

Non neoplastic disorders of WBCs

WBCs= leukocytes

Leukopenia

Decrease in number "low count"

Neutropenia "Agranulocytosis"

*Most common leukopenia

Usually neutrophils count equal 1500 so:
Mild decrease (1000-1500)
Moderate decrease (500-1000)
Severe decrease <500

Neutrophils usually encounter bacteria and fungal infections so **neutropenic patients will have severe fatal bacterial and fungal infections**

Causes:

Decrease production

- BM hypoplasia with chemotherapy or radiation therapy
- Medications (Some drugs suppress BM)
- Replacing BM (by tumors as leukemia)
- Neoplastic lymphocytic proliferation involving BM

Increase destruction

- Autoimmune destruction (ex: SLE)
- Overwhelming bacterial, fungal and rickettsial infections (في حالات ال severe infection يتم استهلاك ال neutrophils . لأنه بصيرليها apoptosis بعد القيام بوظيفتها)
- Splenomegaly (sequestration and accelerated removal of neutrophils) (يسبب تضخم ال spleen ال neutrophils صارت تدخل في spleen و تعلق فيها و هذا زاد من destruction)

Lymphopenia " much less common"

Causes:

- Congenital immunodeficiency disease
- HIV
- High doses of corticosteroids

-Viral infections leading to **lymphocytes redistribution** " To lymph nodes and increase adherence to endothelial cells "

إنه الفيروسات يجبروا ال lymphocytes على الذهاب إلى nodes و بالتالي زح يقل مستواهم في serum

Leukocytosis

Increase in number
"proliferation may be reactive or neoplasms"

-Are relatively nonspecific.

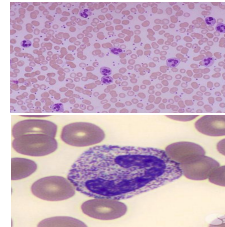
عشان هيك لما يكون فيها زيادة بنشوف إيش هو الاشي الي زاد بالزبط :

Neutrophilic

Causes:

Bacterial

Non-bacterial Sterile inflammation (necrosis, Burns)



Some morphological changes:

- 1- Cytoplasmic vacuoles
- 2-Toxic granules (Coarser "أخشن" and darker)
- 3- Döhle bodies (Dilated ER; sky blue cytoplasmic puddles)

Causes:

- 1- Allergic disorders
- 2- Parasitic infestation (يسبب ال major basic protein الذي يهاجم ال parasites)
- 3-Drugs (as steroids)

Basophilic "Rare"

Cause:

Myeloproliferative disease

Monocytosis

Causes:

- 1- Chronic infection (TB)
- 2- Inflammatory bowel disease
- 3- Autoimmune disorders

Lymphocytosis

Causes:

- 1- Chronic immunologic stimulation (TB, brucellosis)
- 2-Viral infections (HAV, CMV and EBV)

Clinical features:

- 1-Fever, chills and malaise
- 2- Mucocutaneous necrotizing ulcers
- 3- Sepsis



Treatment:

- 1- broad spectrum antibiotics
- 2- G-CSF

Special reactions

Leukomoid reaction

" WBCs count increase up to 25000 "

Causes:

Very severe infection stimulate BM to produce many WBCs, that lead to have immature granulocytes in blood (mimicking leukemia)

Differentiated from leukemia by:

- 1- Younger age patients
- 2- No BCR/ABL fusion gene
- 3- Subsides after treatment

Infectious mononucleosis

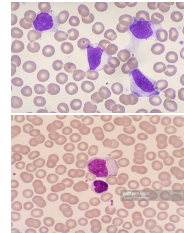
" Acute, self limited , transient "

Cause:

EBV invade B cells increasing there proliferation so CD8+ cells also increase in number to respond against B-cells

Clinical features:

- 1- Fever, sore throat and lymphadenitis
- 2- Lymphocytosis of activated CD8+ (up to 18000)

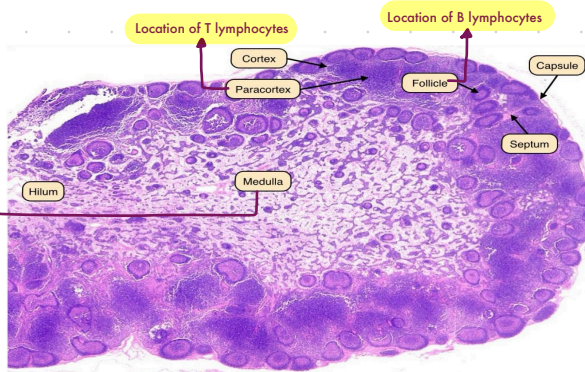


Morphological changes:

Atypical lymphocytes

- 1- Nucleus
(oval, indented or folded)
- 2- Abundant cytoplasm
- 3- azurophilic granules

Reactive lymphadenitis



Lymph node structure

Medulla contains sinusoids that have macrophages and plasma cells

Follicles

Primary follicles

Contain naive B cells
(لسا ما تعرضوا ل antigen)

Secondary follicles

Contain germinal center
(منطقة فاتحة في الوسيط)
And
Activated B cells that will form
plasma cells

Reactive lymphadenitis

Acute nonspecific lymphadenitis

Localized

Generalized

" systemic infection "

- After controlling infection -> Revert to normal nodes
- If damaged -> undergo scarring

Histologic changes in lymph nodes:

Cortex

Paracortex

Medulla

Primary follicles will become Secondary follicles containing **Germinal center**

Will contain neutrophils, necrosis and pus formation

Sinus enlargement with histocytes

Undergo mitosis and contain macrophages

Chronic nonspecific lymphadenitis " Depending on causative agents "

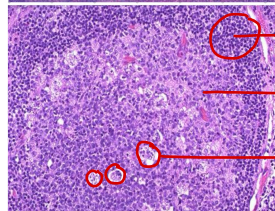
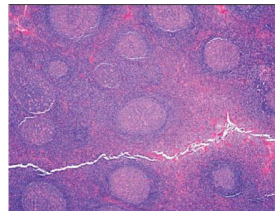
Follicular hyperplasia

Features:

- Activate humoral immunity
- Large germinal centers (Secondary follicles)
- Tingible body macrophages

Causes:

- Rheumatoid arthritis
- Toxoplasmosis
- Early stages of HIV



B cells

Plasma cells

Tingible macrophages

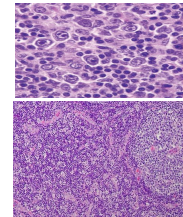
Paracortical hyperplasia

Features:

- Activate T cell region
- T cell transform into large immunoblast

Causes:

- Viral infections
- Vaccination (ex: Smallpox)
- Drugs induced immune reactions (phenytoin)



Sinus histiocytosis

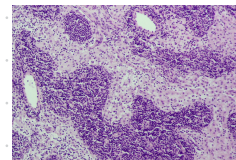
Features:

- Distention of sinusoids due to: hypertrophy of lining endothelium

-Infiltration of macrophages

Causes:

- draining tumors
- immune response to tumors or its products



Note:

Follicular hyperplasia differentiated from follicular lymphoma by:

- 1-Preservation of architecture
- 2-Variation in shape and size of follicles
- 3-GC mitotic figures, macrophages and recognizable light and dark zones