BENIGN BREAST TUMORS

- Fibroadenoma
- Intraductal Papilloma
- Benign phyllodes tumor
- Lactating Adenoma
- Myoepithelioma
- Hamartoma
- Hemangioma
- Hemangiopericytoma
- Lipoma
- Granular cell tumor
- Benign stromal spindle cell tumors

CLASSIFICATION

BREAST CARCINOMA

Carcinoma *in situ*

1. Ductal Carcinoma *in situ*
2. lobular Carcinoma *in situ*

Invasive Carcinoma

1. Invasive ductal carcinoma
2. Invasive lobular carcinoma
3. Medullary carcinoma
4. Mucinous (colloid) carcinoma
5. Papillary carcinoma
6. Tubular carcinoma
7. Paget's disease of the nipple
8. Rare cancers (adenoidcystic, squamous cell, apocrine)

SKIN

Basal cell carcinoma	Sequmaus cell carcinoma
➢ It is the **most common** skin malignancy	➢ It is the **second most common** skin malignancy.
➢ It is also known as **"Rodent Ulcer"**	➢ Squamous cell carcinoma arising from a scar known as **a" Marjolin's scar".**
➢ **Ultraviolet radiation** is the most important Risk factor.	
➢ Over **90%** occur on the face above a *line from the lobe of ear to the corner of mouth.*	➢ There is usually an ulcerative or *cauliflower* like proliferative lesion with an **everted edges**.
	➢ SCC **spread by all three method** i-e local invasion , lymphatic spread and blood borne metastasis.
➢ The most common presentation is as an **ulcer that never heals.**	
➢ The ulcer has **raised and beaded edges.**	➢ SCC is strongly associated with chronic inflammation , chronic sinus trace and pre-exciting scar
➢ BCS is locally aggressive and very rarely metastasis by blood but **never metastasis through lymphatics**	➢ Treatment is surgical excision.

BASAL CELL CARCINOMA
VERSUS
SQUAMOUS CELL CARCINOMA

Characteristic	Basal Cell Carcinoma	Squamous Cell Carcinoma
Tumor origination	The deep layer of the epidermis; in the basal cells	The superficial layer of the epidermis; in the keratinocytes
Occurrence on the body	Only occurrence is in the skin	Can occur in places other than the skin. Including: lungs, thyroid, and esophagus
Genes involved in tumor expression	Proto-oncogenes: c-*fos*, c-*myc*, H-*ras*, and N-*ras*, among others.	Cell-cylce regulator genes, tyrosine kinase receptor genes, RAS/MAPK and PI3K signaling pathway genes, among others.
Tumor location on body	Mainly on ears and nose	On ears and neck, but also on trunk, lips
Incidence	Most common nonmelanoma	Second-most common nonmelanoma

Gleason Scale

Well differentiated

Small, uniform glands

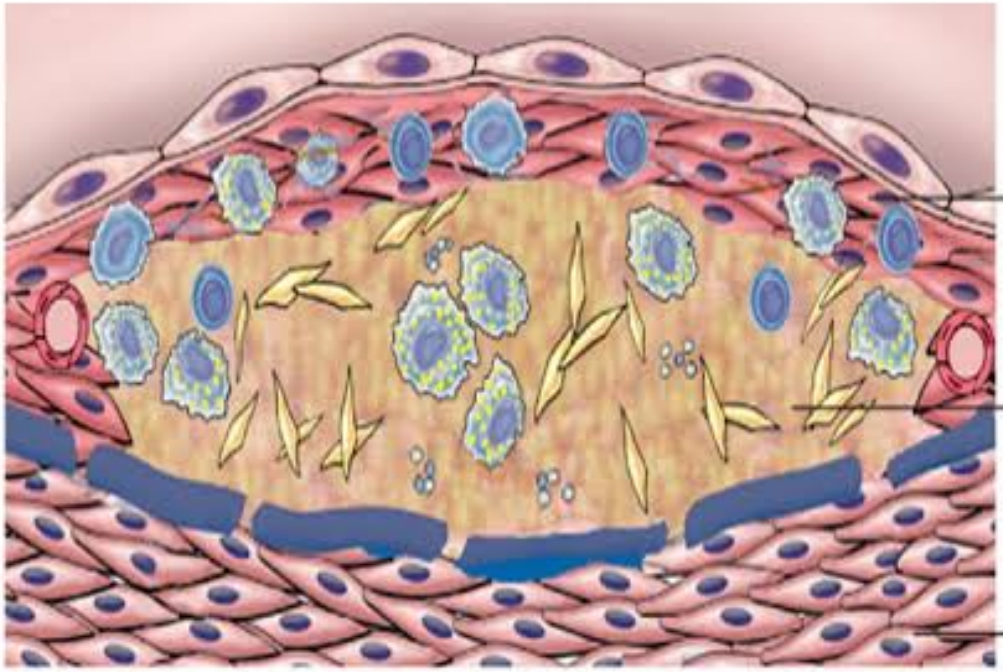
More space between glands

Infiltration of cells from glands at margins

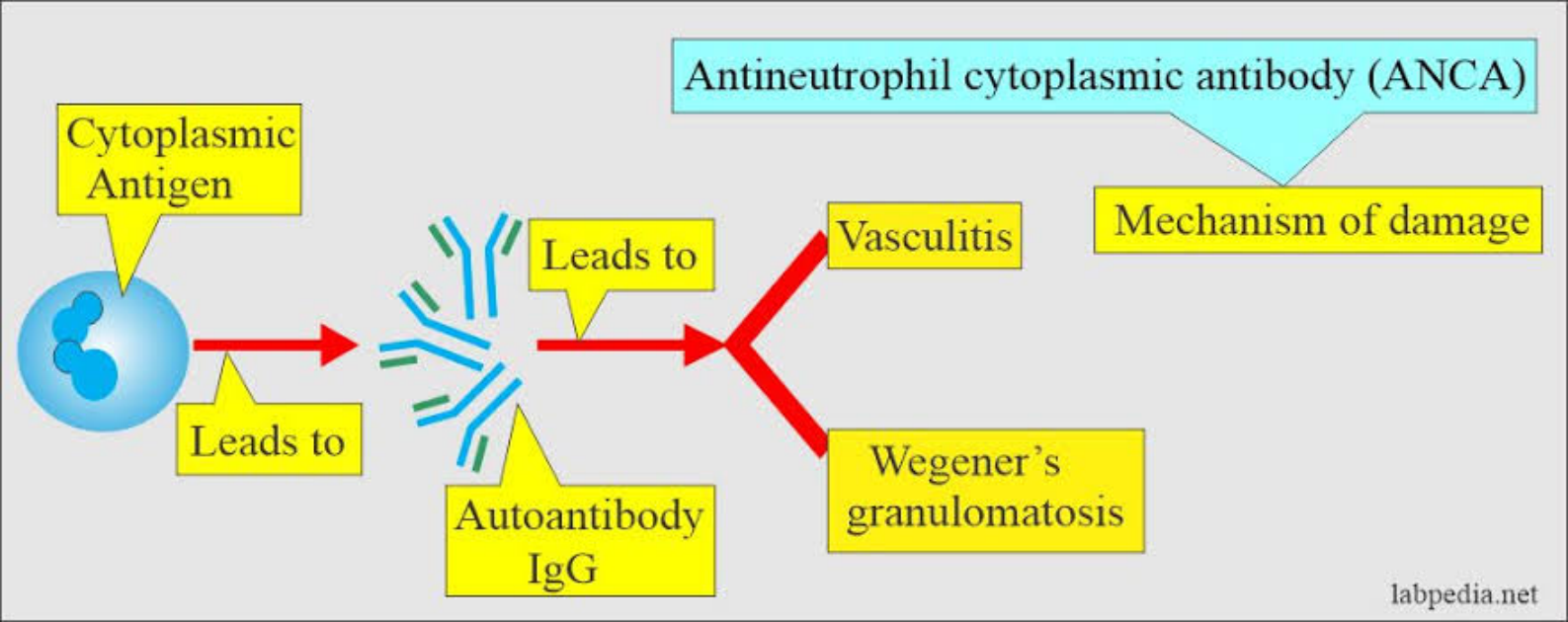
Irregular masses of cells with few glands

Lack of glands, sheets of cells

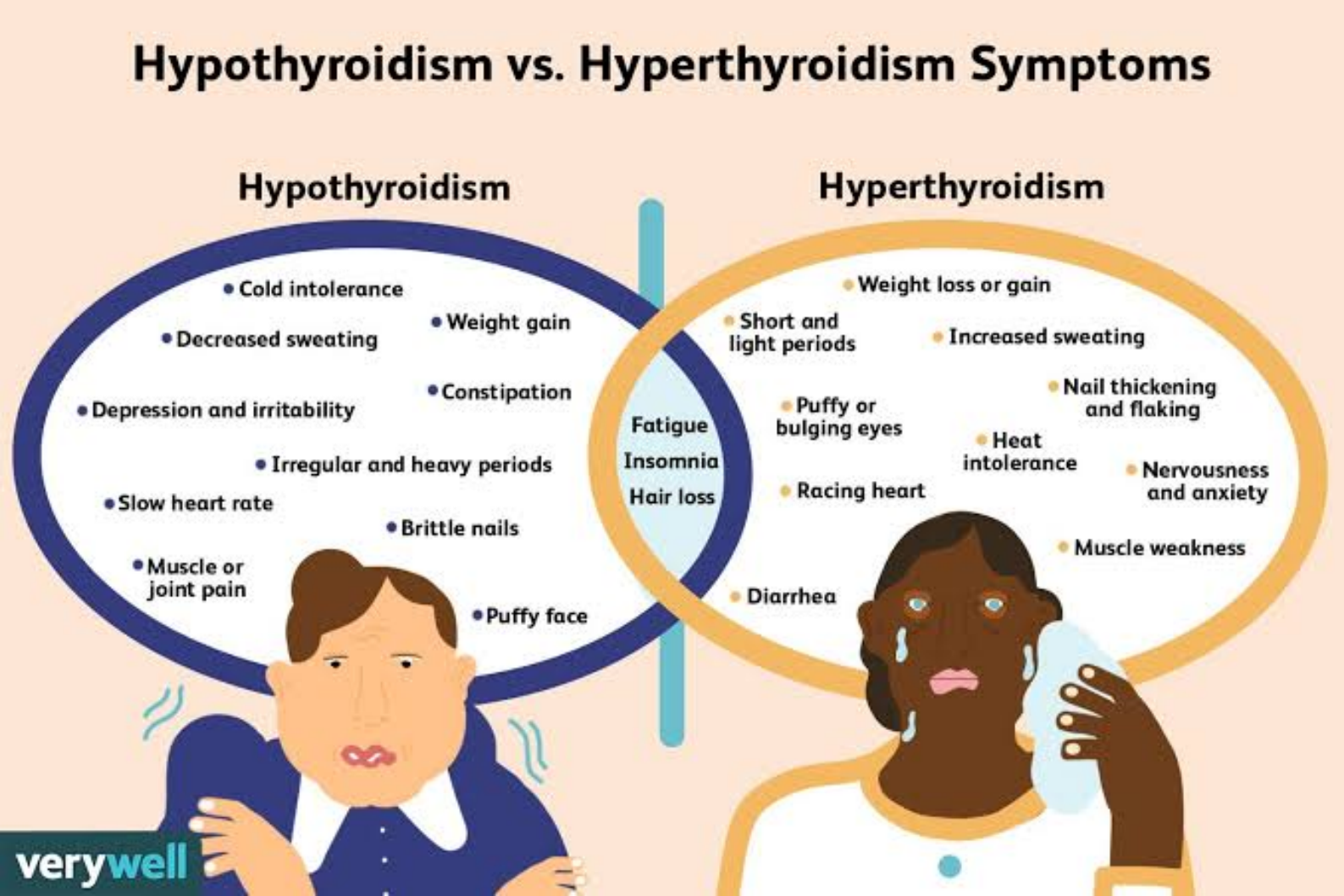
Poorly differentiated



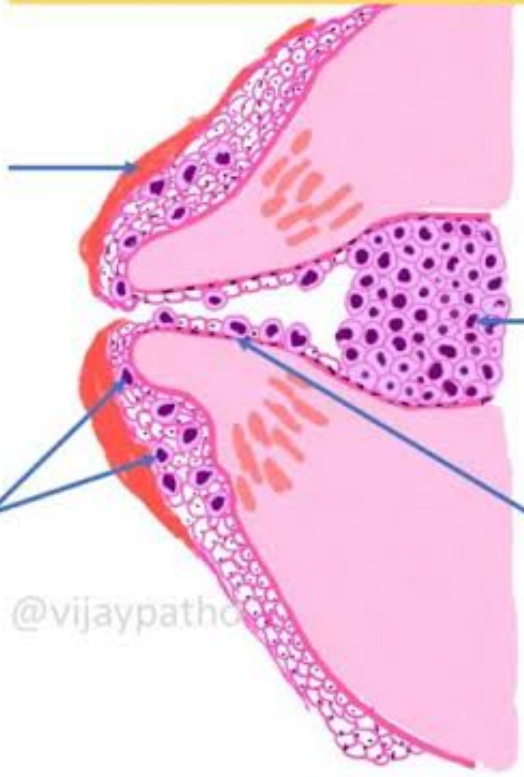
ATHEROMATOUS PLAQUE


Antineutrophil cytoplasmic antibody (ANCA)
Mechanism of damage
Cytoplasmic Antigen
Leads to
Leads to
Autoantibody IgG
Vasculitis
Wegener's granulomatosis
labpedia.net

Hypothyroidism vs. Hyperthyroidism Symptoms



PAGET'S disease of nipple



Crusting on the surface of the nipple

Underlying ductal carcinoma in situ

Paget cells within the surface epithelium of the nipple

Extension of malignant cells via lactiferous duct (basement membrane intact)

Complications

- **Anemia:** Erosive gastritis can cause chronic bleeding which, in turn, can lead to anemia
- **Atrophic gastritis:** Chronic inflammation in the stomach can cause the loss of both the stomach lining and glands
- **Peptic ulcers:** Ulcers can form in the lining of the stomach and duodenum
- **Growths in the stomach lining:** The risk of both benign and malignant growths increases in people with gastritis. If *Helicobacter pylori* (*H. pylori*) bacteria cause gastritis, they also increase the risk of a specific form of cancer known as gastric mucosa-associated lymphoid tissue (MALT) lymphoma.

Complications of Chronic Gastritis

- PEPTIC ULCER DISEASE
- MUCOSAL ATROPHY AND INTESTINAL METAPLASIA
- DYSPLASIA
- GASTRITIS CYSTICA

Kidney tumors in infants and children (WHO):

Nephroblastic tumors:
- Nephroblastoma
- Nephrogenic rests and nephroblastomatosis
- Cystic nephroma and cystic partially differentiated nephroblastoma
- Metanephric tumors
  - Metanephric adenoma
  - Metanephric adenofibroma
  - Metanephric stromal tumor

•Mesoblastic nephroma:

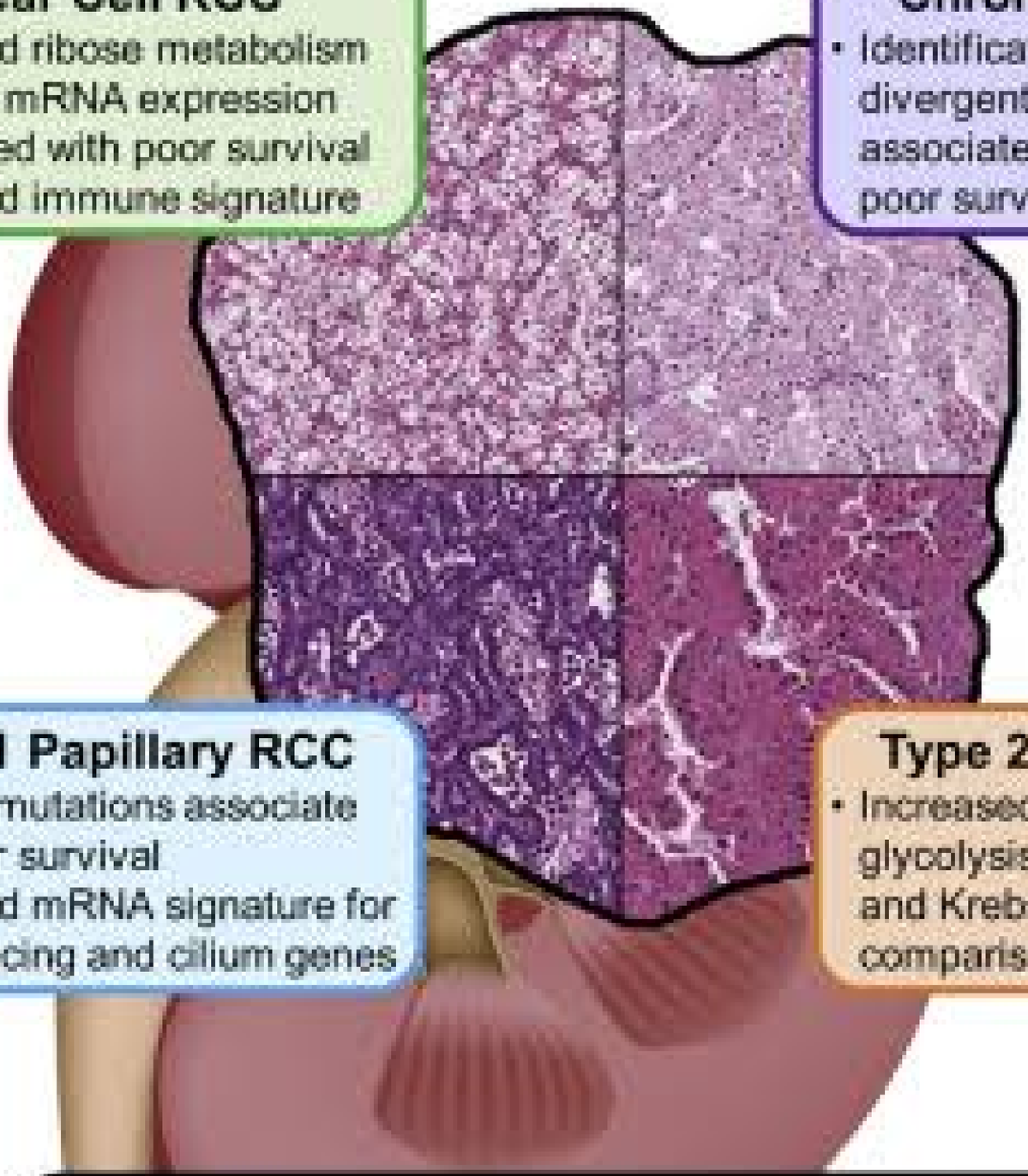
•Clear cell sarcoma

•Rhabdoid tumor of kidney

•Renal epithelial tumors of childhood:
- Papillary renal cell carcinoma
- Renal medullary carcinoma
- Translocation associated RCC (Xp11.2 / t(6;11) translocations)

•Rare tumors:
- Ossifying renal tumor of infancy
- Angiomyolipoma

TCGA Pan-Kidney Cancer Analysis (n=843)



Clear Cell RCC

- Increased ribose metabolism pathway mRNA expression associated with poor survival
- Increased immune signature

Chromophobe RCC

- Identification of metabolically divergent (MD-) ChRCCs associated with extremely poor survival

Type 1 Papillary RCC

- *PBRM1* mutations associate with poor survival
- Increased mRNA signature for RNA splicing and cilium genes

Type 2 Papillary RCC

- Increased expression of the glycolysis, ribose metabolism, and Krebs cycle genes in comparison to Type 1 PRCC

Renal Cell Carcinoma (RCC)

- Increased DNA hypermethylation and CDKN2A alterations associate with poor prognosis in all RCC subtypes
- Increased Th2 immune signature within each RCC subtype associates with poor survival

Familial renal cancer

Malignant renal cell tumors

- Clear cell RCC
- Papillary RCC
- Chromophobe RCC
- Carcinoma of the collecting ducts of Bellini
- Tubulocystic carcinoma
- Renal medulary carcinoma
- Renal carcinoma associated with Xp11.2 translocations/TFE3 gene fusions (MiTF/TFE family translocation carcinomas)
- RCC in long-term survivors after neuroblastoma
- Mucinous tubular and spindle cell carcinoma
- Thyroid follicular carcinoma-like tumor of kidney
- RCC unclassified

Renal cell neoplasms in end-stage renal disease

- RCC associated to acquiered cystic disease
- Clear cell papillary RCC

Renal cell neoplasms of low malignant potential

- Multilocular cystic renal cell neoplasm of low malignant potential (multilocular clear cell RCC)

Benign renal cell tumors

- Papillary adenoma
- Oncocytoma
- Metanephric adenoma and adenofibroma

Mixed stromal and epithelial tumors (renal epithelial and stromal tumor)

- Cystic nephroma
- Mixed epithelial and stromal tumor

RCC, renal cell carcinoma.

Gastric carcinoma

CLASSIFICATION

WHO Classification:

1. Adenocarcinoma:
   a. Papillary adenocarcinoma
   b. Tubular adenocarcinoma
   c. Mucinous adenocarcinoma
   d. Signet-ring cell carcinoma
2. Adenosquamous carcinoma
3. Squamous cell CA
4. Small cell CA
5. Undifferentiated CA
6. Others

Lauren Classification:

1. Intestinal type (53%)
2. Diffuse type (33%)
3. Unclassified (14%)

Ming Classification:

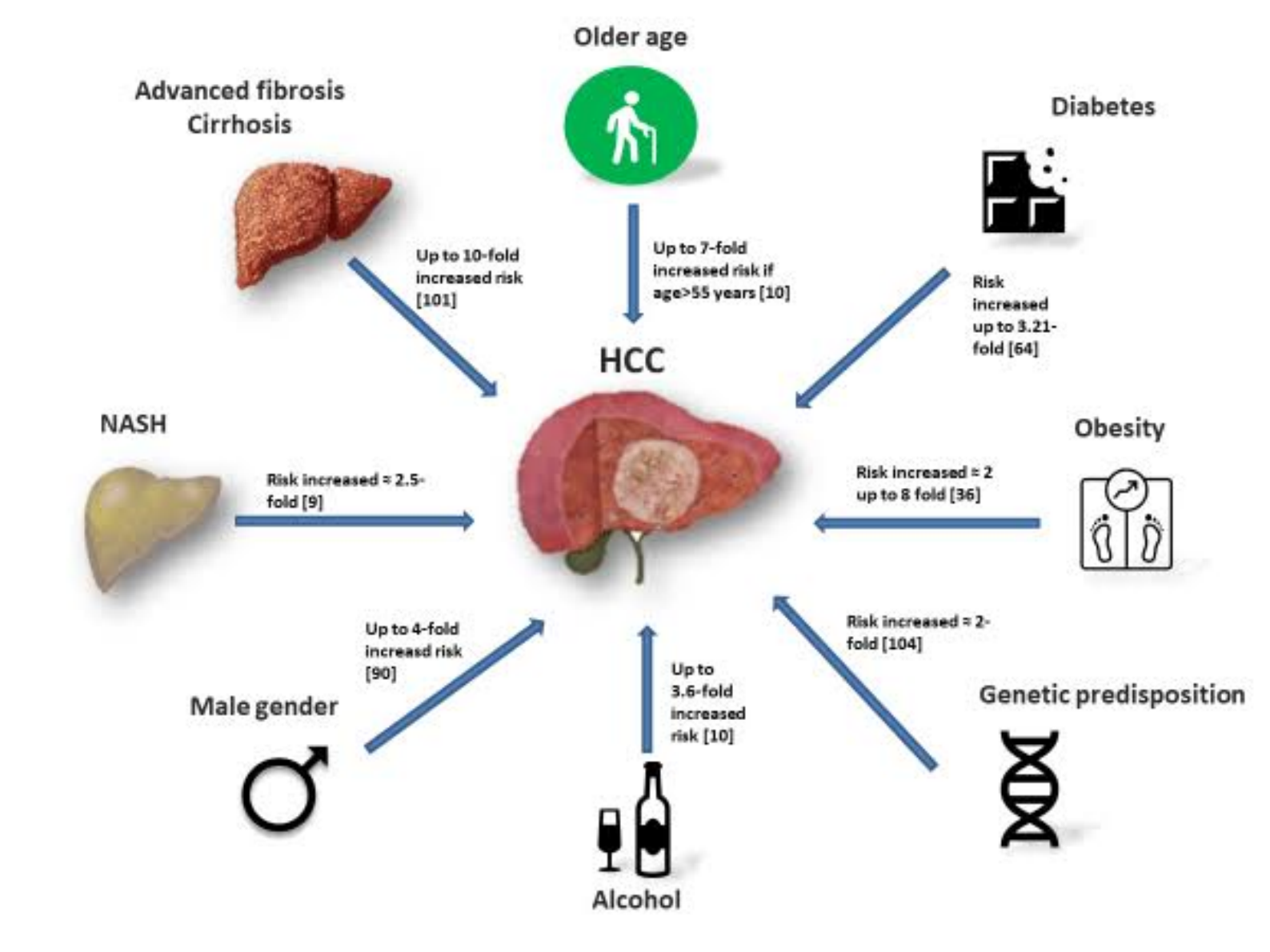
1. Expanding type (67%)
2. Infiltrative type (33%)

Causes of aplastic anemia

🇦🇷 **LIONEL MESSI** 🏟️⚽

- **L**ong term exposure to Toxins
- **I**onizing radiation
- **O**thers(thymoma, pregnancy, GVHD)
- **N**octurnal hemoglobinuria(~PNH)
- Epstein-Barr virus
- **L**upus erythematosus

- **M**edication(NSAIDs, Anti-seizure agents, Antibiotics, Anti-thyroid)
- + **M**yelodysplastic syndromes
- **E**osinophilic fasciitis, **E**BV
- **S**eronegative (non-A through -G) hepatitis
- **S**hwachman-Diamond syndrome and other inherited genetic abnormalities
- **I**diopathic


Older age
Advanced fibrosis
Cirrhosis
Diabetes
Up to 10-fold
increased risk
[101]
Up to 7-fold
increased risk if
age>55 years [10]
Risk
increased
up to 3.21-
fold [64]
HCC
NASH
Obesity
Risk increased ≈ 2.5-
fold [9]
Risk increased ≈ 2
up to 8 fold [36]
Up to 4-fold
increasd risk
[90]
Up to
3.6-fold
increased
risk [10]
Risk increased ≈ 2-
fold [104]
Male gender
Genetic predisposition
Alcohol

Cirrhosis:
Pathogenesis and Complications

Author:
Yan Yu
Reviewers:
Paul Ratti
Amy Maghera
Sam Lee*
** Indicates faculty member at time of publication*

Infections
- Hepatitis

Auto-Immune
- AIH, PBC, PSC

Toxin
- Ethanol

Metabolic Dx
- NAFLD

Genetic Dx
- HH, Wilson's, A1AT

Note on abbreviations:
- **-AIH:** Auto-Immune Hepatitis
- **-PBC:** Primary Biliary Cirrhosis
- **-PSC:** Primary Sclerosing Cholangitis
- **-NAFLD:** Non-Alcoholic Fatty Liver Disease
- **-HH:** Hereditary Hemochromatosis
- **-A1AT:** Alpha-1 Anti-Trypsin

(These disorders, as well as all other causes of Liver Cirrhosis, will be described in subsequent slides)

Death of hepatocytes, inflammatory destruction of normal hepatic architecture → Scarring, fibrosis

Liver is highly regenerative, but here it must regenerate within extensively scarred/fibrotic tissue, forming nodules of poorly-functioning cells → disrupting hepatic vasculature, biliary production/excretion, and other liver functions.

Cirrhosis

↑ resistance to blood flow through fibrotic liver

Hepatocellular carcinoma (HCC) (85% of HCCs occur in background of cirrhosis)

↓ liver function **("liver insufficiency")**

Portal hypertension:
↑ blood pressure in the hepatic circulation

Kideys retain more water & Na+ to ↑ blood volume

Into esophageal varices:
- Hemorrhage could cause an **Upper GI Bleed!**

Blood backs up into the collateral venous system

↑ hydrostatic pressure in abdominal capillaries

↓ effective blood volume felt by kidneys

↑ vasodilators (NO, CO)

Into the spleen:
Splenomegaly
(In turn, a congested, enlarged spleen could lead to ↑ trapping of blood cells within, which could cause pancytopenia)

Into rectal varices:
- Hemorrhage could cause an **Lower GI Bleed!**

Fluid exudes from plasma in the capillaries

Fluid exudation into peritoneal cavity→ **Ascites**

Low Albumin Synthesis

↓ oncotic pressure in systemic capillaries

Fluid exudes from plasma in the capillaries into interstitial tissues

Edema

Liver unable to synthesize clotting factors or anti-coagulant proteins

Low measured clotting factors 10, 5, 2, etc; low Protein C, Protein S, antithrombin, etc

Usually the ↓ in pro- and anti-coagulants are in balance, no coagulopathy results

Liver unable to remove toxins from body

Toxins (i.e. NH3) build up, cross blood-brain-barrier

Encephalopathy
(degeneration of neurological function: confusion, asterixis, etc)

- ↓ conjugation of bilirubin
- ↓ secretion of conjugated bilirubin into bile duct canaliculi
- ↓ drainage of conjugated bilirubin out of the ducts

When bilirubin > 40-50:
Jaundice,
Sclera icterus

Legend: = pathophysiology = mechanism = sign/symptom/lab finding = complications

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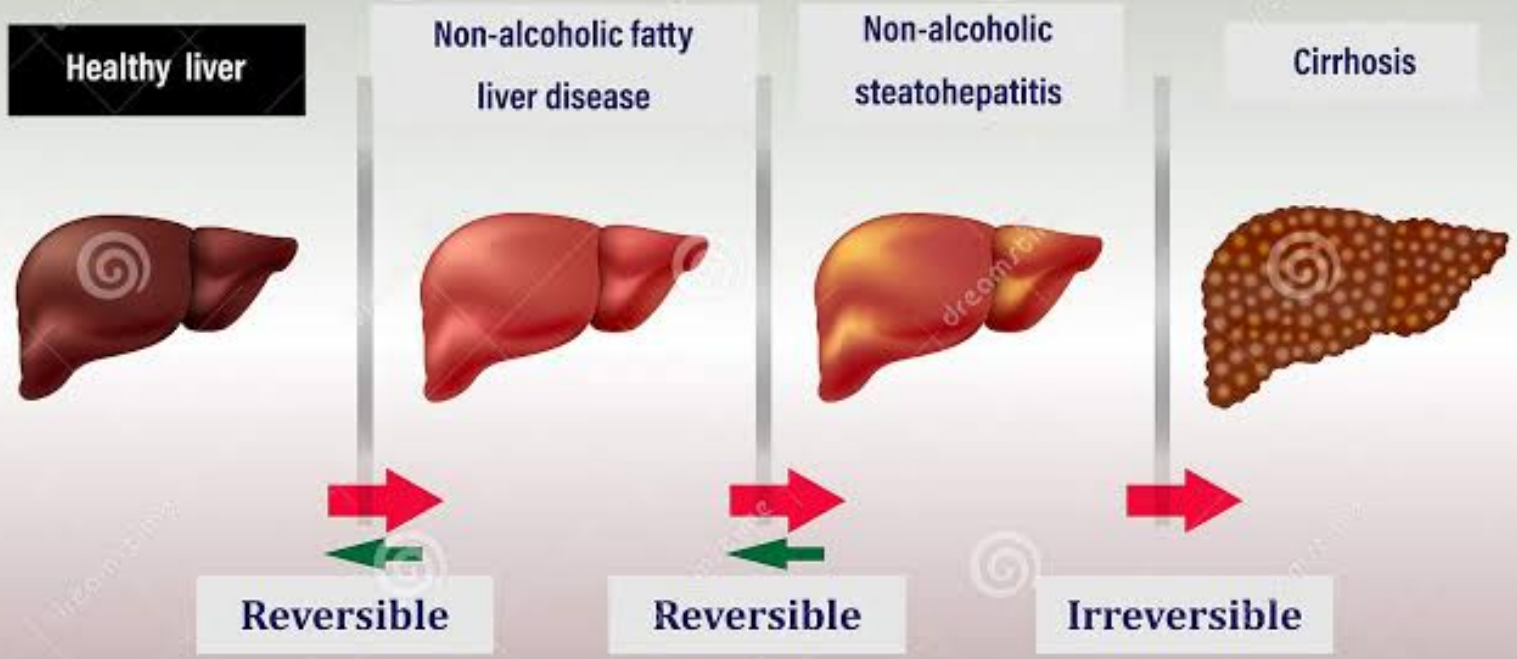
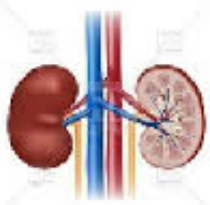
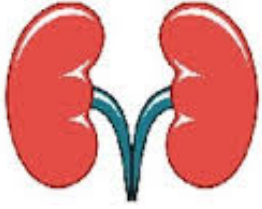


Pathologic Classification of Colorectal Polyps

Neoplastic (Adenomatous) Polyps	Non-Neoplastic Polyps	Submucosal Polyps
Benign	Hyperplastic	Lymphoid
Mild dysplasia	Mucosal	Lipoma
Moderate dysplasia	Inflammatory	Other
High-grade dysplasia	Hamartomatous	
Severe dysplasia	Juvenile	
Carcinoma in situ	Peutz-Jeghers	
Malignant		
Invasive carcinoma		

Kidney Function Tests





- The tests that are part of the Kidney Function test panel are:
- (a) Urine examination
- (b) Serum Urea
- (c) Serum creatinine
- (d) Blood urea nitrogen (BUN)
- (e) Calcium
- (f) Phosphorus
- (g) Protein
- (h) Albumin
- (i) Creatinine clearance
- (j) Urea clearance
- (k) Inulin clearance
- (l) Dilution and Concentration test
- (l) Serum electrolyte levels

medcrine

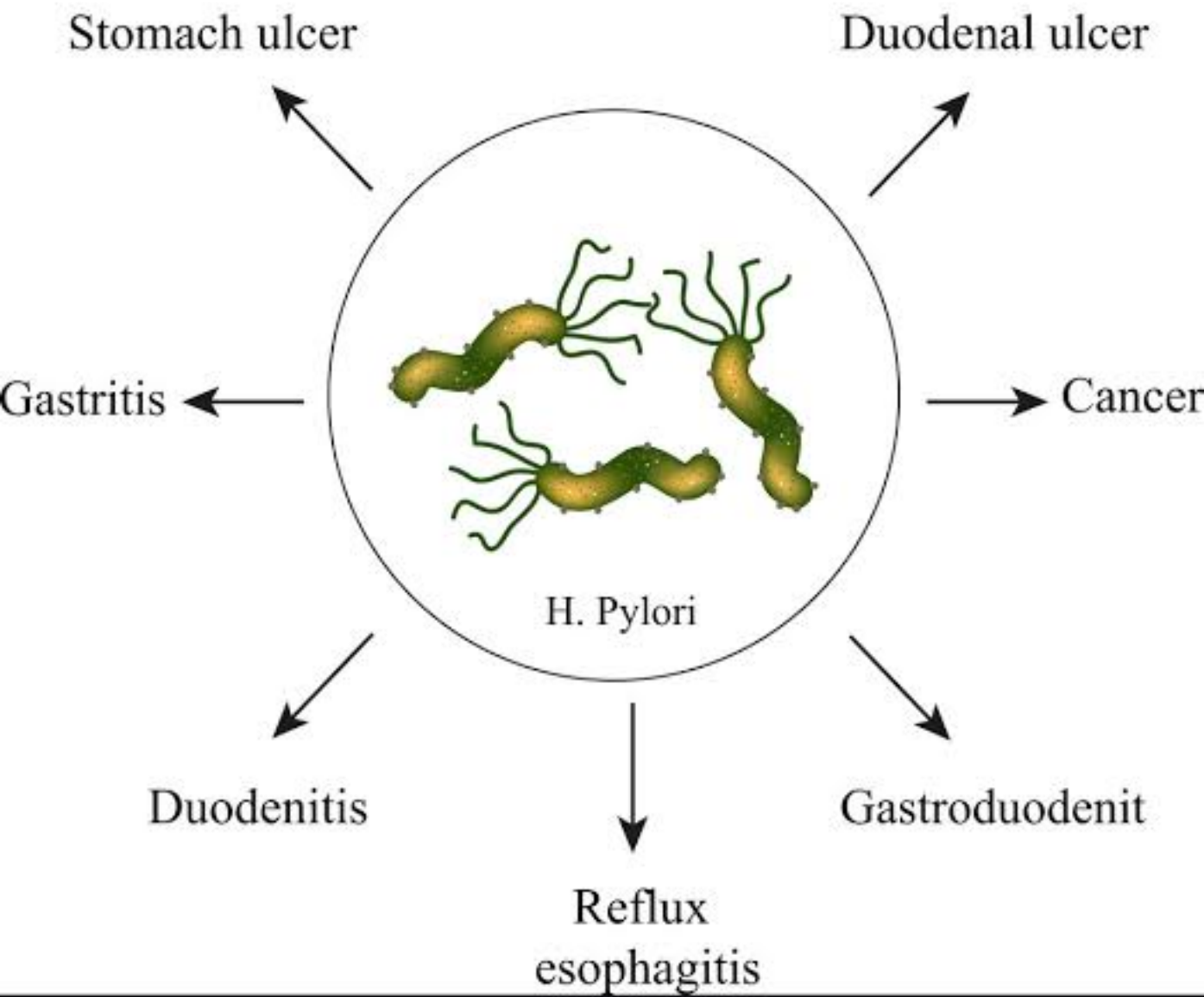
NEPHRITIC vs NEPHROTIC SYNDROME

	NEPHRITIC SYNDROME	NEPHROTIC SYNDROME
ONSET	Abrupt	Insidious
PATHOPHYSIOLOGY	Disruption of glomerular basement membrane due to inflammation	Damage to podocyte foot processes
GFR	Decreased	Normal or decreased
BLOOD	Azotemia	Low serum albumin Hyperlipidemia
URINE	Proteinuria < 3.5 Hematuria Pyuria RBC casts	Proteinuria > 3.5 Fatty casts or none
MANIFESTATIONS	Hypertension Oliguria Edema	Hypotension Edema Risk of thromboembolism or infection
DISEASES	Rapidly progressive glomerulonephritis Alport syndrome Poststreptococcal glomerulonephritis IgA nephropathy (Berger disease) Diffuse proliferative glomerulonephritis Membranoproliferative glomerulonephritis	Focal segmental glomerulosclerosis Membranous nephropathy Minimal change disease Amyloidosis Diabetic glomerulonephropathy

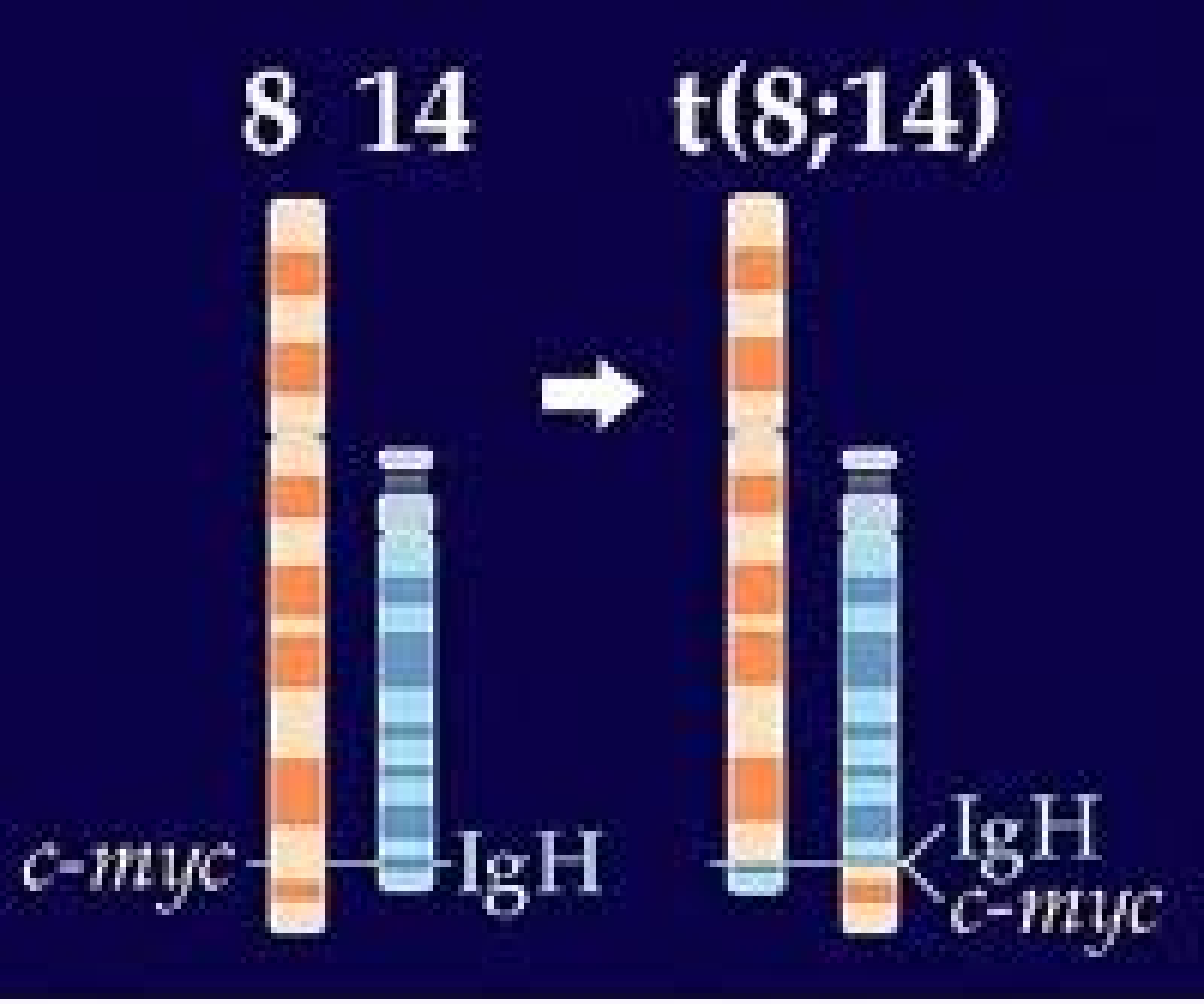
Table 1. Differences between Nephrotic and Nephritic Syndromes

	NEPHROTIC SYNDROME	NEPHRITIC SYNDROME
URINE CASTS	Fatty casts	RBC casts; cola/smoky urine
PROTEINURIA	>3.5 g/day	<3.5 g/day
HEMATURIA	+/-	++
CLINICAL FEATURES	Generalized edema, periorbital edema, HTN	HTN, edema

Helicobacter pylori



Histologic Type	Paraneoplastic Syndrome
Adenocarcinoma	Hypertrophic pulmonary osteoarthropathy Trousseau's syndrome
Squamous cell carcinoma	Humoral hypercalcemia of malignancy (PTHrP) Pancoast syndrome
Small (oat) cell carcinoma	SIADH Lambert–Eaton myasthenic syndrome Cerebellar degeneration



In Burkitt lymphoma, Myc, which is normally found on chromosome 8, is transferred to chromosome 14. This is known as chromosome translocation and can be characteristic of a cancer type. [image credit: Gregory Schuler, NCBI, NLM, NIH.]

DIAGNOSTIC CRITERIA

	CLINICAL FEATURES	LABORATORY FEATURES
THALASSEMIA MAJOR	• Anemia • Hepatosplenomegaly • Growth failure	• Hb : < 7 g/dL • HbF : > 90% • HbA2 : normal or high • HbA : usually absent
THALASSEMIA INTERMEDIA	• Milder anemia • Thalassemia facies • Hepatosplenomegaly	• Hb : < 8-10 g/dL • HbF : > 10% • HbA2 : 4-9%, if > 10% suggests HbE • HbA : 5-90%
β THALASSEMIA TRAIT	• Normal to mild anemia • No organomegaly	• Hb : < 10 g/dL • MCH : < 27 pg • HbF : > 2.5-5% • HbA2 : 4-9%, if >20% suggests HbE trait • HbA : > 90%

SUMMARY

RISK FACTORS FOR ATHEROSCLEROSIS

NON MODIFIABLE

- Increasing age
- Gender
- Genetic factors

MODIFIABLE

- Hyperlipidemia
- Hypertension
- Cigarette smoking
- Diabetes mellitus

ADDITIONAL RISK FACTORS

- Inflammation
- Hyperhomocysteinemia
- Metabolic syndrome
- Abnormal apoprotiens
- Lipoprotein (a)
- Factors affecting hemostatsis
- Lack of exercise
- Stressful life style
- Obesity
- Use of exogenous hormones

Table 1. MIAMI Mnemonic for Differential Diagnosis of Lymphadenopathy

Malignancies

Kaposi sarcoma, leukemias, lymphomas, metastases, skin neoplasms

Infections

Bacterial: brucellosis, cat-scratch disease (*Bartonella*), chancroid, cutaneous infections (staphylococcal or streptococcal), lymphogranuloma venereum, primary and secondary syphilis, tuberculosis, tularemia, typhoid fever

Granulomatous: berylliosis, coccidioidomycosis, cryptococcosis, histoplasmosis, silicosis

Viral: adenovirus, cytomegalovirus, hepatitis, herpes zoster, human immunodeficiency virus, infectious mononucleosis (Epstein-Barr virus), rubella

Other: fungal, helminthic, Lyme disease, rickettsial, scrub typhus, toxoplasmosis

Autoimmune disorders

Dermatomyositis, rheumatoid arthritis, Sjögren syndrome, Still disease, systemic lupus erythematosus

Miscellaneous/unusual conditions

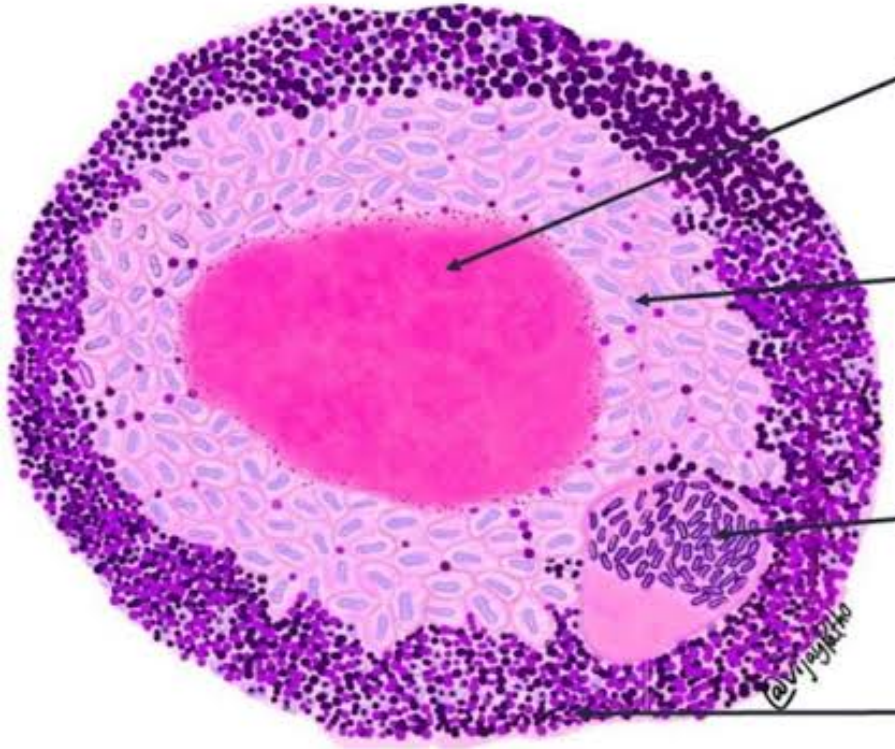
Angiofollicular lymph node hyperplasia (Castleman disease), histiocytosis, Kawasaki disease, Kikuchi lymphadenitis, Kimura disease, sarcoidosis

Iatrogenic causes

Medications, serum sickness

Information from references 2 and 3.

TUBERCULOUS LYMPHADENITIS: **Necrotizing granuloma**



Amorphous granular eosinophilic debritic material
CASEOUS NECROSIS

Modified macrophages with abundant cytoplasm and pale staining "slipper" shaped nuclei
EPITHELOID CELLS

Multinucleated giant cell
LANGHAN GIANT CELL

Collar of lymphocytes surrounding epitheloid cell aggregates

PATHOGENESIS OF INTESTINAL TUBERCULOSIS

Ingestion of infected milk OR sputum

→ Entry of bacilli into the mucosa of the GI tract

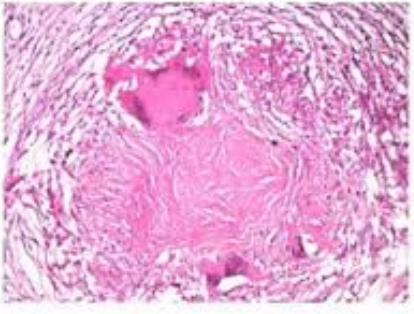
→ Granulomas in the submucosa

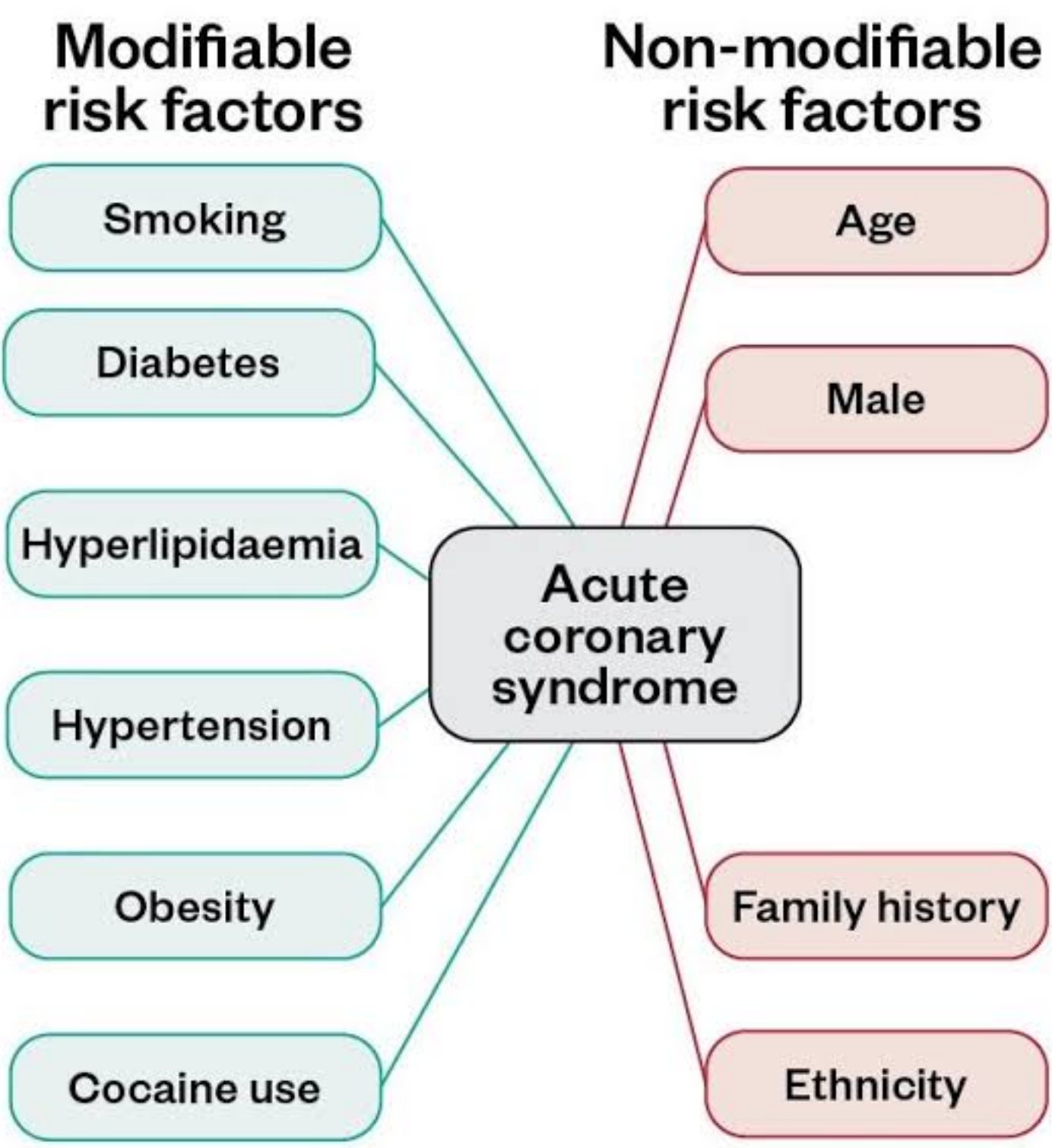
↓ Caseation necrosis

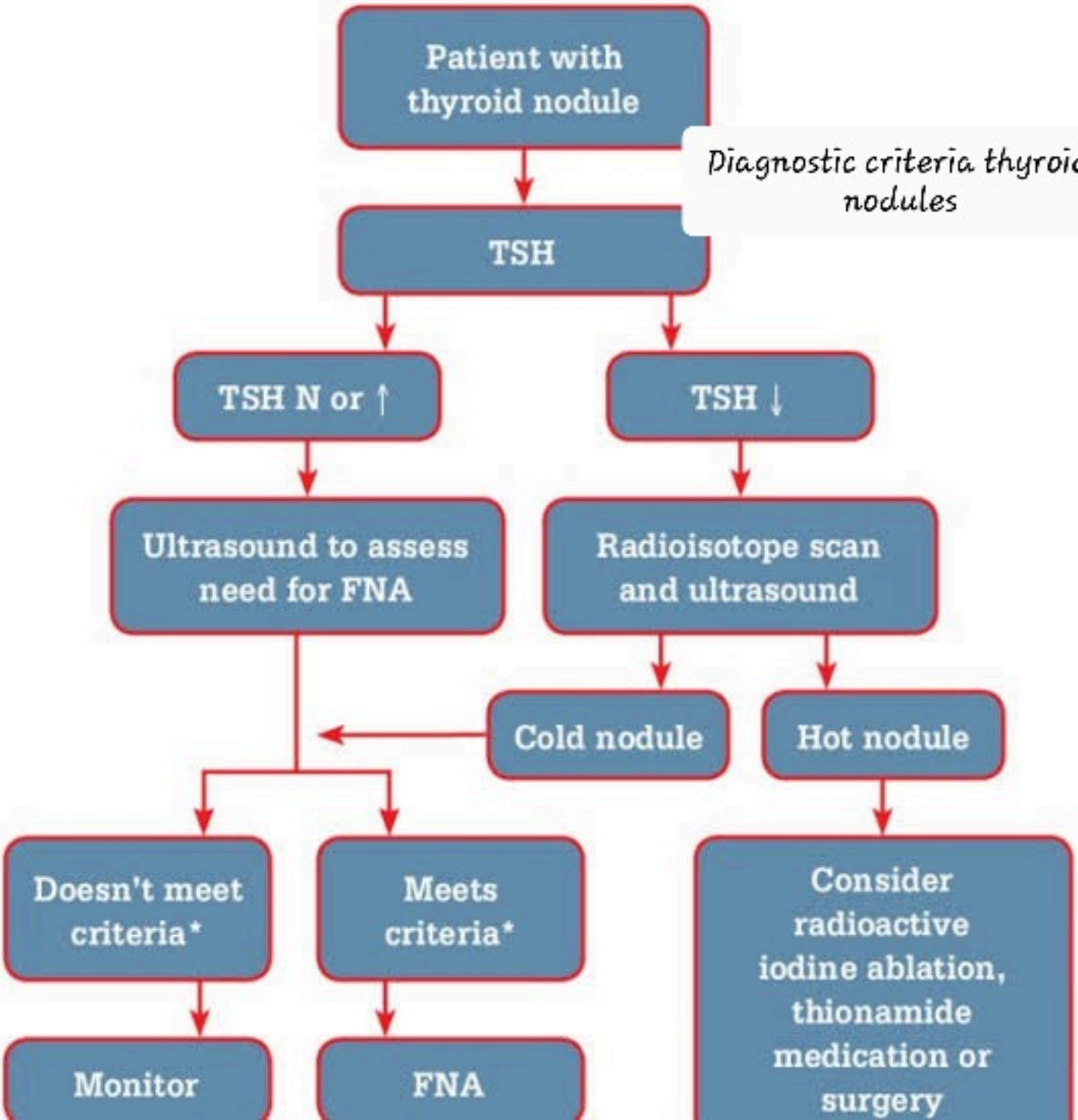
↓ Spread into deeper layers of intestine

↓ Spread into adjacent lymph nodes & Peritoneum

← Ulceration of the overlying mucosa



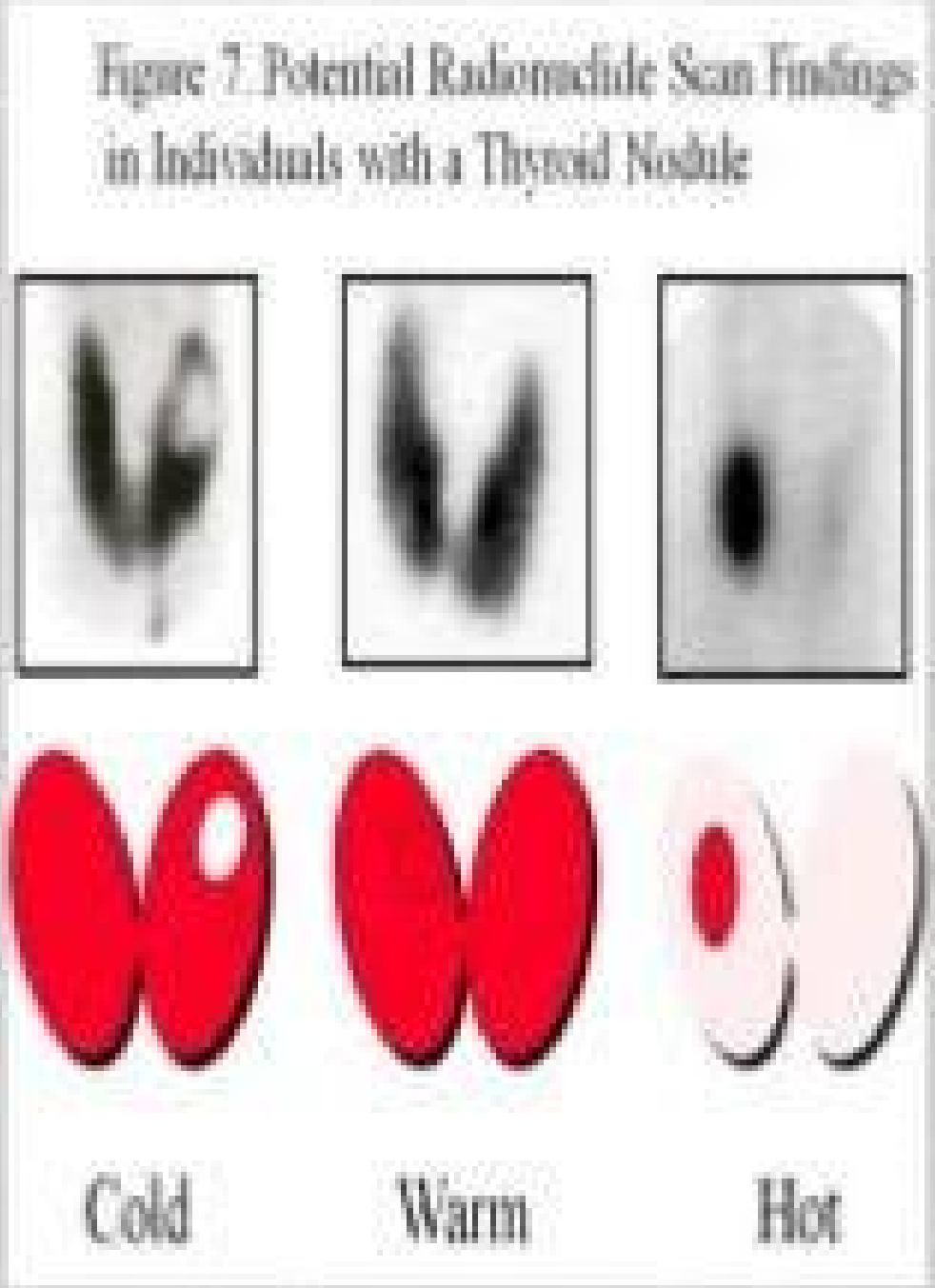

Modifiable risk factors
Non-modifiable risk factors
Smoking
Diabetes
Hyperlipidaemia
Hypertension
Obesity
Cocaine use
Age
Male
Family history
Ethnicity
Acute coronary syndrome


Patient with thyroid nodule
Diagnostic criteria thyroid nodules
TSH
TSH N or ↑
TSH ↓
Ultrasound to assess need for FNA
Radioisotope scan and ultrasound
Cold nodule
Hot nodule
Doesn't meet criteria*
Meets criteria*
Consider radioactive iodine ablation, thionamide medication or surgery
Monitor
FNA

- Hyper functioning - "HOT"
  tracer uptake is greater than surrounding thyroid (~5% *malignant*)

- Iso-functioning - "WARM"-
  tracer uptake is equal to surrounding thyroid (~10% *malignant*)

- Non-functioning - "COLD"
  uptake less than surrounding thyroid (~20% *malignant*)



HYPOTHYROIDISM VERSUS HYPERTHYROIDISM

HYPOTHYROIDISM	HYPERTHYROIDISM
Hyperthyroidism occurs due to excessive production of thyroid hormone	Hyperthyroidism occurs due to insufficient production of thyroid hormone
Indicate signs of slow metabolism	Indicate signs of increased metabolism
Hashimoto's disease is a common cause	Grave's disease is a common cause
Characterized by an increased weight despite a poor appetite	Characterized by loss of weight despite an increased appetite
Characterized by cold intolerance	Characterized by heat intolerance
TSH levels will be elevated	TSH levels will be normal or reduced
Usually treated with Levothyroxine	Can be treated with pharmaceutical agents, radiotherapy or surgical removal of the thyroid gland

Table 3: Causes of Chronic Renal Failure

Pathologic	Physiologic
Glomerulonephritis	Hypertension
Interstitial nephritis	Diabetes mellitus
Tumors	Chronic urinary tract infections
Transplant rejection	Congenital abnormalities
Congenital disease	Vascular disease
	HIV

	Appearance	Opening Pressure mmHg	WBC (cell/μL)	Protein (mg/dl)	Glucose (mg/dL)
Normal	Clear	90-180	< 8	15-45	50-80
Bacterial Meningitis	Turbid	Elevated	>1000-2000	>200	<40
Viral Meningitis	Clear	Normal	<300; Lymphocytic predominance	<200	Normal
Fungal Meningitis	Clear	Normal-elevated	<500	>200	Normal - Low

OBSTRUCTIVE VS. RESTRICTIVE

Obstructive disorders

- **Characterized by:** reduction in airflow.
- So, shortness of breath → in exhaling air.

(the air will remain inside the lung after full expiration)

1. COPD
2. Asthma
3. Bronchiectasis

Restrictive disorders

- **Characterized by** a reduction in lung volume.
- So, Difficulty in taking air inside the lung.

(DUE TO stiffness inside the lung tissue or chest wall cavity)

1. Interstitial lung disease.
2. Scoliosis
3. Neuromuscular cause
4. Marked obesity

TABLE 80.2

Results of Pulmonary Function Tests in Obstructive and Restrictive Lung Disease

Value	Obstructive	Restrictive
FVC	Normal or decreased	Decreased
FEV_1/FVC	Decreased	Normal or increased
MMEFR (FEF_{25-75})	Decreased	Normal
MBC	Decreased	Normal
TLC	Normal or increased	Decreased
RV	Increased	Decreased
DL_{CO}	Decreased in COPD Normal in asthma	Decreased

FVC, forced vital capacity; FEV_1, forced expired volume in the first second; MMEFR, mid-maximal expiratory flow rate; FEF_{25-75}, forced expiratory flow between 25 and 75% of the FVC; MBC, maximum breathing capacity; TLC, total lung capacity; RV, residual volume; DL_{CO}, diffusion capacity of the lung for carbon monoxide; COPD, chronic obstructive pulmonary disease.

Categories of Disease

	OBSTRUCTIVE	MIXED	RESTRICTIVE
FEV1/FVC	↓70%	↓ 70-79%	Normal or ↑
FEV1	↓(marked)	↓	Normal or ↓(slight)
FVC	Normal or ↓	↓	↓
PEFR	↓	↓	N or ↑ with linear decrease in flow versus lung volume
MVV	↓	↓	Normal or ↓
TLC	Normal or ↑	↓	↓

Morphologic changes

Diabetic Nephropathy & Chronic Renal Failure

- Glomeruli:
  - increase GBM thickening
  - Mesangial expansion
  - nodular (Kimmelstiel-Wilson) & diffuse forms of intercapillary glomerulosclerosis
  - capsular drop lesion
  - fibrin cap lesion
- Tubulointerstitium,& tubular functional defects
  - Interstitial scarring
  - Impaired tubular reabsorption of low MW proteins and albumin
  - Increased Na reabsorption leading to volume expansion
  - Hypercalciuria
  - Impaired excretion of H & K ions
- Vascular, hyaline thickening of the arteriolar wall
- Glomerular haemodynamic changes
  - Decreasing Pglom: ACE-I, ARB, low protein diet

Stage	Definition	Subdivision
Tumor stage		
T0	No evidence of primary tumor	
T1	< 7 cm in greatest dimension, confined to the kidney	1a: < 4 cm (►**Fig. 1**) 1b: > 4 cm and < 7 cm
T2	> 7 cm in greatest dimension, confined to the kidney	2a: > 7 cm < 10 cm (►**Fig. 2**) 2b: > 10 cm
T3	Extends into major veins or perinephric tissues but not into the ipsilateral adrenal gland or beyond Gerota fascia	3a: Tumor extends into renal vein branches, or invades perirenal and/or renal sinus fat (►**Fig. 3**)
		3b: Tumor extends into the subdiaphragmatic inferior vena cava
		3c: Tumor extends into the supradiaphragmatic inferior vena cava
T4	Tumor invades beyond the Gerota fascia and/or contiguous extension into the ipsilateral adrenal gland (►**Figs. 4** and **5**)	
Regional lymph nodes		
N0	No regional lymph node metastasis	
N1	Metastasis to regional lymph nodes	
Distant metastasis		
M0	No distant metastasis	
M1	Distant metastasis	

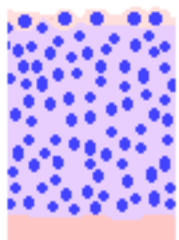
RCC TNM Staging

The Papillary Lesions

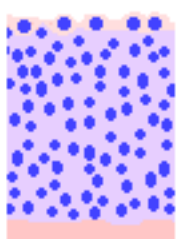
New System

Urothelial
Papilloma

Papillary
Urothelial
Hyperplasia

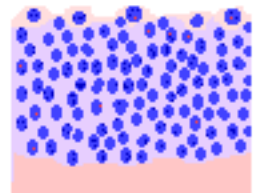


No anaplasia

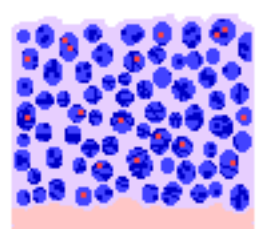


No or minimal
anaplasia

Papillary Urothelial Tumor
of Low Malignant Potential



Low-Grade Papillary
Urothelial Carcinoma



High-Grade Papillary
Urothelial Carcinoma

- Crohn's disease
- NSAIDs
- Lymphoma
- Infections: tuberculosis or cytomegalovirus
- Induced by drugs: gold, potassium, contraceptives or thiazides
- Zollinger-Ellison Syndrome
- Vasculopathies: antithrombin III deficiency, systemic erythematosus lupus, polyarteritis nodosa and Behcet's disease
- Nonspecific ulcers: stenosing multifocal ulcerative enteritis

Intestinal Ulcers Causes. H. pylori +

peptic ulcer

ameobic dysentery, Shigella
E. coli

Test	*Bacterial*	*Viral*	*Fungal*	*Tubercular*
Opening pressure	Elevated	Usually normal	Variable	Variable
White blood cell count	≥1,000 per mm^3	<100 per mm^3	Variable	Variable
Cell differential	Predominance of PMNs*	Predominance of lymphocytes†	Predominance of lymphocytes	Predominance of lymphocytes
Protein	Mild to marked elevation	Normal to elevated	Elevated	Elevated
CSF-to-serum glucose ratio	Normal to marked decrease	Usually normal	Low	Low

CSF = cerebrospinal fluid; PMNs = polymorphonucleocytes.

**—Lymphocytosis present 10 percent of the time.*
†*—PMNs may predominate early in the course.*

Information from references 2, 10, 17, and 20.

Table 1. Differences between Nephrotic and Nephritic Syndromes

	NEPHROTIC SYNDROME	NEPHRITIC SYNDROME
URINE CASTS	Fatty casts	RBC casts; cola/smoky urine
PROTEINURIA	>3.5 g/day	<3.5 g/day
HEMATURIA	+/-	++
CLINICAL FEATURES	Generalized edema, periorbital edema, HTN	HTN, edema

Nephrotic vs Nephritic Syndrome

Nephrotic Syndrome		Nephritic Syndrome
• Low serum albumin (<30g/L) • Proteinuria (>3.5g/day) • Oedema • Dyslipidaemia • Hypercoagulability (loss of antithrombin III) • Reduced immunity (loss of immunoglobulins)	**Characteristics**	• Haematuria • Hypertension • Mild proteinuria (<3.5g/day) • Mild oedema • Temporary oliguria and uraemia
Peripheral oedema (adults), facial oedema (children), frothy urine, fatigue, recurrent infections	**Symptoms**	Haematuria (frank/microscopic), mild oedema, oliguria, signs of uraemia (fatigue, pruritus, nausea)
++++	**Proteinuria**	++
May or may not be present	**Haematuria**	+++
Absent	**Red blood cell casts**	Present

• Minimal change disease (most common in children)
• Membranous nephropathy (most common in adults)
• Focal segmental glomerulosclerosis
	Causes	• IgA nephropathy
		• Post-streptococcal glomerulonephritis
		• Rapid progressive glomerulonephritis (RPGN):
		  • Anti-GBM glomerulonephritis
		  • ANCA Vasculitis

Familial renal cancer

Malignant renal cell tumors

- Clear cell RCC
- Papillary RCC
- Chromophobe RCC
- Carcinoma of the collecting ducts of Bellini
- Tubulocystic carcinoma
- Renal medulary carcinoma
- Renal carcinoma associated with Xp11.2 translocations/TFE3 gene fusions (MiTF/TFE family translocation carcinomas)
- RCC in long-term survivors after neuroblastoma
- Mucinous tubular and spindle cell carcinoma
- Thyroid follicular carcinoma-like tumor of kidney
- RCC unclassified

Renal cell neoplasms in end-stage renal disease

- RCC associated to acquiered cystic disease
- Clear cell papillary RCC

Renal cell neoplasms of low malignant potential

- Multilocular cystic renal cell neoplasm of low malignant potential (multilocular clear cell RCC)

Benign renal cell tumors

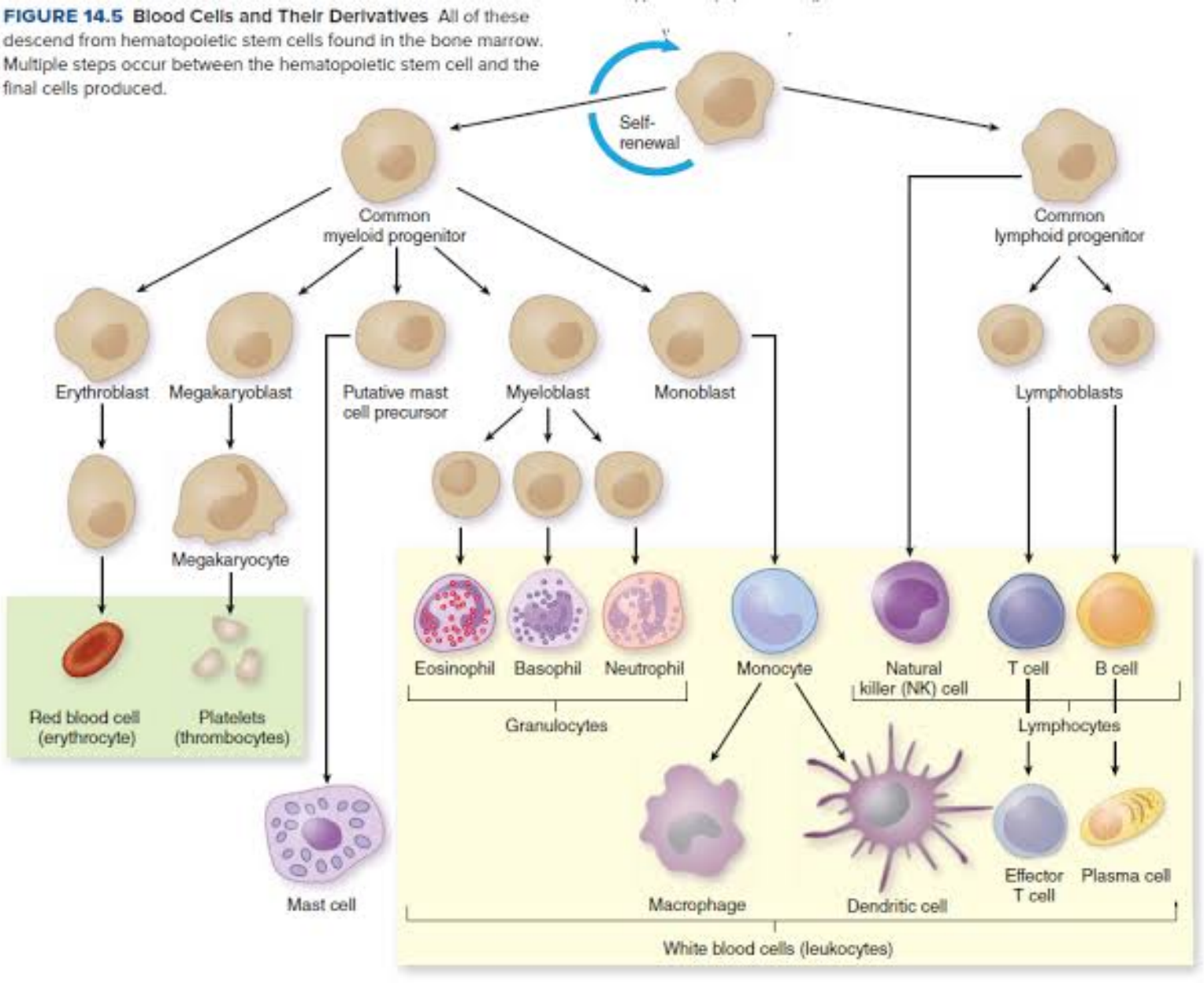
- Papillary adenoma
- Oncocytoma
- Metanephric adenoma and adenofibroma

Mixed stromal and epithelial tumors (renal epithelial and stromal tumor)

- Cystic nephroma
- Mixed epithelial and stromal tumor

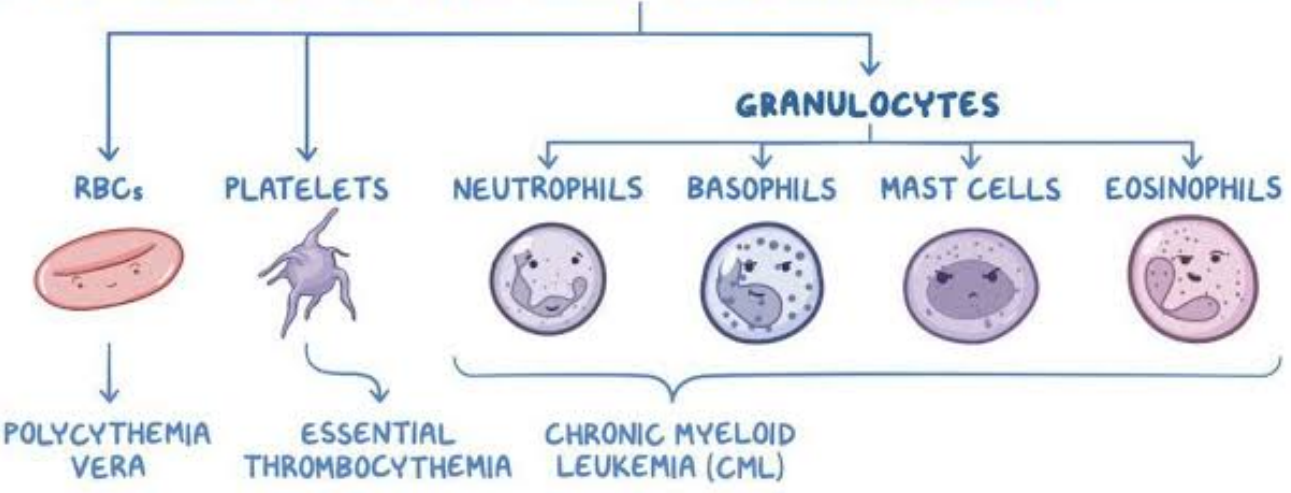
RCC, renal cell carcinoma.

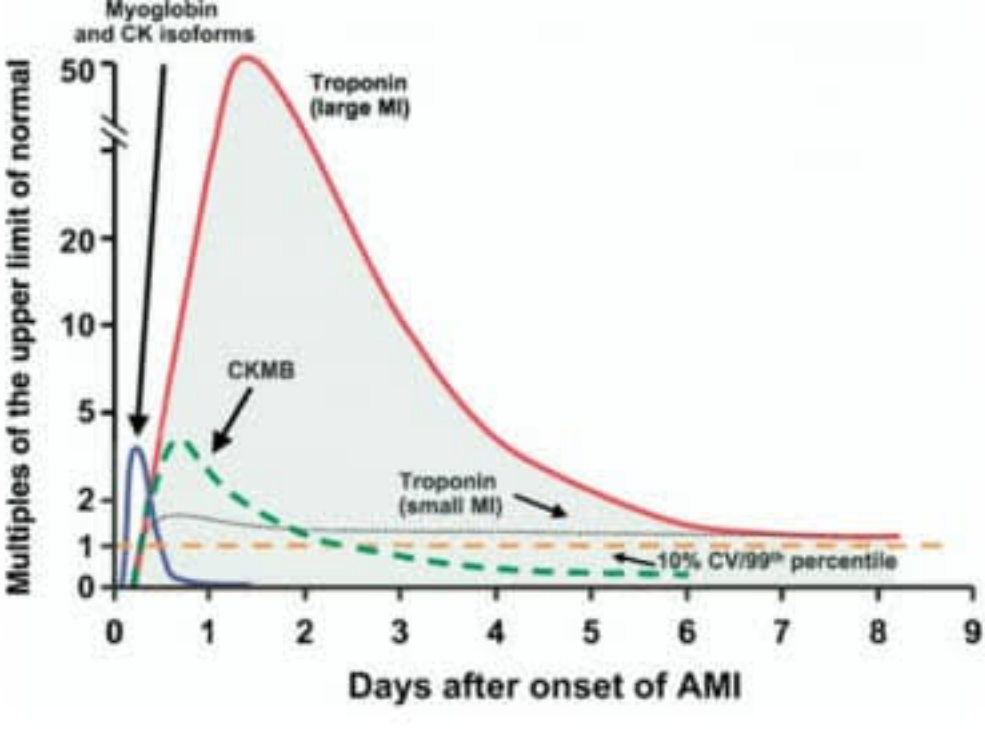
FIGURE 14.5 Blood Cells and Their Derivatives All of these descend from hematopoietic stem cells found in the bone marrow. Multiple steps occur between the hematopoietic stem cell and the final cells produced.

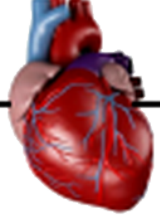


MYELOPROLIFERATIVE NEOPLASMS

* CLASSIFIED BASED on DOMINANT CELL LINE INVOLVED




Myoglobin
and CK isoforms
Troponin
(large MI)
CKMB
Troponin
(small MI)
10% CV/99th percentile
Multiples of the upper limit of normal
50
20
10
5
2
1
0
0
1
2
3
4
5
6
7
8
9
Days after onset of AMI

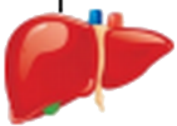


Right heart failure

Left heart failure

Congestion of peripheral tissues

Oedema and ascites



Liver congestion

Impaired liver function

GI tract congestion

Anorexia, GI distress, weight loss

Decreased cardiac output

Activity intolerance
Signs of decreased tissue perfusion

Cyanosis and signs of hypoxia

Pulmonary congestion

orthopnea

Cough with frothy sputum

Paroxysmal nocturnal dyspnea

Effect of Amino Acid Change—Sickle Cell Anemia