



Pathology

Subject :

Lec no : 11

Done By : Shahd Abu-Tariah

وَقُلْ رَبِّ زِدْنِي عِلْمًا

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طباعة المحاضرة : شهيد أبو هريرة

تجدون في guidance مادة الباثو على موقع النادي :

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ROBBINS

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شرح المادة

يوجد شرح للمادة كاملة من إرشيف دةمة آثو ، مع العلم ان الوحدة الثالثة كانت تغطي من قبل الدكتور غادة

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شرح قديم للفريق العلمي

تفاريغ

يمكن الاستفادة من تفاريغ الدقع السابقة

ATHAR PATHOLOGY NOTES

VEIN PATHO. NOTES

تفاريغ دقعتي اثر و وريد قويات جدا

QUIZZES

كويزات للدكاترة

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للوصل الى guidance الباثو و تفاريغ
المادة كاملة :



كل اعمال الفريق العلمي تنشر على قناة
التيليغرام



MORPHOLOGIC PATTERNS OF ACUTE

INFLAMMATION: There are 4 patterns.

1. Serous inflammation.
2. Fibrous inflammation.
3. Suppurative inflammation.
4. Membranous or pseudomembranous

Will be affected by :

- A. The **cause** of inflammation .
- B. **severity** of inflammation . **mild or severe**
- C. the **type of tissue involved**, can all modify the basic morphologic patterns of acute inflammation, producing distinctive appearances.

1- Serous inflammation :

This is characterized by outpouring of thin watery fluid called effusion which is protein – poor fluid that is either derived from the blood^① (serum) or^② the secretion of mesothelial cells^a of pleura , peritoneum^b , pericardium^c or the synovial cells^d lining the joint spaces.

This serous fluid accumulates in body cavities as seen in TB infection ..

Skin blister that results from burn or viral infection is also an example of serous inflammation.

Transudate is a clear serous fluid that has low protein content, low specific gravity and a low cellular content

Effusion is the **accumulation** of fluid.

Serous fluid is a clear, serum-like fluid containing small amounts of protein which occurs in acute stages of inflammation.

Serous fluid has low gravity, light weight, clear color, and low protein content.

Its color is light yellow and has a low specific gravity due to the low protein and cells content.

Q. What is the first response in acute inflammation?

Ans: **Homeostasis**.



Serous drainage

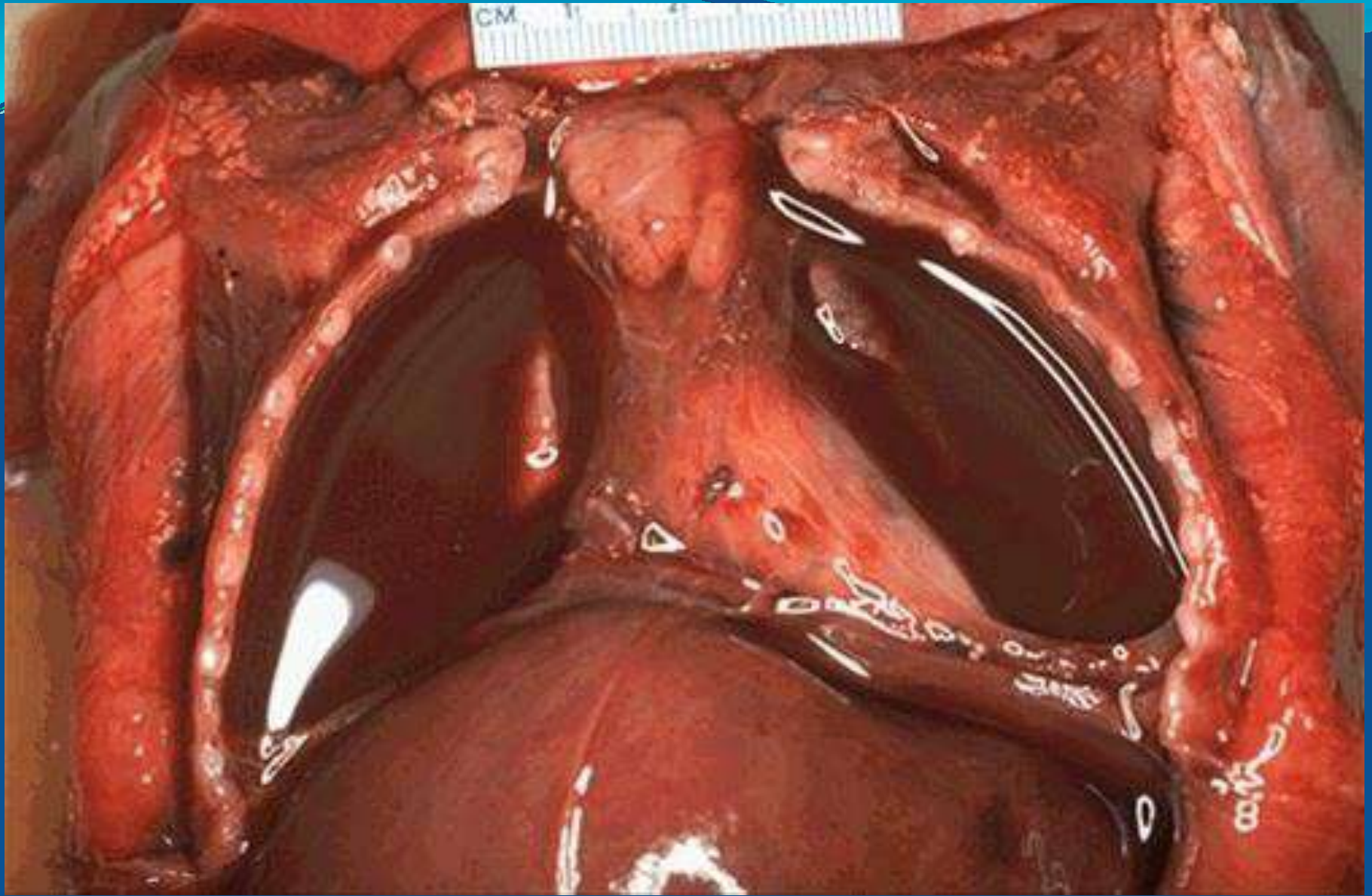
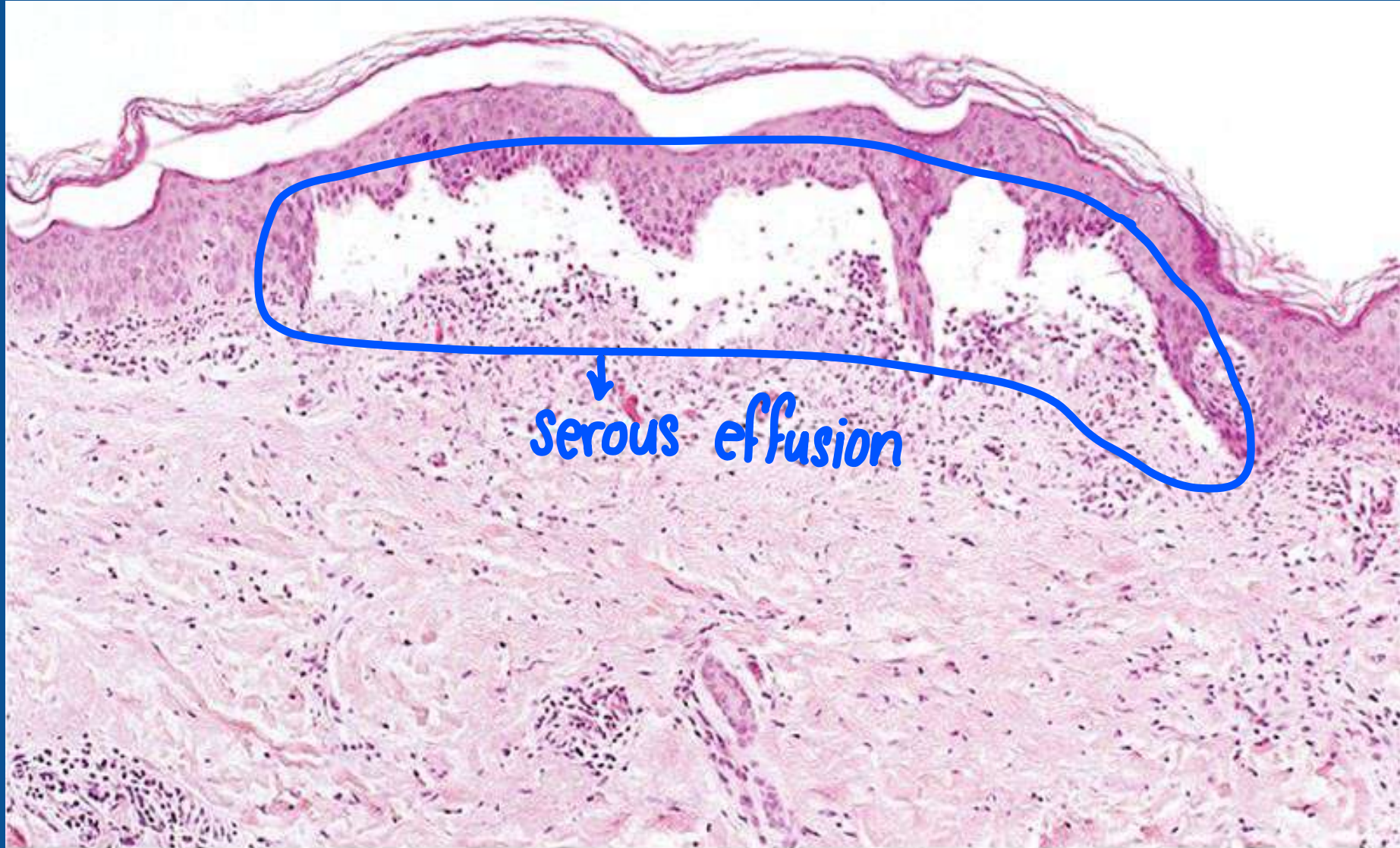


Figure 25 : Gross view of bilateral pleural clear serous fluid , example of serous inflammation .



Figure 26 : Serous inflammation of skin in burn showing bullae filled with serous fluid .

F 27 : Serous inflammation: Subepidermal bullous. The epidermis is separated from the dermis by a focal collection of serous effusion.



2- Fibrinous inflammation :

In this type of inflammation there is exudation of large amount of plasma proteins including fibrinogen with subsequent precipitation of masses of fibrin.

This is characteristic of certain sever inflammatory responses ^{why} increasing vascular permeability ^{to} allow larger molecules in blood like fibrinogen to pass the endothelial barrier with subsequent precipitation of masses of fibrin.

The accumulated extravascular fibrin appear as an eosinophilic mesh-work of threads on linings of body cavities or on meninges .

Such fibrin may be degraded by fibrinolysis and the debris removed by macrophages resulting in resolution.

يعني يتم التخلص من هذه المشكلة
بالكامل.

In severe inflammation, vascular permeability increases

(نفاذية جدار الوعاء الدموي تزيد).

زيادة النفاذية تؤدي إلى مرور جزيئات كبيرة في الدم كال **fibrinogen** الذي سيتحول إلى **fibrin** الذي يسبب تخثر (clotting) في الدم.

تجمع ال **fibrin** رح يكون على شكل شبكة (mesh-work of threads) موجودة بال **linings of body cavities** أو بال **meninges**.

ال **fibrin** يتم تحطيمه عن طريق ال **fibrinolysis** و يتم ازالة بقايا ال **fibrin** من قِبَل ال **macrophages** و ينتج عنه زهاب التخثر.

Sometimes the inflammation can be too severe to the point where macrophages can't get rid of the large amounts of fibrin. This causes the conversion of fibrin to **fibrous tissue.**

يعني الالتهاب قد يكون شديد جدا لدرجة ال **macrophages** ما رح يقدرُوا يتخلصوا من الكميات الهائلة من ال **fibrin** مما يؤدي إلى تحول ال **fibrin** إلى **fibrous tissue**.

In **rheumatic pericarditis** , the pericardial space may become filled with large masses of fibrin , when the epicardium is stripped from the pericardium, the rubbery adherent fibrin coats **both surfaces** and simulating the appearance of **bread and butter** .

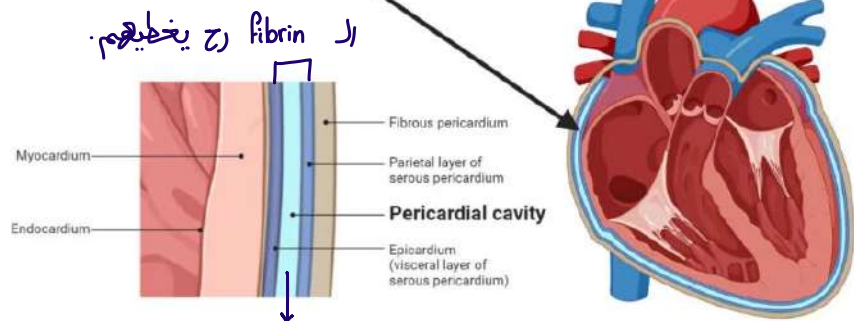
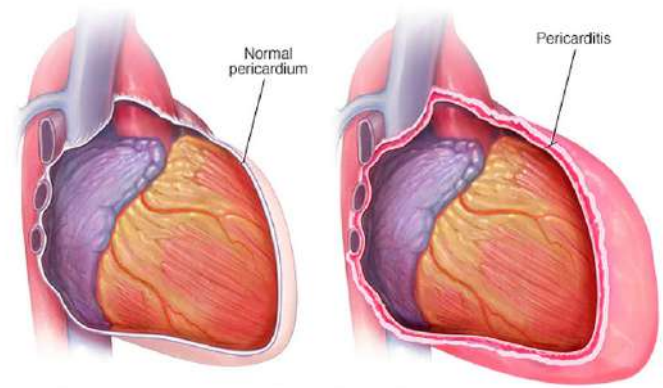
Parietal layer + visceral layer (epicardium)

Organization of fibrinous exudates by formation of new capillaries with fibroblasts leading to **scarring** and consequently obliterate the **pericardial cavity** .

* في حال الالتهاب الشديد، رح يكون حبيب على الجسم التخلص من الـ fibrin ما يؤدي إلى قوله إلى fibrous tissue و بعد رح يهبر في Scarring و الـ pericardial cavity رح يختفي.

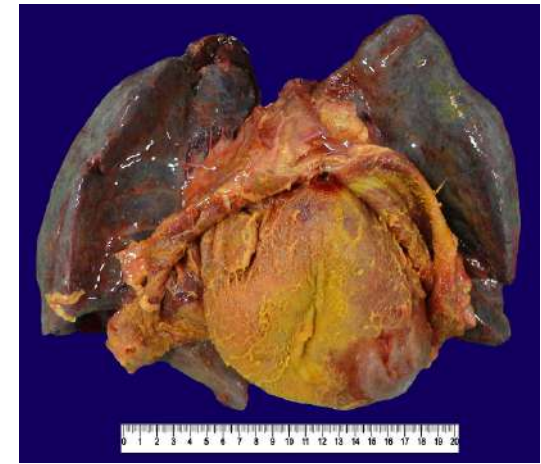
حجرة تنوعية لـ pericardium .

Pericardium

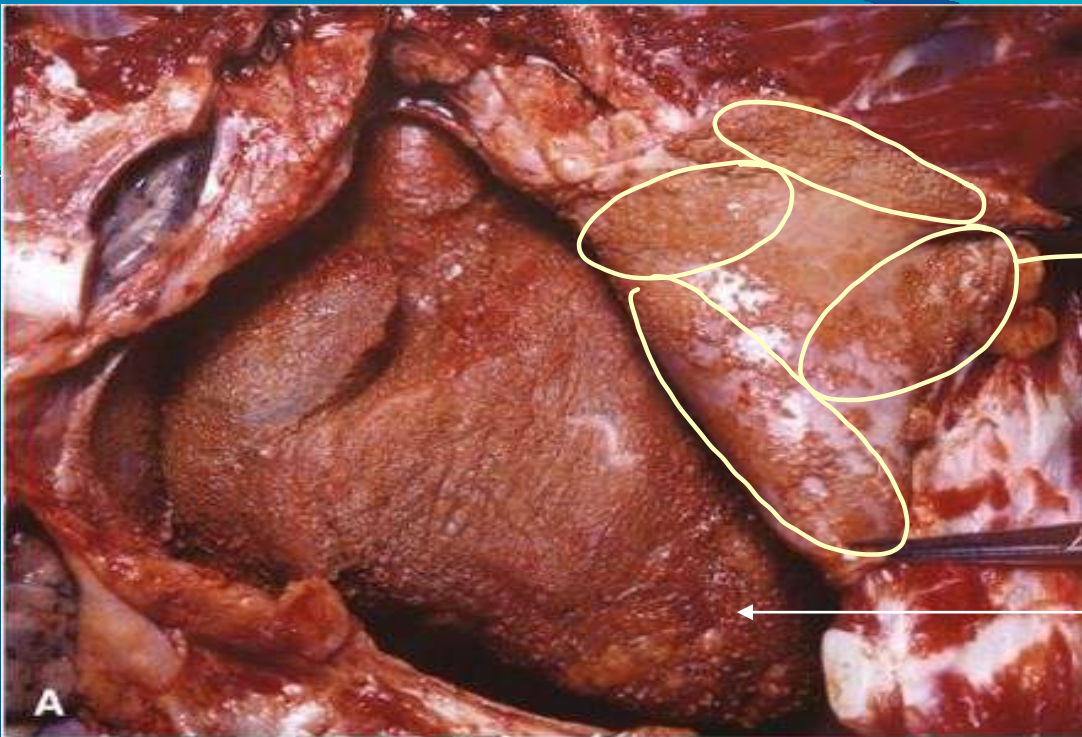


فبرين راح يخطيهم
صوت راح يكون
فبرين راح

* اسفة عالمقر.

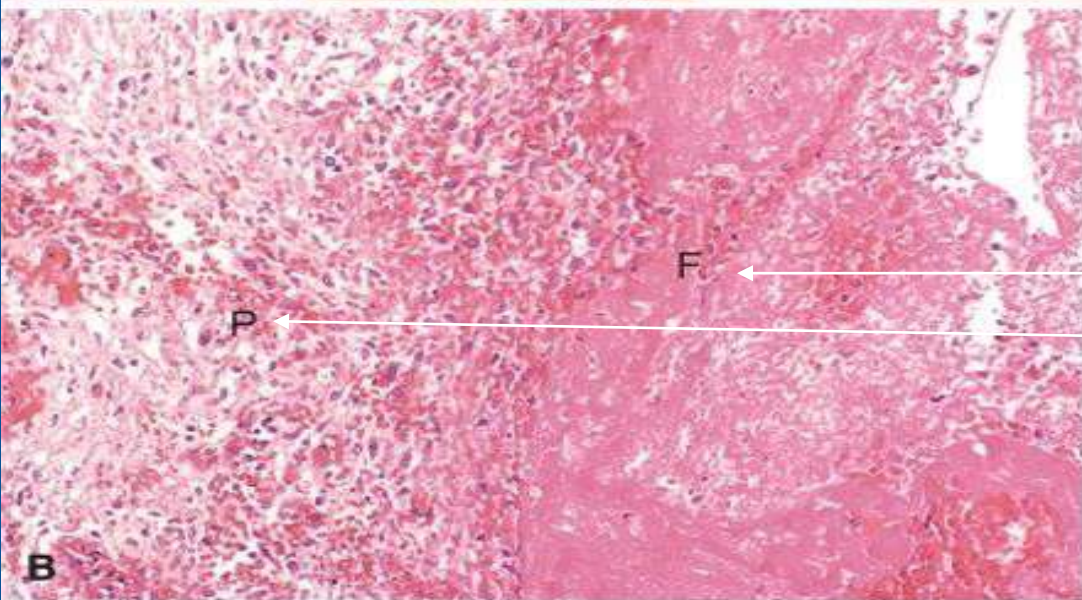


هيك راح يكون شكل القلب بعد ما راح يغطي الـ pericardium
"Bread & butter" appearance.

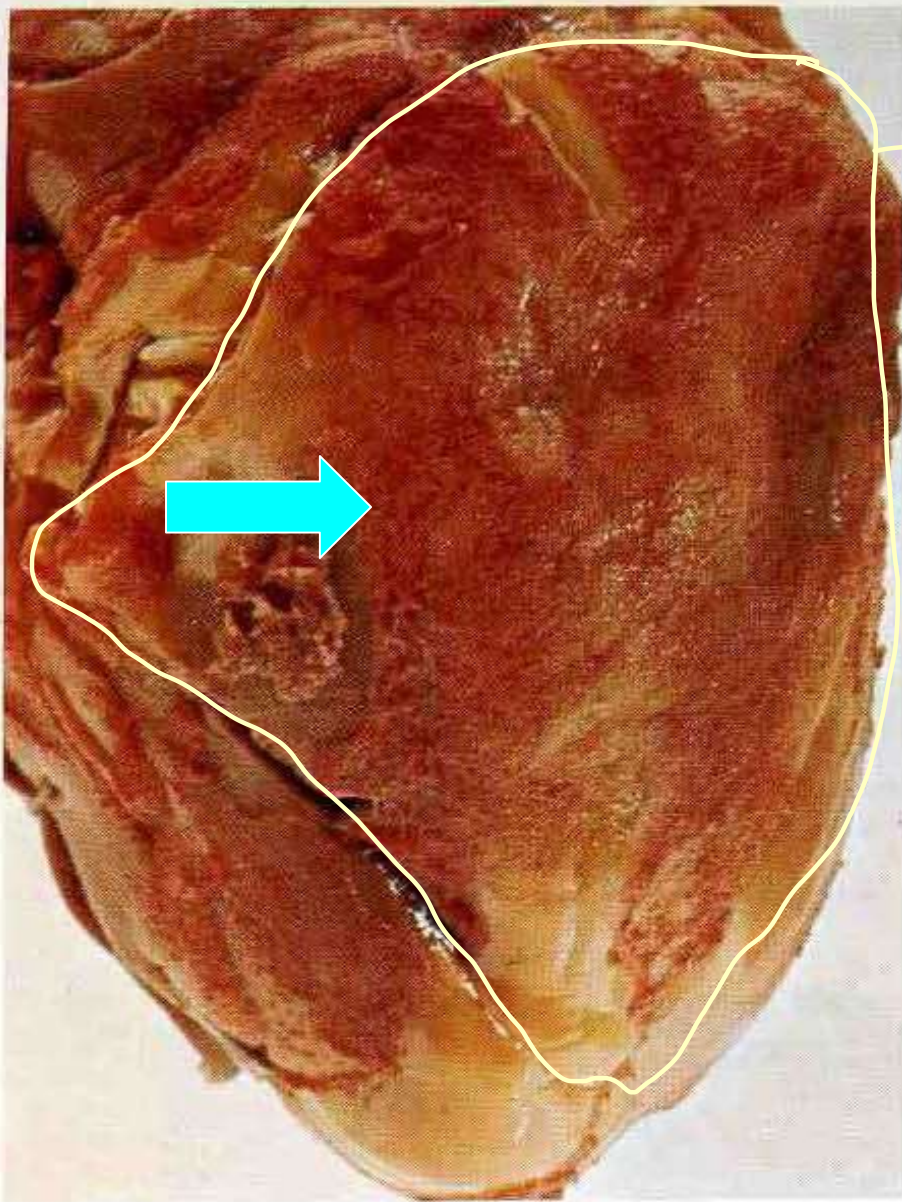


→ mesh-work of fibrin exudate

F 28 : Fibrinous pericarditis. A, Deposits of fibrin on the pericardium.



B, Pink meshwork of fibrin exudate (F) overlies pericardial surface (P).



→ epicardium.

F 29 : Acute Fibrinous pericarditis.

The epicardial surface of the heart is covered with a fibrinous exudate.

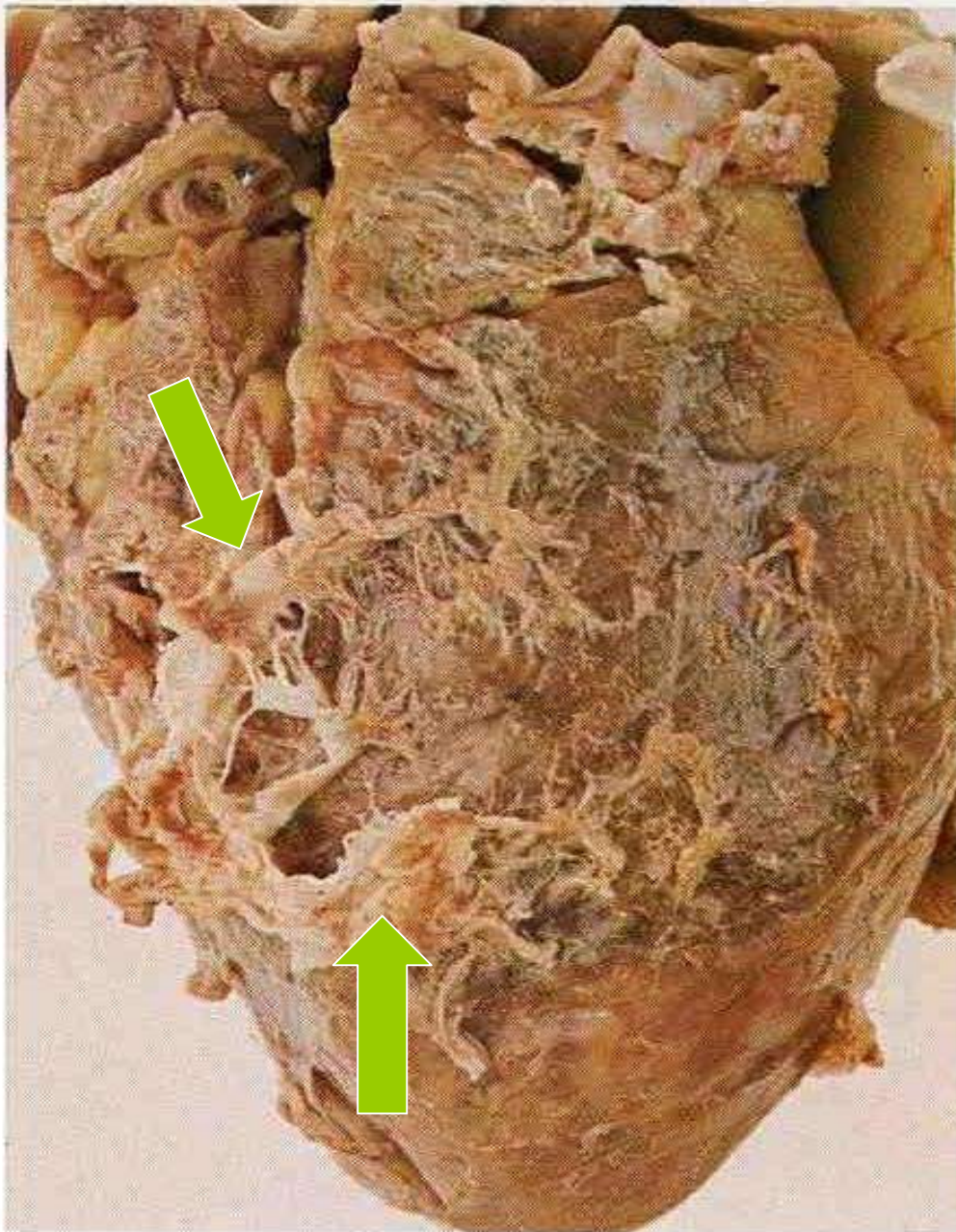
A protein-rich fluid was also present in the pericardial sac.

1.1 Acute fibrinous pericarditis

معاي الصوره
دائماً بتيجي بالافتحار



Figure 30 : Gross view of chronic fibrinous pericarditis, showing bread & butter appearance.



6.15 Uraemic pericarditis: heart

F 31 : Uraemic
pericarditis: heart.
fibrinous pericarditis.

The epicardial surface is
covered with grey-white
strands of fibrin some of
which ^{why} appear contracted &
white [?] as a result of
organization (so-called,
bread & butter
appearance).

fibrin became fibrous tissue.

3- Suppurative inflammation :

This is characterized by production of large amount of **pus** (or **purulent exudate**).

Infection with **Staphylococci** produce localized suppuration as **the skin pustule**.



الزائدة الدودية

In **suppurative appendicitis**, there is **pus** within the **lumen** and an intensive **infiltration of polymorph neutrophils** that are present in the **mucosa , submucosa , muscularis & the serosa of the appendix**.

In some cases localized collection of pus will lead to **abscess formation**.

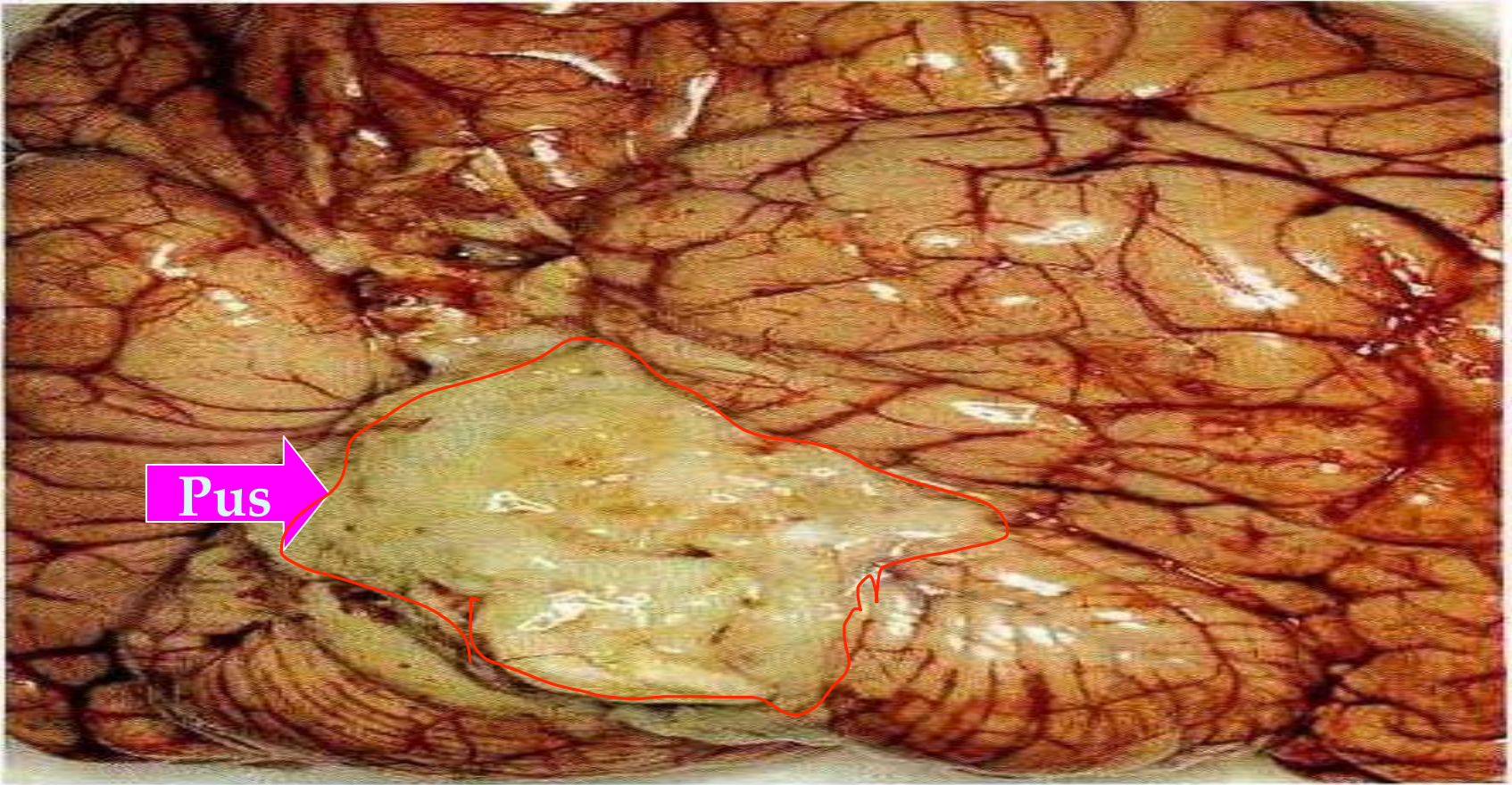
↳ Occurs in severe inflammation cases.

Pus: a thick yellowish, whitish, or greenish fluid made up of dead white blood cells (dead neutrophils), dead tissues, and dead bacteria or fungi. It is produced as part of the body's response to infection.

When staphylococci (a type of bacteria **اللي اخدناها بالميكرو) infect the skin, skin pustule is produced.**

Neutrophils (WBCs) will release enzymes to fight the staphylococci. This causes the accumulation of neutrophils and bacteria in the form of pus.

Abscess is a collection of pus.



1.6 Purulent meningitis

Figure 32 : **Purulent meningitis**. The under surface of the brain is shown . A thick green purulent exudate Pus fills the subarachnoid space over the brain-stem & cerebellum. The patient had acute meningitis caused by **staphylococcus aureus**.

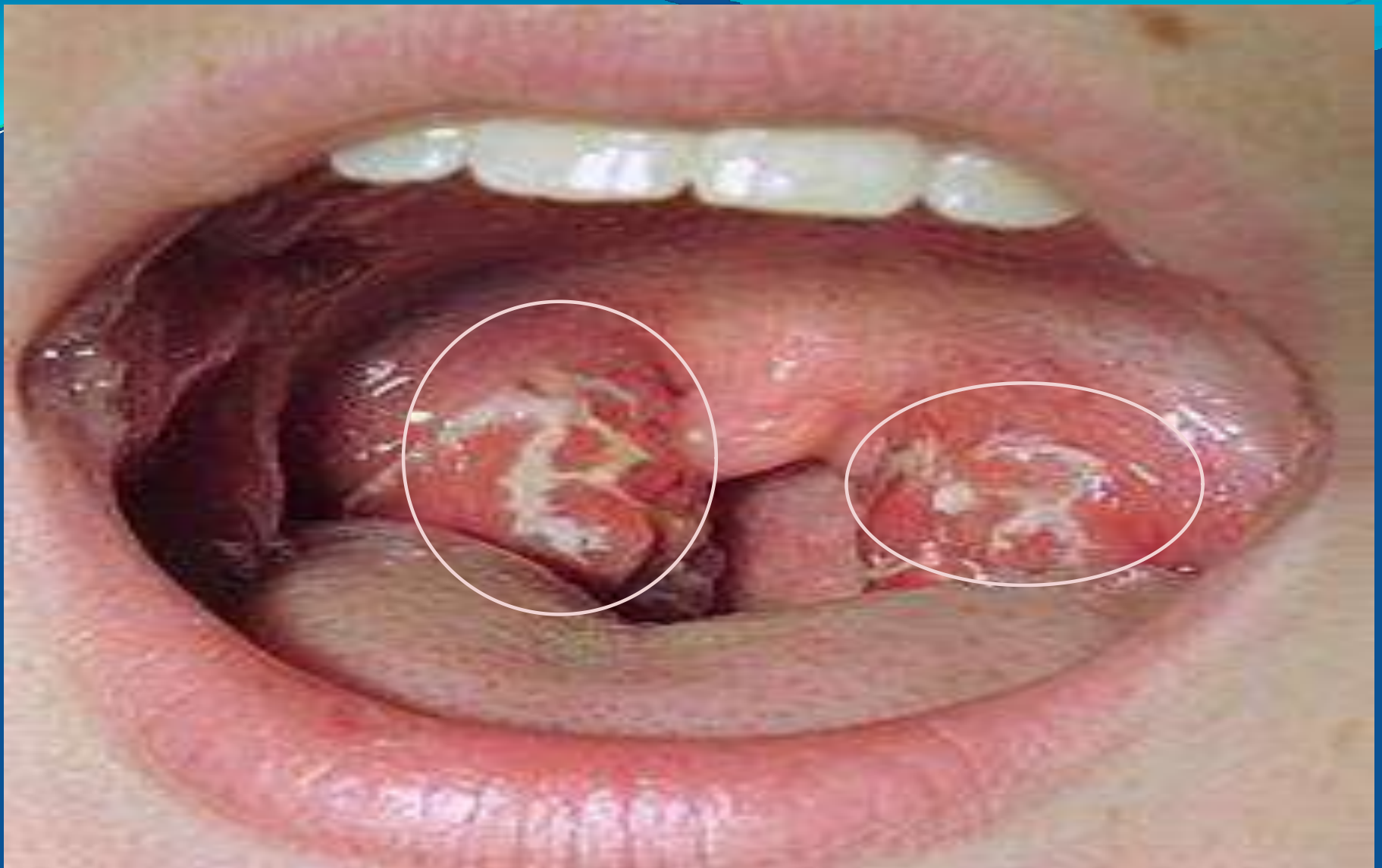


Figure 33 : Acute suppurative tonsillitis, the tonsils being covered by whitish yellowish material (pus) .

F 34 : Microscopic view of Purulent inflammation.

A, Multiple **bacterial abscesses** in the lung (arrows) in a case of bronchopneumonia.

B, The abscess contains **neutrophils + cellular debris = Pus**, & is surrounded by **congested blood vessels**.

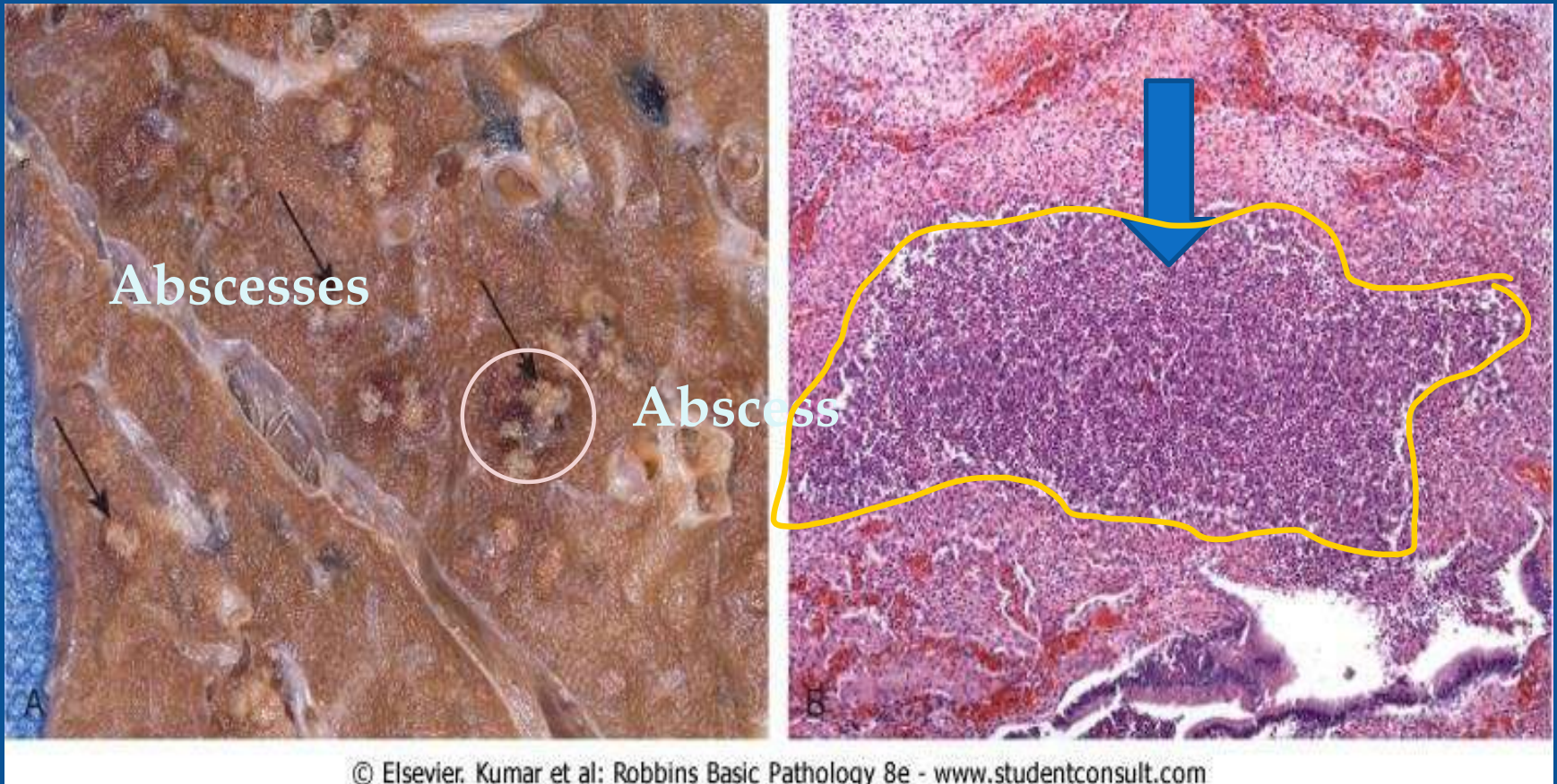
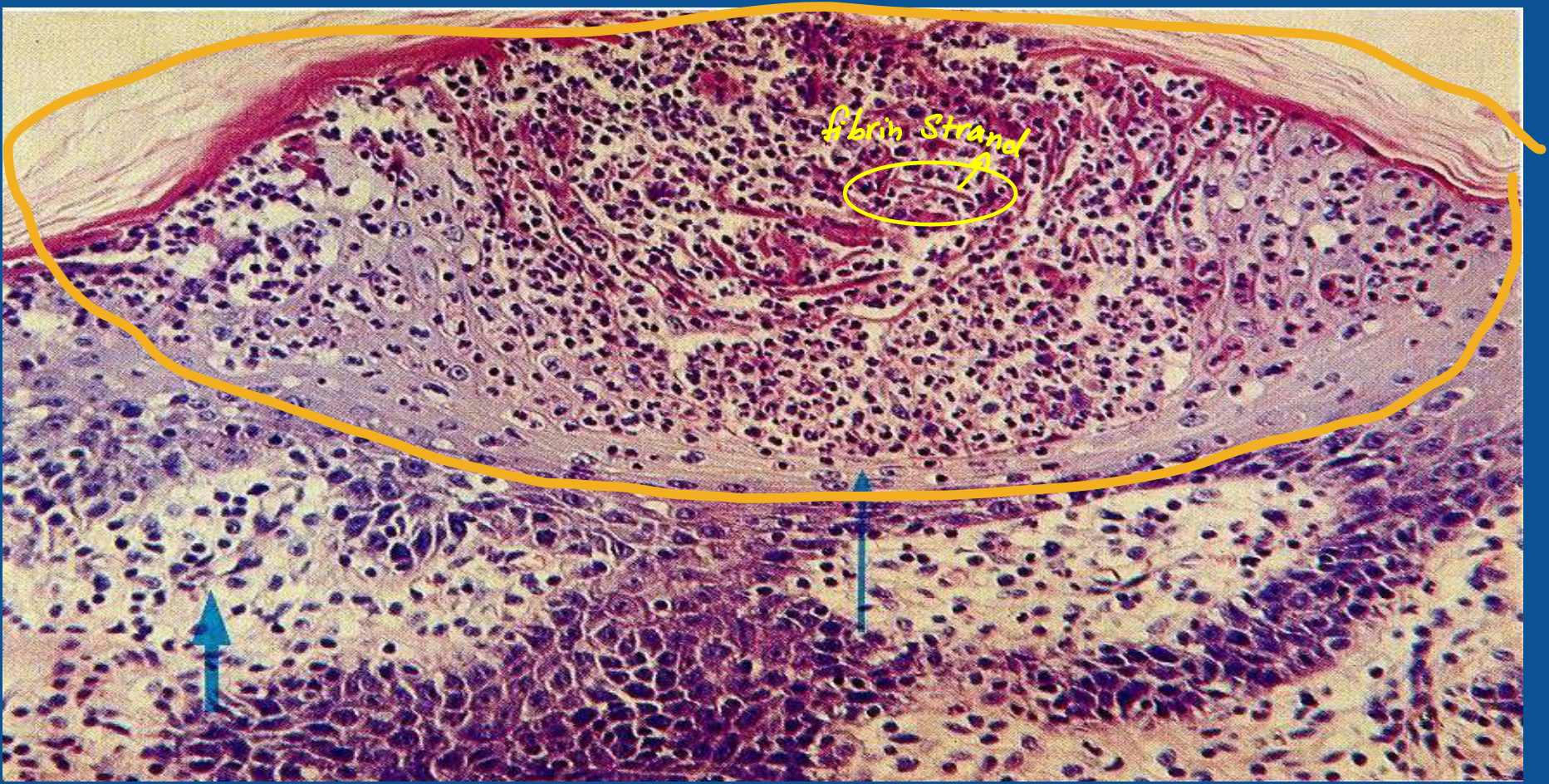


Figure 35 : Acute inflammation : Pustule = skin abscess . Small ovoid abscess (thin arrow) within the upper epidermis causing the skin surface . The main constituent of the abscess is neutrophils with necrotic squamous cells.



4- Membranous or pseudomembranous inflammation :

Pseudomembrane → الغشاء الكاذب

This is a form of inflammatory reaction that is characterized by the formation of a membrane or more correctly a pseudo-membrane.

It is usually made up of precipitated fibrin, necrotic epithelium & inflammatory leukocytes.

When does This occur? *imp.* when the inflammation is so severe as to cause epithelial necrosis and sloughing.

An example of this pattern is seen with Diphtheria affecting the larynx & pharynx.

بالقولون It may also affect the large bowel causing pseudomembranous colitis. The latter is caused by Clostridium difficile infection → *مسبب التهاب*

In this type of inflammation there is an extensive confluent → *تجمع هائل* necrosis of the surface epithelium or mucosa & sever inflammation of the underlying tissue.

كيف يتكون الغشاء الكاذب Fibrinogen coagulates within necrotic tissue & together with polymorph neutrophils, red cells, bacteria & debris of dead tissue produce the false membrane over the inflamed surfaces.

هذا النوع يكون **severe inflammation**.

لما ال **epithelium** يموت و يكون تحته فيه **severe inflammation** رح يطلع بروتينات من ضمنها ال **fibrin**.

ال **fibrin** و ال **necrotic epithelium** و ال **leukocytes** بيعملولي **structure** بيشبه ال **membrane**.



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Figure 36 : Gross view of colon showing Pseudomembranous colitis , multiple yellowish patches of necrotic material seen on mucosal surface .



Figure 37 : Microscopic appearance of pseudomembranous colitis , showing mushroom like membrane over ulcerated mucosa of the colon .

Serous inflammation

- . Effusion
- . from blood
- . mesothelial cells of pleura, pericardium or synovial cells in joints.
- . like in TB infection.
- . And skin blisters in burns or viral infection.

Suppurative inflammation

- . Production of pus.
- . infection with staphylococci.
- . like skin pustule.
- . massive infiltration of neutrophils.
- . may form abscess.

Fibrinous inflammation

- . Extensive fibrin exudate.
- . in severe response.
- . Eosinophilic meshwork in body cavities & meninges.
- . Resolution by fibrinolysis.

Pseudomembraneous inflammation.

- . Formation of a structure that resembles a membrane.
- . from fibrin, necrotic tissue & leukocytes.
- . When the inflammation is too severe.
- . Diphtheria affecting larynx & pharynx.
- . Clostridium difficile affecting large bowel.

Outcomes of inflammation

تلاخيس جھوینتے

Resolution
تھانی کمال

- .limited/short duration.
- .no or minimal damage.
- .Capability of replacement.

Chronic

Acute $\xrightarrow{\text{بنتحول ہی}}$ Chronic

- .Agent is not removed.
- .in viral infection.
- .in autoimmune disease.

inflammation

Scarring

تکڑن ندبہ

- .in tissues that don't regenerate.
- .in massive destruction.
- ↳ severe damage in support structures.
- .in extensive fibrinous exudate that can't be completely absorbed.

Abscess

تکڑن

OUTCOMES of acute inflammation are : 4 outcomes

(I) Resolution, with complete recovery :

with restoration of normal structure & function, occurs only if all the following conditions are satisfied .

This occurs :

- (1) When the injury is limited or short-lived → إذا كان الـ injury طويلاً الـ اعادة رح يصير Chronic و يجب علاجه
- (2) when there has been no, or minimal tissue damage.
- (3) when the tissue is capable of replacing any necrotic cells. → Only stable & Labile cells NOT Permanent cells!!

(II)- Progression to chronic inflammation Acute → Chronic

May follow acute inflammation, if the offending agent is

not removed. In some instances (viral infections or immune responses to self-antigens), signs of chronic inflammation may be present at the onset of injury.

Depending on the extent of the initial & continuing tissue injury, as well as the capacity of the affected tissues to re-grow .

When the agent is so resistant and can't be removed like in autoimmune diseases and viral infections, the injury will become chronic.

This depends on:

1. The extent of the initial injury.
2. The ability of the injured tissues to regrow.

(III) - Scarring or fibrosis results:

(1) If inflammation occurs in tissues that do not regenerate

(e.g., skeletal & myocardial muscles; & neurons). → *Permanent cells.*

(2) After substantial tissue destruction (if the supporting structures of the tissues are severely damaged)

even stable cells like hepatocytes won't be able to regenerate if they're severely damaged.

(3) In extensive fibrinous exudates which can not be completely absorbed & therefore, it is organized by ingrowth of connective tissue & resultant fibrosis.

(IV) - Abscess formation (suppuration) → *Localized formation of pus surrounded by fibrous capsule.*

May occur in the setting of extensive neutrophilic infiltrate (in pyogenic or "pus forming" bacterial or fungal infections).

Due to the extensive tissue destruction as seen in abscess (including the extracellular matrix).

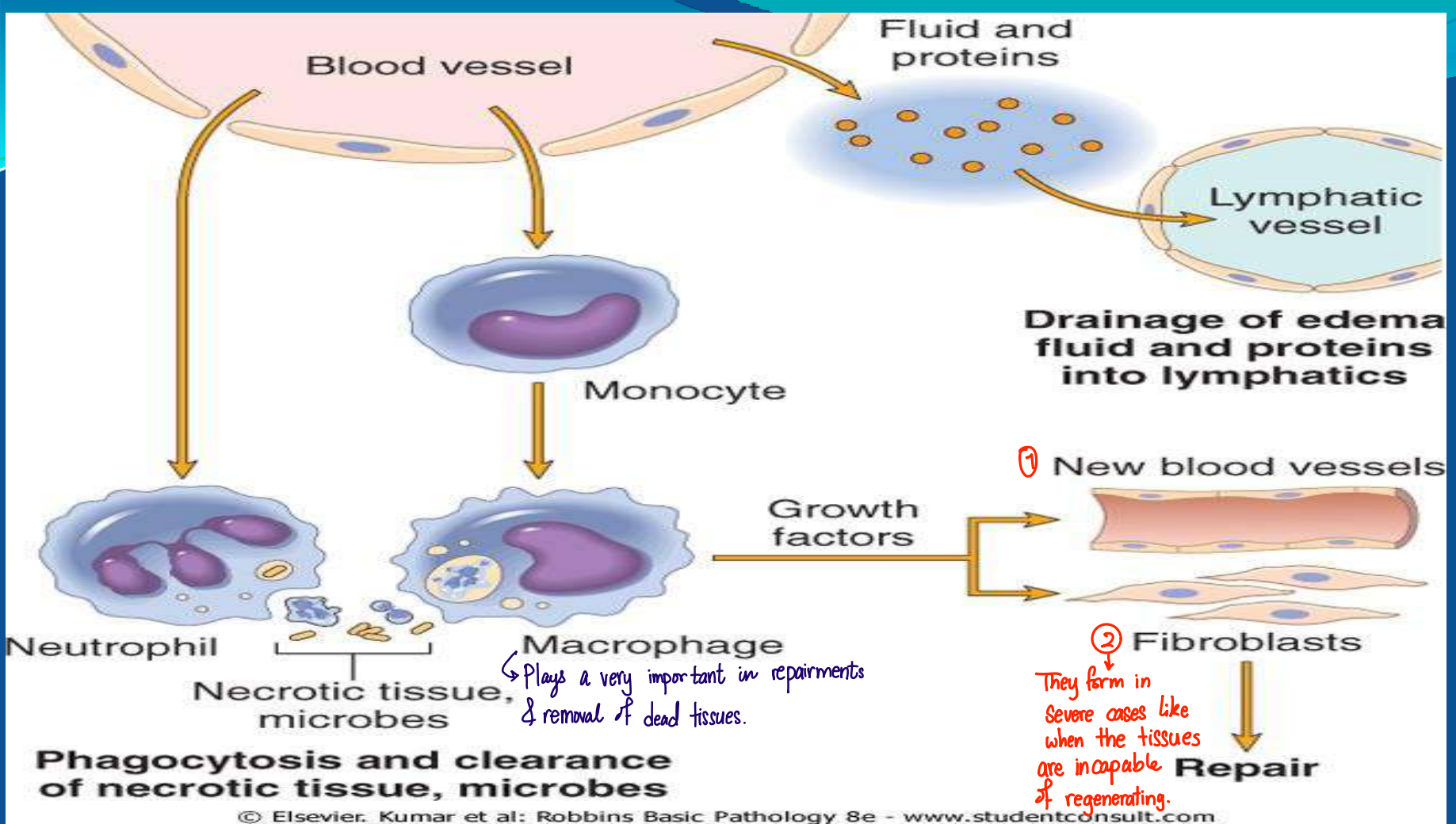


Figure 38 : Events in the resolution of inflammation. Phagocytes clear the fluid, WBCs & dead tissue, & fluid & proteins are removed by lymphatic drainage

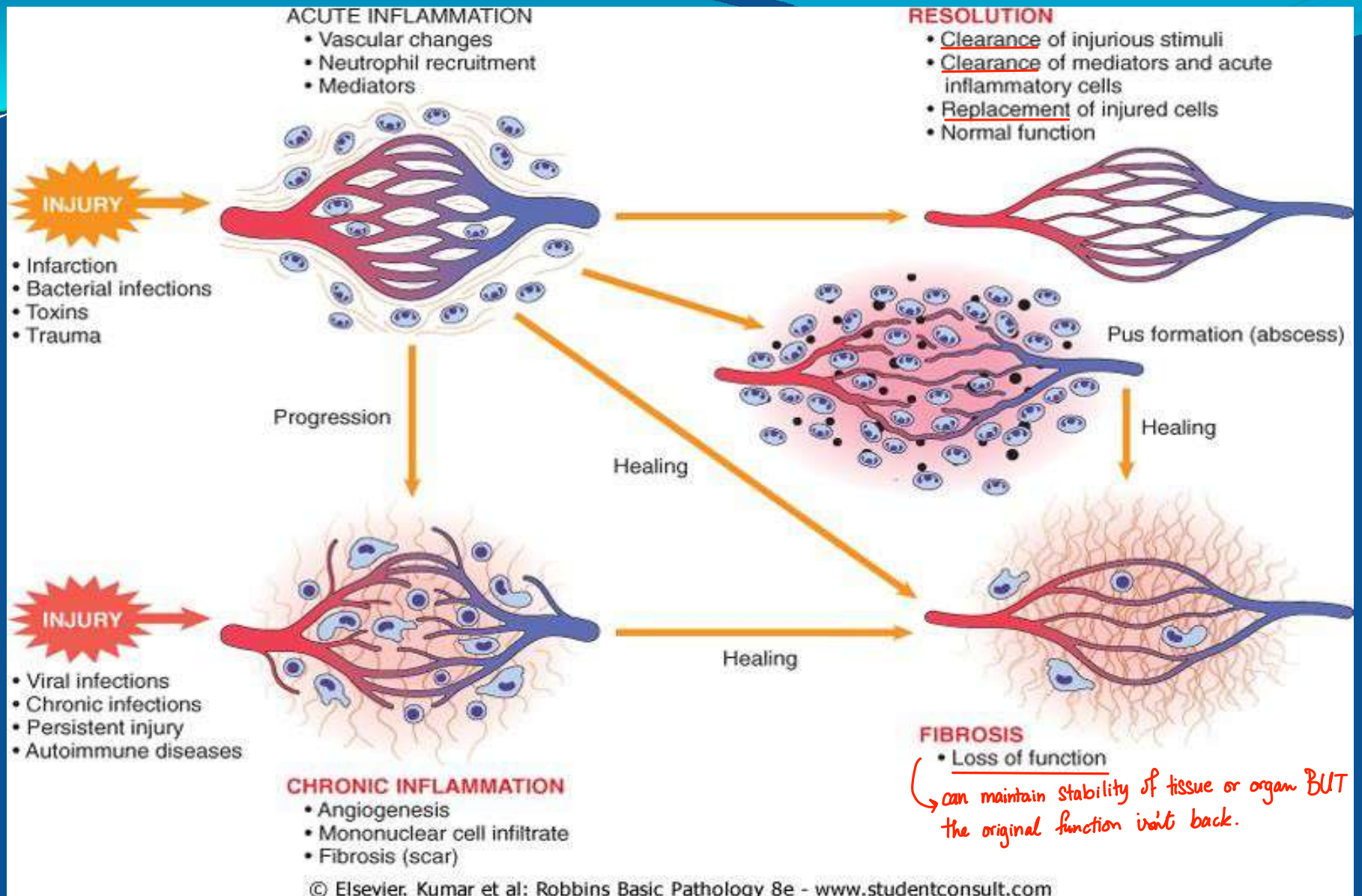


Figure 39 : Outcomes of acute inflammation: resolution, healing by scarring or chronic inflammation

Cause of chronic inflammation:

1. Progression of acute → injurious agent ما راح.
 ↳ problems in healing.

2. viral infections: مش أي خلية بتقدر تتهاجم و تقضي على الفيروسات، إلا لما يدخل جوا الخلية و يقتل اخلية كاملة مع الفيروس اللي جواها. و الخلايا هاي **Chronic inf. cells** حق أول ما دخل الفيروس ما في neutrophils تزد بتصرف معاه، بتروح ال-lymphocytes و macrophages و هاي **Chronic inf. cells**.

3. Persistent infection: ↓ immunity

4. immune-mediated: مثل ال autoimmune disease، مهاجمة شئ طبيعي موجود بجسمك و رح تظلم المناعة تتهاجمه. عشان هيك **Chronic**.

5. Prolonged exposure to toxins: زي عمال المناجم لما يتعرضوا للكربون مثلاً ألياف الأسبست

CHRONIC INFLAMMATION

Inflammation of prolonged duration (weeks, months to years) in which active inflammation, tissue injury, & healing proceed simultaneously. → *الالتهاب و الإحماية و الشفاء يكونوا بنفس الوقت.*

Chronic inflammation is characterized by :

التهاب الى مكان الإلتعاب

1. Infiltration with mononuclear chronic inflammatory cells, including macrophages^①, lymphocytes^②, & plasma cells^③.

2. Tissue destruction, largely directed by the inflammatory cells → *Cells die due to the toxins secreted by inflammatory cells.*

3. Repair, involving new vessel proliferation (angiogenesis) & fibrosis .

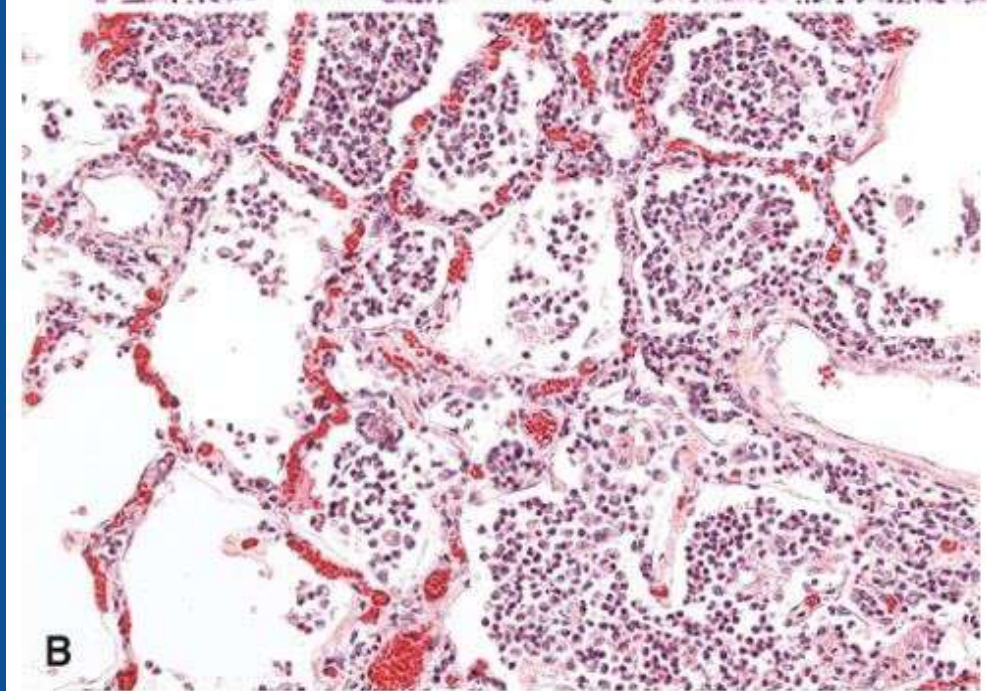
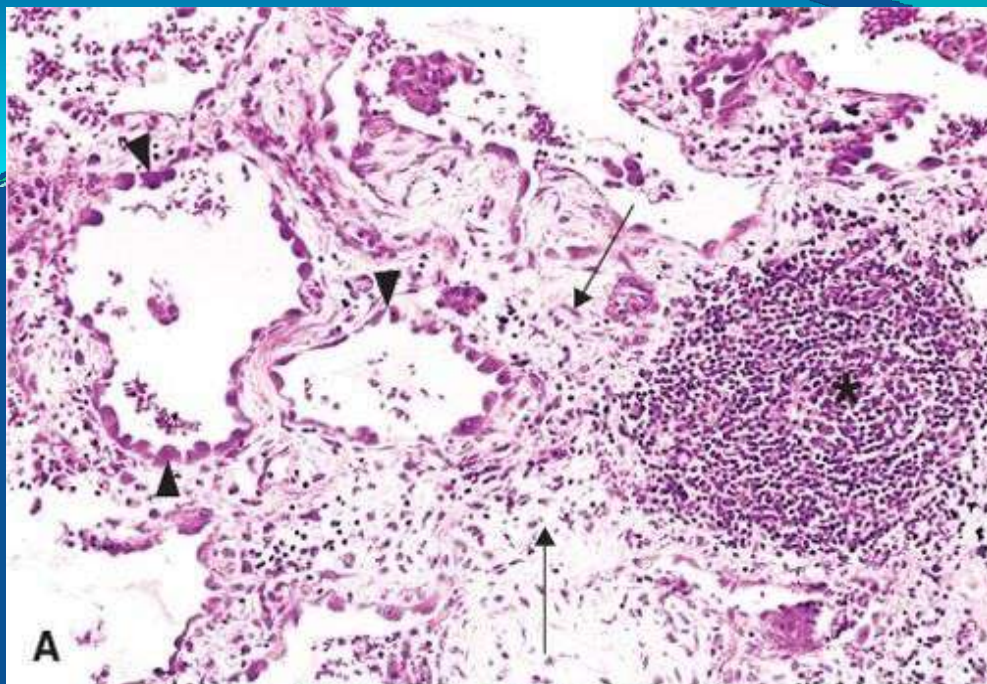


Figure 40 :

A, Chronic lung inflammation, showing collection of chronic inflammatory cells (asterisk) + destruction of parenchyma (normal alveoli are replaced by spaces lined by cubical epithelium, arrowheads), + fibrosis (arrows).

B, Acute bronchopneumonia. Showing **neutrophils** filling the alveolar spaces with **congested blood vessels** .

Causes of chronic inflammation

- Progression of acute to chronic inflammation.
- Viral infections.
- Persistent infections.
- immune-mediated inflammatory diseases.
- prolonged exposure to potentially toxic agents.

1- Progression of acute to chronic inflammation :

occurs when the acute response cannot be resolved, either:

- (a) because of the persistence of the injurious agent, or
- (b) because of interference in the normal process of healing.

For example, a peptic ulcer of the stomach or duodenum initially shows acute^① inflammation followed by the beginning stages^② of resolution and healing^③ process

هجمات متكررة

However, recurrent attacks of duodenal epithelial injury interrupt this process, & result in chronic peptic ulcer.

الهجمات المتكررة تعمل على مقاطعة عملية الشفاء (healing process) مما يؤدي إلى تحول الالتهاب الـ acute إلى chronic.

اول مسبب للالتهاب المزمن هو **تطور الالتهاب الحاد إلى إلتهاب مزمن**.
هذا يحصل بسبب عدم المقدرة على معالجة الإلتهاب الحاد إما بسبب:
١. **صمود مسبب الالتهاب (injurious agent)**
٢. **تدخل عمليات الشفاء الطبيعية**. يعني تكرار الإصابة بالالتهاب الحاد
مما يؤدي إلى إلتهاب مزمن و هذا مثال على ال **peptic ulcer**.

ثاني سبب هو الإصابة ب **viral infections** (موجود تحت)
هذا يتطلب تدخل ال **mononuclear chronic inflammatory cells** من
البداية حتى لو كان **acute**.
يعني ال **viral infection** من البداية **chronic**.

(2) **Viral infections:**

Intracellular infections of any kind typically require a response that involves chronic inflammatory cells (lymphocytes & macrophages) from the ^{beginning} onset in order to identify, & eradicate infected cells ^{تخطيط}, as in viral hepatitis..

(3) **Persistent infections** by microbes that are difficult to eradicate, e.g., tubercle bacilli of T.B.; Treponema pallidum of syphilis, certain viruses, & fungi; all of which tend to established persistent infections & elicit a T lymphocyte-mediated immune response, called (delayed hypersensitivity reaction) i.e. Type IV.

السبب الثالث هو **persistent infections by microbes** يعني السبب هو البكتيريا أو فيروس أو فطريات. هذول عندهم مقاومة جدا قوية ضد مناعة الجسم و رح تظل موجودة لفترة طويلة. ينتج عنها **delayed hypersensitivity reaction** أو ال **T-lymphocyte-mediated immune response**.

Delayed hypersensitivity is a common immune response that occurs through direct action of sensitized T cells when stimulated by contact with antigen.

Immune-mediated inflammatory diseases: موجود تحت
في أمراض بتخلي مناعة الجسم تهاجم على أنسجة الجسم السليمة مما يؤدي إلى ضرر مزمن بهذول الأنسجة. هذول الأمراض اسمهم **autoimmune diseases**. زي مرض ال **rheumatoid arthritis**. و في كمان الربو و الحساسية ضد المواد البيئية مثل الغبار يعتبروا أمراض مناعية.

(4) Immune-mediated inflammatory diseases, or hypersensitivity diseases.

Immune reactions may develop against the individual's own tissues, leading to **autoimmune diseases**, resulting in chronic tissue damage & inflammation e.g., **rheumatoid arthritis** .

Immune responses against common environmental substances are the cause of **allergic diseases**, such as bronchial asthma.

(5) Prolonged exposure to potentially toxic agents

غير قابلة للتحلل

e.g., non-degradable exogenous material such as inhaled **silica & asbestos**, which can induce a chronic inflammatory responses in the lungs called **silicosis & asbestosis** respectively .



Asbestos



Asbestosis

(long-term inflammation & scarring of the lung caused by asbestos)

*imp for cases:

. شخص معه peptic ulcer و أعطوه دواء يساعد على ال healing. بعدين رجع مهار معه peptic ulcer مرة ثانية ورجعوا أعطوه الدواء.. وصعدنا.. ← صاي الحالة صارت Chronic.

لما يبجي مريض معه ألم في المعدة من ٦ أشهر ← هذا Chronic.

لما يبجي مريض ويحكك اخذت aspirin ومارعندي ألم في المعدة ← acute