

Morphologic patterns of inflammation

لماذا يكونون أشياء Special زيادة عن General Features

لماذا يشوبهم؟ ع (ع) أشياء :-

1. Severity of the Reaction
2. Specific cause
3. Particular tissue
4. Site

لماذا مكانه خلاصهم غير مراد Underlying cause

Type	Definition and Mechanism	Examples/Subtypes	Notes
Serous inflammation	<ul style="list-style-type: none"> Exudation of cell poor fluid into spaces created by injury to surface epithelial or into body cavities such as peritoneal, pleural, or pericardial cavities. 	<ol style="list-style-type: none"> Effusion: Accumulation of fluid in body cavities SKIN BLISTER (Bullae): <ul style="list-style-type: none"> ✓ Resulting from a burn or viral infection. ✓ Represents accumulation of serous fluid within or immediately beneath the damaged epidermis of the skin 	<ul style="list-style-type: none"> Not infected by destructive organisms and does not contain large numbers of leukocytes
Fibrinous inflammation*	<ul style="list-style-type: none"> Develops when the vascular leaks are large or there is a local procoagulant stimulus. 		<ul style="list-style-type: none"> Characteristic of inflammation in the lining of body cavities, such as the meninges, pericardium and pleura.

Type	Definition and Mechanism	Examples/Subtypes	Notes
Purulent (Suppurative) inflammation	<ul style="list-style-type: none"> Production of pus, an exudate consisting of neutrophils, the liquefied debris of necrotic cells, and edema fluid. 	<ul style="list-style-type: none"> Abscess: Localized collections of pus caused by suppuration buried in tissue, an organ, or a confined space → They are produced by seeding of pyogenic bacteria into a tissue. In time the abscess may become walled off and ultimately replaced by connective tissue 	<ul style="list-style-type: none"> The most frequent cause is infection with pyogenic (pus producing) bacteria, such as staphylococci ACUTE APPENDICITIS
Ulcer	<ul style="list-style-type: none"> Local defect, or excavation, of the surface of an organ or tissue that is produced by the sloughing (shedding) of inflamed necrotic tissue. 	<ol style="list-style-type: none"> Acute stage: <ul style="list-style-type: none"> ✓ Intense polymorphonuclear infiltration and vascular dilation in the margins of the defect. With chronicity: <ul style="list-style-type: none"> ✓ the margins and base of the ulcer develop fibroblast proliferation, scarring, and the accumulation of lymphocytes, macrophages, and plasma cells. 	<ul style="list-style-type: none"> The mucosa of the mouth, stomach, intestines, or genitourinary tract. The skin and subcutaneous tissue of the lower extremities in older persons

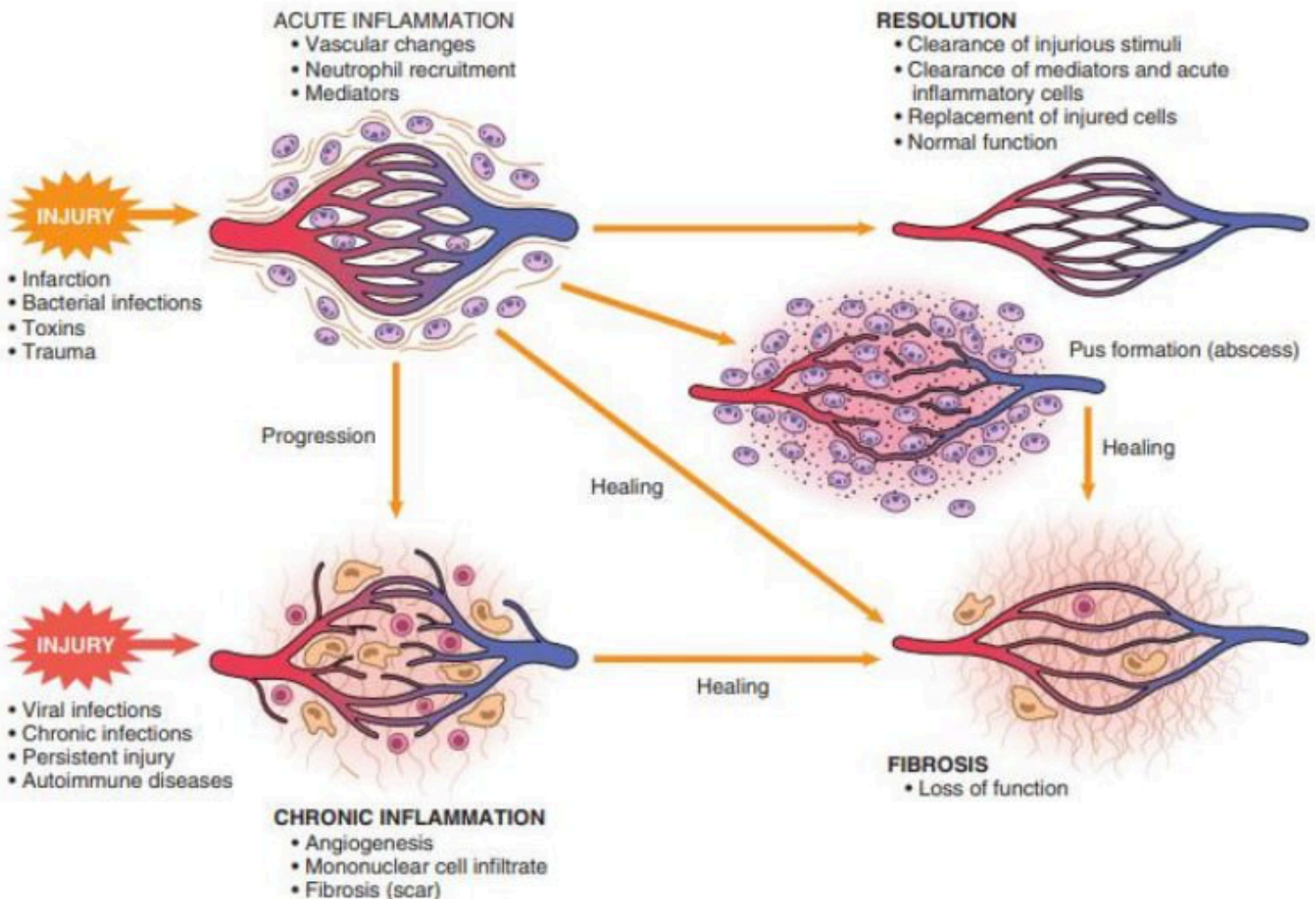
* In fibrinous inflammation →

Large ↑ in vascular permeability → Higher molecular weight such as FIBRINOGEN can pass out of the blood → FIBRIN is formed & deposited in the extracellular space!

Abscesses have multiple areas:

1. Central region with necrotic leukocytes and tissue cells.
2. Zone of preserved neutrophils around this necrotic focus.
3. Vascular dilation, parenchymal and fibroblastic proliferation.

Outcomes of acute inflammation



1. Complete resolution:

- Occur when the injury is limited or short-lived or when there has been little tissue destruction and the damaged parenchymal cells can regenerate.
- Resolution involves removal of cellular debris and microbes by macrophages, and resorption of edema fluid by lymphatics.

2. Healing by connective tissue replacement (scarring, or fibrosis):

- Occurs after substantial tissue destruction, when the inflammatory injury involves tissues that are incapable of regeneration, or when there is abundant fibrin exudation.
- Connective tissue grows into the area of damage or exudate, converting it into a mass of fibrous tissue.

3. Progression of the response to chronic inflammation.

- Occurs when the acute inflammatory response cannot be resolved, as a result of either:

A. the persistence of the injurious agent

B. interference with the normal process of healing

CHRONIC INFLAMMATION

لے جیوں 🐣 های یا بتجی کماها سر لیدای (Begin insidiously)
اؤ تکیو تکلمه ل acute

❖ Chronic جسمک باره طویل یعنی سین ویکور ۹۰

inflammation.
tissue injury.
attempts of repair. } coexist,
in varying combinations.

← (أهم أسبابها) :-

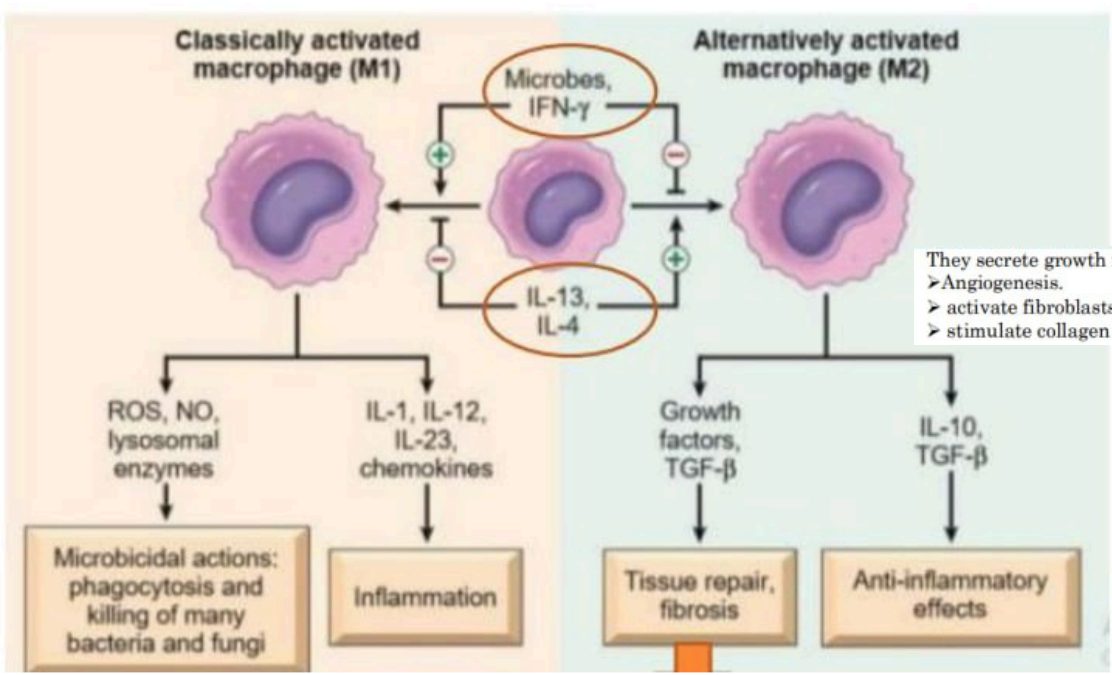
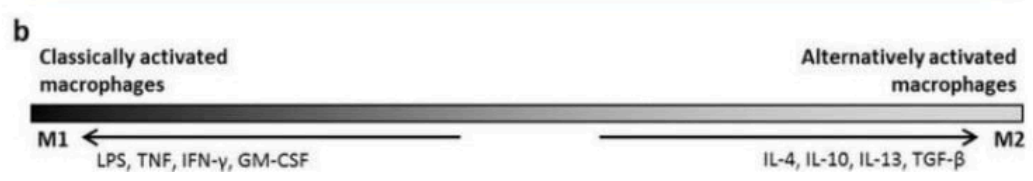
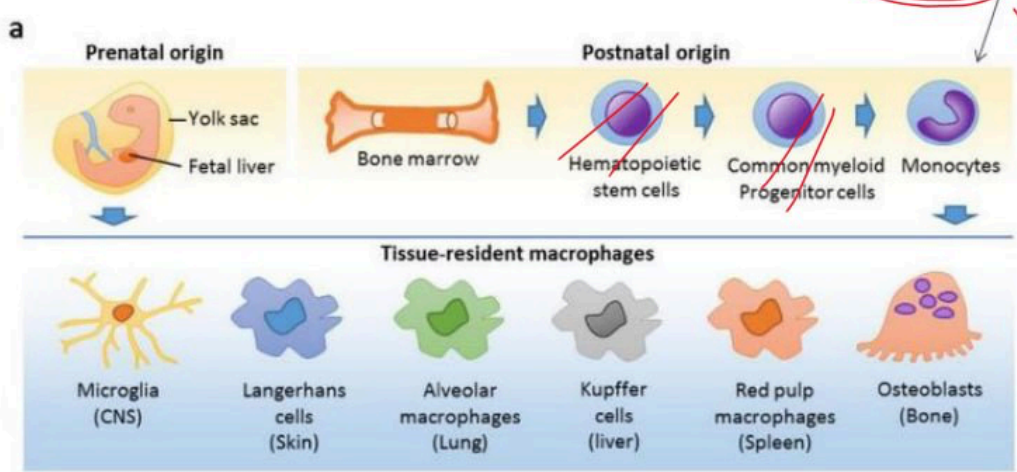
1. Persistent infections, e.g (TB & HIV) → هس قادر بره علیها
2. Hypersensitivity diseases.
3. Autoimmune disease. → جسمک بره م نفعه
4. Allergic diseases. → حساسیة
5. Prolonged exposure to potentially toxic agents, e.g Silica →

1. Macrophage ← کتلا یا تکیو

2. Lymphocyte

1. Macrophage
 → Dominant in Chronic
 → Two Activation Pathways
 → Classical → Destruction
 → Alternative → initiate Tissue Repair
 → Products of Activated Macrophage
 → Eliminate injurious agent
 → Initiate process of repair
 → Responsible for much of tissue injury in chronic inflammation.

Macrophages are professional phagocytes.

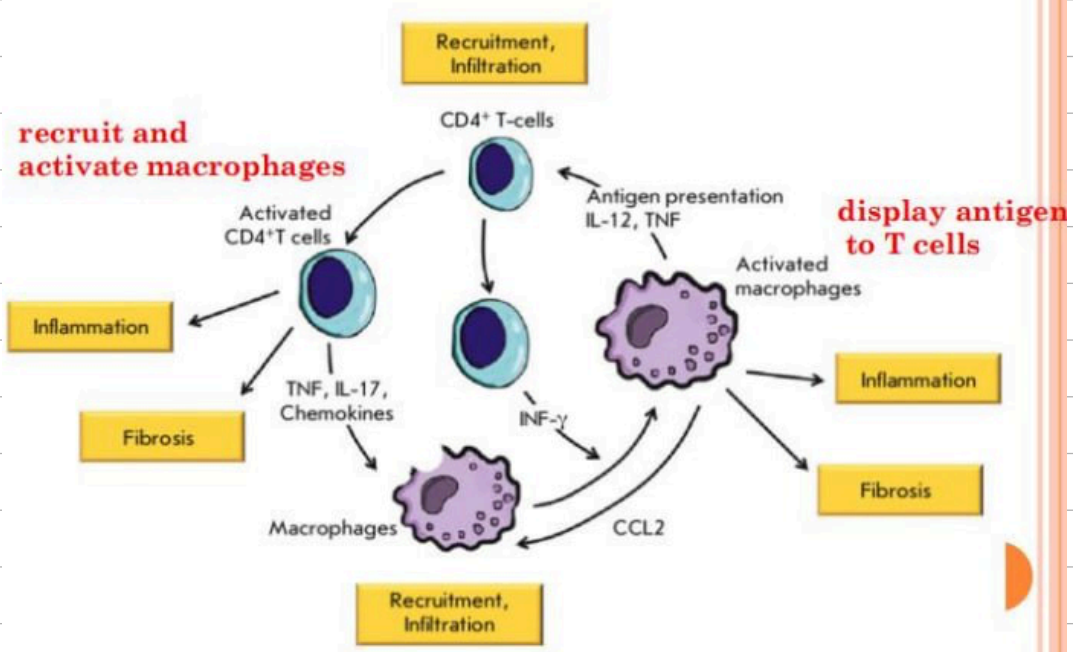


They secrete growth factors that promote
 > Angiogenesis.
 > activate fibroblasts.
 > stimulate collagen synthesis.

LYMPHOCYTES:

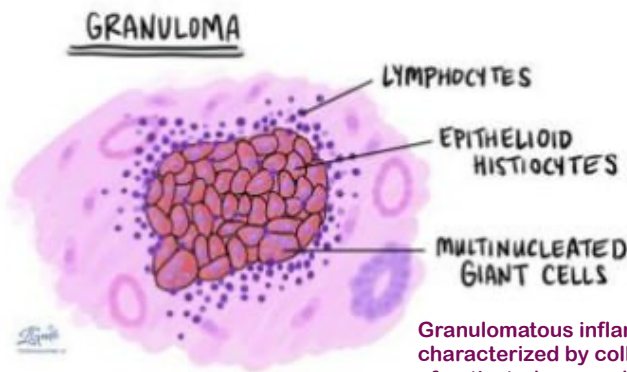
- Microbes and other environmental antigen activate T and B lymphocytes, which amplify and propagate chronic inflammation.
- Some of the strongest chronic inflammatory reactions, such as granulomatous inflammation, are dependent on lymphocyte responses.

LYMPHOCYTES AND MACROPHAGES INTERACT IN A BIDIRECTIONAL WAY.



ما قفنا ناكل منه في بتحصير

Granulomatous inflammation



Granulomatous inflammation is a form of chronic inflammation characterized by collections of activated macrophages, often with T lymphocytes. ✓ Granuloma formation is a cellular attempt to contain an offending agent that is difficult to eradicate

1. Immune granulomas:

- Caused by persistent T cell-mediated immune response, when the inciting agent cannot be readily eliminated.

2. Foreign body granulomas:

- Seen in response to inert foreign bodies, in the absence of T cell-mediated immune responses.
- May form around materials such as talc (associated with intravenous drug abuse), sutures, or other fibers

Disease	Cause
Tuberculosis	<i>Mycobacterium tuberculosis</i>
Leprosy	<i>Mycobacterium leprae</i>
Syphilis	<i>Treponema pallidum</i>
Cat-scratch disease	Gram-negative bacillus
Sarcoidosis	Unknown etiology
Crohn disease (inflammatory bowel disease)	Immune reaction against undefined gut microbes and, possibly, self antigens

SYSTEMIC EFFECTS OF INFLAMMATION

- Inflammation is associated with cytokine-induced systemic reactions that are collectively called the acute-phase response.
- The cytokines TNF, IL-1, and IL-6 are important mediators of the acute phase reaction

SS

- THE ACUTE-PHASE RESPONSE CONSISTS OF SEVERAL CLINICAL AND PATHOLOGIC CHANGES:

1. Fever:

- ✓ Substances that induce fever are called **pyrogens**
- ✓ Caused by prostaglandins especially **PGE2** that are produced in the vascular and perivascular cells of the **hypothalamus**.

2. Acute-phase proteins

3. Leukocytosis

