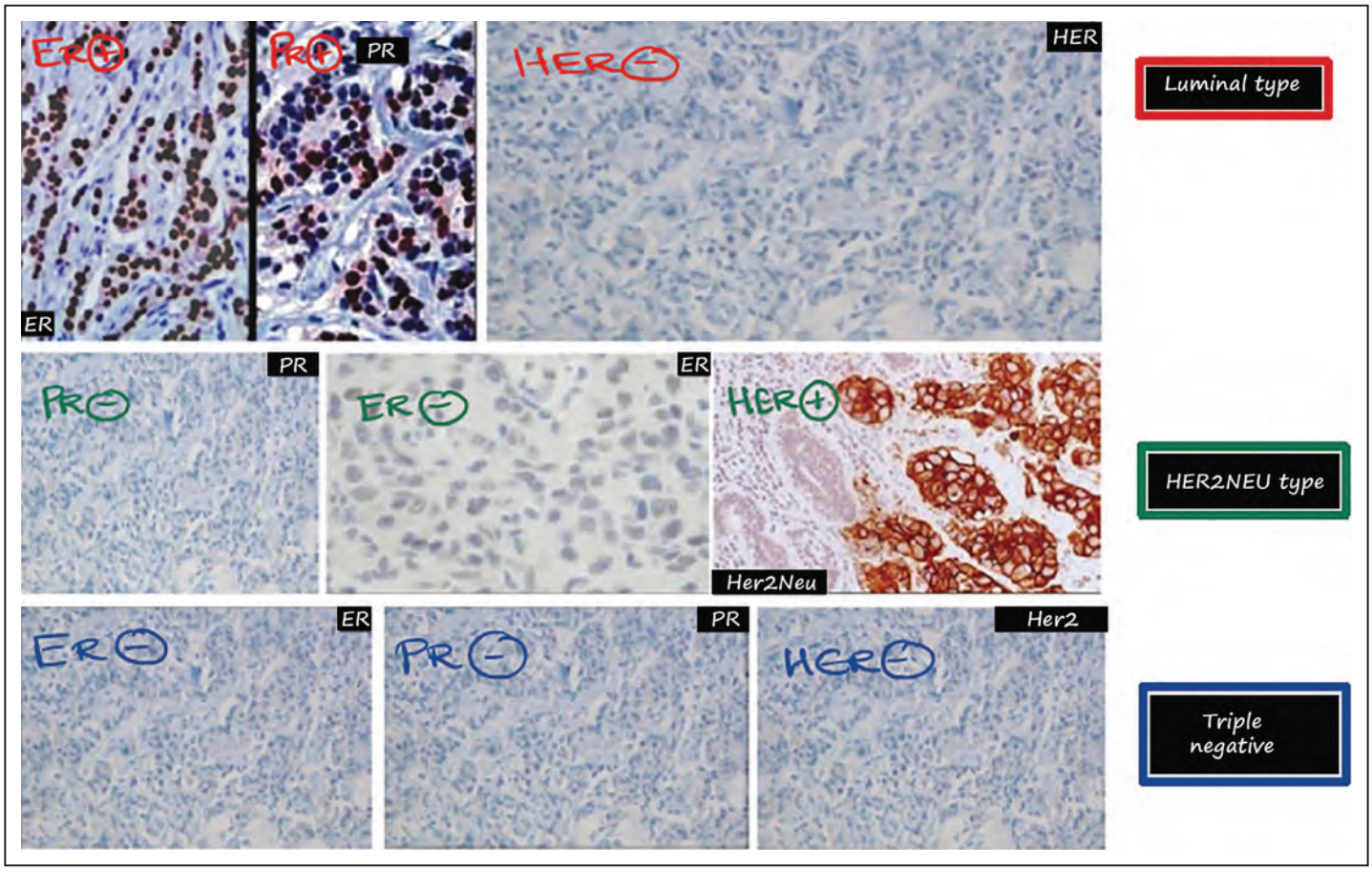
- Every cell has antigen
- Antibody is coated with DAB: Di Amino benzidine (Brown color compound)

ANTIBODY - ANTIGEN COMPLEX ⟶ BROWN COLOR - POSITIVE REACTION

Nuclear positive - ER+, PR+

Cytoplasm positive

Membrane positive- Her-2-Neu



CLAUDIN TYPE: MARKER FOR EPITHELIAL MESENCHYMAL TRANSITION
EMT type of breast cancer- Metastasis is faster

- HER2-positive cancers arise through a pathway that is strongly associated with amplification of the HER2 gene on chromosome 17q
- Most common subtype of breast cancer in patients with germline TP53 mutations (Li-Fraumeni syndrome).

PROGNOSTIC FACTORS FOR INVASIVE BREAST CARCINOMA

Prognostic factors	Comments
Elements of AJCC 8th Edition Staging	
Distant metastasis (M)	Metastasis beyond regional lymph nodes is the most important prognostic factor.
Regional lymph nodes (N)	Nodal metastasis (including the number of involved nodes) is the second most important prognostic factor.
Tumor (T)	Size, involvement of skin (e.g., ulceration or dermal metastasis), invasion into chest wall, and presentation as inflammatory carcinoma are important features.
Histologic grade	Survival diminishes with higher histologic grade.
Expression of ER, PR, and HER2	Survival is highest for the most favorable combination (high ER and PR and absent HER2) and is lowest for the least favorable combination (absent ER, PR, and HER2).
Other Prognostic Factors	
Lymphovascular invasion	Tumor cells seen in vascular spaces at the periphery of carcinomas are a poor prognostic factor.
Special histologic types	Some histologic types of cancer are strongly correlated with very favorable survival (e.g., tubular, adenoid cystic).
Response to chemotherapy	The degree of response is a strong prognostic factor for TNBC and HER2 cancers, but not the majority of luminal cancers.
Gene expression profiling	The most important clinical value of these assays is to identify patients with antiestrogen-responsive cancers who do not need chemotherapy.

STAGING OF BREAST CANCER

Stage[b]	T: Primary cancer (tumor)	N: Lymph nodes	M: Distant metastasis	10-year survival (%)
0	Ductal carcinoma in situ	No metastases	Absent	97
I	Invasive carcinoma ≤2 cm	No metastases or only micrometastases	Absent	87
II	Invasive carcinoma >2 cm Invasive carcinoma >5 cm but ≤5 cm	1–3 positive LNs 0–3 positive LNs	Absent Absent	65
III	Invasive carcinoma >5 cm Any size invasive carcinoma Invasive carcinoma with skin or chest wall involvement or inflammatory carcinoma	Negative or positive LNs ≥4 positive LNs Negative or positive LNs	Absent Absent Absent	40
IV	Any size invasive carcinoma	Negative or positive LNs	Present	5

American Joint Committee on Cancer 8th Edition: Anatomic Stage

ONE Touch Pathology

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