



Pathology

Doctor 2018 | Medicine | JU

● Sheet

○ Slides

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Hold Your VISION , Track the PROCESS to your GOALS and NOW
let's start our new topic .



What organ do you see in this picture?

It's an abnormal, congested ,and **inflamed** appendix ; as it appears swollen (due to edema) and has a red color (the normal one is whiter and thinner) . SO, WHAT IS INFLAMMATION???

Inflammation

It is the response of **vascularized tissue** to injury (infections or tissue damage) including the recruitment of cells and molecules from circulation to the sites where there is a need to eliminate the offending agent that cause the injury .
(EX. on offending agents ; bacteria , virus , heat...etc)

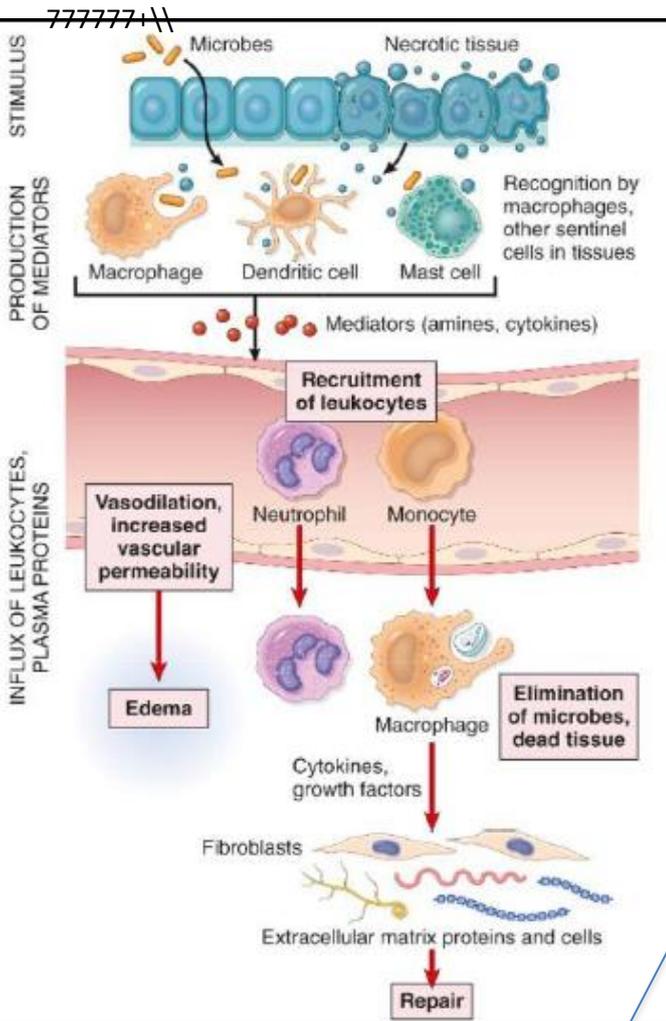
[**VASCULARIZED** indicate that it is **alive tissue** with a blood supply] so if it is dead no inflammation can occur .

SO ,Why we have inflammatory response and what its importance ??

- **It supposed to be a protective mechanism HOW??** (when the offending agent attack a tissue in our body the infl. response work to eliminate it and repair the tissue back to its normal state, but in some cases the offending agent can be strong enough not to be eliminated)
- **proper healing** ; without inflammation--> infection can be fatal , wound would never heal and injured tissue may sustain permanent damage .

>>EXAMPLE on the importance of infl. : patients with severe immune diseases OR cancer patients who lack the ability for inflammatory response , died earlier due to chronic diseases and deficiencies in immune responses .

General view for inflammation mechanism and the 5 Rs steps



Microbes or any offending agent **recognition** by different immune cells and molecules

Immune cells release mediators to initiate infl. (mainly amines and cytokines)

Then they enter the blood vessels at the site of injury and cause vasodilation ,increasing their permeability and then edema [**the early vascular phase of inflammation**]

Recruit plasma proteins and WBCs (mainly and initially neutrophils and monocytes that are the major players of inflammation) **in the site of injury.**
-RECALL that when a monocyte enter the tissue it is called a macrophage

PL. proteins and WBCs work together to destroy and eliminate(**Remove**) the enemy

Rxn is then controlled, and terminated (**Regulation**)

MORE mediators (cytokines and growth factors) are released to stimulate fibroblasts, and **repair** of damaged tissues occur (by regeneration and fibrosis)

Do you remember the inflamed appendix?? YESS this is the reason for its red and swollen appearance

Imp: Those steps happen in the previous order but they can overlap (they may occur in nearby times).

NOTE : The inflammatory response **NORMALLY** produced in the right amounts and localized in the inflamed tissue (limited and containment), but if the inflammatory mediators (that are **normally** produced to kill microbes) leak to normal tissues can cause their damage (**collateral damage**) .

The main classification of inflammation: Acute and Chronic

TABLE 3.1 Features of Acute and Chronic Inflammation

Feature	Acute	Chronic
Onset	Fast: minutes or hours	Slow: days or months
Cellular infiltrate	Mainly neutrophils	Monocytes/macrophages and lymphocytes And sometimes plasma cells
Tissue injury, fibrosis	Usually mild and self-limited	May be severe and progressive
Local and systemic signs	Prominent	Less

IMPORTANT notes on the previous table !!

- In **acute inflammation**

-- the tissue (or cellular) infiltrate cells are **mainly** neutrophils and another cells can participate like: mast cells and macrophages .

RECALL! that neutrophils are called polymorphonuclear leukocytes (3 lobules nucleus) and their **life span is short** (about 6 hours).

>> **SO, acute inflammation** : is a short time process , appearing for minutes or hours.

-- Usually cause low and limited damage .
[FOR EXAMPLE : Acute Flu that ” comes fast and goes fast”] .

--prominent : has a strong effect and symptoms
ex : fever, loss of appetite and pain (It declares itself) [As in** Acute Meningitis (مرض السحايا)and Acute Appendicitis]

we diagnose **MIS
(Meningeal Irritation Signs)
by testing **the Lumbar vertebrae functions** and
Nuchal rigidity (the ability to move the nape of his/her neck up and down)

• While in **chronic inflammation** :

-- the major players are monocytes ,macrophages, lymphocytes and sometimes plasma cells.

-- **Declaration is much less (less prominent, and insidious)** as it can be unnoticed for long time while damaging an important organ and when noticed it will be too late .

>>so often you should search for the **Chronic inflammation symptoms'** .

→ **USUALLY Acute inflammation** cause lower fibrosis and lower injury compared to **chronic inflammation** that can cause death .

{**FOR EXTRA clarification** : although acute inflammation declares itself and prominent but it is treated faster so it doesn't cause that significance damage compared to chronic infl. }

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- In many cases an overlapping between these two types may occur , and an unwanted outcome results when the acute infl. becomes chronic infl. Or **acute on the top of chronic inflammation.**

acute on the top of chronic inflammation can occur in

- **pancreas** (acute on the top of chronic pancreatitis)
 - **Gallbladder** due to Gallstones (Cholecystitis that cause sudden abdominal pain and weight loss)
 - **Liver** steatosis patients usually have a recurrence turning between Acute and acute on the top of chronic inflammations that at the end cause severe liver damage
- <<You should take care of acute infl. so that it won't turn to chronic infl. >>



***Cardinal signs of Acute inflammation**

1. heat(**calor*) remember the energy unit calorie.
2. redness(**rubor*) full of blood vessels.
3. swelling (***tumor*).
4. pain(**dolor*).
5. loss of function (**functio laesa*) when there is an inflammation in the ankle (arthritis) the joint loses its function.

***: a Latin term**

*tumor :something large not necessary cancer ,it may be edema or swelling .

*EXTRA: cardinal signs means essential/ important/reacting signs.

Although inflammation is normally protective

(when inflammatory mediators are produced in normal levels and the damaged tissue is fully repaired), **it can cause damages, severe diseases and even death, that when there is :**

- **Too much and exaggerated inflammation**
- **Too little defective inflammation**
- **Misdirected inflammation** : when the mediators that are produced inside our bodies affect normal ,harmless cells and damaging them [FOR EXAMPLE: :rheumatoid arthritis, and systemic lupus erythematosus and other autoimmune diseases and allergies].
- **Chronic inflammation → chronic disease**: it is one of the unwanted outcomes of acute inflammation

when there is recurrent episodes of acute inflammation that weaken the organ ,as well as when the offending agent is still found in the organ and non-eliminated ,then the acute inflammation turned to be chronic .

Examples on acute and chronic diseases

TABLE 3.2 Disorders Caused by Inflammatory Reactions

Disorders	Cells and Molecules Involved in Injury
Acute	
Acute respiratory distress syndrome	Neutrophils
Asthma	Eosinophils; IgE antibodies
Glomerulonephritis	Antibodies and complement; neutrophils, monocytes
Septic shock	Cytokines
Chronic	
Arthritis	Lymphocytes, macrophages; antibodies?
Asthma	Eosinophils; IgE antibodies
Atherosclerosis	Macrophages; lymphocytes
Pulmonary fibrosis	Macrophages; fibroblasts

What are the major players in each disorder ?
What is the main cell infiltrate in each disorder?

Listed are selected examples of diseases in which the inflammatory response plays a significant role in tissue injury. Some, such as asthma, can present with acute inflammation or a chronic illness with repeated bouts of acute exacerbation. These diseases and their pathogenesis are discussed in relevant chapters.

NOTE: THE DOCTOR REPEAT THIS POINT SO FOCUS ON IT
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Acute disorders

1. Acute Respiratory Distress *Syndrome (ARDS)

The cause: when too much neutrophils are induced by an injury ,they damage the alveolar cells in the lung wall and produce a white lung .

- pneumonia may complicate to ARDS .

It was a cause for high mortality (>75%) but it has been improved recently (thanks for better supportive measures and treatments) .

is it is a clinical syndrome not a pathological term .

{**EXTRA NOTE:*** syndrome is a set of medical signs , symptoms and collection of diseases which are not correlated with each other and often associated with a particular disease or disorder.}

2. Asthma (bronchial asthma):

the major players are Eosinophils and IgE antibodies so it is a reaction that cause **allergy** .

-Characterized by Bronchospasm (تشنج قصبي) and edema .

symptoms : shortness of breath ,coughing ,chest tightness and wheezing (الصفير).

3. Glomerulonephritis :due to a cute inflammation in the kidney

the major players are antibodies , **complement** , neutrophils and monocytes.

Complement :Part of plasma proteins that is produced from the mediators .

Don't worry please , it will be discussed later in details ^_^ .

- 4. Septic shock** : it is a very dangerous disease due to bacterial infection that cause severe fever ,leukocytosis and blood culture with gram negative bacteria .
- Cytokines which are produced by leukocytes are the major players in septic shock .

Chronic disorders

1. Arthritis

The most common types of **arthritis** are **osteoarthritis** (which is not considered an inflammation but it is a joint disease) and **rheumatoid arthritis**.

the major players are lymphocytes , macrophages and antibodies .

2. Chronic Asthma

the major players are Eosinophils and IgE antibodies.

3. Atherosclerosis

The cells involved in Atherosclerosis progress are endothelial cells , macrophages, lymphocytes ,masts cells , smooth muscles of vessels wall and platelets.

4. Pulmonary fibrosis (non- functional lungs)

the major players are macrophages and fibroblasts .

To Sum UP!

Acute or Chronic asthma? : it depends on the strength of inflammation , for example ; if the patient already have a chronic asthma that is not prominent and he used to take an asthma drug then if he forget to take the drug he will have a high ability for severe , acute attack .

Finally, always remember that ” **You don’t have to be smart to be a good physician; but you need to be thorough** ” Thomas Eskin

<I Wish GOOD LUCK For You All >