

Practical Pathology

cell injury and inflammation

second-Year

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طب وجراحة عامة

البورد عربي (دكتوراه) تشخيص الأورام الحميدة
والسرطانية والنسيج المرضي
فرع الامراض / كلية الطب / جامعة تكريت
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Practical pathology

cell injury , inflammation and repair

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كلية الطب / فرع الامراض

طب وجراحة عامة- كلية طب

تكريت

M.B.CH.B.TUCOM.ABH.

شهادة البورد العربي (دكتوراه) تشخيص الأورام الحميدة والخبيثة والنسيج المرضي بالابرة

القاطعة والدقيقة والهرمونات السرطانية

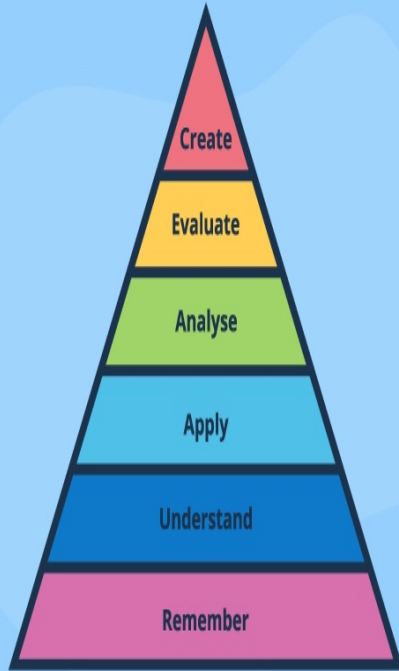
بغداد/الاردن

Anatomical Pathology, ABH (Arab Board Of Histopathology)

Diagnosis of histopathology by Tru-cut biopsy , FNA (Fine Needle Aspiration) and
Immunohistochemistry (IHC)

Baghdad/Jordan

Bloom- Toxonomy



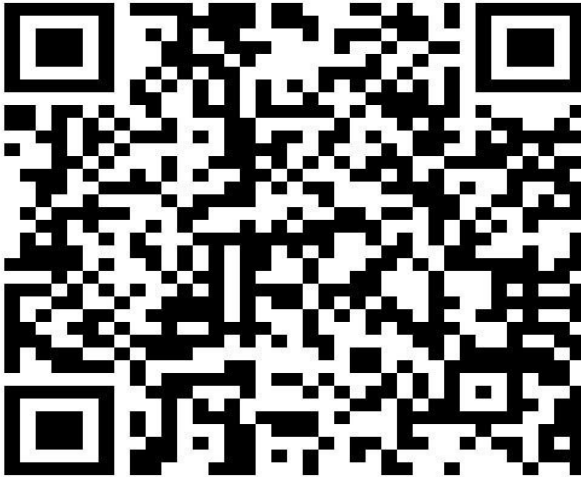
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What is the apoptosis

what your information about **microscopic** feature

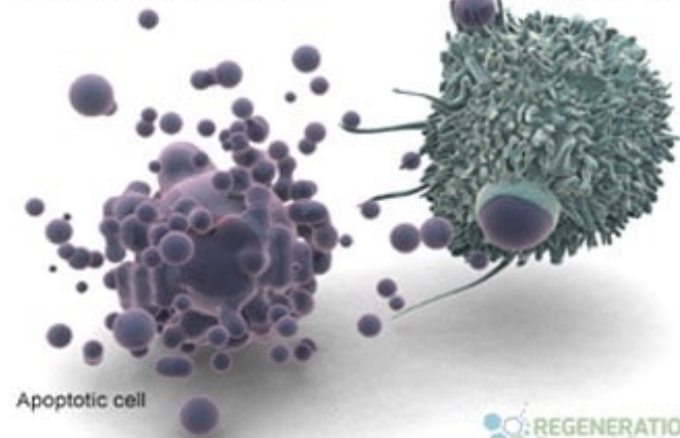
what is the role of **apoptosis** with development of **cancer** and different diasease

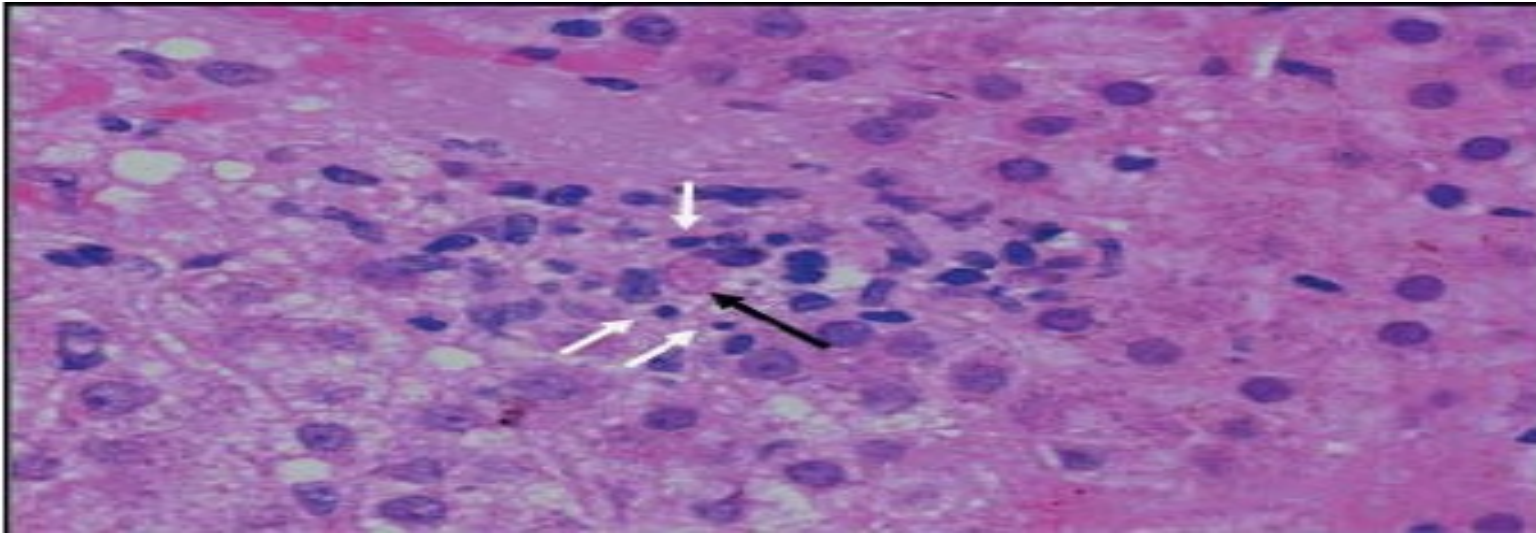
How can you correlate basic information with **clinical information**



Final stage of apoptosis

White blood cell



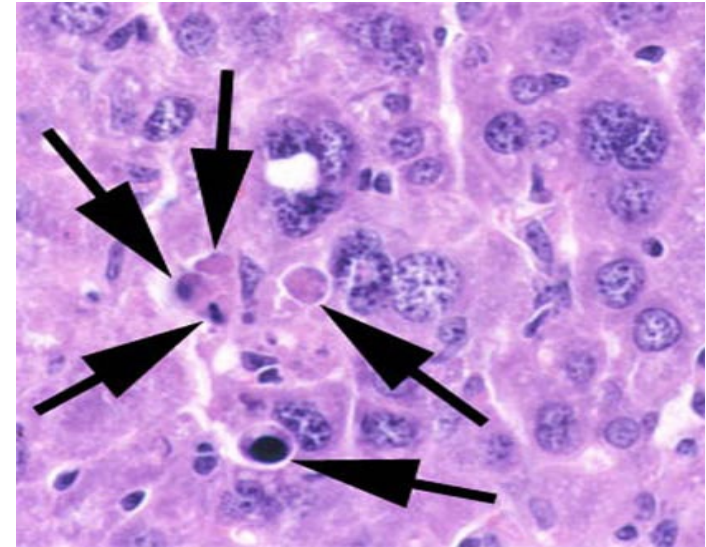


Evidence of hepatocyte apoptosis in vivo. Histopathological examination of a liver section from a patient with hepatitis C virus by conventional haematoxylin-eosin staining shows an apoptotic body (also known as Councilman body, black arrow) surrounded by immune cells, such as T lymphocytes (white arrows) and macrophages.

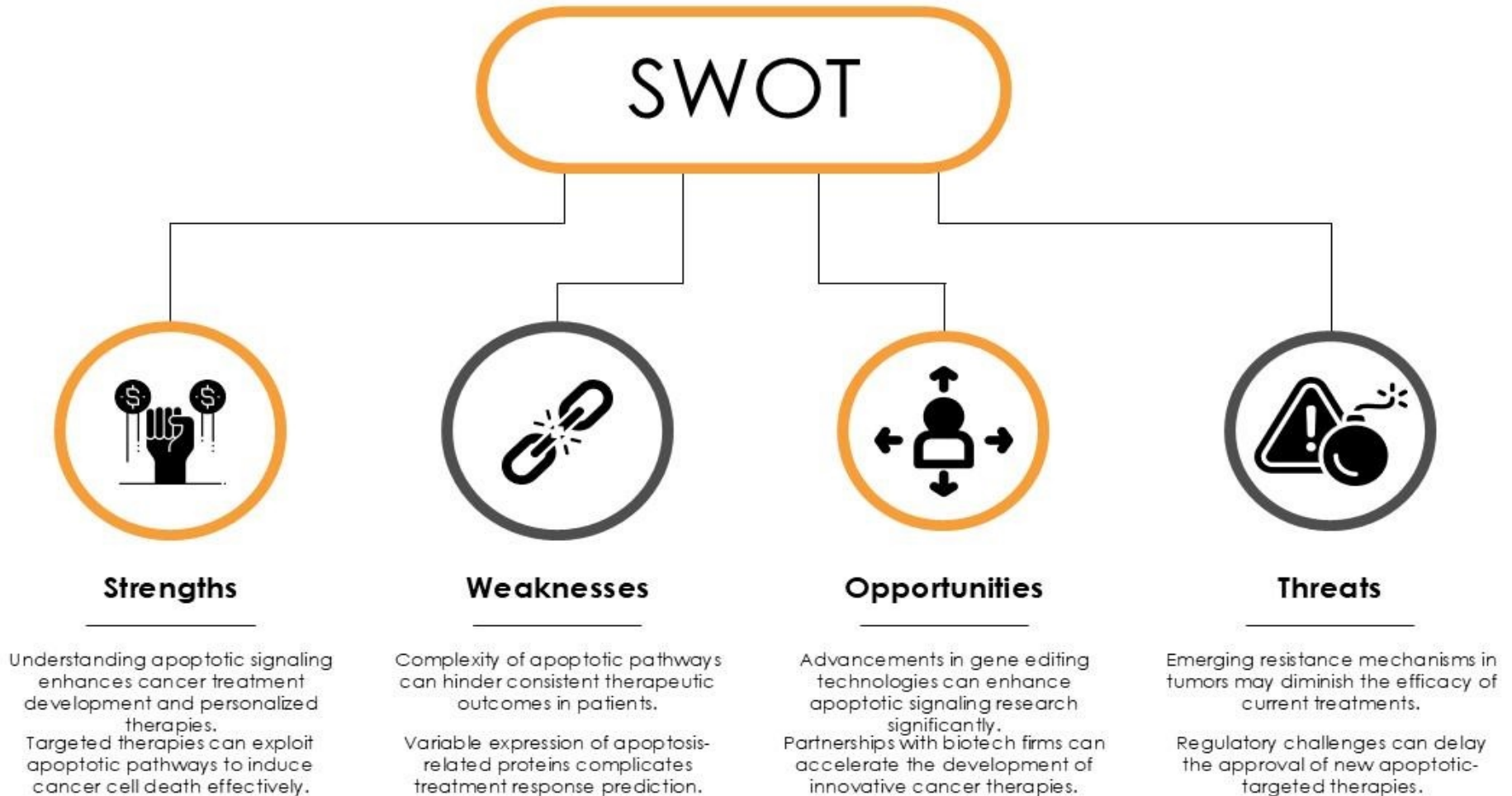
In Greek, **apoptosis** translates to the "falling off" of leaves from a tree. Cormack, professor of Greek language, reintroduced the term for medical use as it had a medical meaning for the Greeks over two thousand years before



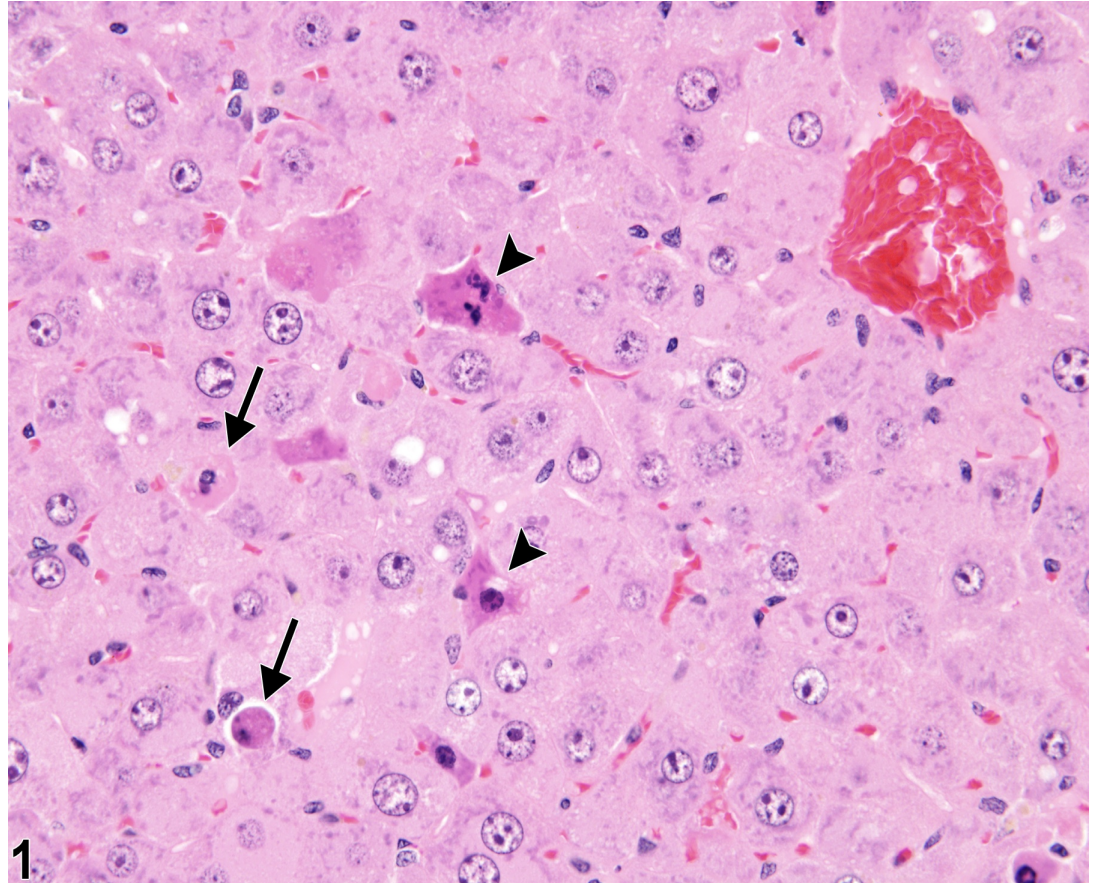
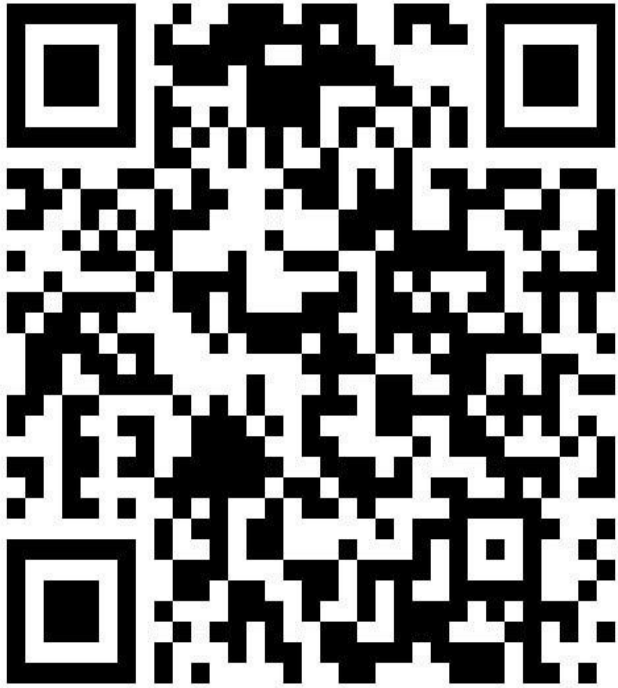
microscopic features of apoptosis:
apoptosis involves single cells or small clusters of cells. The apoptotic cell appears as a round or oval mass with dark eosinophilic cytoplasm and dense purple nuclear chromatin fragments (Figure 1).



Apoptosis and Cancer Connection



What is the diagnosis?

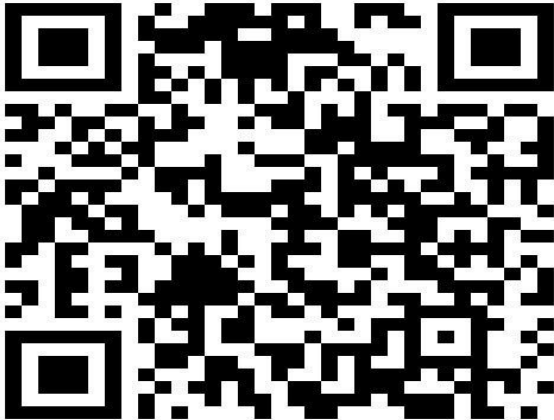


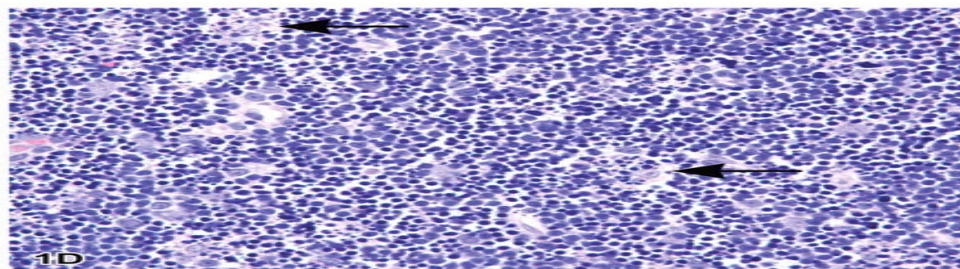
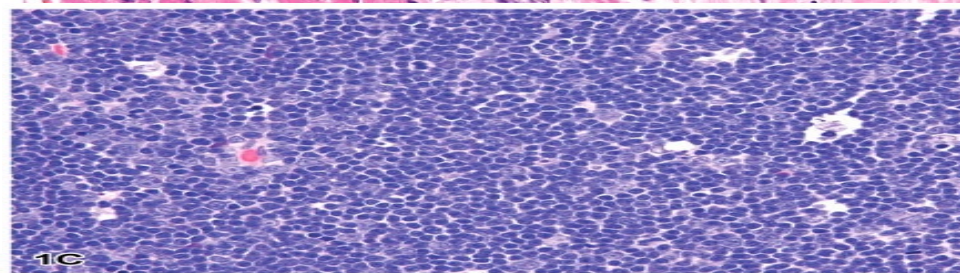
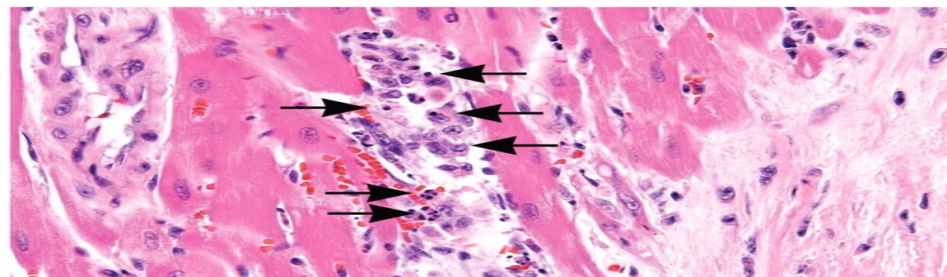
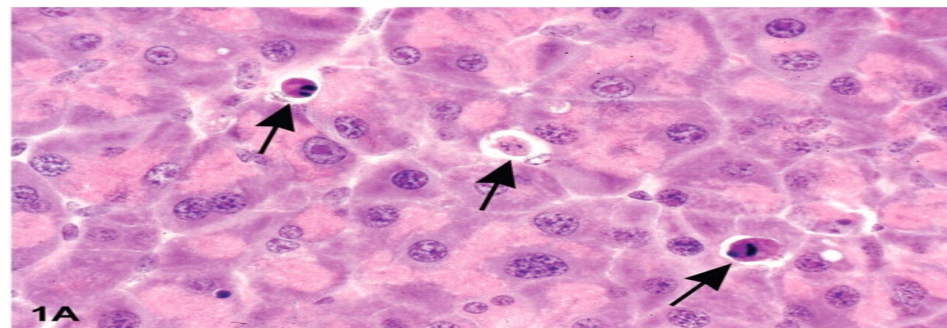
Team based learning (divided to sub-group)(G1-G6)(each group with 25 students):

- 1. Summarize microscopic features of apoptosis
- 2. Discuss clinical aspect of apoptosis
- 3. Discuss role of apoptosis in treatment of cancer



Form of Collaborative Projects & Assignments: Team-Based Learning





For more information about
Apoptosis please see video by visit
below link....

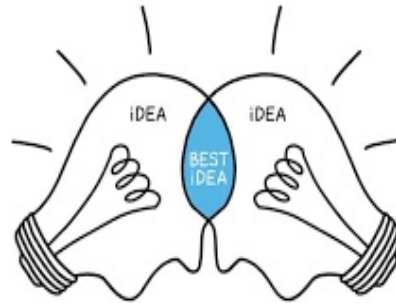
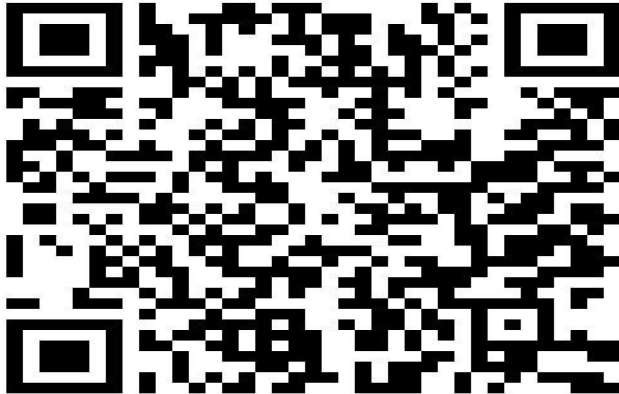


[https://www.youtube.com/watch?v=-vmtK
-bAC5E](https://www.youtube.com/watch?v=-vmtK-bAC5E)

What is **Necrosis**

what is the clinical application of Necrosis

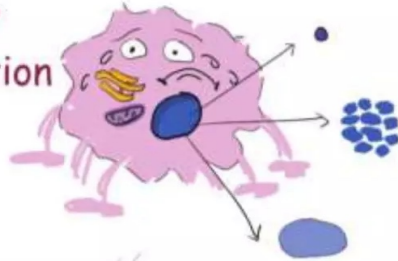
what is the clinical benefit of Necrosis and
correlation with **cancer**



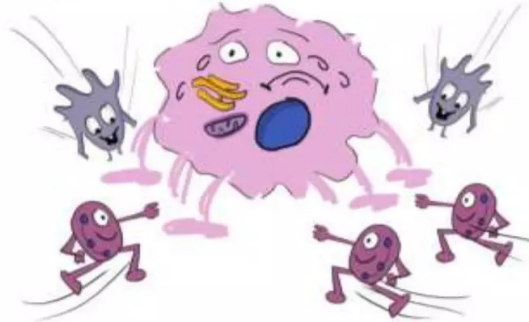
Team-Based
Learning

Necrosis

6. Nuclear
Degeneration



5. Inflammation

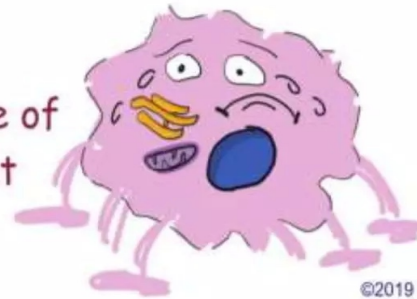


1.
Always Pathological



2.
Cell Enlargement

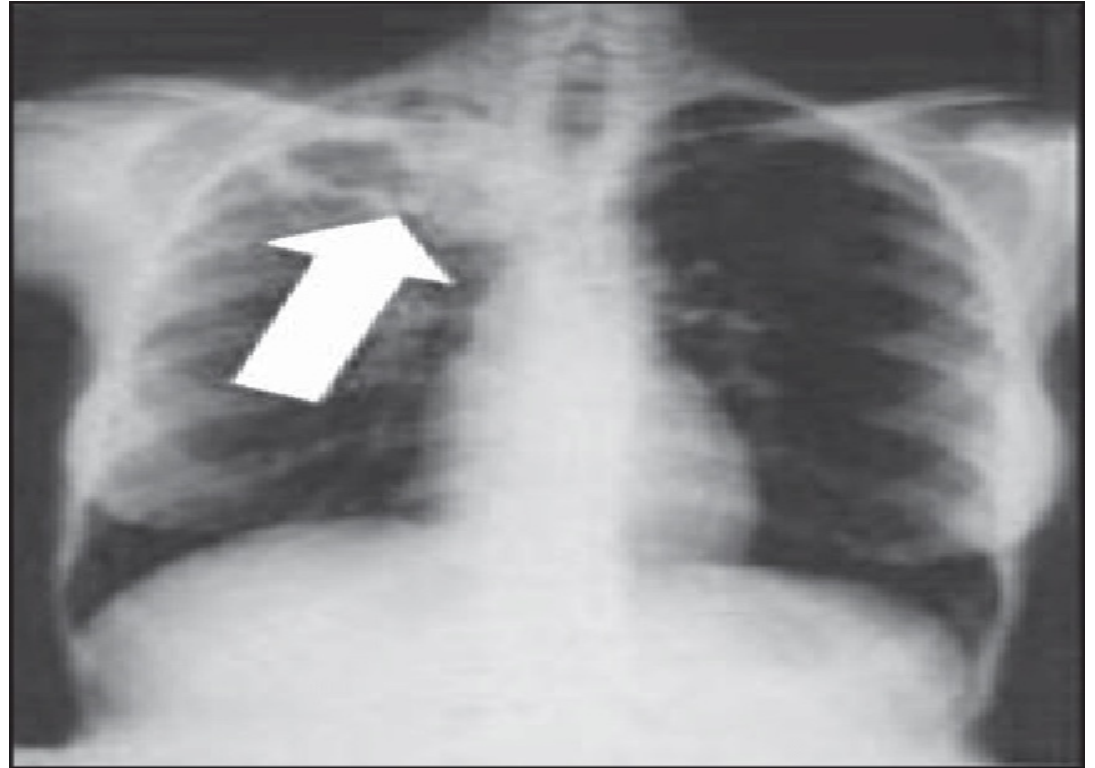
4.
Leakage of
Content



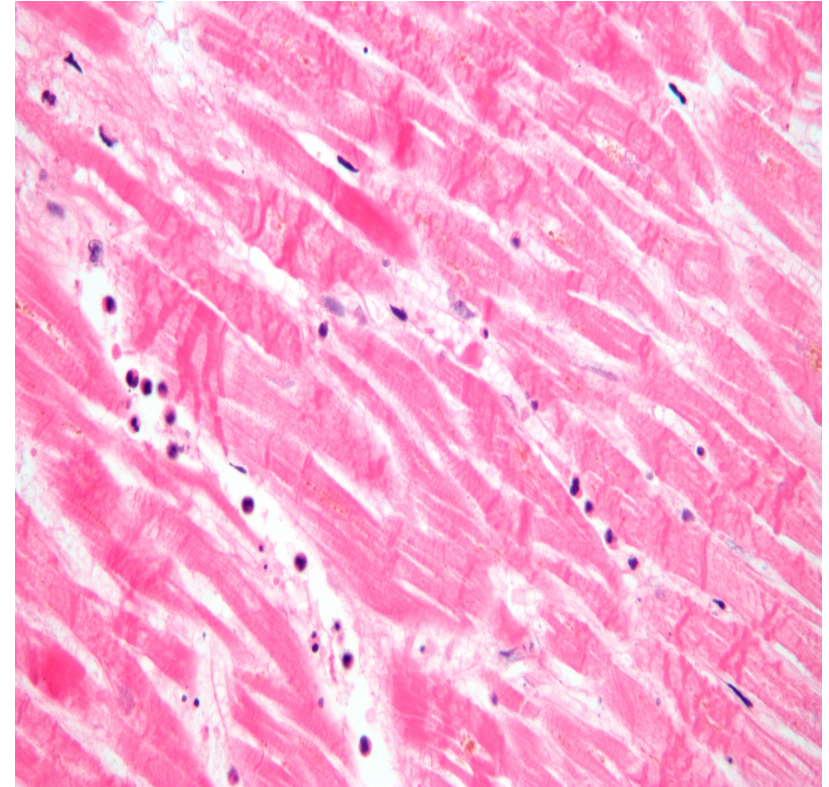
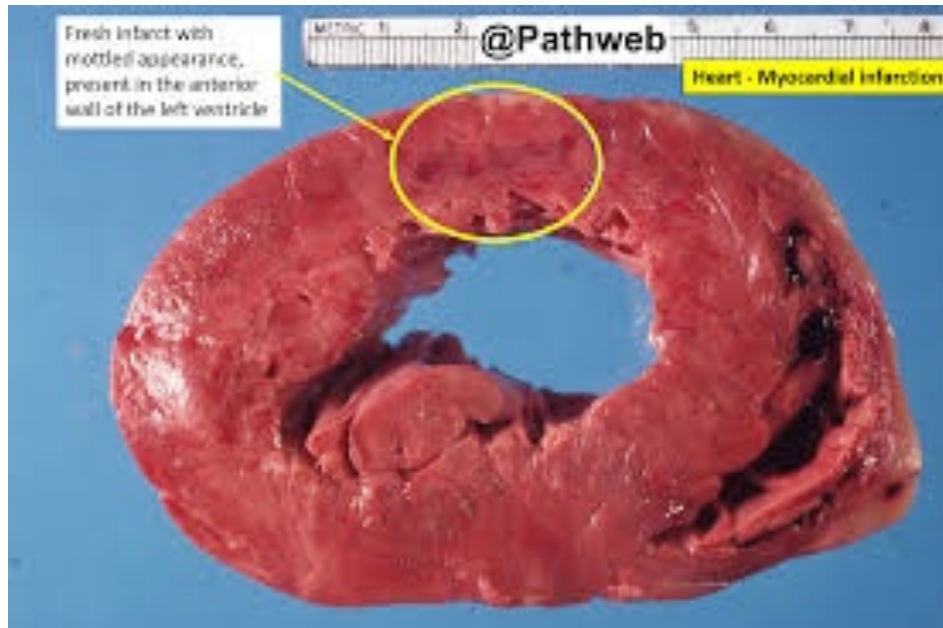
3. Loss of
Membrane Integrity



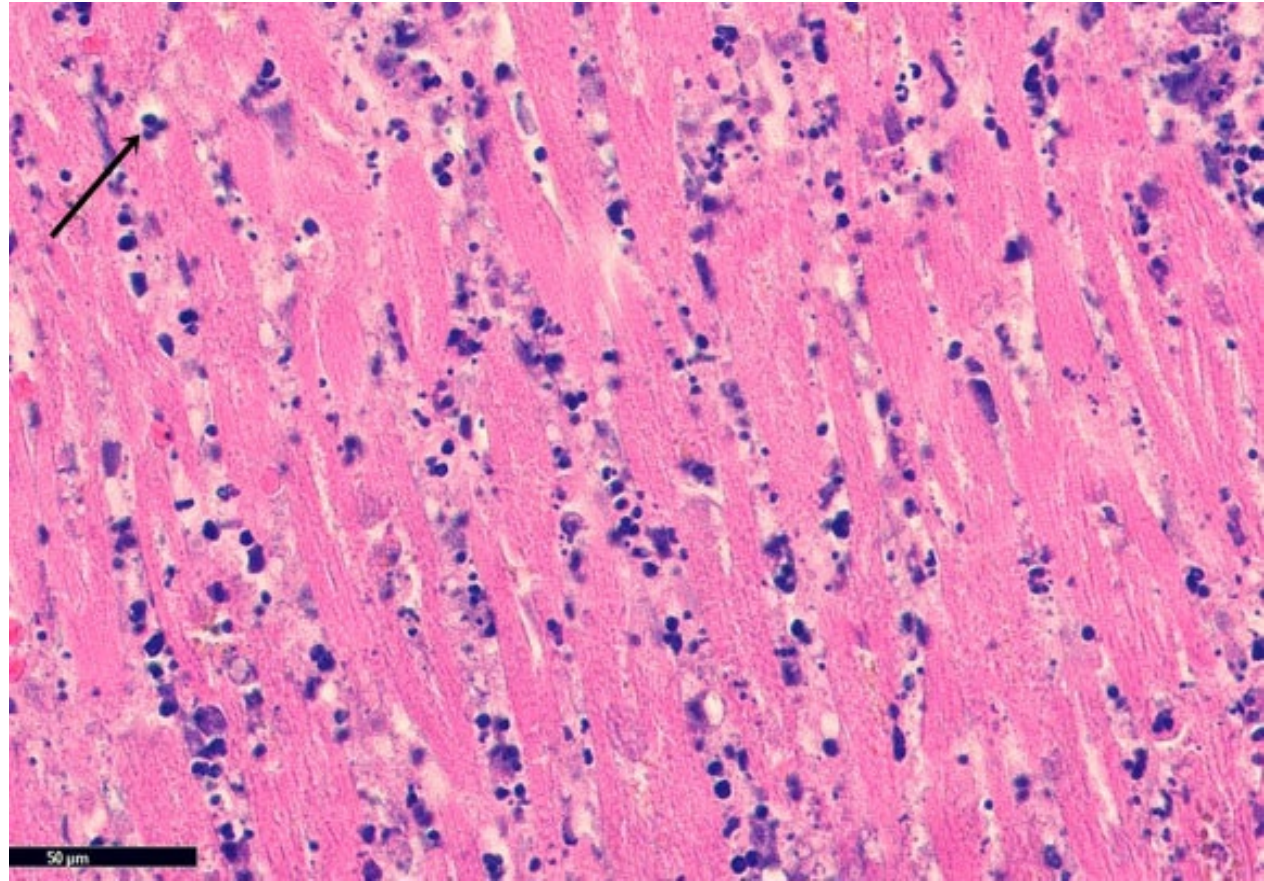
Necrosis comes from the Greek origin **nekrōsis** meaning “death” and later moved to modern Latin to necrosis. Necrosis can be described as a pathological process of cell death which could have been resulted from infections, hypoxia, trauma or toxins. Unlike apoptosis, necrosis is uncontrolled and release lots of chemicals from the dying cell to which causes damage to surrounding cells. Inflammation is often initiated due to necrosis. There are many types of morphological patterns that necrosis can present itself. These are **coagulative**, **liquefactive**, **caseous**, gangrenous which can be dry or wet, **fat** and **fibrinoid**.



60 year old male presented to emergency room with severe chest pain radiating to his left arm after 24hr of treatment and investigation he is passing out, his heart send for forensic medicine for further investigation and histopathology of heart showing as in the below picture



solid organs which allow
preservation of cell shape by
coagulation of cell proteins
e.g.) heart, liver, kidney
Except (**Brain**)



Coagulative necrosis of the myocytes
with absent nuclei and an interstitial
neutrophilic infiltrate (black arrow).

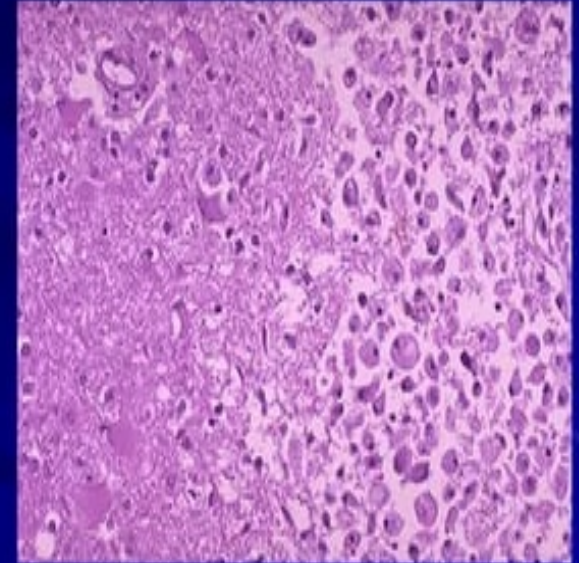
- 70 year old male presented to emergency room complain from seizure attack ,CT-scan showing oedema within temporal area after 3hrs he is died by due to brain ischemia, his brain tissue showing this microscopic picture what is your diagnosis.



LIQUEFACTIVE NECROSIS BRAIN



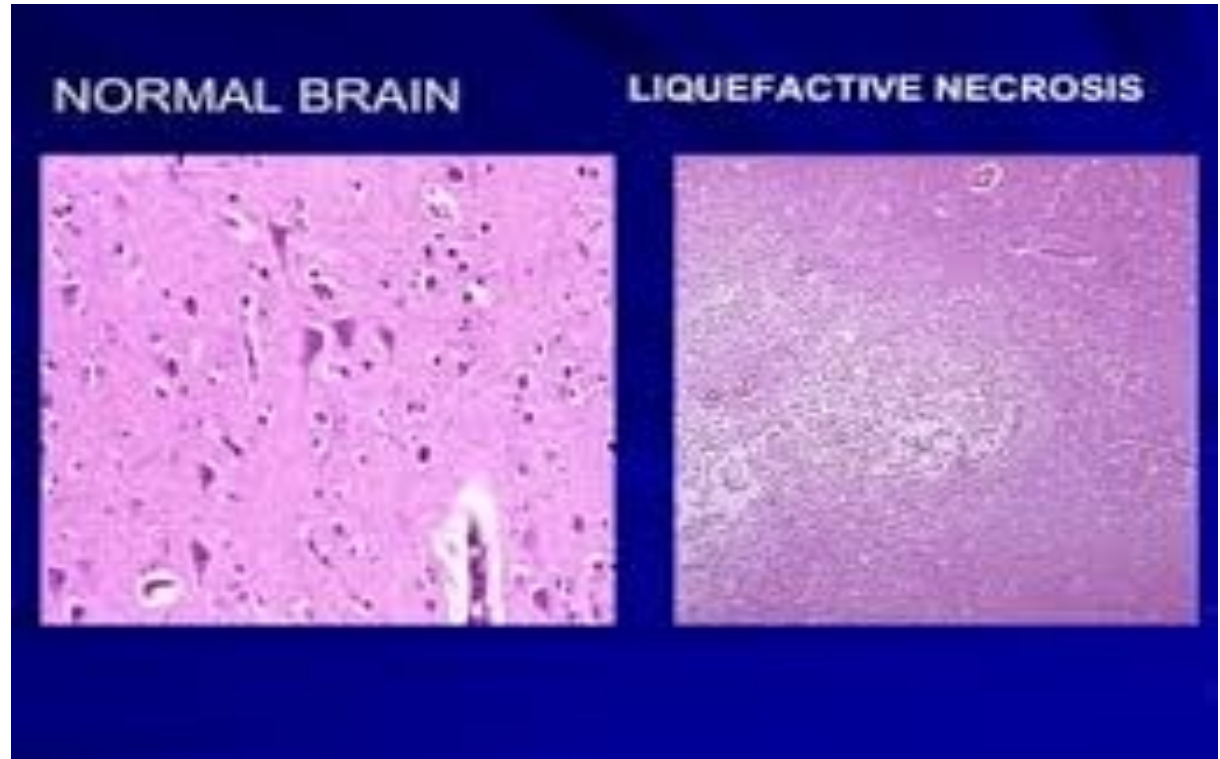
LIQUEFACTIVE NECROSIS BRAIN



soft organs which allow lysis of cells and
surrounding proteins
e.g.) brain

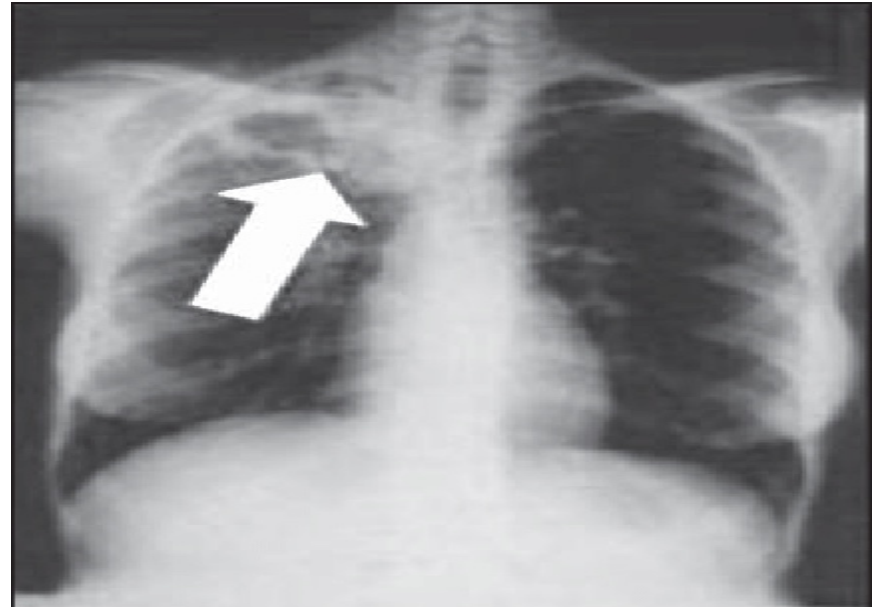
Where/when:
infections, **brain**,
abscess.

Microscopic: nothing
left; pink on H&E.

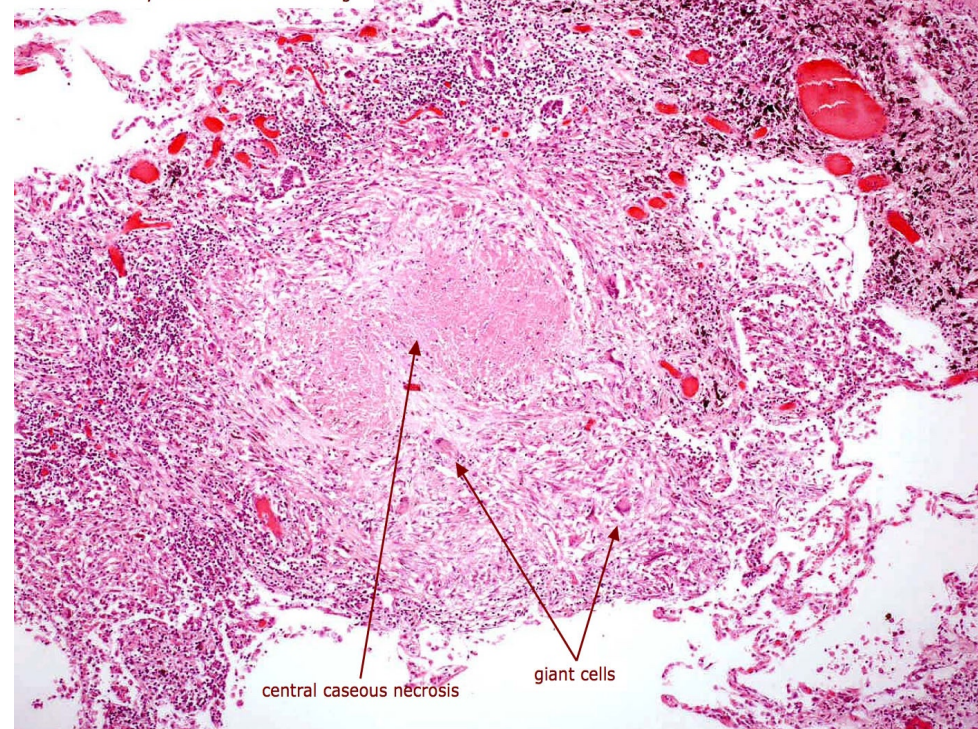
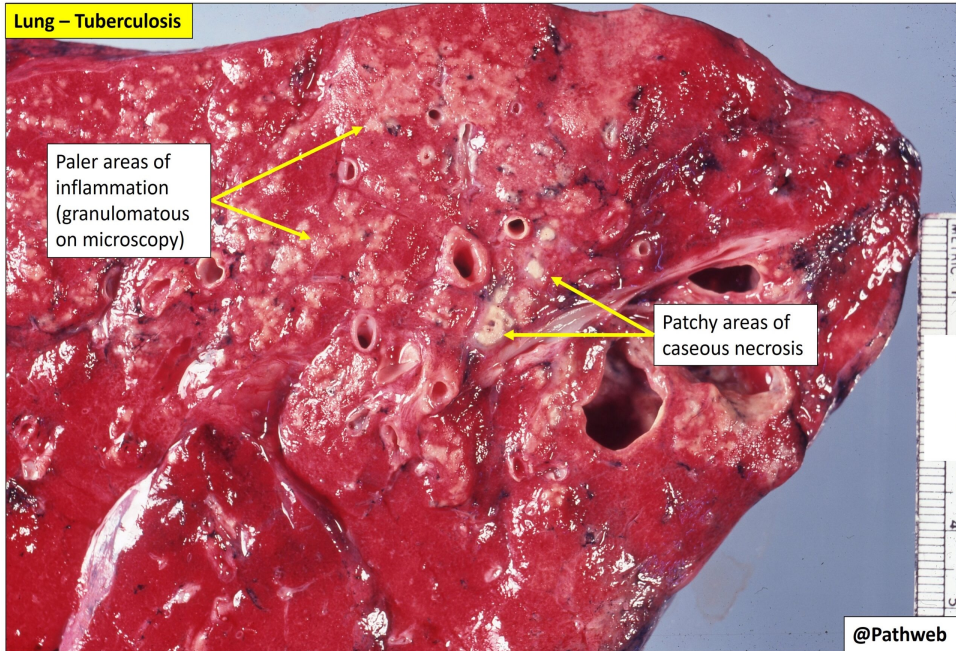


- 40year old female complain from cough ,fever and night sweating for 3months , after proper investigation her chest x-ray showing as below what is your diagnosis. Answer by classroom

- <https://classroom.google.com/c/NzIzMzQwNjIzODcx/p/NzE0OTY4NzE0MjEw/details>

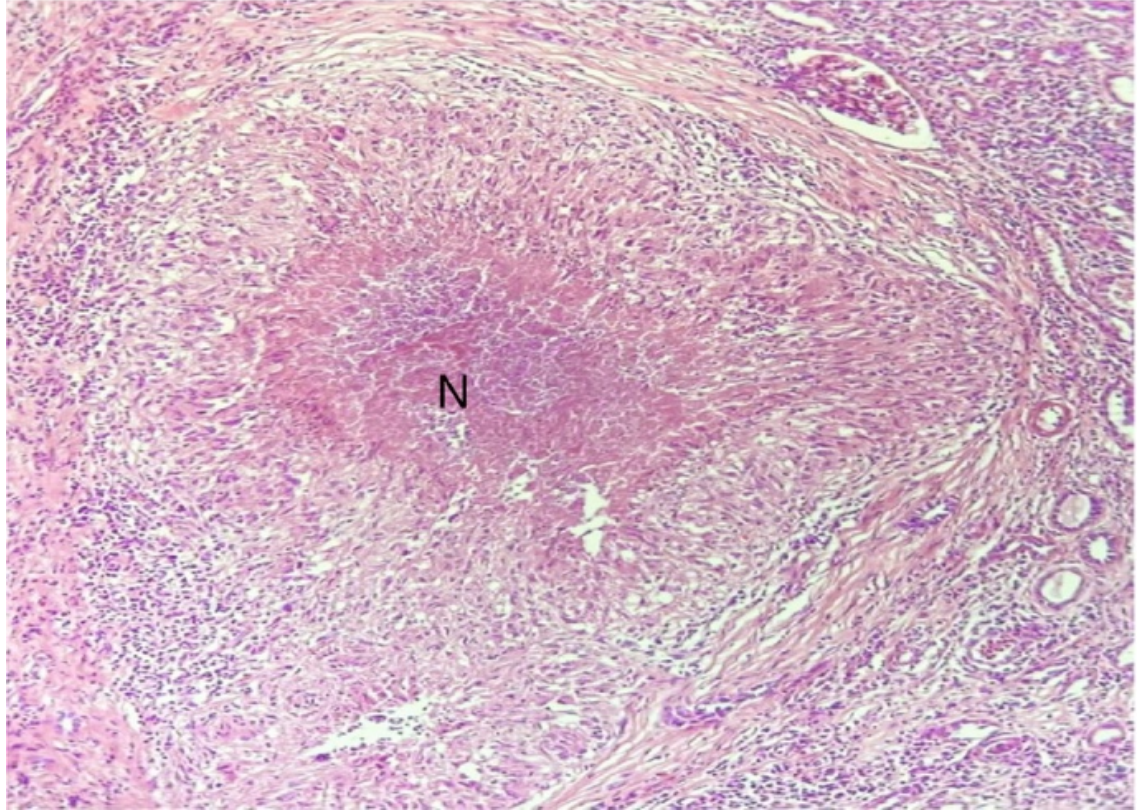
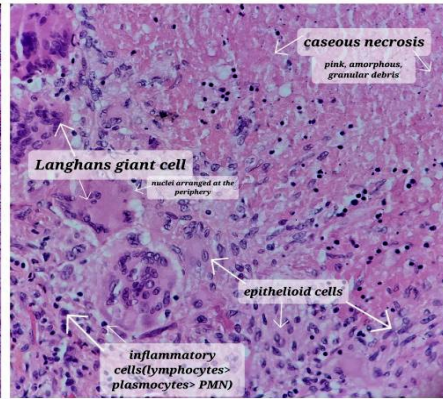
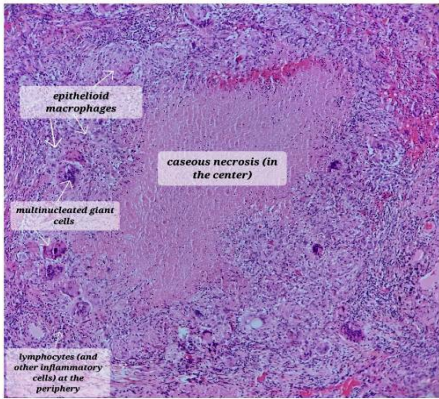
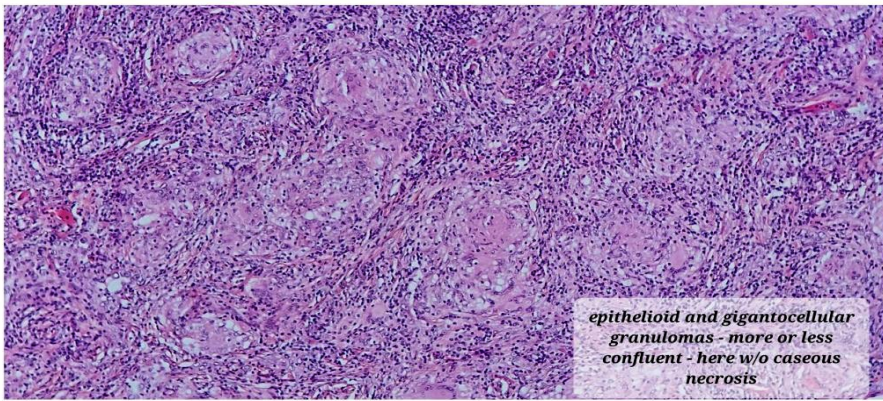


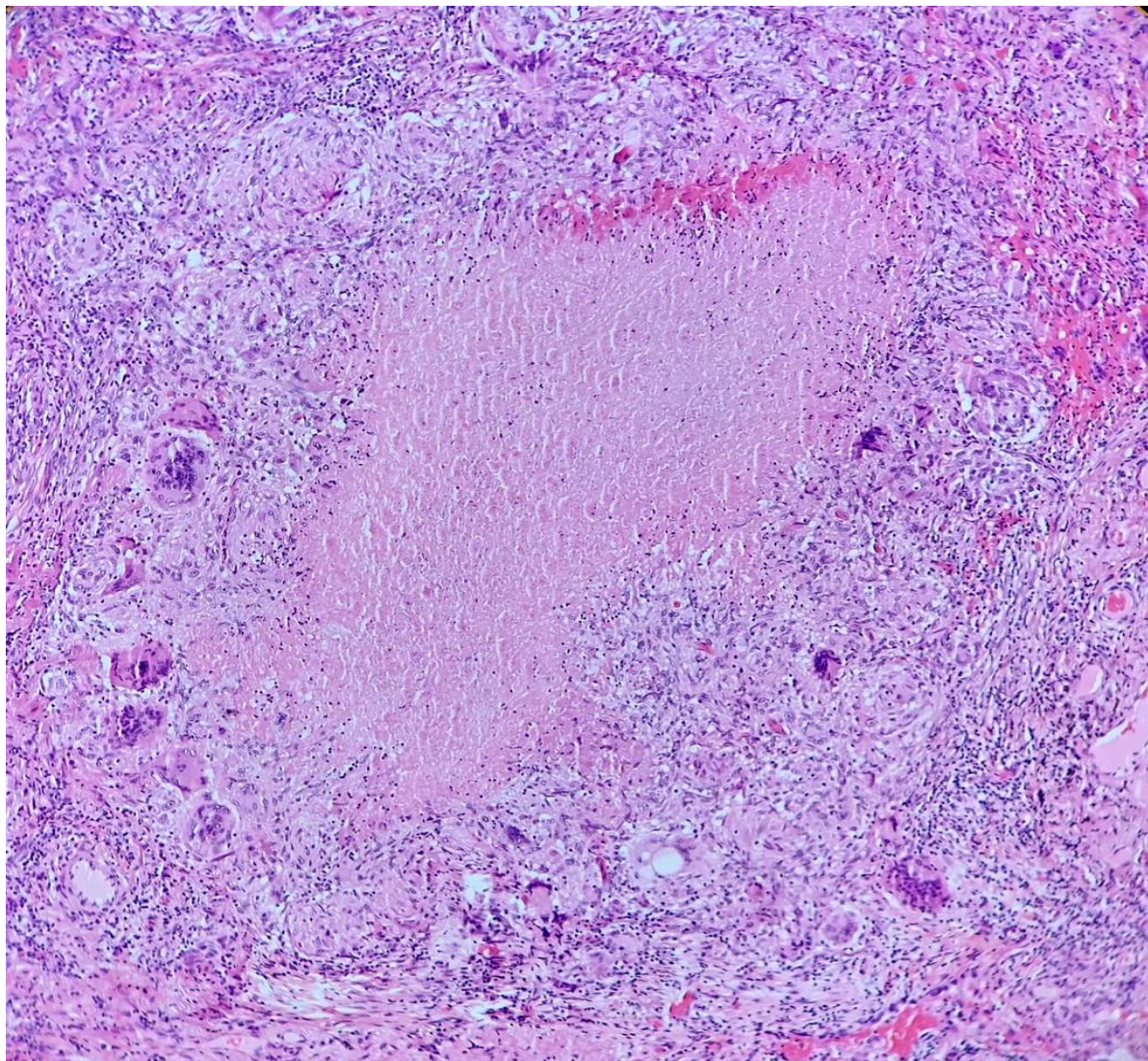
Lung – Tuberculosis



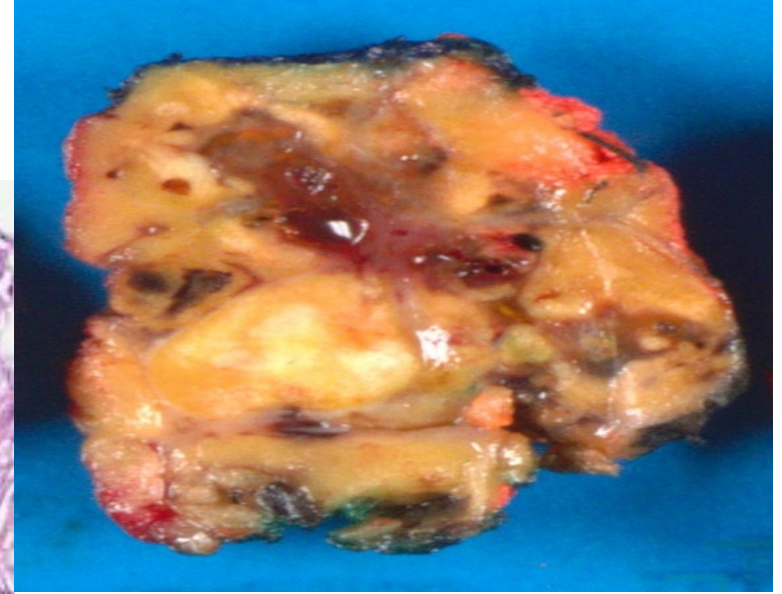
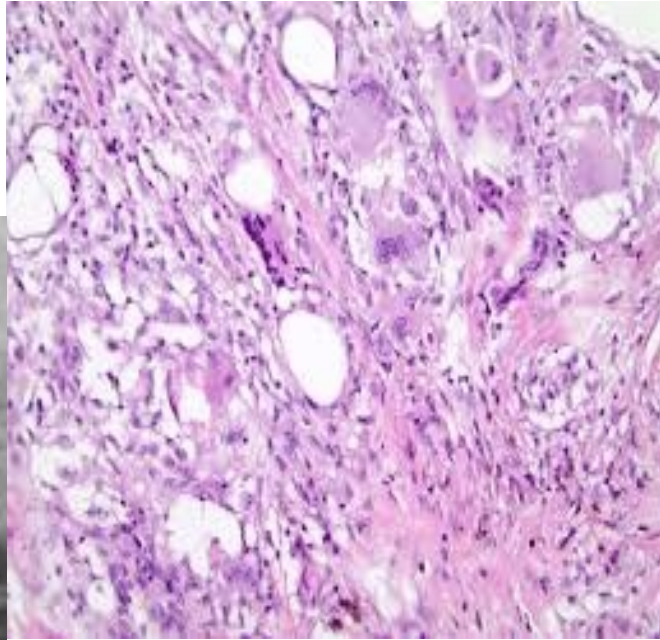
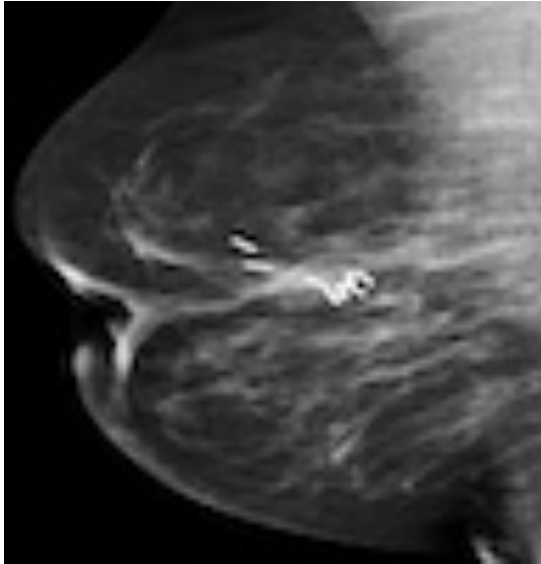
presence of fatty cell walled
organisms creates a cheese-like
consistency
e.g.) tuberculosis, fungi



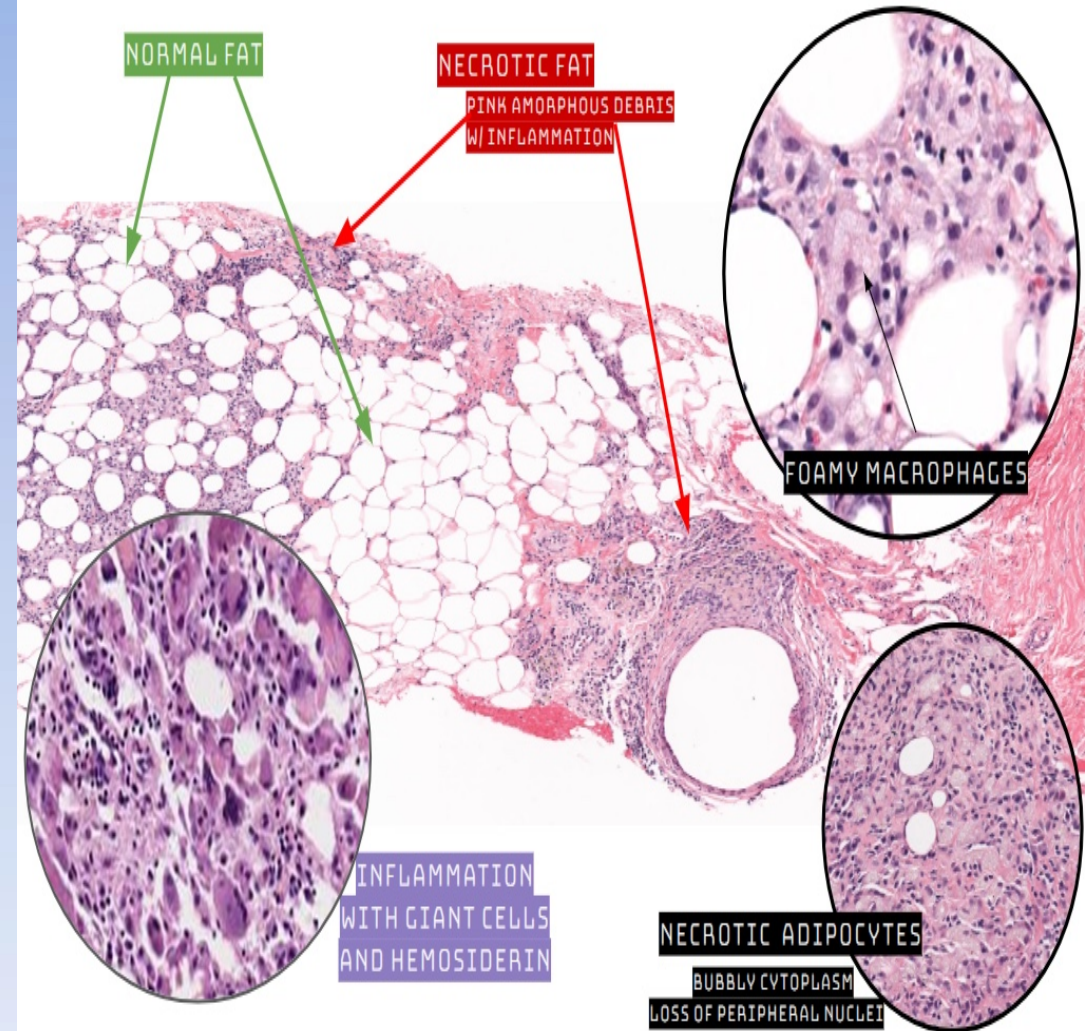


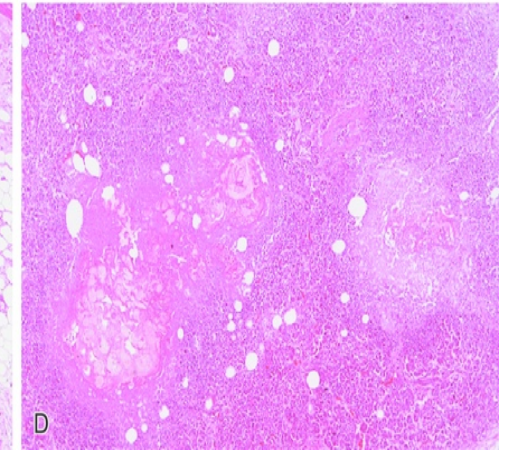
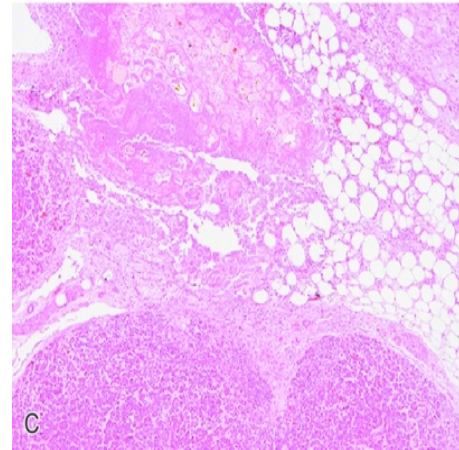
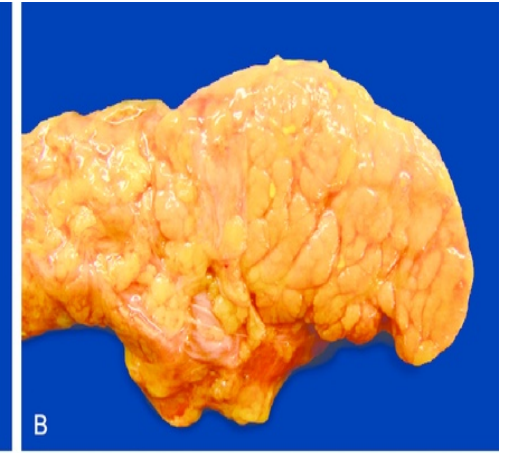
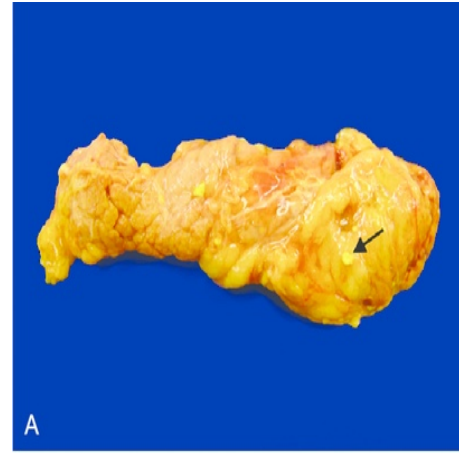


50 year old female complain from left breast mass after 6months history of trauma to her breast and after triple breast assessment(proper clinical examination ,radiological examination and histopathology biopsy), the histopathology showing in the below picture

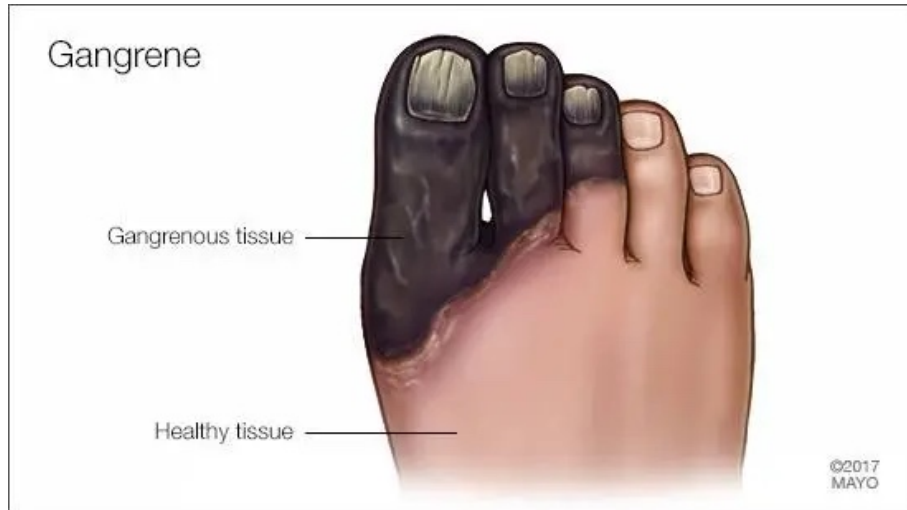


- **Fat necrosis:** It is specialized necrosis of fat tissue, resulting from the action of activated lipases on fatty tissues such as the pancreas, breast.
- In the pancreas it leads to acute pancreatitis, a condition where the pancreatic enzymes leak out into the peritoneal cavity, and liquefy the membrane by splitting the triglyceride esters into fatty acids through fat saponification.
- Calcium, magnesium or sodium may bind to these lesions to produce a chalky-white substance.

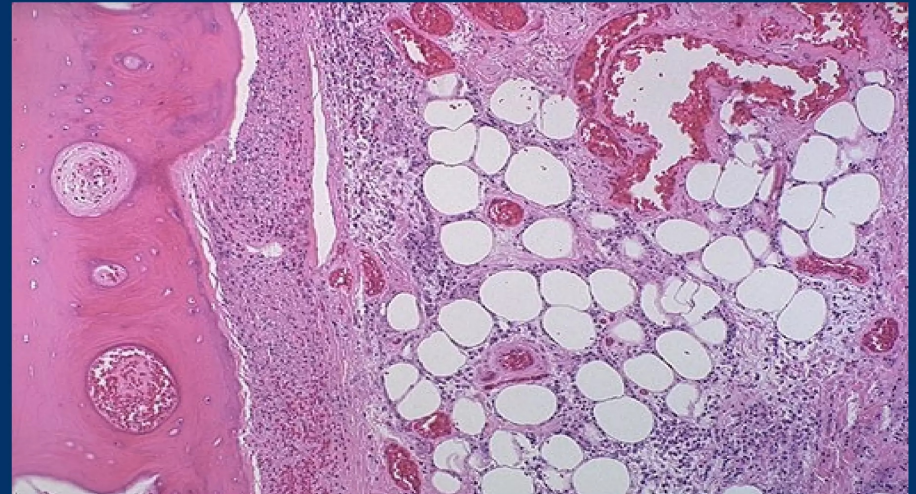




year old male presented to diabetic foot unit complain from black 50•
big toe as show in the below pictures



GANGRENOUS NECROSIS



- **Gangrenous necrosis:** It can be considered a **type of coagulative necrosis** that resembles mummified tissue.
- It is characteristic of ischemia of lower limb and the gastrointestinal tracts.
- **Types of gangrene:**
 - **Dry gangrene:** form of coagulative necrosis and due to peripheral artery disease. Occured in foot and toes.
 - **Wet gangrene:** tissue infected by m.or., cause swelling and foul odour, develop due to blockage of arterial blood flow. Occured in moist tissue such as mouth, cervix, lungs, diabetic foot, bed sore.
 - **Gas gangrene:** produced gas within tissue by bacterial infection. It is fatal.



DRY GANGRENE



GAS GANGRENE

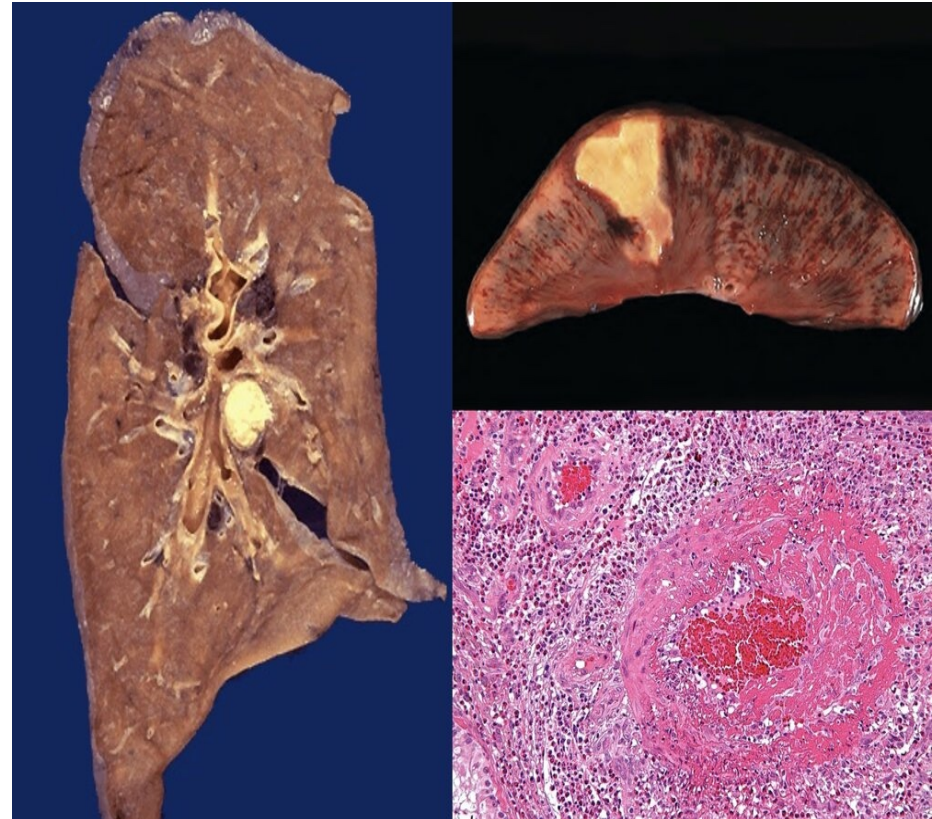


wet gangrene of the intestine



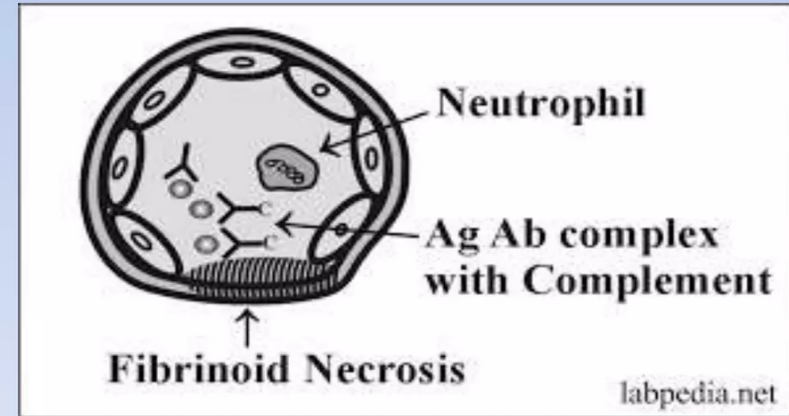
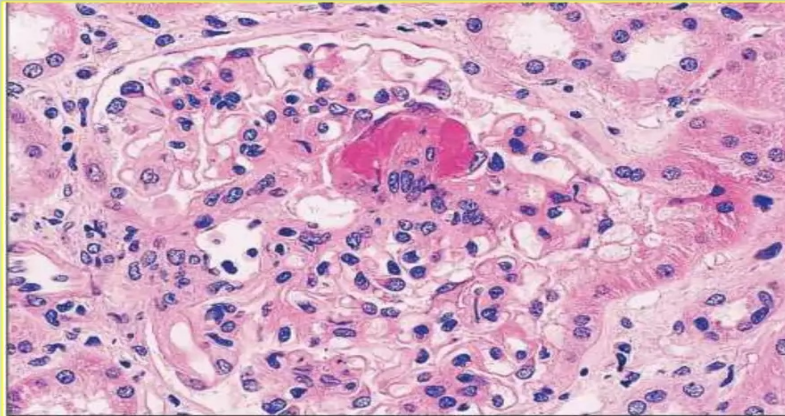
WET GANGRENE

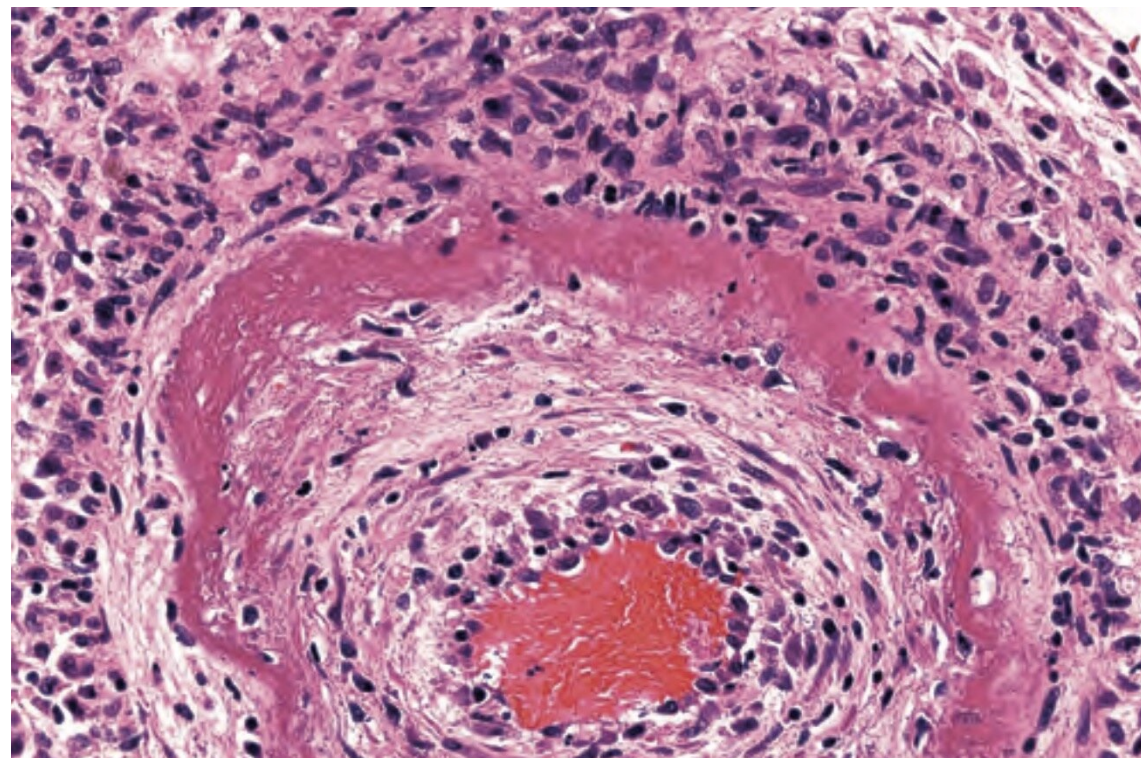
30 year old female complain from malar rash ,fever and joint pain ,she is passing out after 10 years of renal failure ,her organs as show in below pictures ,what is your diagnosis?



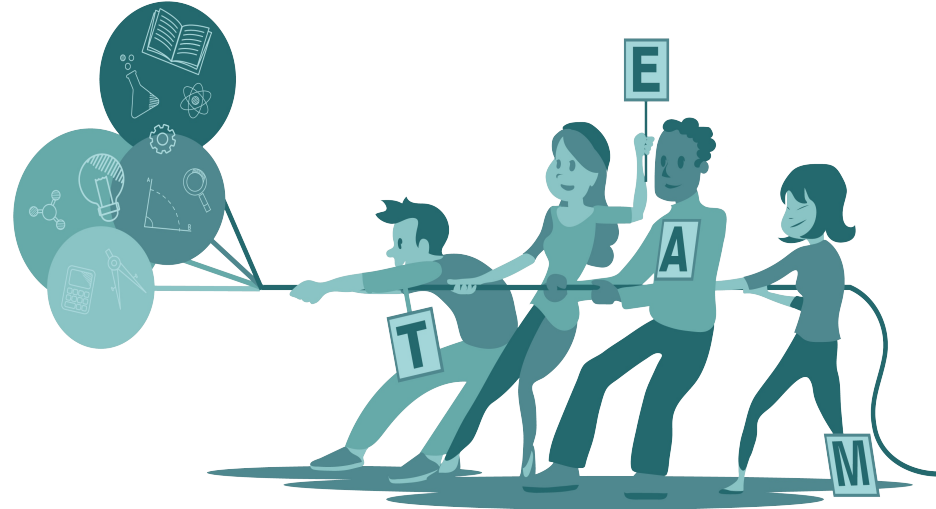
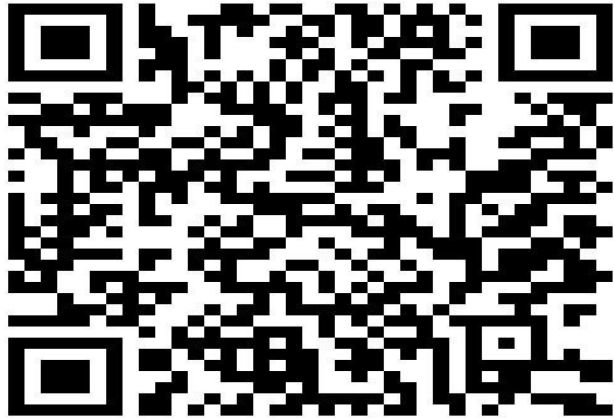
Fibrinoid necrosis: It is a special form of necrosis usually caused by immune-mediated vascular damage.

It is marked by complexes of antigen and antibodies, referred to as immune complexes deposited within arterial walls together with fibrin.





Team-based learning
from previous information can
you summarize clinical aspect of
necrosis



For more information about **Necrosis** see
.....below video by visit the following link

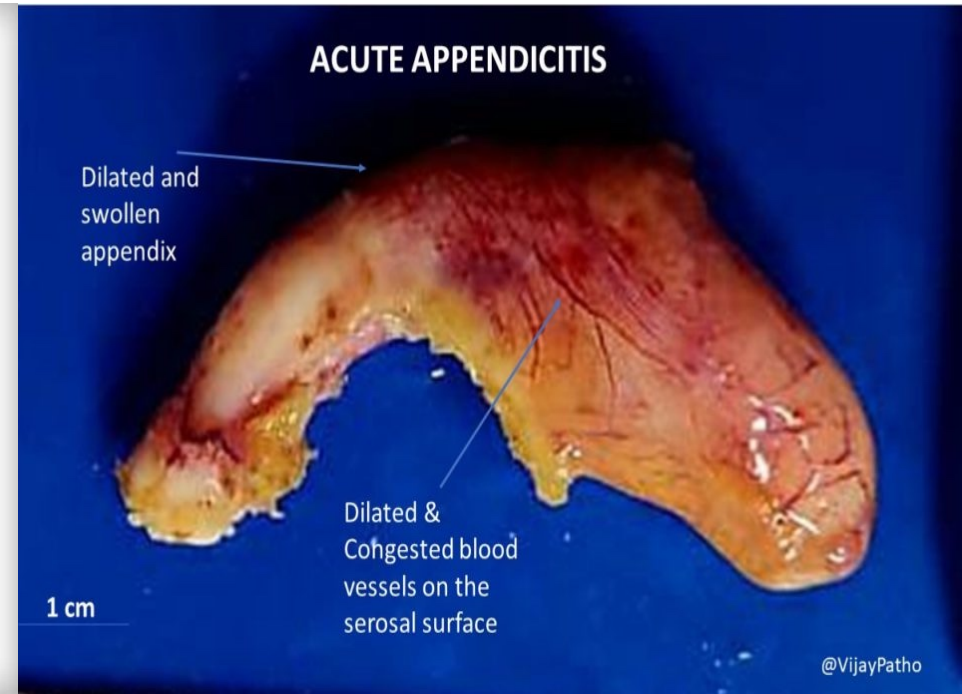
https://www.osmosis.org/video/Necrosis_and_apoptosis•

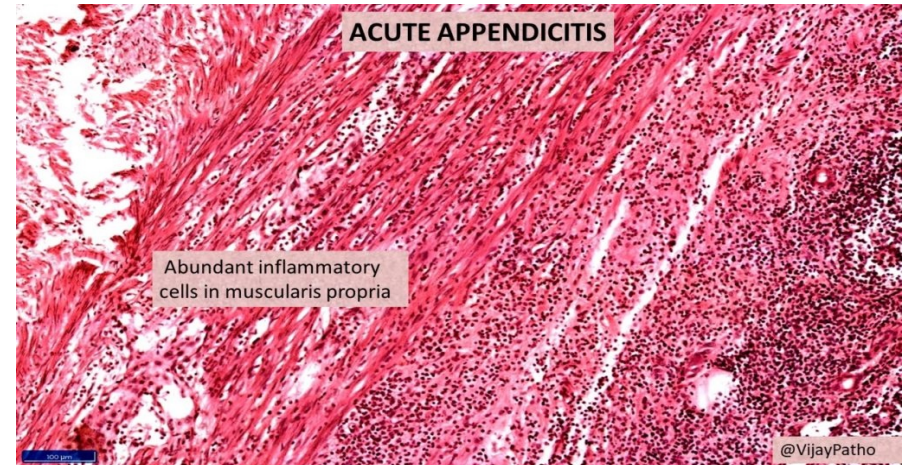
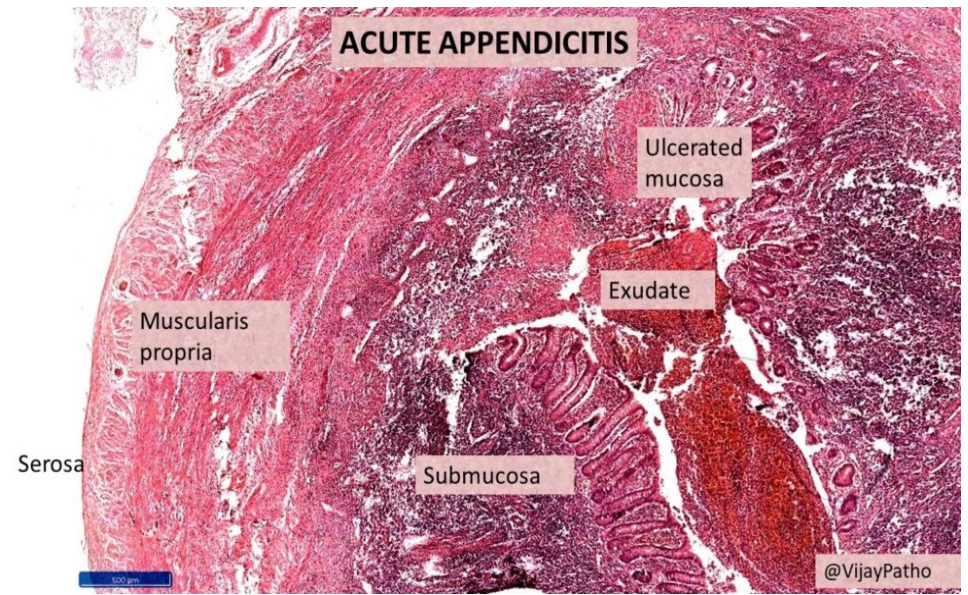
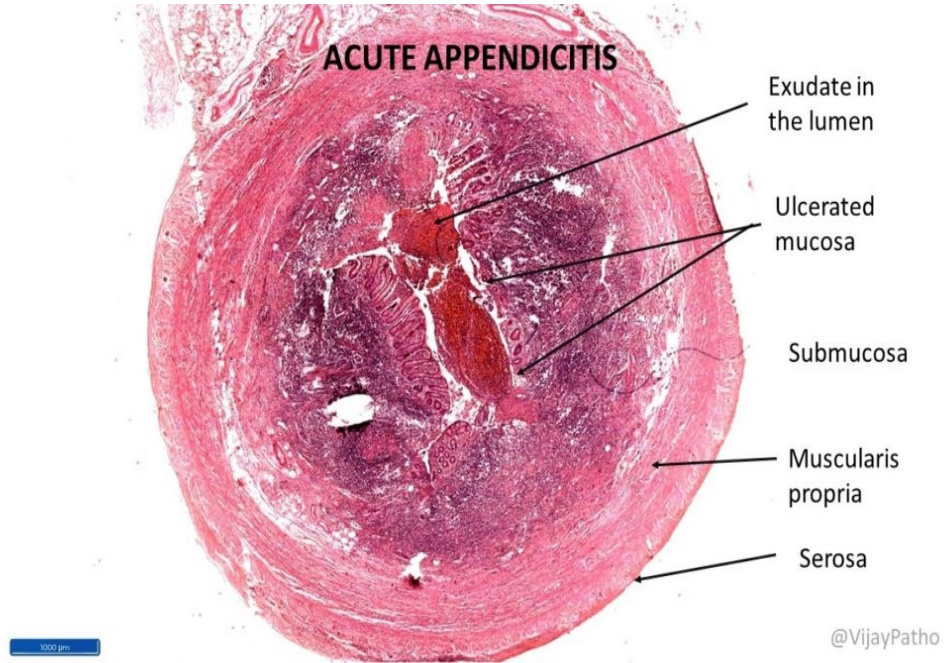


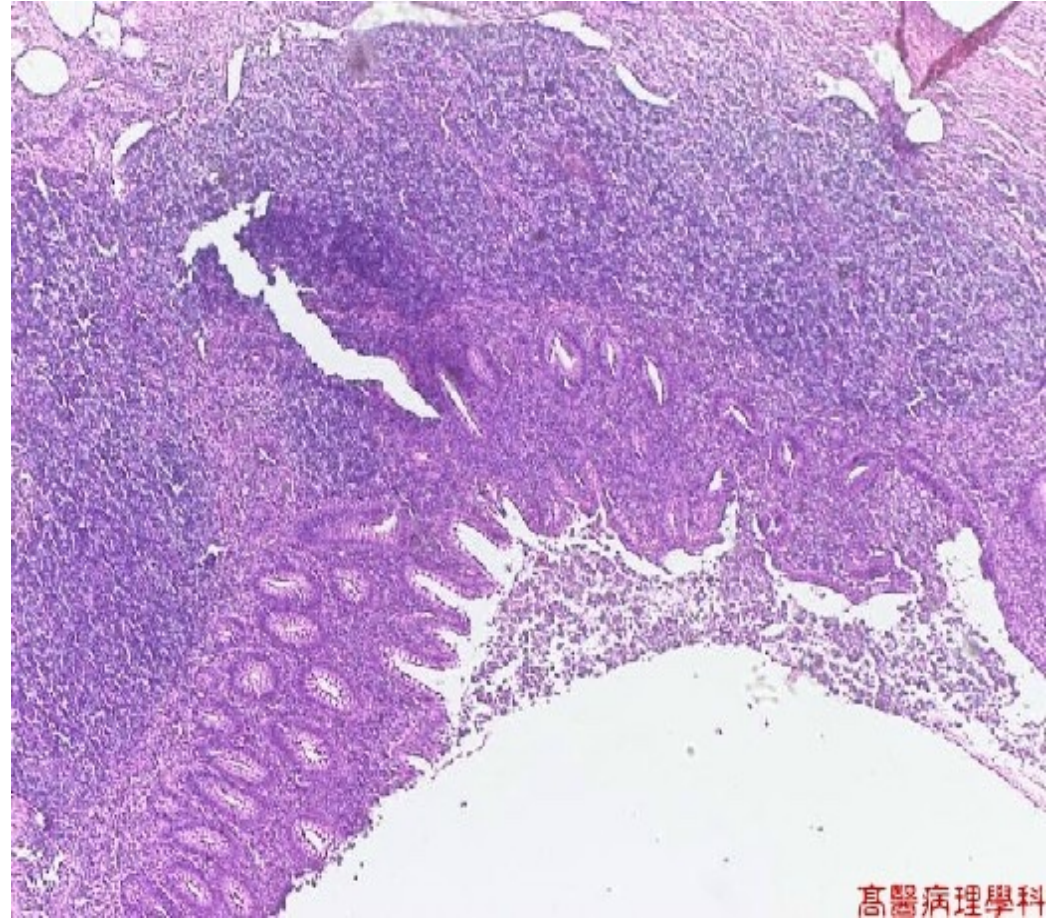
- What is your information about acute and **chronic inflammation**



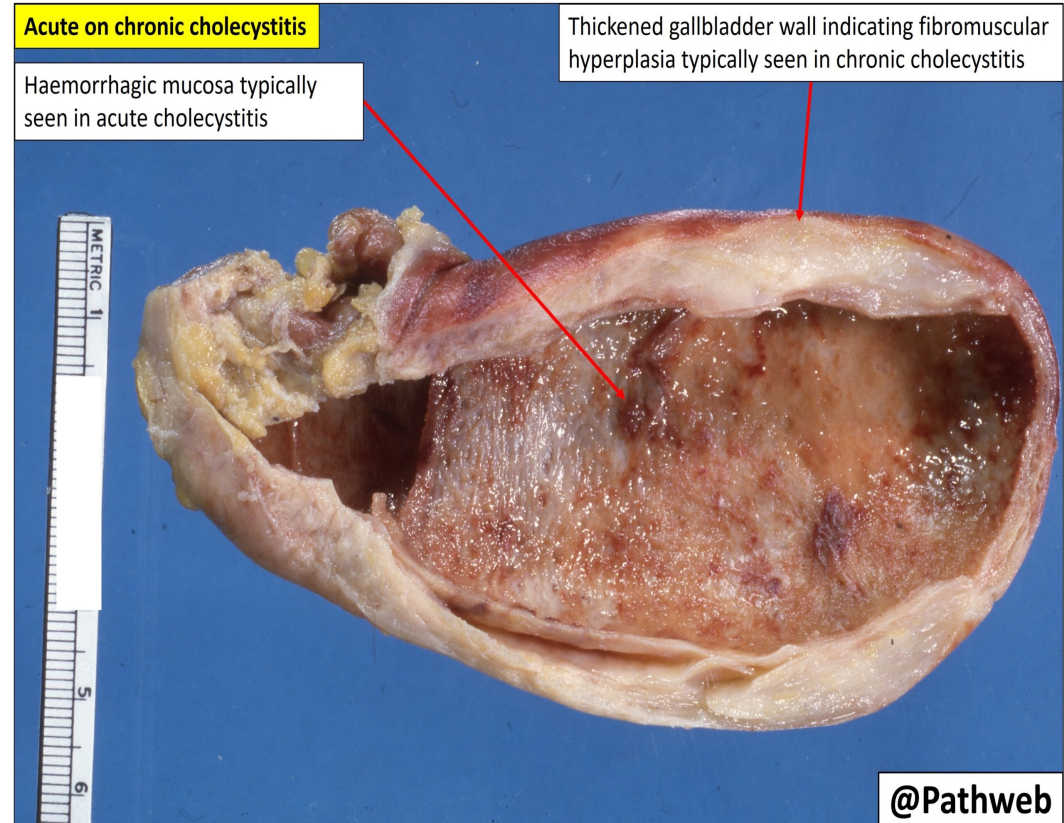
- 30year old male presented to emergency department complain from right lower abdominal quadrant pain not response for treatment ,the pain start within peri-umbilicus area and shifted to right lower abdominal area ,laproscopic surgery showing congested appendix ,what is your diagnosis.





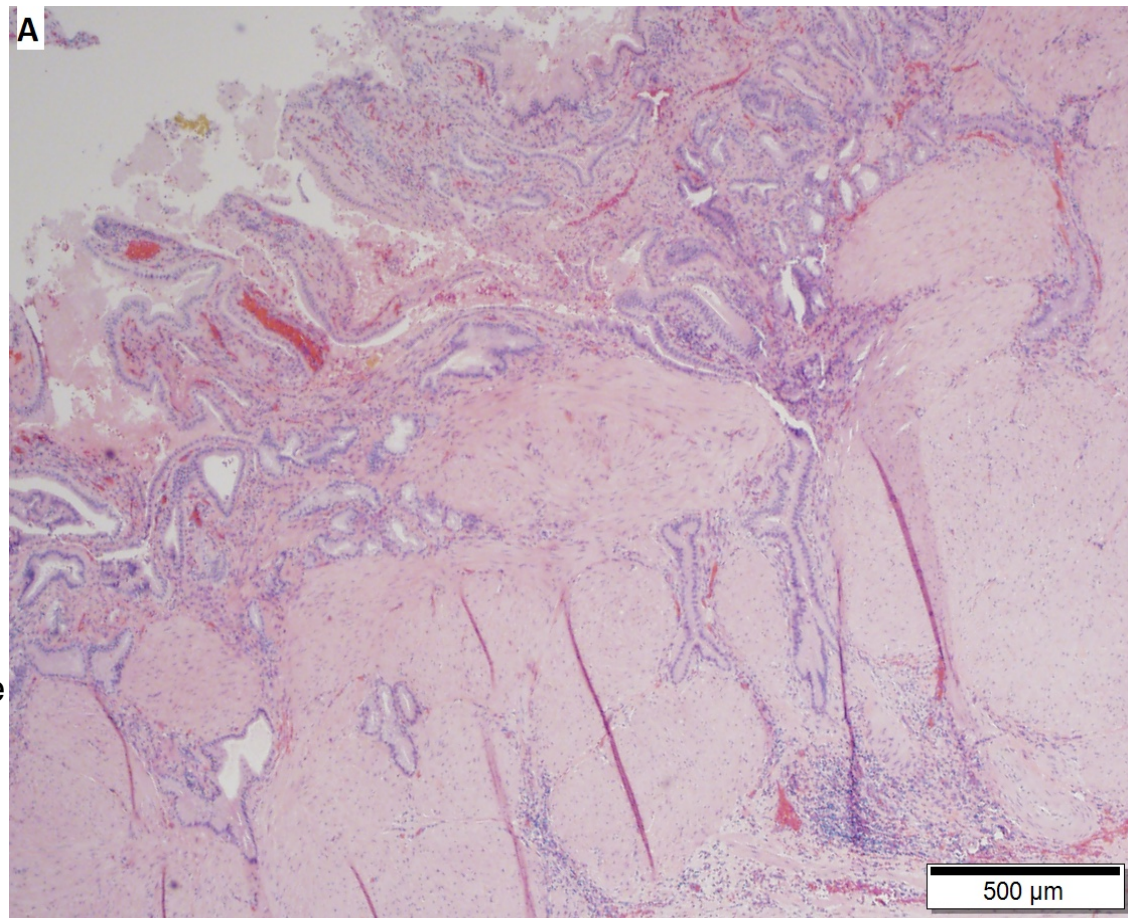


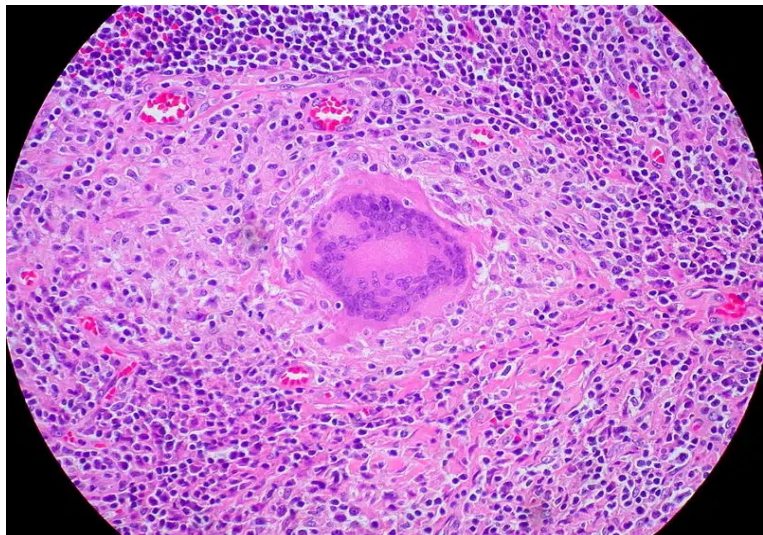
- 50 year female presented with chronic episode attack of right upper quadrant colicky abdominal pain ,laproscopic surgery show contracted gallbladder what is your diagnosis ?



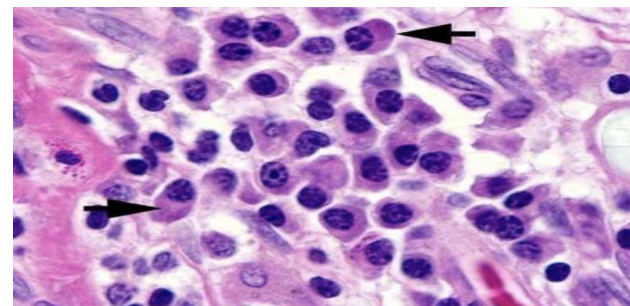
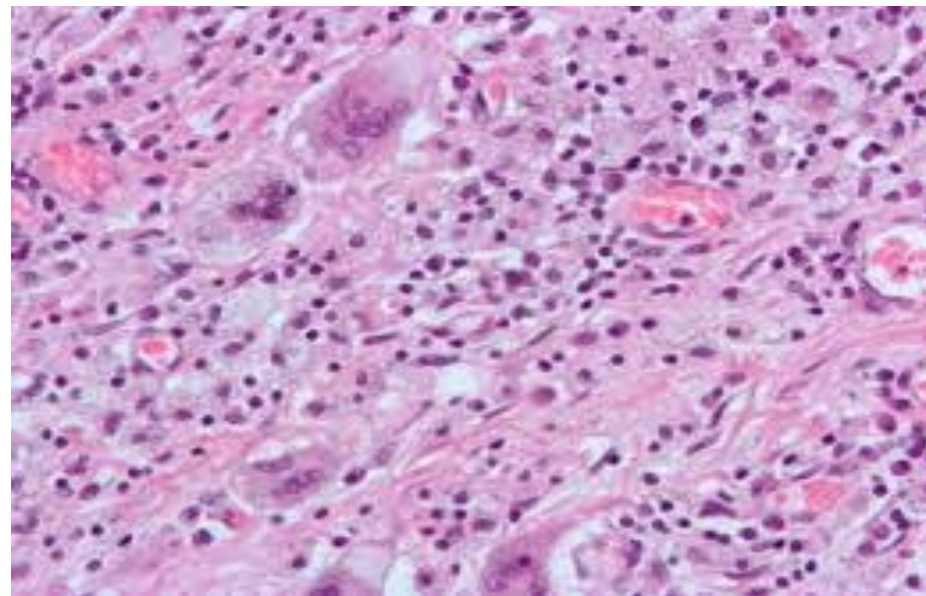
Microscopic
Features:[2]

Thickening of the gallbladder wall - due to fibrosis/muscular hypertrophy - key feature.
Chronic inflammatory cells - usu. "minimal".
Lymphocytes - most common.
Rokitansky-Aschoff sinuses - common.[3]
Entrapped epithelial crypts -- pockets of epithelium in the wall of the gallbladder.
+/-Foamy macrophages in the lamina propria (cholesterolosis of the gallbladder).

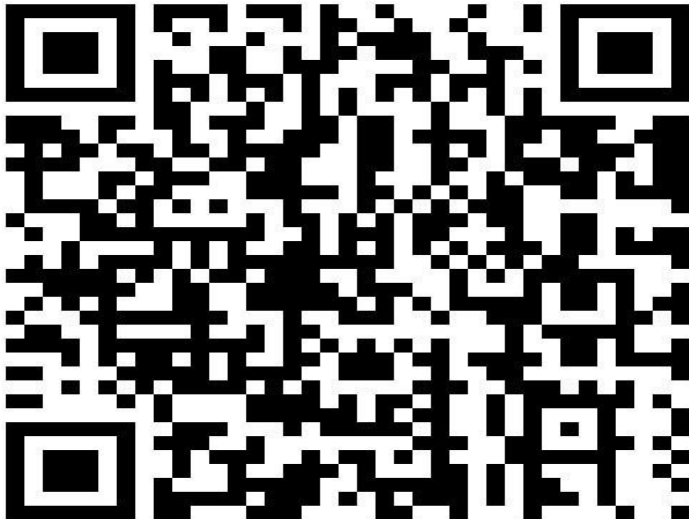




<https://classroom.google.com/c/NzIzMzQwNjIz/ODcx/p/NzE0OTY4NzE0MjEw/details>



- Team –based learning
- Summers microscopic features of acute and chronic inflammation

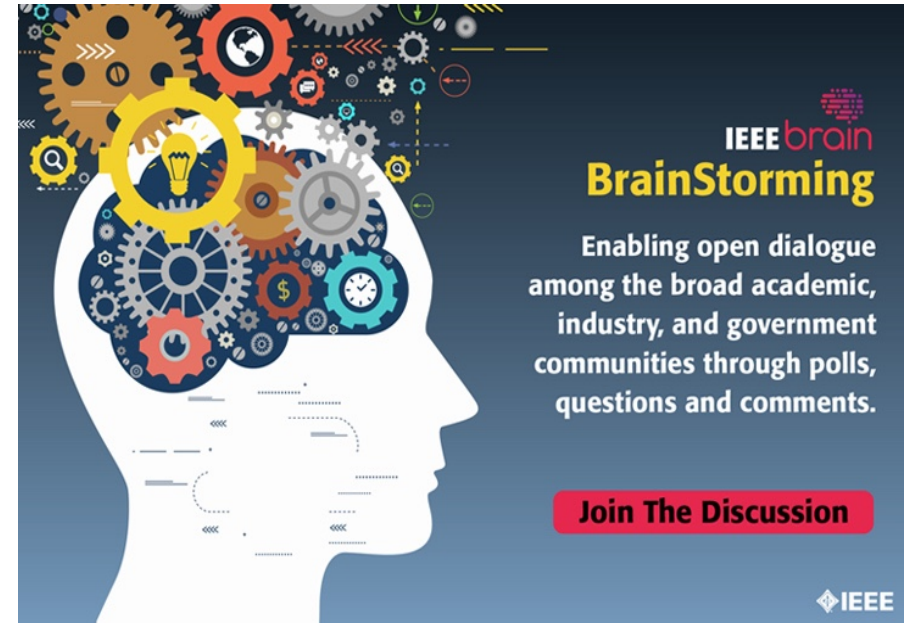


For more information about acute and chronic inflammation please visit below link

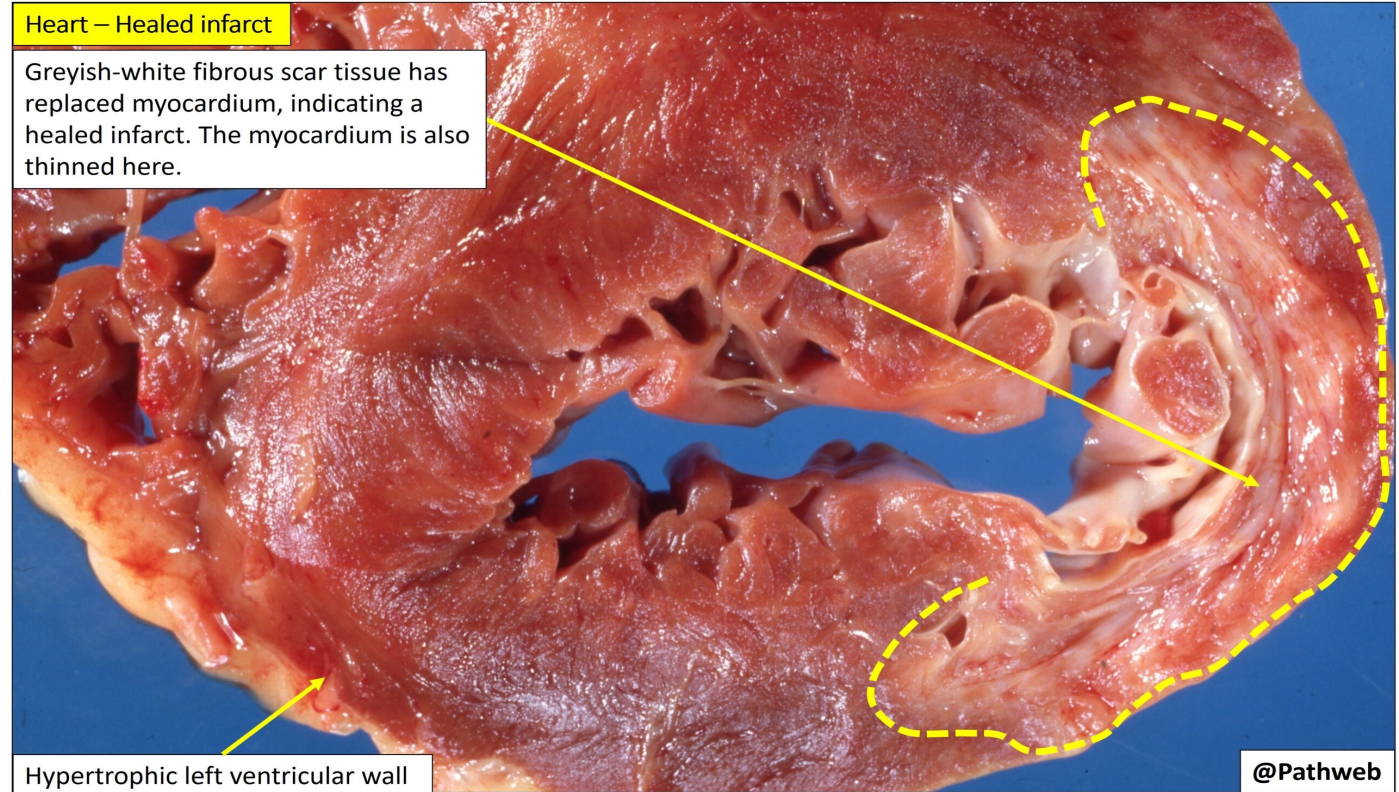
- https://www.youtube.com/watch?v=IfVVMIm_RKU



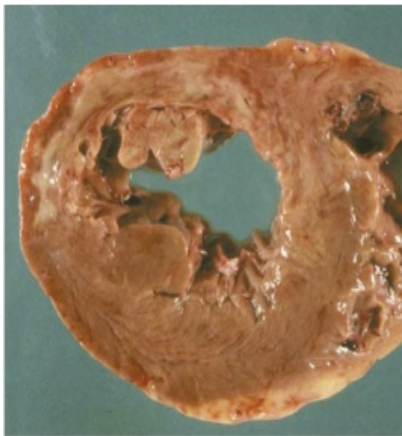
- What is your information of clinical aspect of repair and healing in different disease and treatment



- 50 year old male presented to emergency department with severe chest pain , ECG showing posterio-inferior Myocardial infraction ,elevated Troponin level within blood after proper treatment (Mona His Hepatitis B) his condition become stable and sending to home after 3 months he is died due to accident ,his heart showing as in below picture



Acute Coronary Occlusion



Myocardial Ischemia



Cell death

Apoptosis
Autophagy
Necrosis

Inflammation

Neoangiogenesis
Myofibroblast proliferation

Regeneration

Granulation tissue

Infarct Healing

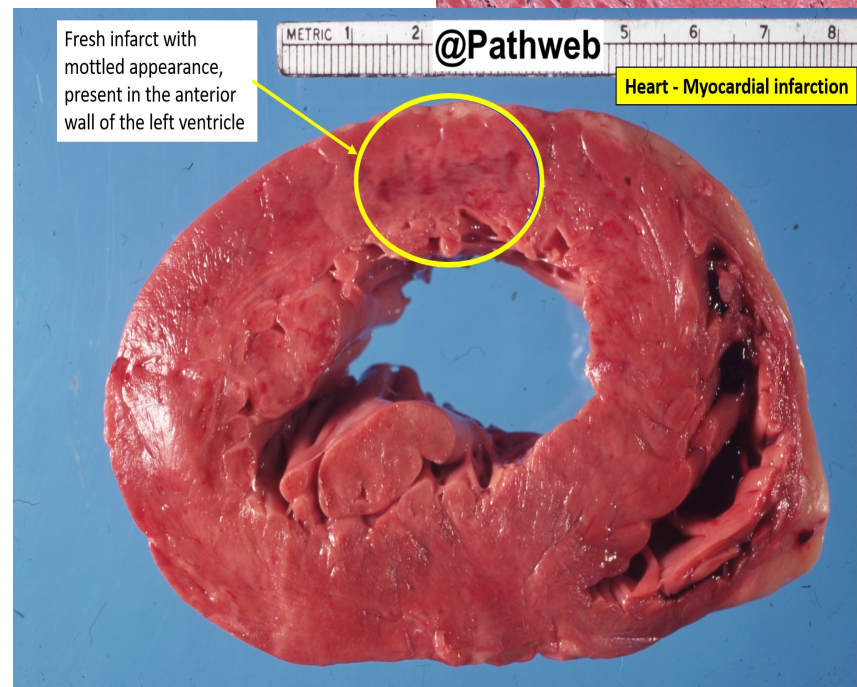
Scar formation



@Pathweb

Heart - Myocardial infarction

Fresh infarct with mottled appearance, present in the anterior wall of the left ventricle

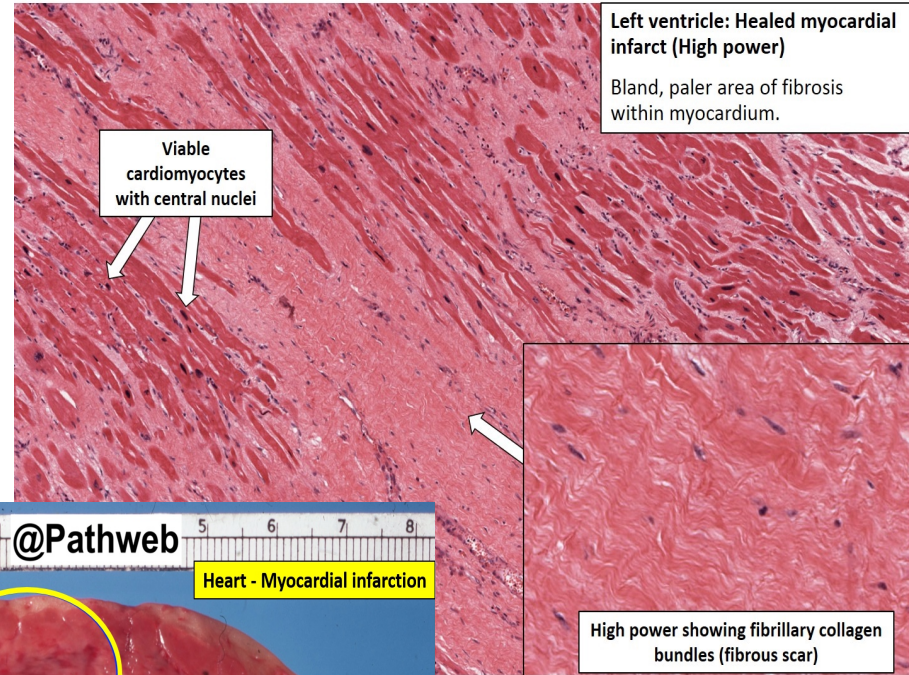


Viable cardiomyocytes with central nuclei

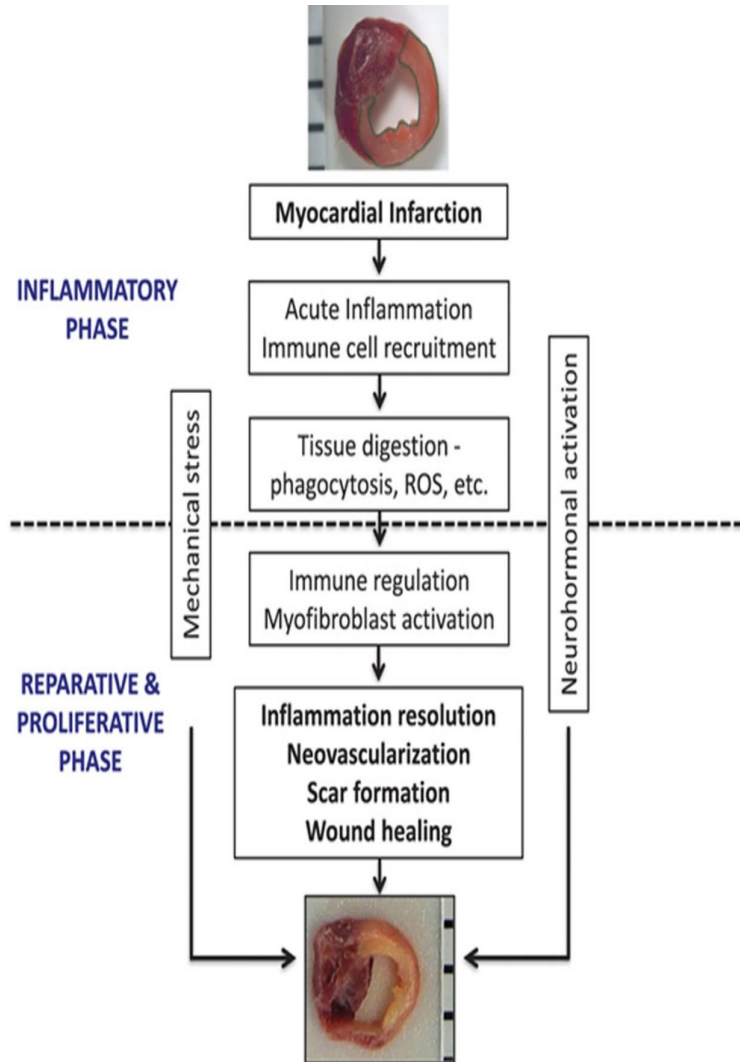


Left ventricle: Healed myocardial infarct (High power)

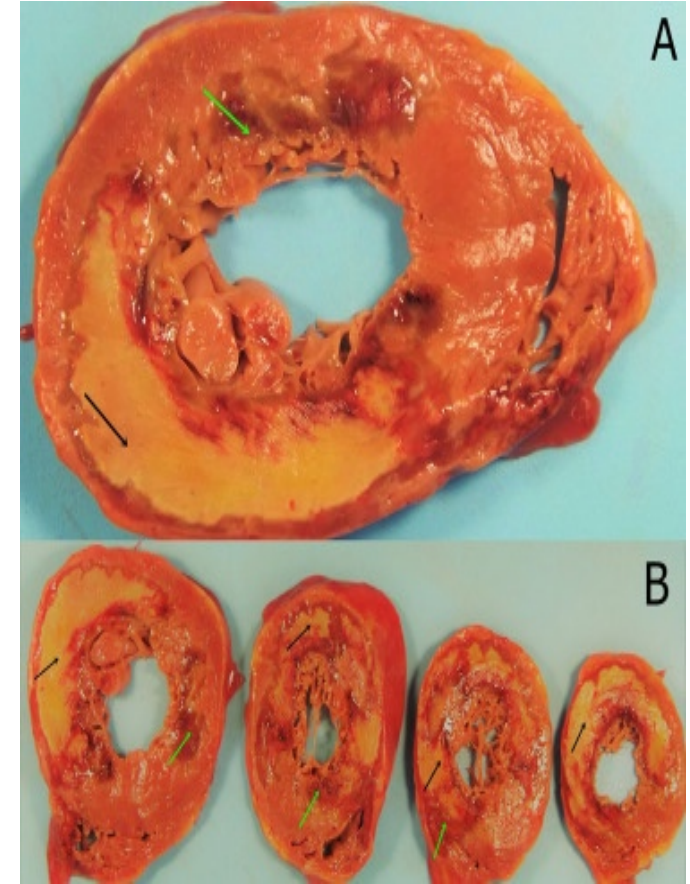
Bland, paler area of fibrosis within myocardium.



High power showing fibrillary collagen bundles (fibrous scar)

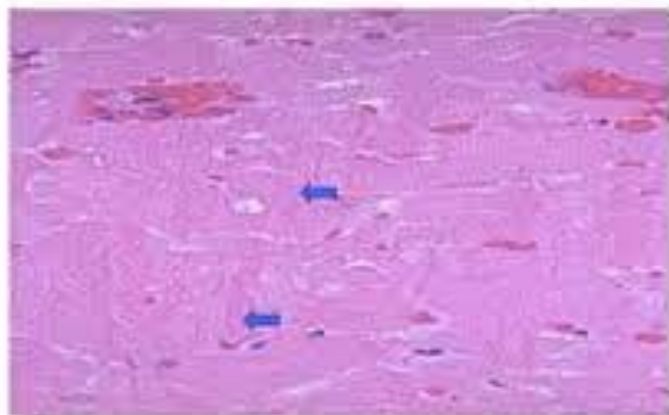


Figures 4. A and B, On gross examination, 2 lesions involving the myocardium and the papillary muscles are seen. Green arrows point to dark mottling, and the black arrows point to a yellow, softened lesion with red-tan borders; these correspond to a myocardial infarction in between 12 to 24 hours and 10 to 14 days, respectively.

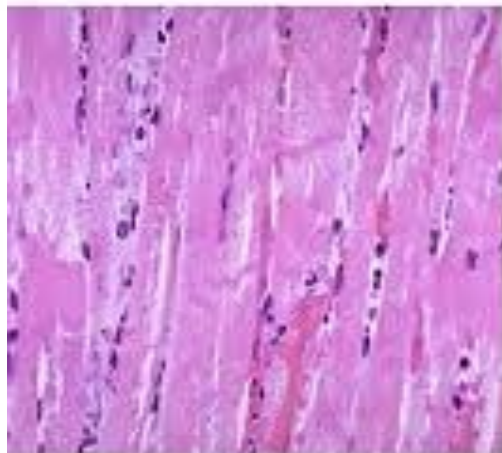




MI 18-24 hr loss of nucleus, contraction bands, coagulative necrosis.



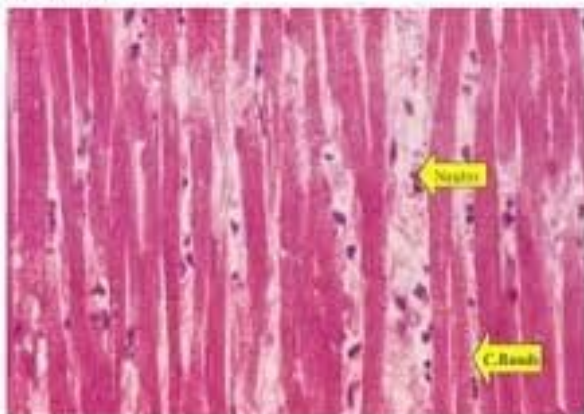
1-2 days old infarct.



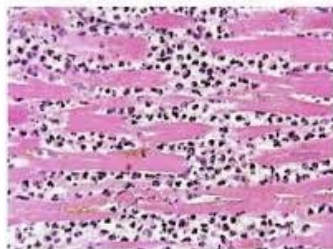
- The myocardial fibers have dark red **contraction bands** and their nuclei disappear.
- **Neutrophils** appear to show acute inflammation.
- Clinically, changes in the ECG and a rise in the MB fraction of creatine kinase can be seen in blood.



MI 1day loss of nucleus, contraction bands, few neutrophils.



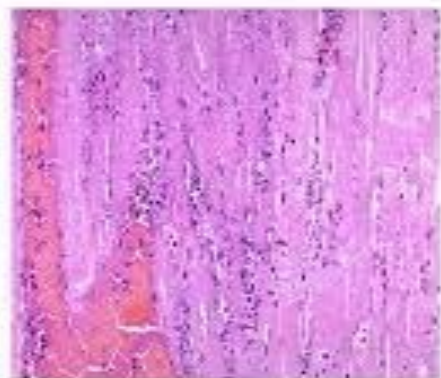
MI 1-3 day – Plenty of Neutrophils.



MI 2-3 day – Marginal inflammation.

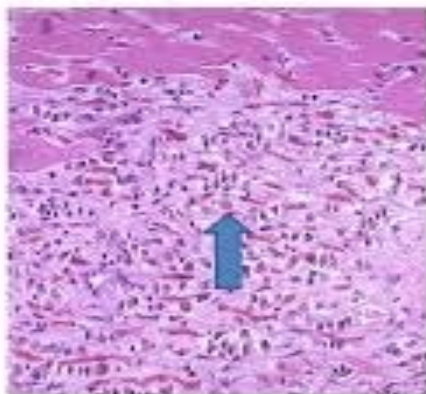


3-4 day old infarct.



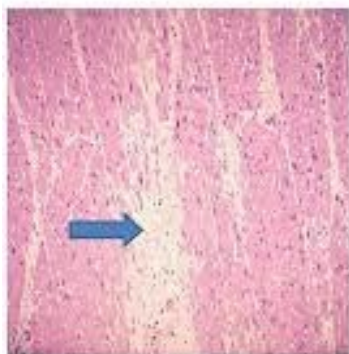
- There is an extensive **acute inflammatory infiltrate** and the myocardial fibers are so **necrotic** that the outlines of them are only barely visible.

7-14 days old infarct.



- Normal myocardial fibers at the top.
- Below these fibers macrophages have appeared along with numerous capillaries and collagen, collectively called **GRANULATION TISSUE**.

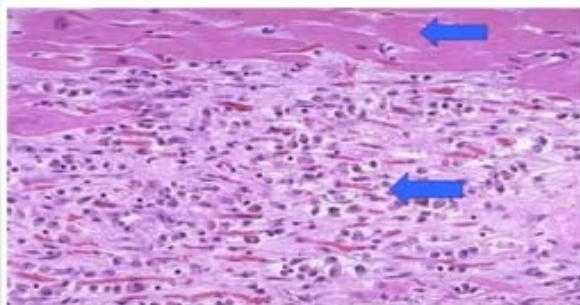
More than 2 week old .



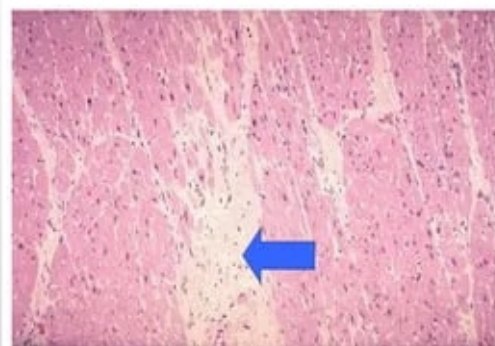
- There is pale white extensive **collagen** deposition within the interstitium between myocardial fibers.
- Inflammation has disappeared.



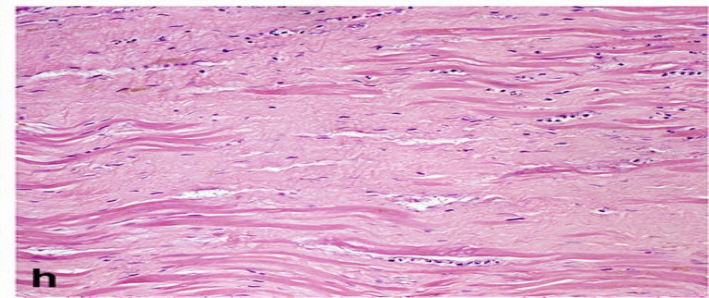
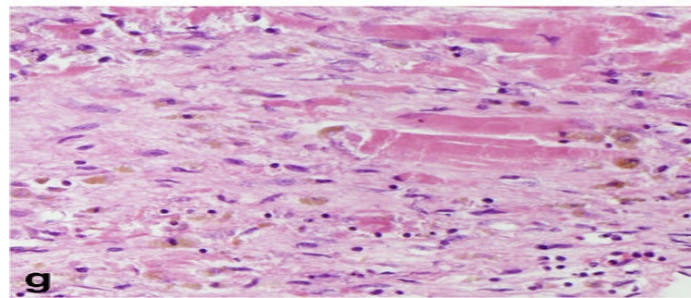
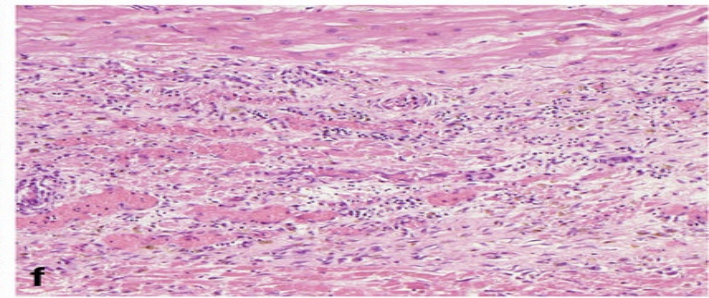
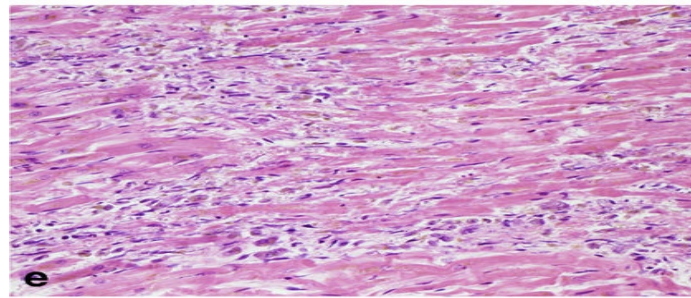
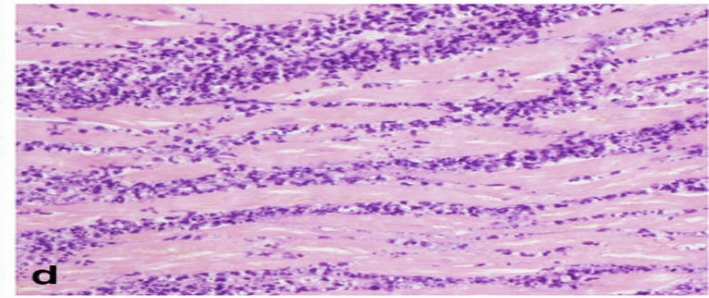
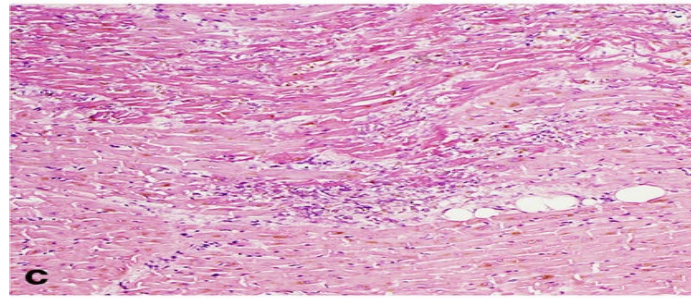
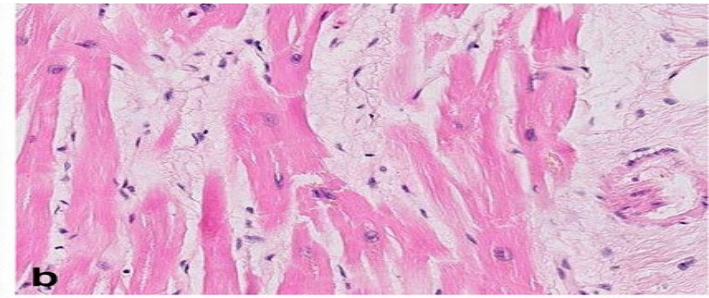
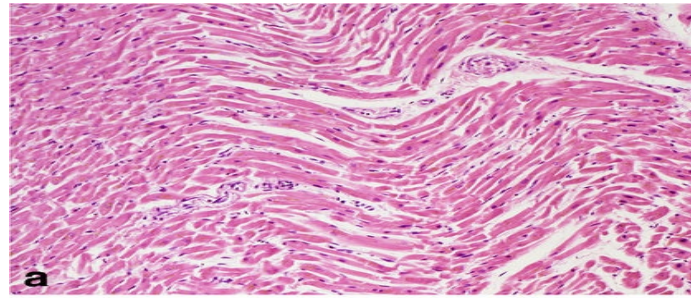
MI 1-3 wk — Granulation tissue, capillaries.

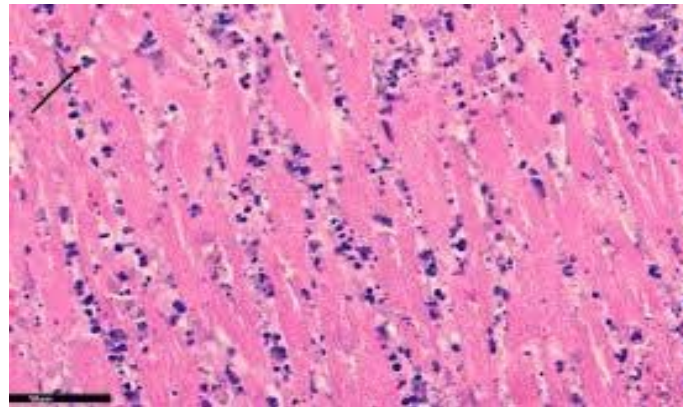
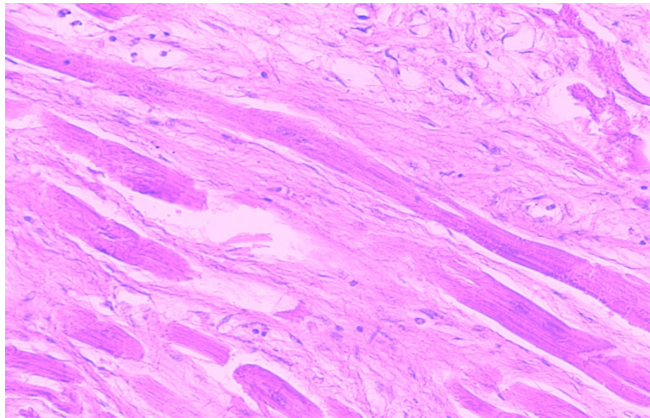
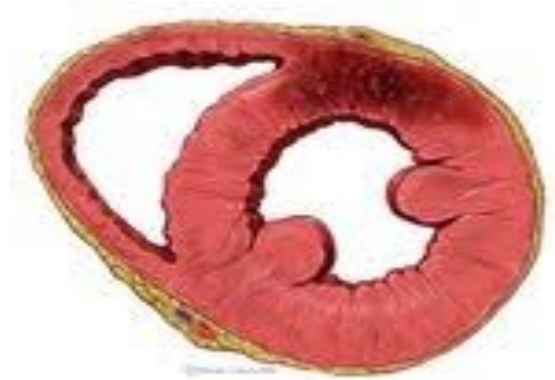


MI >6-Years - Collagen Scar no inflammation.



Histological features of MI at different stages, without reperfusion; **myofiber waviness (a)**; **interstitial oedema (b)**; hypereosinophilia and coagulative necrosis of cardiomyocytes (c); heavy granulocyte infiltration with karyorrhexis (d); macrophages and lymphocyte infiltration with early removal of necrotic debris (e); granulation tissue with formation of microvessels (f); fibroblast proliferation and early collagen deposition (g); dense fibrous scar replacing myocyte loss (h). All sections are stained with haematoxylin and eosin





Team –based learning

Discuss clinical aspect of healing
process of myocardial infraction
with anti-hypertensive
group(ARBs,ACE)



Team-Based
Learning™
Collaborative

For more information about repair by see video

- <https://www.youtube.com/watch?v=t-5EjIS6qjk>



Take Home message

- 1 .Apoptosis ,necrosis , inflammation , healing and repair process all of these mechanism play vital role in our body to prevent different complications.
- 2.Basic information about Apoptosis, Necrosis , inflammation ,healing and repair must be correlate with **clinical information for proper teaching approach** .



Evaluate and create please

- What is your feedback about this leature



استبيان



Reference

1. <https://www.pathologyoutlines.com>
2. https://books.google.com/books/about/Robbins_Basic_Pathology_E_Book.html?id=jheBzf17C7YC

