Pathology

INFLAMMATION

LECTURE (2)

As a doctor you should know what can threaten your patient's life
you should know what makes your patient suffers from pain
THAT'S WHY YOU LEARN PATHOLOGY

Definition: BLUE Examples:GREEN Important: RED Extra explanation:GRAY Disease names: **UNDERLINE**.

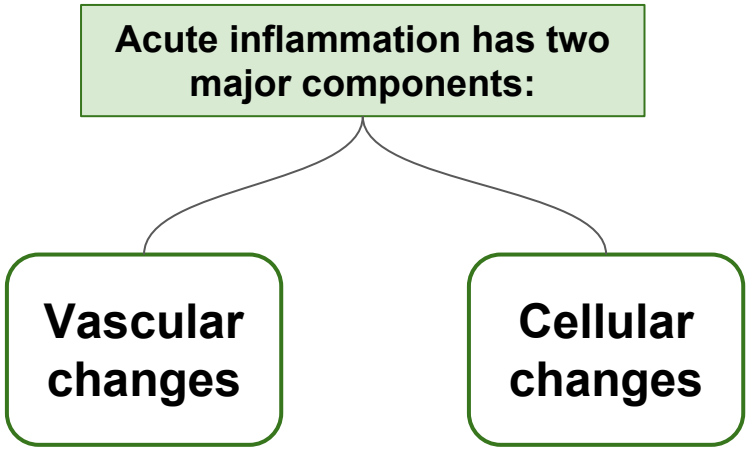
Lecture(2) Outlines

- Acute inflammation.
- Vascular Changes.
- Cellular Changes.
- Morphological patterns of acute inflammation.

Definition of acute inflammation.

Definitions:

Overview: The acute inflammatory response rapidly delivers leukocytes and plasma proteins to sites of injury. Once there, leukocytes clear the invaders and begin the process of digesting and getting rid of necrotic tissues.



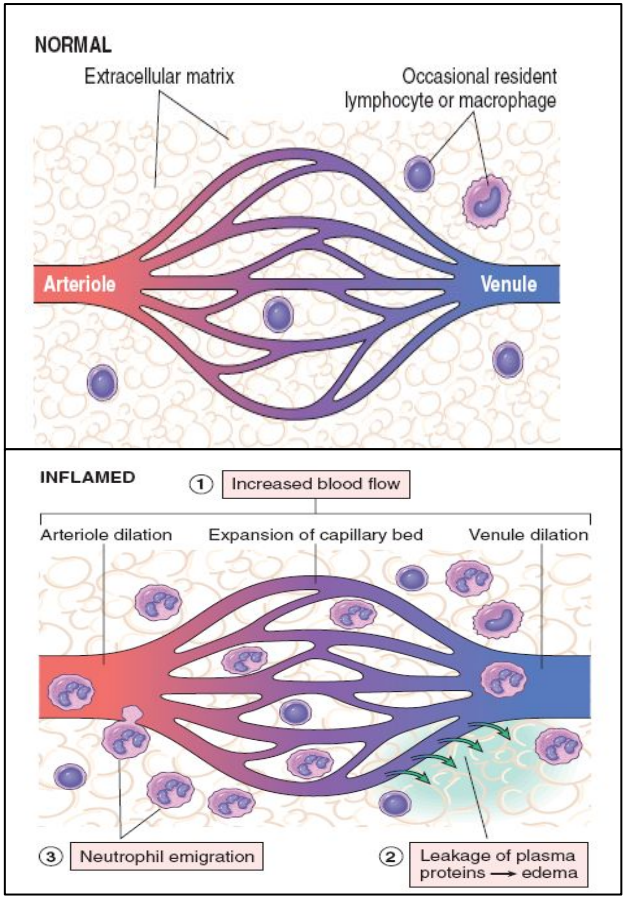
Vascular changes

The main vascular reactions of acute inflammation are:

Increased blood flow secondary to vasodilation.

Increased vascular permeability.

BOTH designed to bring blood cells and proteins to sites of infection or injury.



Extra Explain

THE FIGURES:
Vascular and cellular reactions of acute inflammation. The major local manifestations of acute inflammation. compared with normal are :
(1) vascular dilation and increased blood flow (causing erythema and warmth).
(2) extravasation of **plasma fluid** and proteins (edema)
(3) Leukocyte (mainly neutrophil) emigration and accumulation.

Vascular changes

Increased blood flow secondary to vasodilation.

Vasodilation :Increased blood flow (because of increased diameter of the blood vessel) Leads to Hemodynamic (means relating to the flow of blood within the organs and tissues of the body) (localized).

- Happens at the arteriole because it has muscles that contract.

Increased vascular permeability.

(caused by histamine) → endothelial cells contraction → more neutrophils → Edema.

Edema:

- **Exudate: rich in protein.**
- **Transudate: poor in protein.**

  Will discuss them later
- Happens at the venule because is it is much thinner that the arteriole.

Changes in Vascular Caliber and Flow

FIRST : When there's an immediate antigen (foreign body e.g. bacteria, viruses, parasites, suture material) invading the body we get a transient **arteriolar vasoconstriction** as an inflammatory reaction (last for seconds or few minutes at most).

THEN: Vasoconstriction is followed by **vasodilation** (occurs by chemical mediators) which will lead to a congestion at the area(so this is why we have redness and warmth and stasis at the sight of inflammation).

After that, As stasis develops, **leukocytes (principally neutrophils)** begin to accumulate along the vascular endothelial surface moving from the center to the periphery of the blood vessel; in a process called **margination.**

More Explanation:

أول ما يتعرض الجسم لأي جسم غريب رح يكون في تضيق في الأوعية الدموية بشكل مؤقت يستمر لمدة بسيطة جداً ما تتجاوز الخمس دقائق.

بعد عملية تضييق الأوعية الدموية بيصير عندي توسع في الأوعية عند طريق ال Chemical mediators.

هذا التوسع يؤدي إلى زيادة تدفق الدم وتوسع للشعيرات الدموية أيضاً , وهذا هو سبب الإحمرار والحرارة اللي تصير عند الاتهابات.

بالإضافة إلى حدوث عملية تسمى:

Stasis:means slow circulation due to dilated small vessels packed with RBC's.

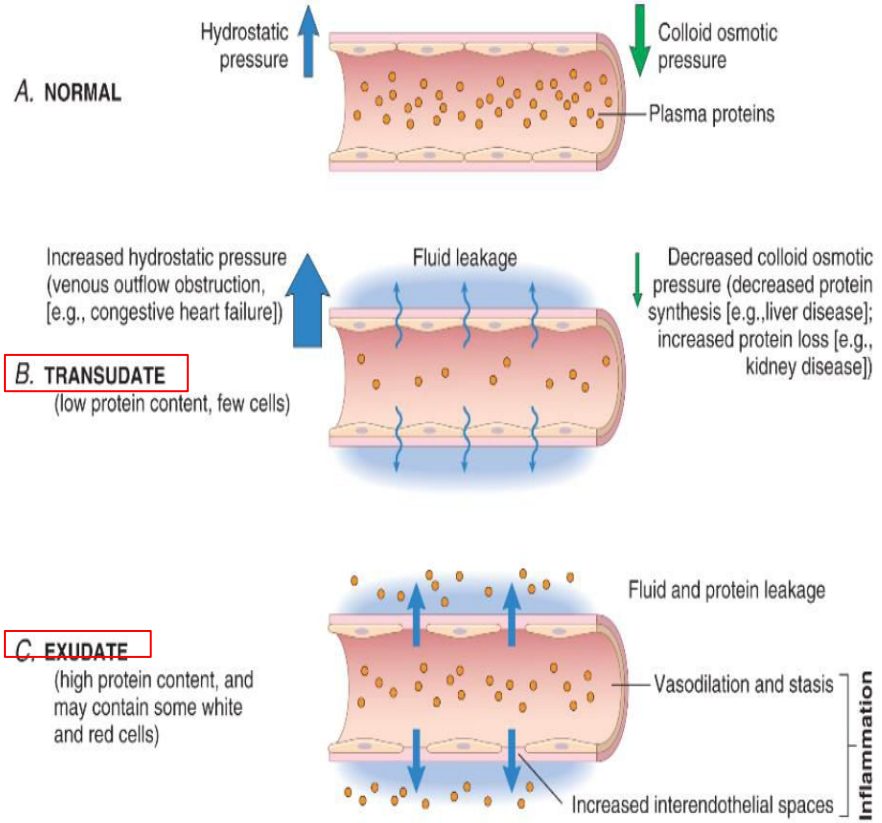
لمن تتطور هذه العملية يجي دور خلايا الدم البيضاء وبالأخص النيتروفيلز,بالعادة هي تكون ماشية بنص الأوعية تروح وتبدا تتجمع على الأطراف وتكون عملية اسمها : Margination.

Increased Vascular Permeability

Increased vascular permeability means:
Endothelial cell contraction caused by (histamine, bradykinin, leukotrienes). The gaps between endothelial cells increases, leads to the movement of **protein-rich fluid** ,and even **blood cells**, to the extravascular tissues. The resulting protein-rich fluid accumulation is called an **exudate.**

After that, fluids we'll escape from the blood vessels to the interstitial tissue because (there are changes in the pressure) and this will cause Edema that lead to a **swelling**.
(One of the causes of pain is that edema fluids compresses the nerve ends).
inflammatory cells = leukocytes = WBCs.

Margination: The movement of inflammatory cells at this state.
Endothelial cells: Cells of the inner wall of blood vessels

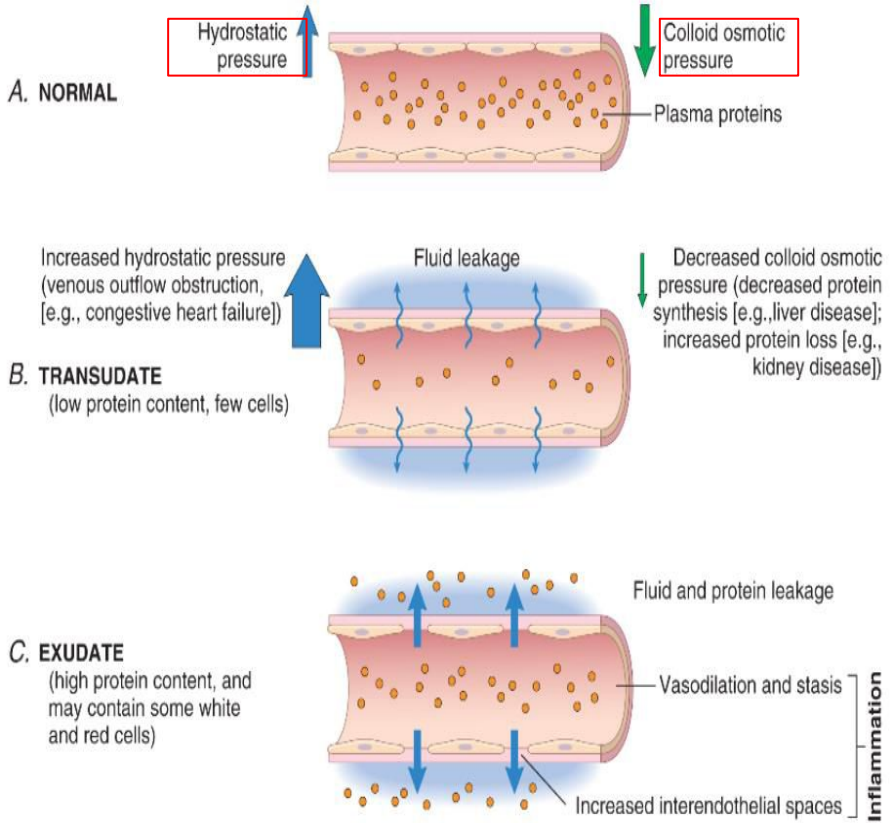


Three types of pressures that control the exudation of fluids:

Osmotic pressure: it happens when there's a difference in the concentration of ions between two different media and (it doesn't play a major role in inflammation).

Hydrostatic pressure: it's the pressure applied from the blood on the walls of blood vessels and it depends on the contraction force of the heart muscle or the pressure from the blood to the vessel wall.

Colloid or oncotic pressure: it's controlled by proteins in the blood (for example if protein levels in the blood are low this will cause edema).



Summary

Fluid type	Condition	Content
Transudate	Increased Hydrostatic pressure or decreased colloid osmotic pressure	Low protein
Exudate	Acute inflammation	High protein
Pus	Acute inflammation	High protein & neutrophils

FROM ROBBINS BOOK

SUMMARY

Vascular Reactions in Acute Inflammation

- Vasodilation is induced by chemical mediators such as histamine (described later) and is the cause of erythema and stasis of blood flow.
- Increased vascular permeability is induced by histamine, kinins, and other mediators that produce gaps between endothelial cells; by direct or leukocyte-induced endothelial injury; and by increased passage of fluids through the endothelium. This increased permeability allows plasma proteins and leukocytes to enter sites of infection or tissue damage; fluid leak through blood vessels results in edema.

Cellular changes

MARGINATION

First step : (the white blood cells move from the center of the blood stream - which is their normal place- to the side of the blood stream (peripheral) this makes them become closer to the vessel wall) this is called **MARGINATION.**

(In inflammation those adhesion molecules are activated by certain chemical mediators, this occurs after the margination of inflammatory cells)

Adhesion molecules

Second step : These cells will adhere to the endothelial cells because there are adhesion molecules called:

Integrin: they cover the outer surface of inflammatory cells.

Selectin: they cover the outer surface of endothelial cells.

Diapedesis

Third step :

Diapedesis: is when inflammatory cells through extension from their cytoplasm and go in between the gaps of endothelial cells to the outside of blood vessels.

Chemotaxis

Third step :

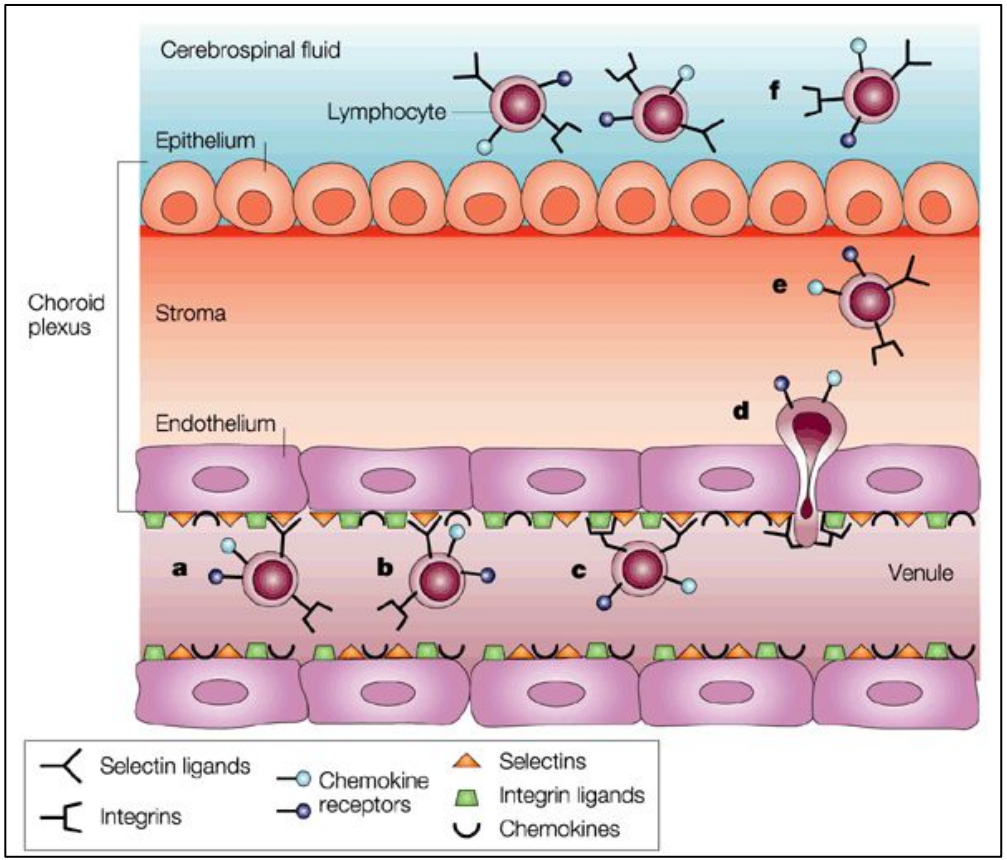
Chemotaxis: is a guided margination of inflammatory cells from the blood vessels to the site of inflammation.

Phagocytosis

Fourth step :

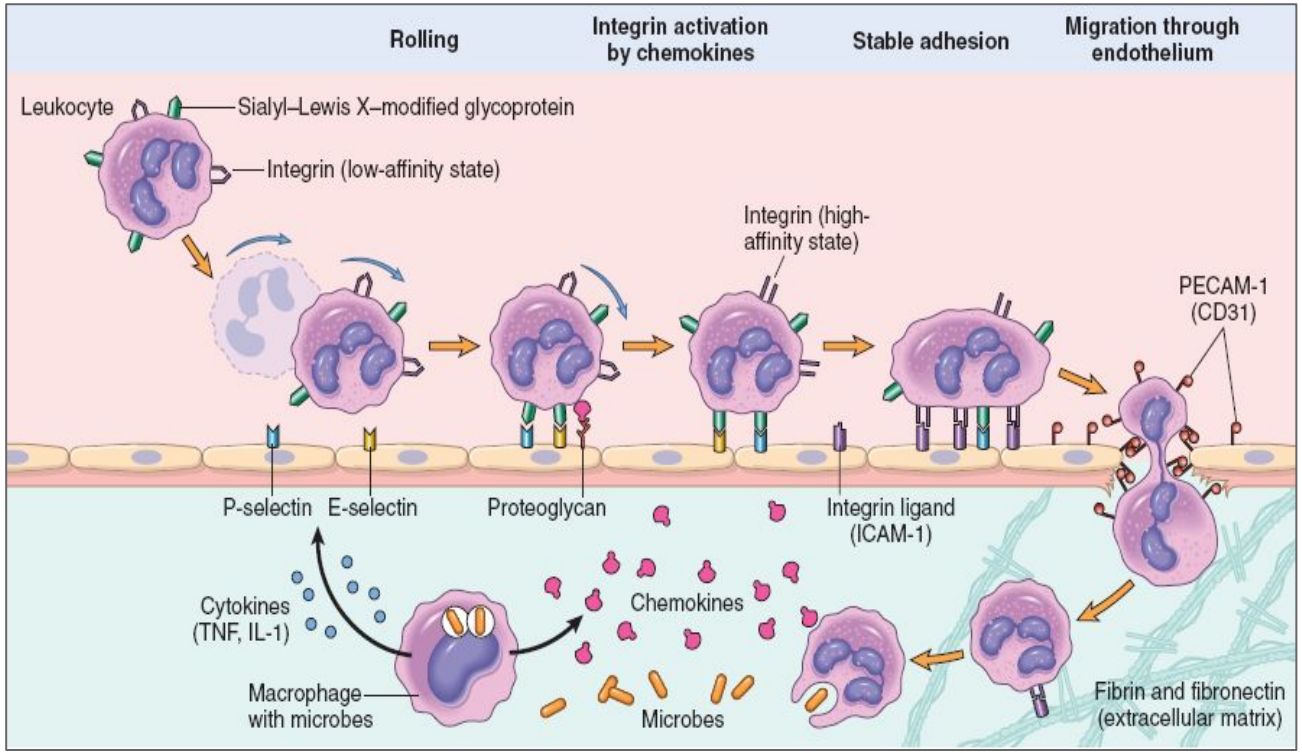
Phagocytosis: When the inflammatory cell phagocytose (eat) the invader or the infected cell and digest it.

Cellular changes



مثال عندنا مرض السل مرض تنفسي بالدرجة الأولى ويعتبر من أمراض الالتهاب المزمن، وهو يحدث في الرئة لأن الباكتيريا تحب الأكسجين ولذلك تجدها هناك، وبسبب تراكم البكتيريا ستذهب الخلايا اللمفاوية للصدر، وسيحدث زيادة نفاذية للأوعية الدموية مما يفسر وجود سائل متراكم في الرئة .

Cellular changes



THE FIGURE:

Mechanisms of leukocyte migration through blood vessels. The leukocytes (neutrophils shown here) first roll, then become activated and adhere to endothelium, then transmigrate across the endothelium, pierce the basement membrane, and migrate toward chemoattractants emanating from the source of injury.

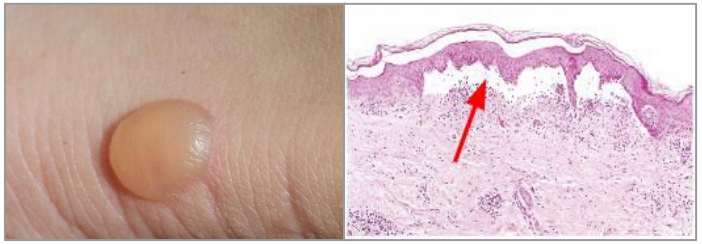
The sequence of events in inflammation:

Injury → vascular constriction → vascular congestion → vascular dilation increased hydrostatic pressure escape of fluid and inflammatory cells to interstitial area
→ margination of inflammatory cells
→ adhesions of inflammatory cells → they go outside of the blood vessels to the site of injury/antigen/foreign body/burn.

Morphological patterns of acute inflammation

1- Serous inflammation:

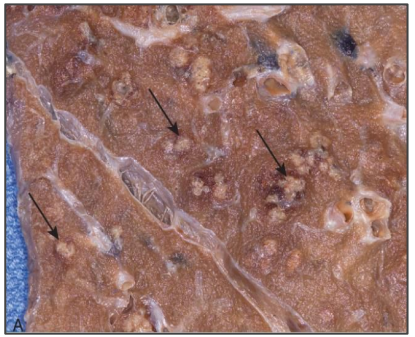
Is marked by the outpouring accumulation of protein-poor fluid (watery) under the epidermis.
derived either from the plasma, or from the secretions of mesothelial cells lining the peritoneal, pleural, and pericardial cavities. Fluid in a serous cavity is called an **effusion.**
it's usually yellowish in color and seen in some diseases and as a result of some burn.
happens in skin
e.g. sunburn, eczema.

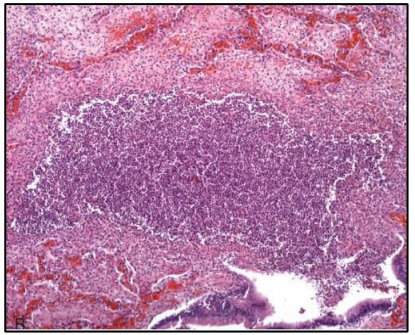


2- Suppurative(purulent) inflammation:

Known by the presence of large amount of purulent exudate (pus)
(usually formed by bacteria, fibrin, nuclear debris (حطام الخلايا)
Abscess consisting of neutrophils, macrophages, necrotic cells, and edema fluid.
It has a yellowish to grey color.

Pyogenic bacteria: Bacteria that produces lots of pus.
Examples: (Staphylococcus aureus and streptococci)





Morphological patterns of acute inflammation

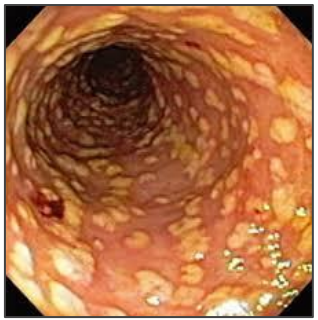
3-Pseudomembranous colitis

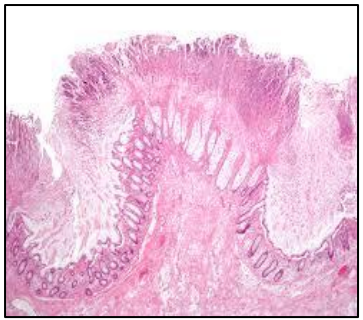
(التهاب القولون الغشائي الكاذب)

NOT very common

For example: a man suffers from diarrhea, blood,mucus that started when he was using antibiotic for a long period of time (e.g. 2 months) now he has new bacteria resistant to all antibiotics.

It affects his large bowel→ pseudomembranous inflammation → caused by clostridium difficile bacteria resistant to ordinary antibiotics.





4-Fibrinous inflammation:

Occurs as a consequence of more severe injuries, resulting in: greater vascular permeability and the exudate (pus)

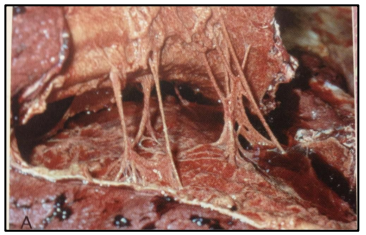
It's also rich of fibrinogen (which will turn to fibrin in the coagulation process).

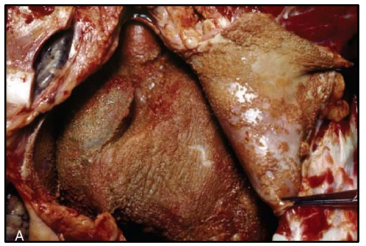
it usually affects the serous cavity.

e.g. pericardium (cavity around the heart)

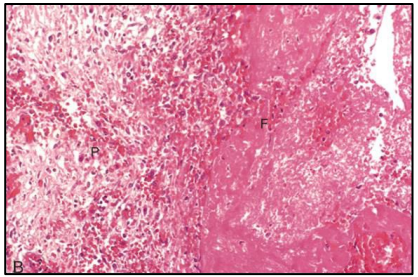
It has yellowish adhesions "bread and butter appearance"

NOTE: fibrin is not related to fibroblast.





Deposits of fibrin on the pericardium.

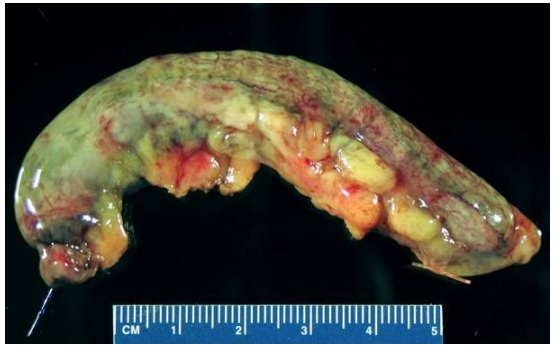


Morphological patterns of acute inflammation

5- Gangrenous inflammation.

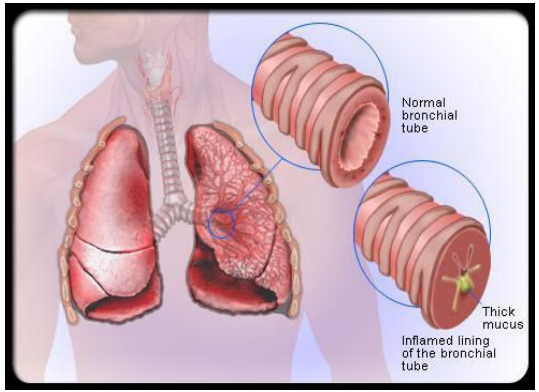
sometimes the inflammation becomes severe and will activate the coagulation cascade, this will result in accumulation of platelets, thrombin, and fibrin and will clot the blood vessels.

If inflammation occurs for a long time, local ischemia happens→ gangrene.
e.g. gangrenous appendicitis



6- Catarrhal inflammation:

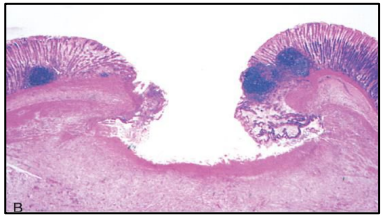
Regular cold where there is increased secretion of mucus.



7- Ulcer

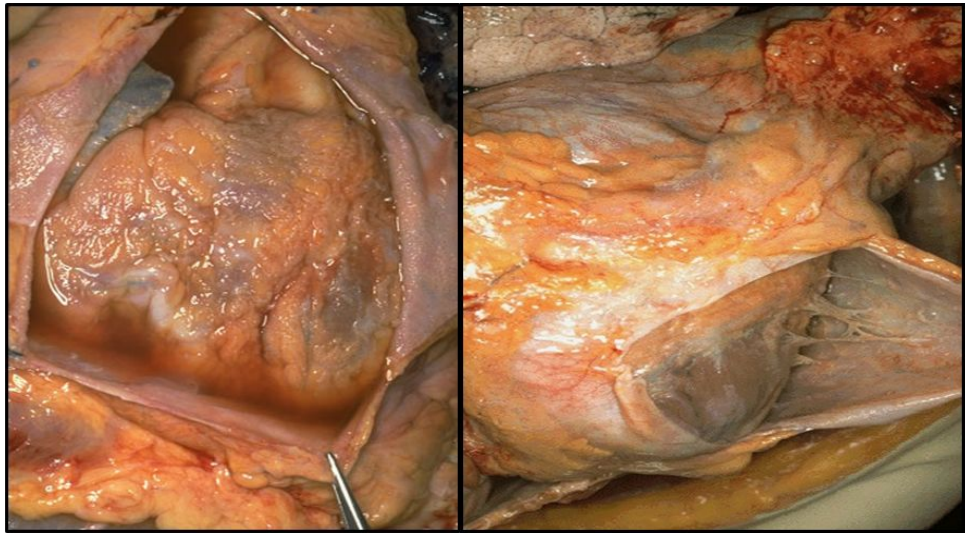
القرحة

A local defect (hole) on the surface of an organ or tissue.
It produces necrotic cells and sloughing(انسلاخ) an exclusion of dead layer)



Clinical Ceases

Fibrinous pericarditis (1)



pink-tan **fibrin have been formed and this explain the paleness and the rough appearance**

causes

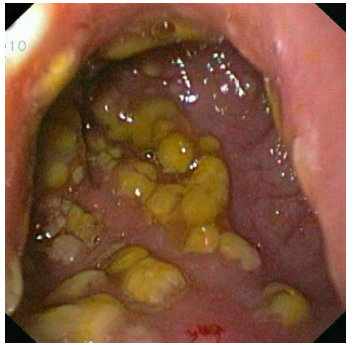
* viral
* tuberculosis
* pyogenic
* myocardial infarction
* chronic renal failure
* connective tissue diseases
* malignant neoplasms

clinical presentation

Pericardial friction rub may be heard.

Clinical Ceases

Pseudomembranous Colitis (2)



symptoms

- Watery diarrhea
- Abdominal pain along with cramping
- Fever
- Mucusy stools
- Nausea
- Dehydration

causes

- if an individual takes excessive antibiotics
- if the balance of harmful and healthy bacteria gets affected causing overgrowth of harmful bacteria

treatment

- stopping the antibiotic which started the symptoms
- treat with another antibiotic which can destroy C. Difficile

HOPEFULLY WE DID A GREAT JOB

MCQS ONLINE TEST

FOR ANY QUESTIONS AND SUGGESTIONS CONTACT US ...

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To make sure that all students are aware of any changes, please check out this link to know if there are any additions or changes.
The same link will be used for all of our work:
(Pathology Edit)

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- Sarah AlHussein

DIFFICULTIES IN YOUR LIFE DON'T COME TO DESTROY YOU.. BEST OF LUCK