

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.

CBSPD

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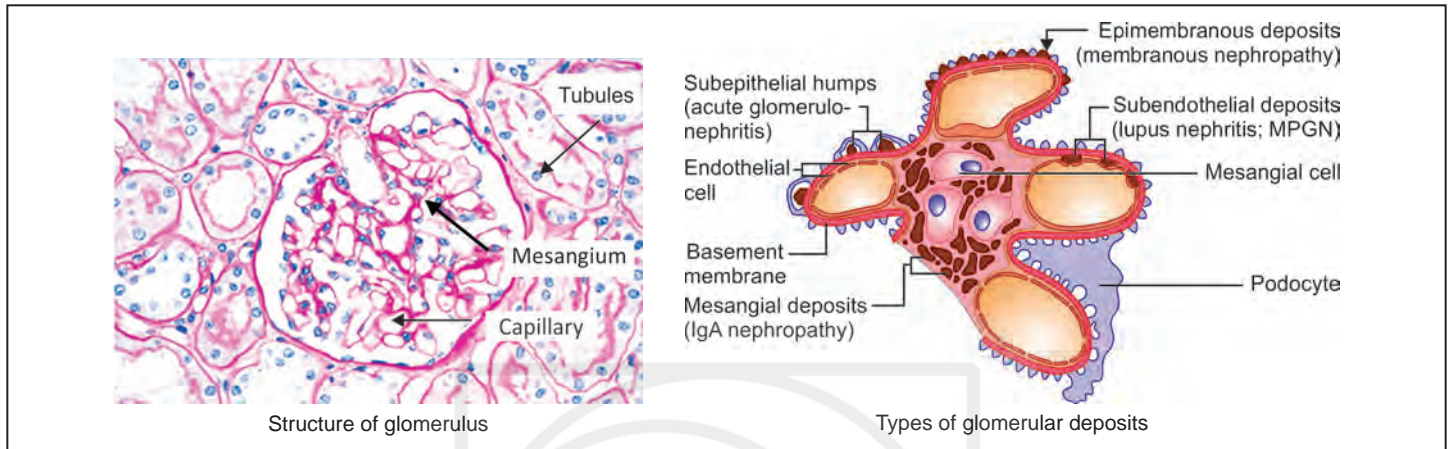
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KIDNEY

GLOMERULUS



PATHOLOGIC RESPONSES OF THE GLOMERULUS TO INJURY

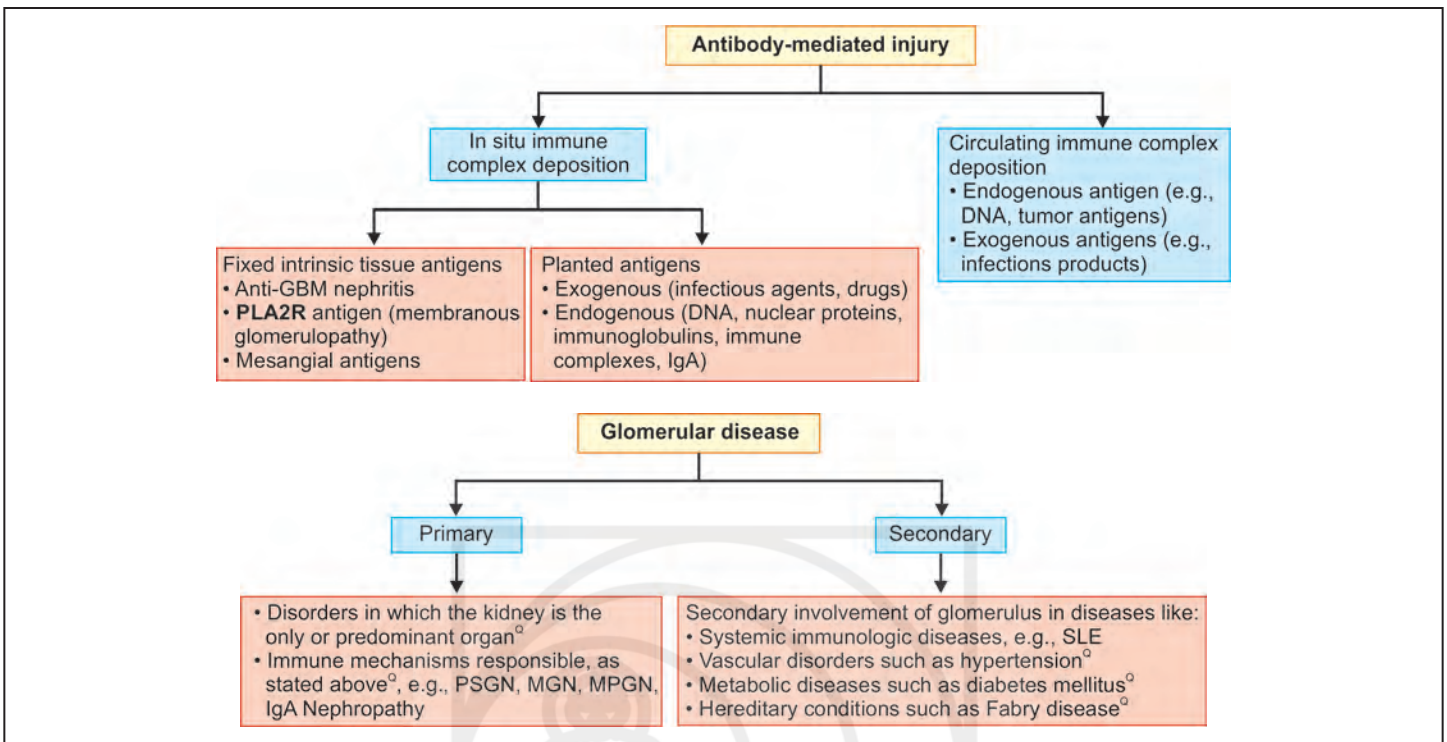
| Site of glomerular deposits | Type of glomerulonephritis |
|-----------------------------|--|
| Subepithelial deposits | <ul style="list-style-type: none"> • PSGN • Membranous GN • RPGN • Heymann Nephritis |
| Subendothelial deposits | <ul style="list-style-type: none"> • Lupus nephritis • MPGN-I |
| Membranous deposits | <ul style="list-style-type: none"> • MPGN II |
| Mesangial deposits | <ul style="list-style-type: none"> • 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



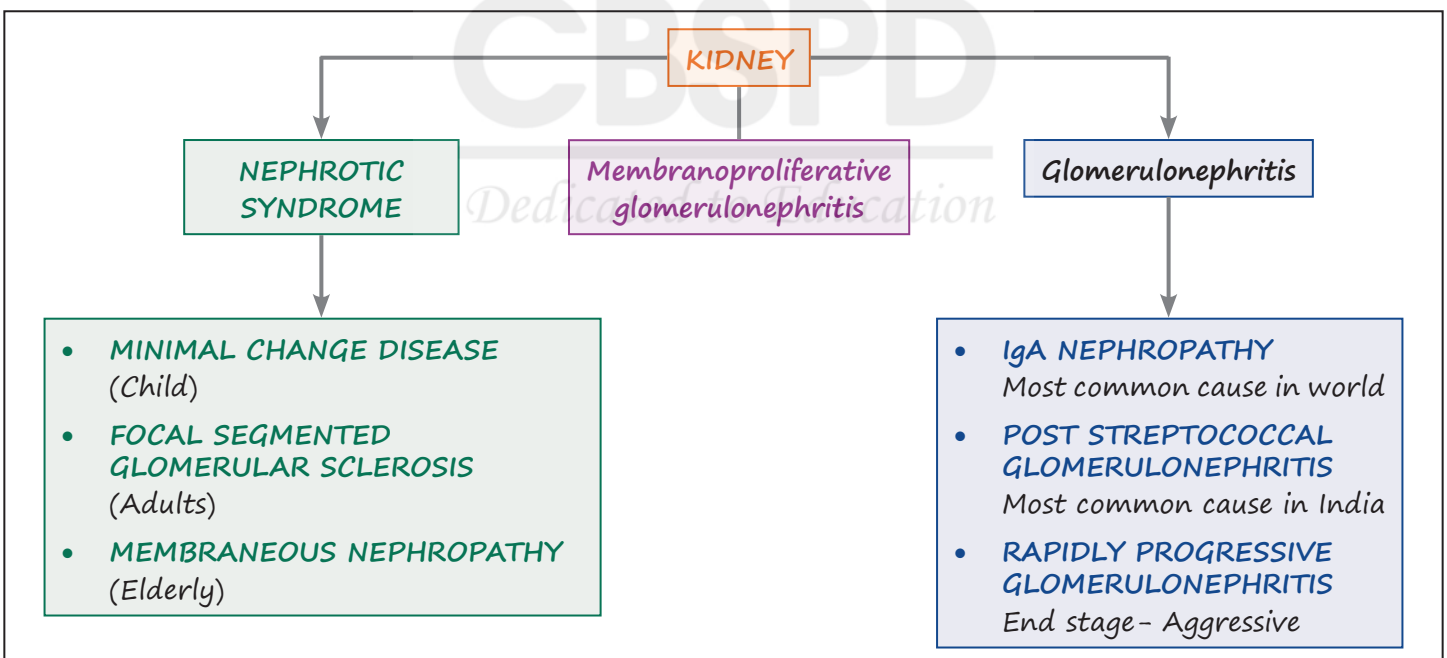
NEPHRON MUTATION

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

PODOCIN

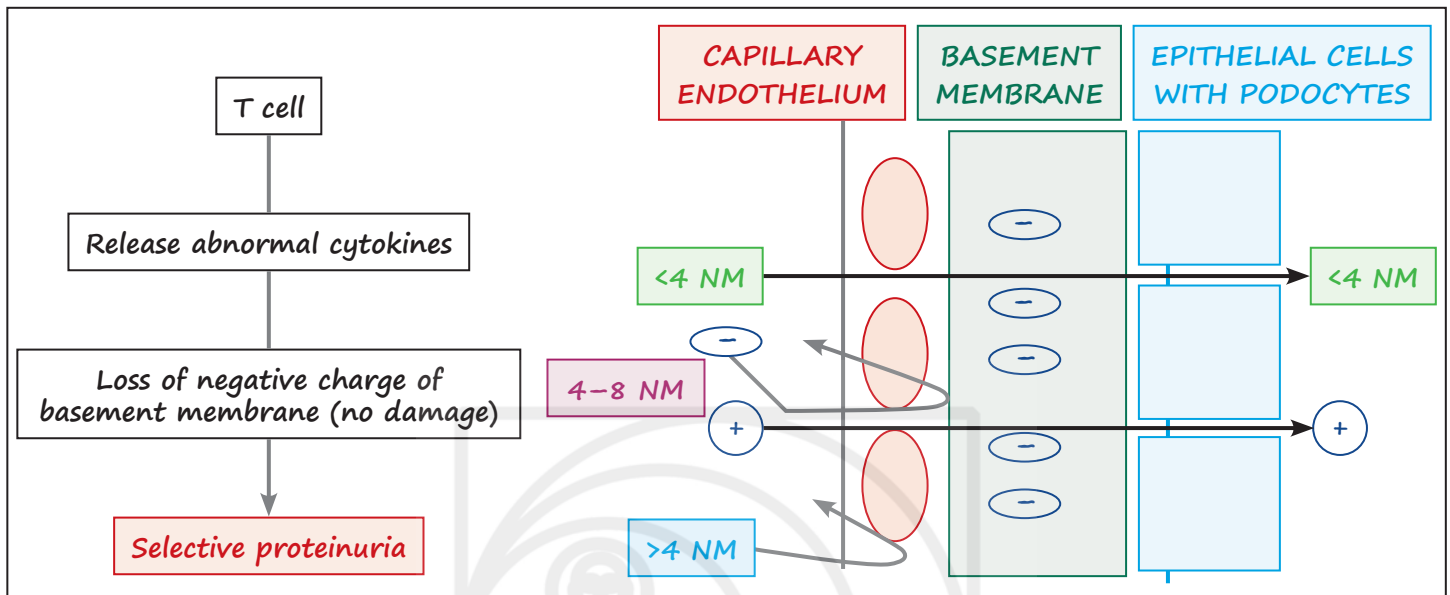
- Coded by NPHS-2
- FSGS like presentation
- Does not respond to steroids

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

Minimal change disease

↓
Flattened podocytes

- **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
- Sick cell anemia

<50% of glomeruli- Focal segmented

Podocytopathies

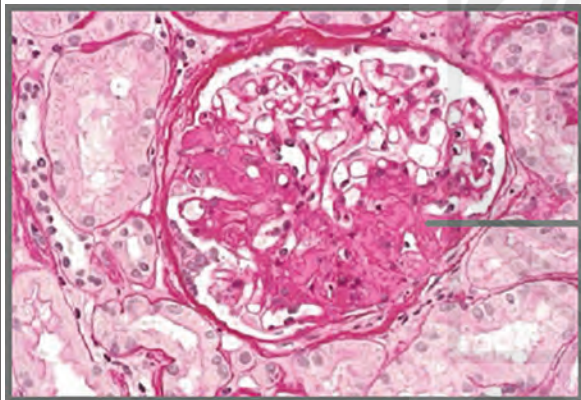
- Minimal change disease
- FSGS

DAMAGED PODOCYTES

SCLEROSIS

HIVAN

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



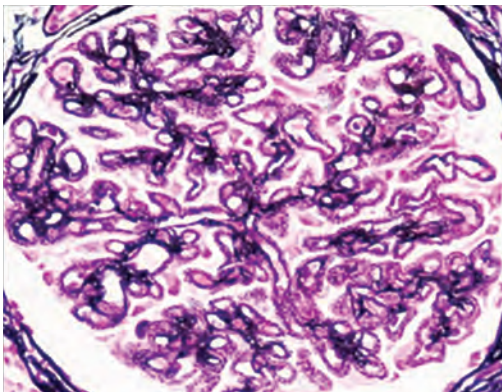
- Morphology:
 - Light microscopy
 - ♦ Collapse of capillary loops in sclerotic areas
 - ♦ Deposition of plasma proteins along capillary wall (hyalinosis)
 - Immunofluorescence microscopy
 - ♦ IgM + C3 deposition in sclerotic areas and/or in mesangium.
 - Electron microscopy
 - ♦ Diffuse effacement of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

MEMBRANOUS NEPHROPATHY

- Immune complex mediated
- Antigen - Phospholipid A2

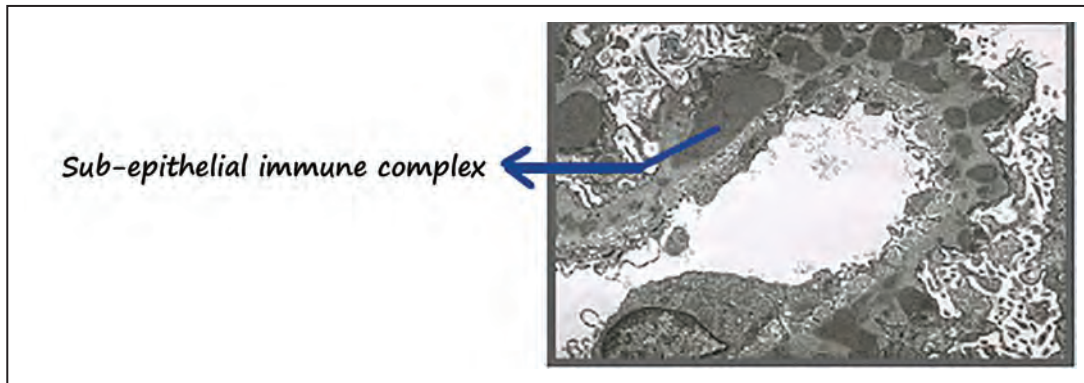
ON LIGHT MICROSCOPY

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



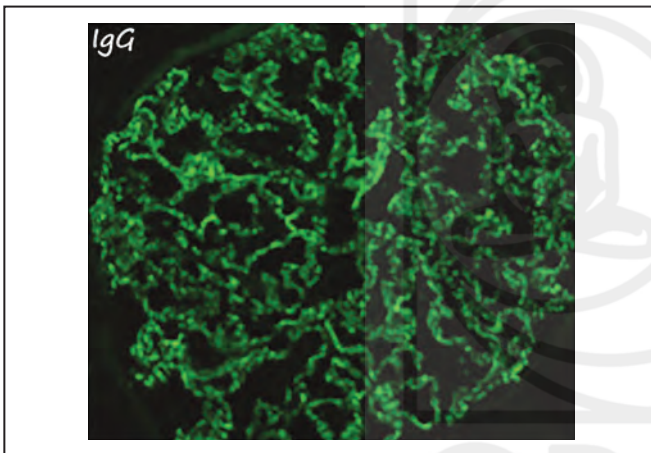
- Morphology:
 - Light microscopy: Uniform, diffuse thickening of the glomerular capillary wall.^a
 - Immunofluorescence microscopy: Granular/Lumpy bumpy^a electron dense immune complexes deposits
 - Electron microscopy: Granular deposits (Ig + complement)
 - ♦ Effacement of podocyte foot processes^a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

ON ELECTRON MICROSCOPY



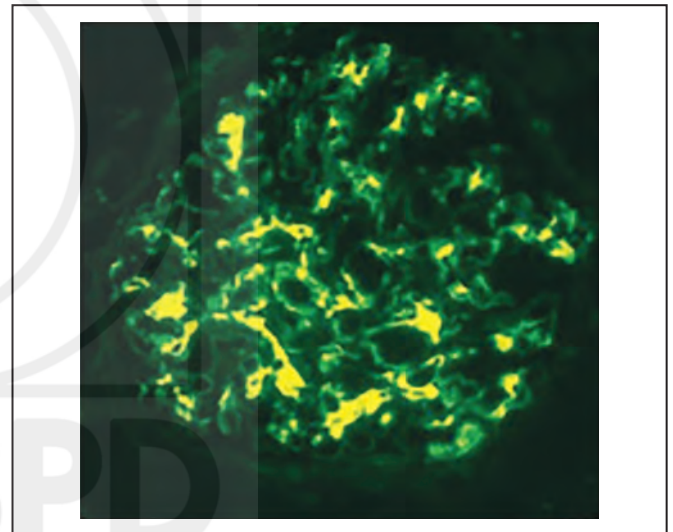
IMMUNOFLUORESCENCE

- Fluoro isothicyanate (green color)
- Granular immunofluorescence



IN IMMUNOFLUORESCENCE

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

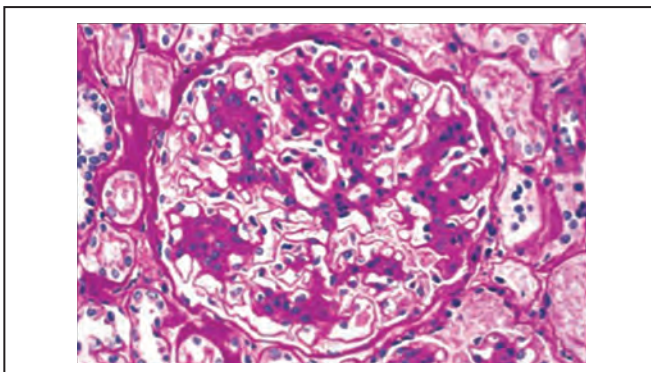


IgA NEPHROPATHY (BERGER'S DISEASE)

- 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



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

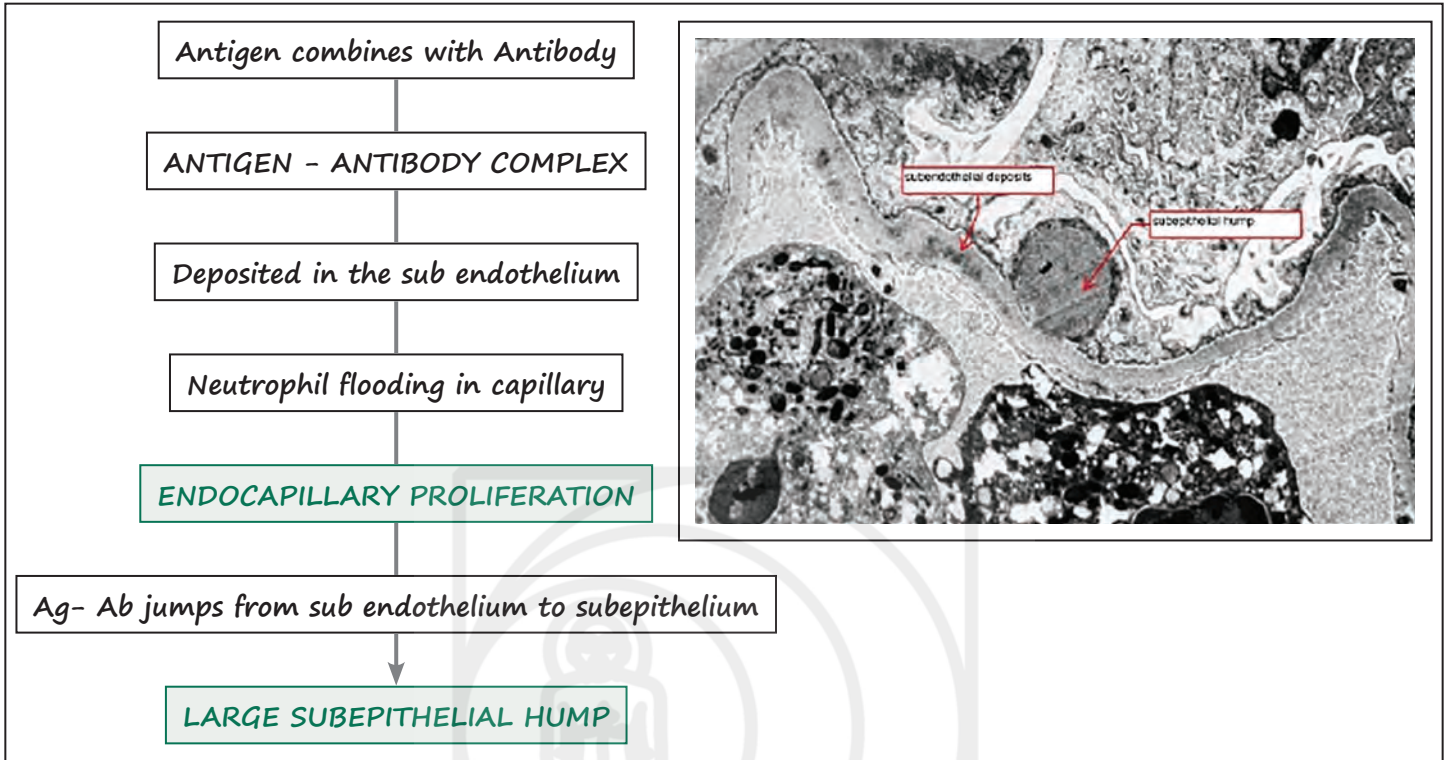
TREATMENT

- Transplantation
- Reoccurs after transplantation

POST STREPTOCOCCAL GLOMERULONEPHRITIS

- Immune complex mediated
- Caused by B hemolytic streptococci

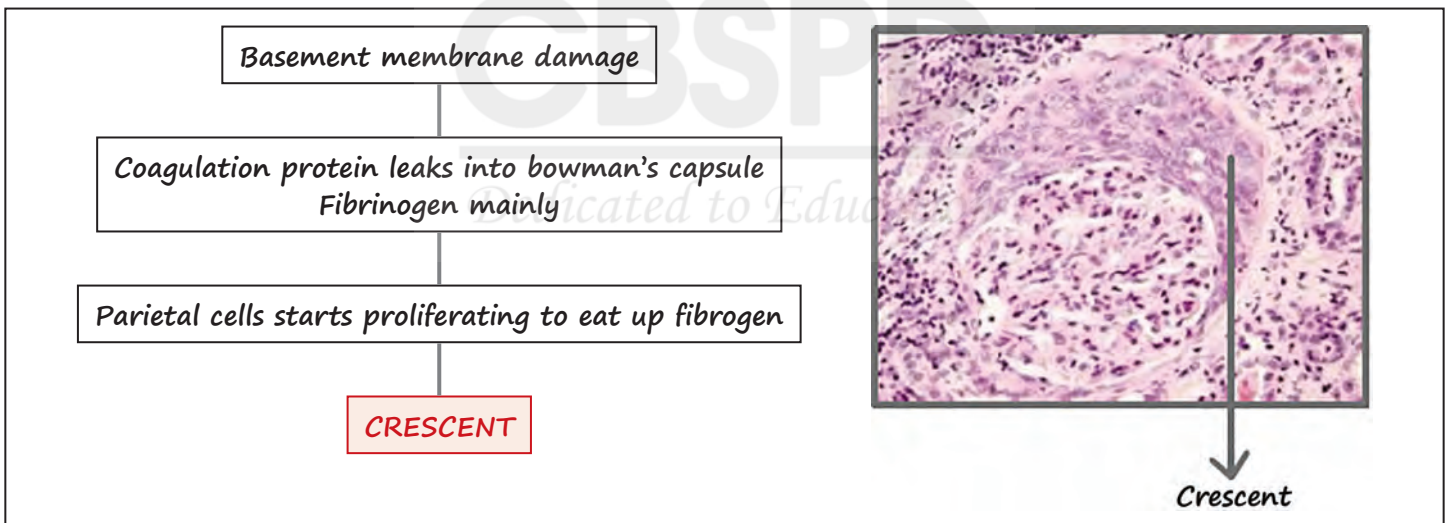
ANTIGEN- STREPTOCOCCAL PROGENITOR EXOTOXIN B - SPE B



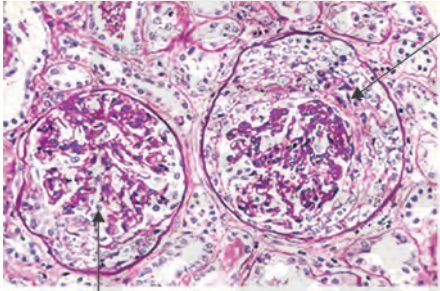
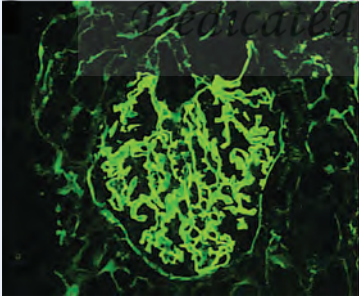
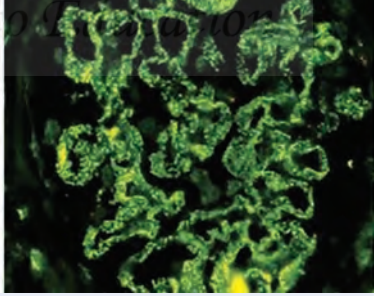
- Within 6 weeks heals by itself
- Complement deposition (C1, C3, C4)

RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS

Hallmark: BASEMENT MEMBRANE DAMAGE



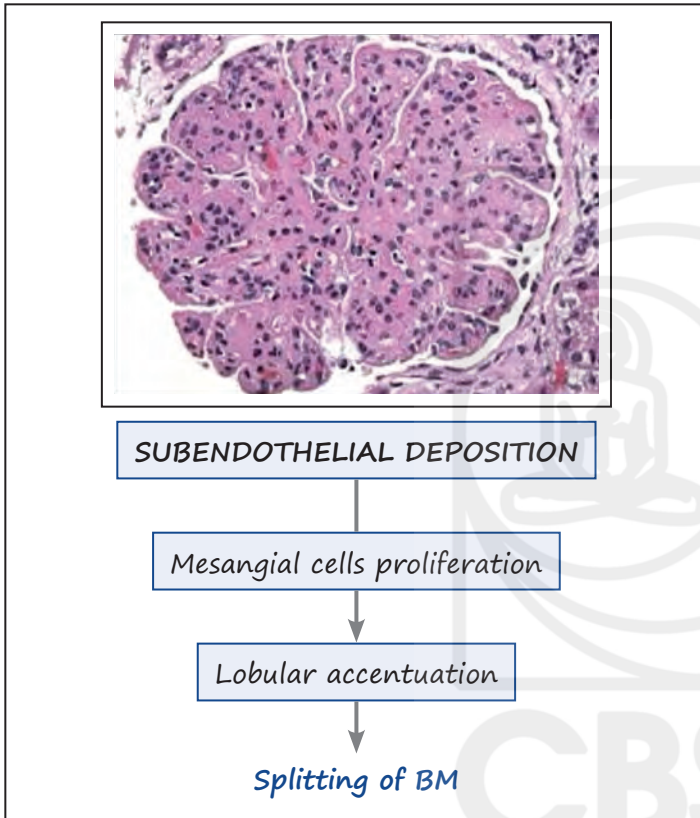
CRESCENT - Fibrinogen, inflammatory cells, parietal cells
 Very big crescent can interact visceral epithelial cells

| 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) | <ul style="list-style-type: none"> • Postinfectious <ul style="list-style-type: none"> ▪ Poststreptococcal glomerulonephritis^Q ▪ Bacterial endocarditis^Q • Noninfectious <ul style="list-style-type: none"> ▪ SLE^Q, HSP^Q ▪ Mixed cryoglobulinemia^Q • Primary renal disease <ul style="list-style-type: none"> ▪ MPGN^Q ▪ IgA nephropathy^Q | ANCA-associated <ul style="list-style-type: none"> • Idiopathic • Granulomatosis with polyangiitis (Wegener granulomatosis)^Q • Microscopic polyangiitis^Q • Hypersensitivity vasculitis^Q |
| Grossly | Kidneys are enlarged and pale, often with petechial hemorrhages on the cortical surfaces. (FLEA-BITTEN KIDNEY) ^Q | | |
| Light m/e | <ul style="list-style-type: none"> • Glomeruli: Crescents are Hallmark^Q. • Focal and segmental necrosis^Q, endothelial and mesangial proliferation^Q. • Pauci-immune: Segmental glomerular necrosis is a feature characteristic^Q. <div style="border: 1px solid black; padding: 5px; margin-top: 10px;">  <p style="text-align: center;">Crescentic glomerulonephritis</p> <p style="color: red; margin-left: 20px;">Crescents—formed by</p> <ul style="list-style-type: none"> • Proliferation of parietal cells^Q • Infiltration by WBCs^Q • Fibrin strands.^Q <p style="margin-left: 20px;">Crescents obliterate the urinary space and compress the glomerular tuft, hence</p> <p style="margin-left: 20px;">More the number of crescents → poorer the prognosis^Q</p> </div> | | |
| Immunofluorescence m/e | Linear GBM fluorescence ^Q  <p style="text-align: center;">Type I RPGN (linear)</p> | Granular immune deposits ^Q  <p style="text-align: center;">Type II RPGN (granular)</p> | No deposition of immune reactants ^Q 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

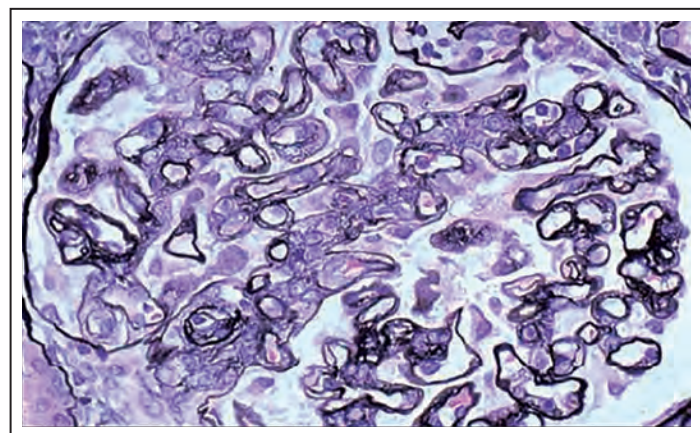
| | | | |
|----------------------------|--|----------------|--------------|
| LIGHT MICROSCOPY | All crescents | | |
| ELECTRON MICROSCOPY | All will show basement membrane damage | | |
| IMMUNOFLUORESCENCE | I Linear | II Granular | III No IM |

MEMBRANOPROLIFERATIVE GLOMERULONEPHRITIS



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

$$C3 \xrightarrow{C3 \text{ convertase}} C3a + C3b$$
- C3 convertase is then broken down by Factor H and Factor I

$$C3 \text{ NeF} \xrightarrow{-} \text{Factor H and Factor I}$$

C3Ne F blocks the action of Factor H and I Which causes more breakdown of C3 to C3a and C3b C3 will be decreased
- Deposition of dense material in laminae densa of BM (ribbon like)

SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

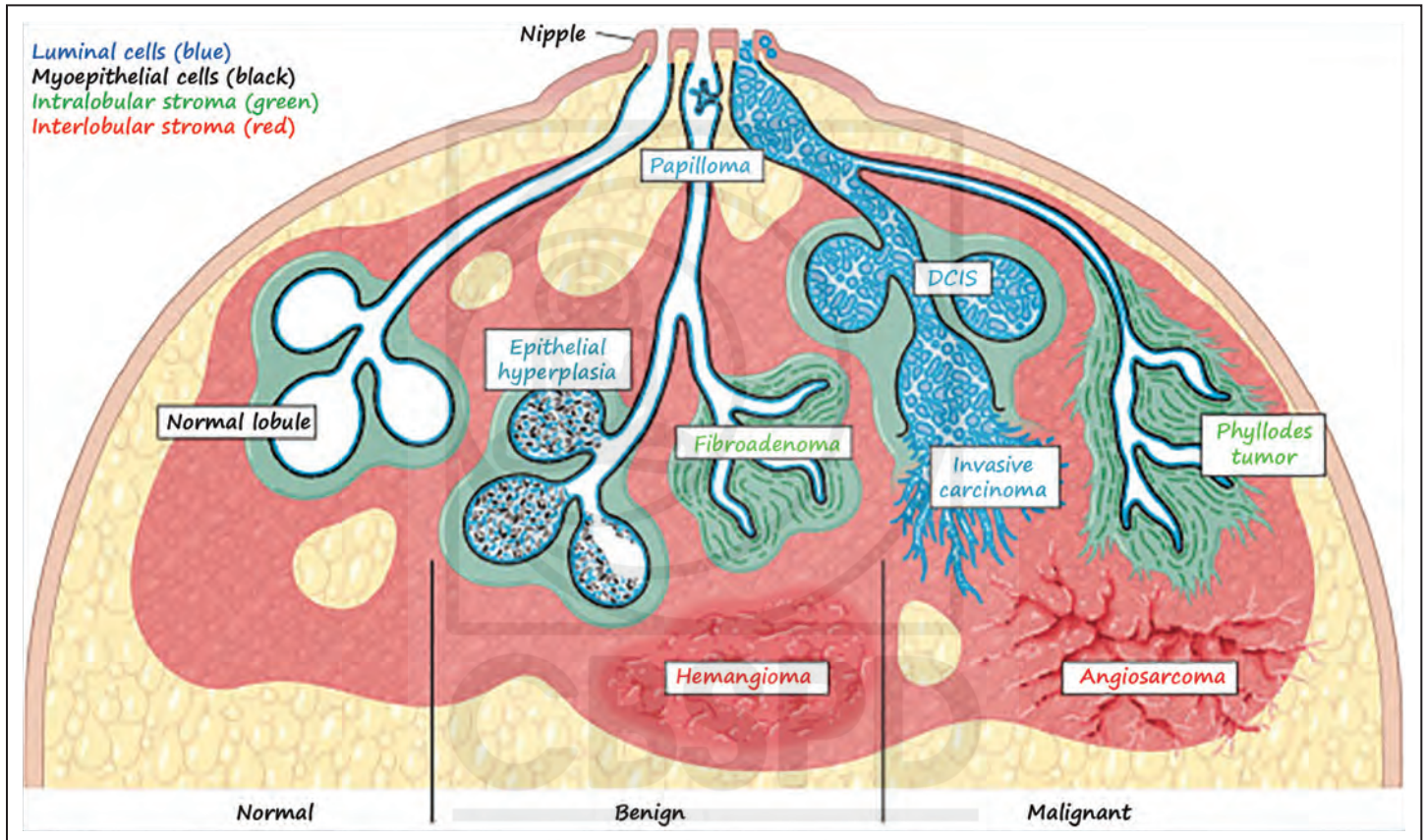
| ISN/RPS (2003) classification of lupus nephritis | |
|--|--|
| Class I | Minimal mesangial LN |
| Class II | Mesangial proliferative LN |
| Class III | Focal LN (<50% of glomeruli) III (A): active lesions III (A/C): active and chronic lesions III (C): chronic lesions |
| Class IV | Diffuse LN (50% 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.



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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

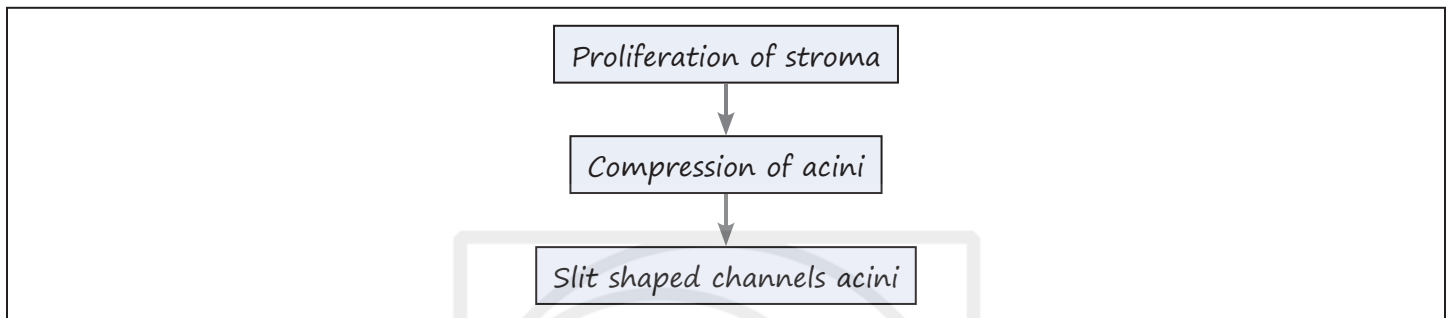
STAINS OF MEC

- P63
- Calponin
- S100
- Smooth muscle actin

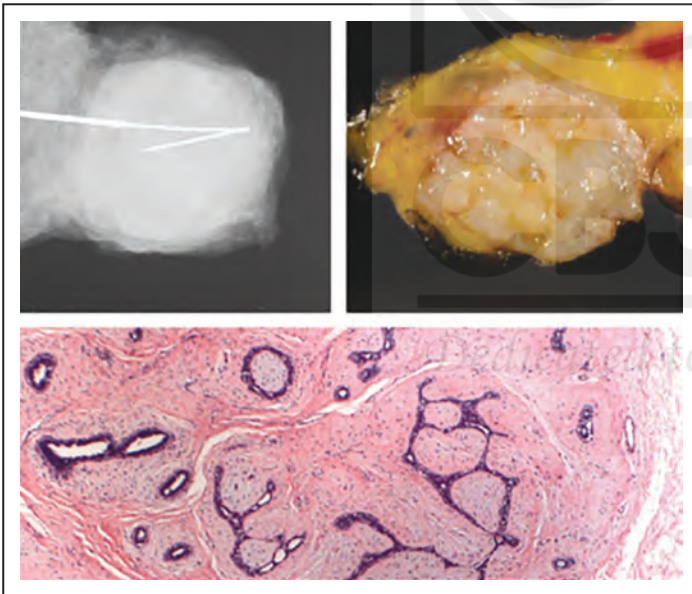
TUMORS OF THE BREAST

FIBROADENOMA

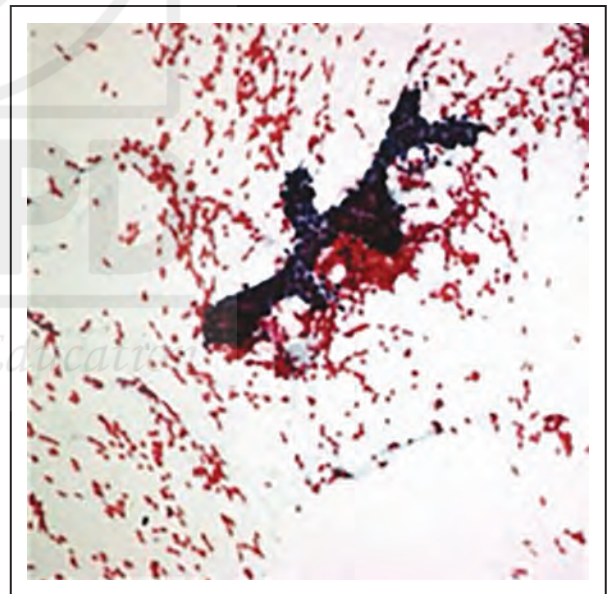
- Most common benign tumor of the female breast.
- Two-thirds of fibroadenomas harbor driver mutations in *MED12*.
- Arises from intralobular stroma
- Always benign, limited to intralobular stroma



- 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

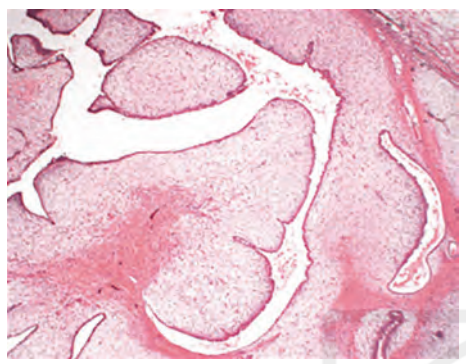


Antler horn pattern seen in FNAC

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.

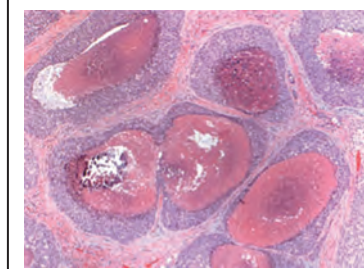
INTERLOBULAR STROMA TUMORS

- 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

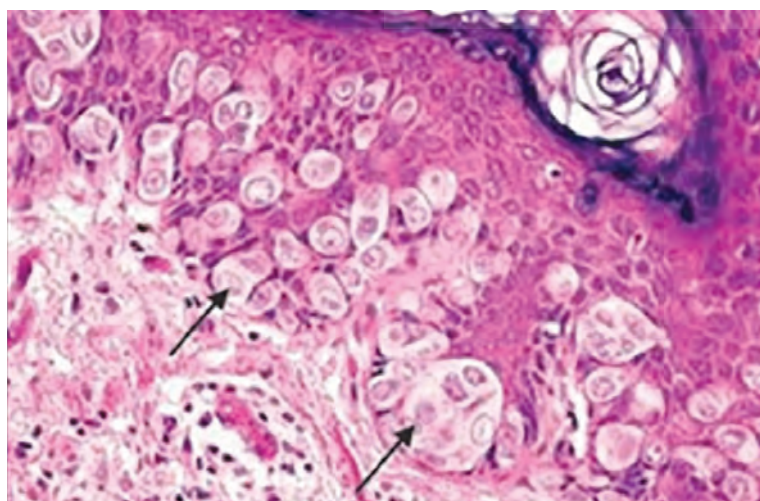


Comedo type DCIS

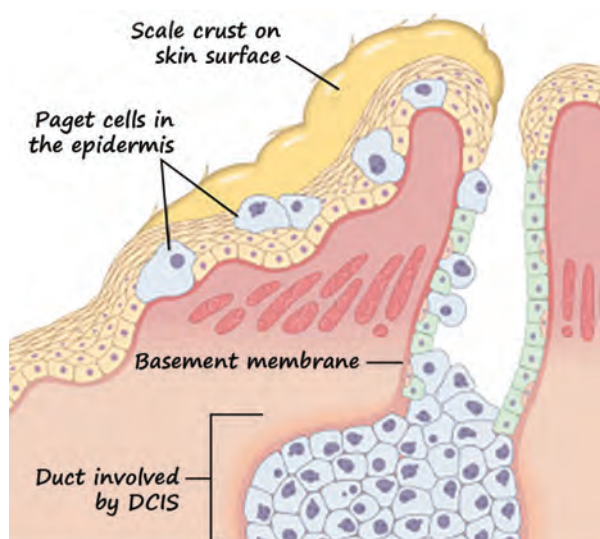
PAGET'S DISEASE OF BREAST

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

Can also occur in vulva → No underlying malignancy

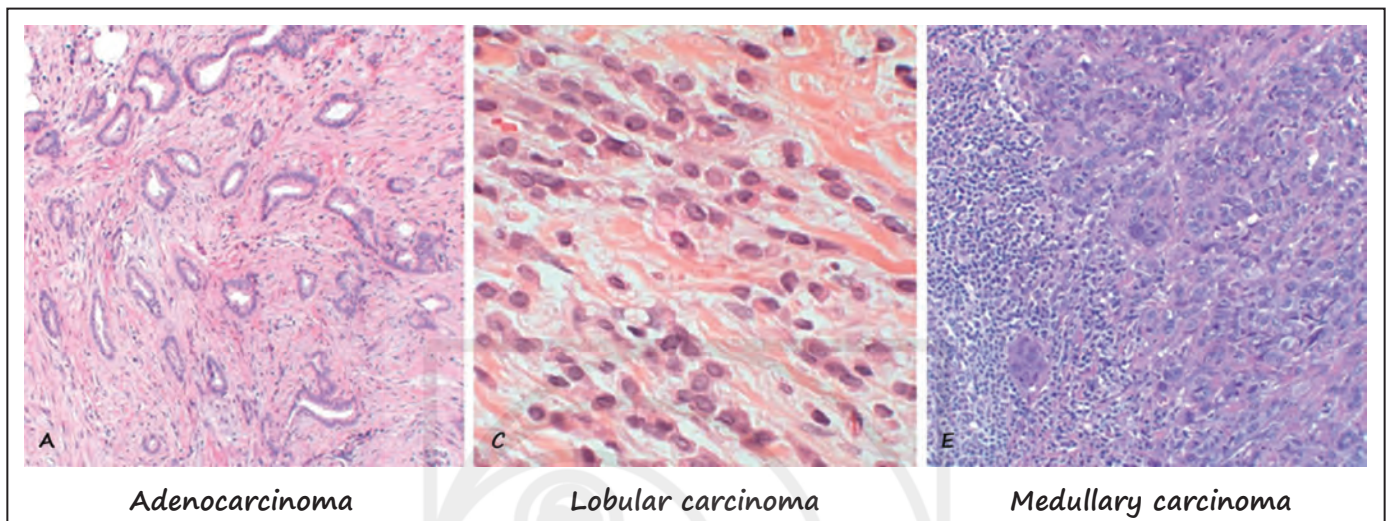


Paget's disease of vulva



MEDULLARY CARCINOMA OF BREAST

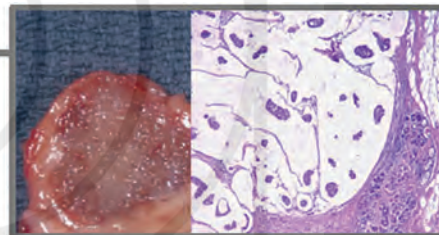
- Mimic fibroadenoma
- Grow as sheets of lymphocytic and plasma infiltrate
- Associated with triple negative mutation



MUCINOUS (COLLOID) CARCINOMA

Seen in menopausal age group

Cancers having good prognosis:
Tubular, Colloid and lobular



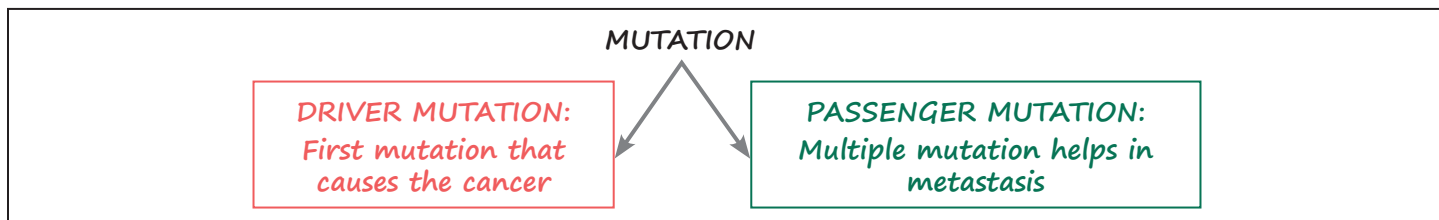
RISK FACTORS FOR DEVELOPING BREAST CANCER

| 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 inhibition; protects against metastasis

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



E cadherin is a passenger mutation.

It is a driver mutation only for Gastric CA, Lobular carcinoma of breast

LOBULAR CARCINOMA OF BREAST

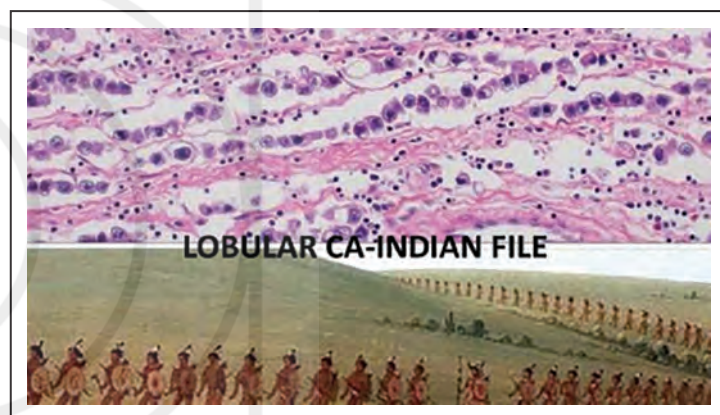
- Single cells infiltrate the breast
- Indian file pattern
- Bilateral breast cancer
- Contains targetoid mucin

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 |
| STK11 (Peutz-Jeghers) | <1% | ~40%–60%, females | Ovarian, colon, pancreas, others | Also associated with benign colon polyps |
| CDH1 (hereditary diffuse gastric cancer) | <1% | ~50%, females | Gastric signet ring cell carcinoma, colon | Majority of cancers are lobular in type |
| PALP2 (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)

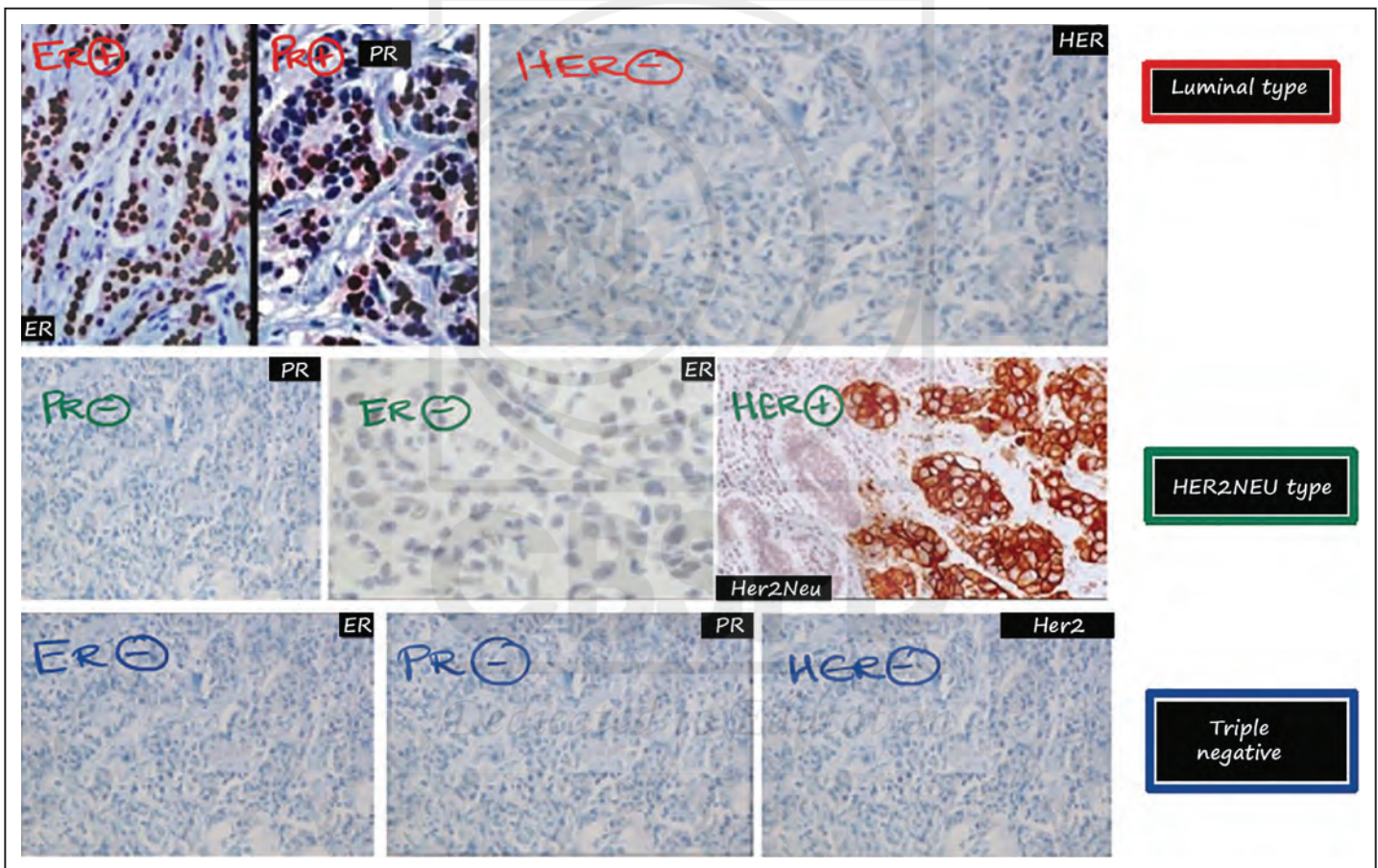
ER-negative, HER2-negative Tumors ("basal-like" triple negative Ca; 15%)–The majority of carcinomas arising in women with BRCA1 mutations

IMMUNO HISTOCHEMISTRY

- Every cell has antigen
- Antibody is coated with DAB: Di Amino benzdine (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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