* L1

* Stimuli → Pathogen, toxins and truma.

*The goal of inflammation → respond to stimuli and restore balance.

* External and enternal factors

- Non-microbial
  - allergies
  - toxic compounds
  - irritants
- microbial
  - virulence factors → help pathogens colonize the tissue
  - PAMPs → Conserved Patterns
    - LPS
    - Peptidoglycan
    - Lepoteichoic acid
    - Mannan

* Enernal factors (endogenus) → DAMPs

PAMPs and DAMPs are recognized by PRRs

① non-specific
└ it can't distinguish specific pathogen from another although they can distinguish between catogries like bacteria and viruses.

② Fast: min - hours.

③ No memory.

Extravasation → In the case of inflammation, it refers to the movement of WBCs through the capillary wall into the surrounding tissues (transmigration).

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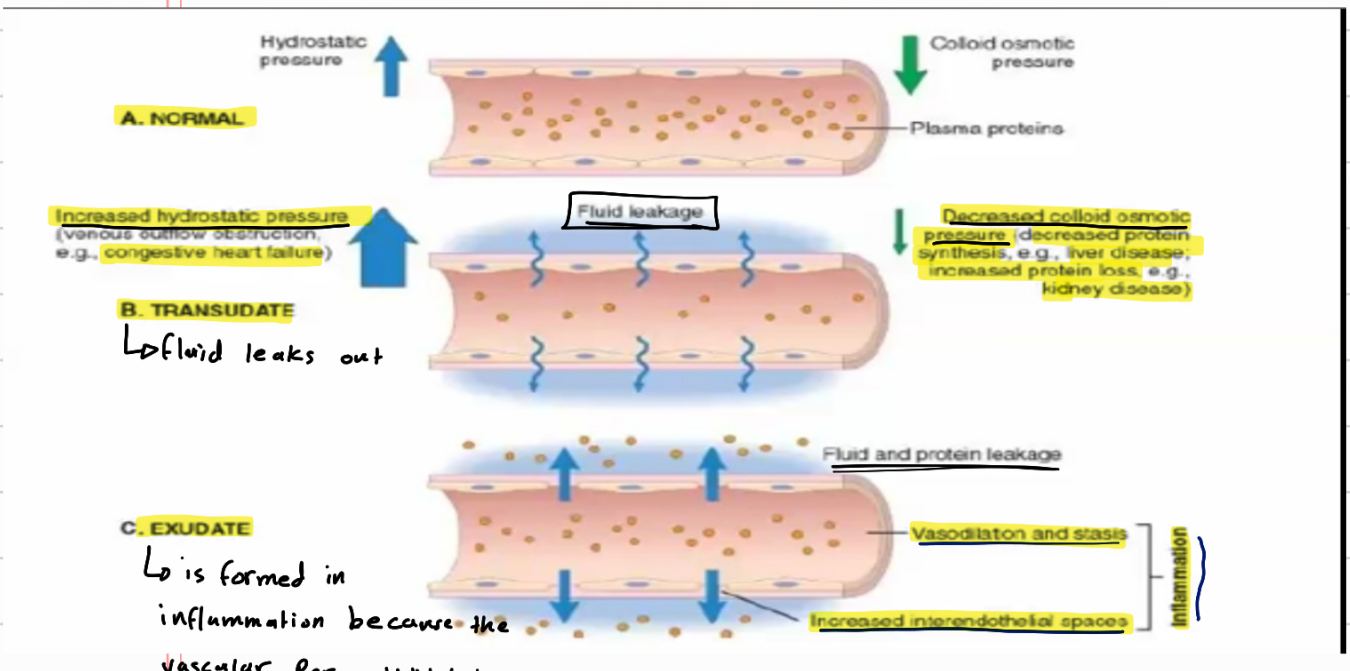
*L2



↳ fluid leaks out

↳ is formed in inflammation because the vascular permeability increases.

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*L3

*Outcomes of inflammation →

① Quick resolution → Complete resolution can occur after a relatively minor trauma.

② Scaring → if there is a greater inflammation

*wounds heal by fibrosis or regeneration.

* regeneration → adjecent healthy cells replace the damaged cells.

*In many wounds you get combination of regeneration and scarring.

③ chronic inflammion → if the inflammation isn't removed.

with abscess formation with the accumlation of Pus.

* Pus → dead granular sites, polymorphonucler sites and macrophages with some living leukocytes and bacterial cells thats why pus can be infectious.

* liquid liquifaction necrosis, so when the cell necrose they liquify and you gen liquifaction (semi-liquid thick solution of pus).

* Suppurative → something that is producing pus.

* Purlent → something contains a lot of pus.

* Empyema → a pus filled cavity, for example:

In sever Cholesystites → empyema in gallbladder.

* If there is a large area of inflammation this can result in a systamic absorption of cytokines and get the systemic inflammatory response syndrome.

④ death → is possible of outcome of a major inflammatory response.

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* Ls

* Complement system → The most important defenses of our bodies.

* it consists of 30 different proteins.

* about of 15,000,000,000,000,000,000 (15 quintillion of them are saturating every fluid in your body right now. سبحان الله الرقم هائل لدرجة لا يمكن تخيلها

* Antibodies → acitivat com. sys

Com. sys:
- ① crippling enemies
- ② Activating immune sys.
- ③ Rips holes in things unitle they dye.

* Comp. Proteins float around in a sort-of passive mode until they get activated and change their shap.

* How comp. sys. works ?

① C3 is activated by other Proteins or antibodies and it will spilte into 2 small Proteins C5 and C3b

② C3b anchors itself very tightly to the surface of the Pathogen and changes its chape many times until give C3 convertase. (thousnds of Proteins cover the bacteria)

③ C3a call the immune system to help, the first immune cells that arrive are Phagocytes.

④ C3b makes it easy for immune cells(Phagocytes) to catch their victims.

⑤ C3 Convertase is converted to C5 convertase which splits into C5a and C5b.

⑥ C5b attach with C6-C9 to make MAC.

*Vaccinia virus → Produces Protein that shuts comp.sys. down, it creates safe zones around the cells it infects.

*Some bacteria can grab molecules from the blood that keep the com.sys calm and make themselves invisible.

* They are smarter than a lot of People

*L6

Granulomatous inflammation → Specific chronic inflammation.

* Macrophages —(IFN-γ, cytokines, microbial products)→ Activated macrophages

Activated macrophages:

Eliminate the injurois agent
- ROS, RNS
- proteases
- cytokines
- Coagulation factors
- A.A metabolites

Initiate the repairing process
- Growth factors (PDGF, FGF, TGF-β)
- Fibrogenic cytokine
- Angeogenic cytokines
- Collegen remodelers

* In acute → macrophages are dying off

* In chronic → macrophages accumlate.

↳ due to
- continous recruitment from blood
- local proliferation
- Immbolization

* Pathogensis → microbes are resistent to be killed

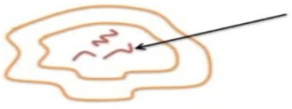
Macrophages → Epithelioid cells
- More killing ability

*Giant cells → fusion of epithelioid cells

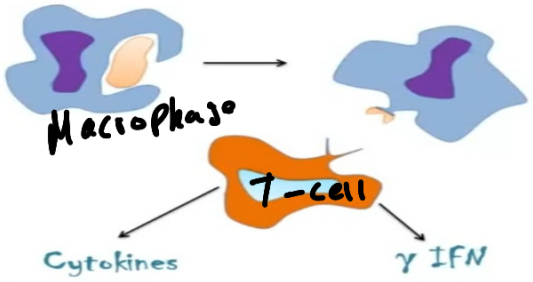
*Granulmats inflammation → more tissue distruction and fibrosis.

2 Types (pathogenesis)

- Foreign body granuloma
  - Talc, suture material, fibers
  - No immune response



- Immune granuloma
  - Poorly degradable



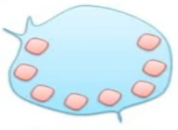
- Digest the foreign agent
- Activate T cells
- Activate Macrophages

Cell types

1. Epithelioid cells :-

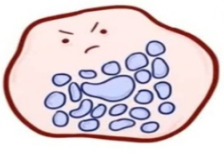
- Pale, eosinophilic cytoplasm
- Dispersed chromatin – Increased synthetic activity
- Elongated nucleus – Footprint shape
- Less phagocytic capability
- Enhanced secretory ability
- More microbial killing ability

2. Langhan's giant cells :-



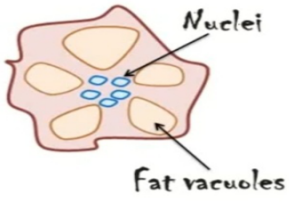
- Nuclei – Periphery f the cell (Horse shoe)
- Tuberculosis

3. Foreign body type giant cells :-



- Nuclei – Arranged randomly
- Foreign material

4. Touton giant cells :-



- Foamy macrophages
- Fat necrosis

Morphological patterns of granulomatous inflammation

1. Caseating Granulomata
   - Center – Caseous necrosis
   - Tuberculosis
2. Non – necrotizing Granulomata
   - No central necrosis
   - Sarcoidosis, Leprosy, Crohn's disease
3. Suppurating Granulomata
   - Centre – Suppurative necrosis with neutrophils
   - Cat scratch disease, Fungal infection, TB (rarely)

*L7 → احضر الفيديو ما بده تلخيص ومفيد بومخلك كيف بتتكون ال non-caseating granuloma ☺

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*L8

*Wound healing →

*The largest organ in our body is Skin.

*After a deep cut or wound, the newly healed skin will look different from the surronding area.

Skin
- → Top layer → epidermis → consists of keratinocytes
- → inner layer → dermis → contains BV, glands and nerve endings

* Regenerative Process →

① Hemostasis → skin's response to 2 immediate threats:

you are losing blood and the physical burrier of the epiderms has been compormised.

*The BVs tighten to minimize the bleeding → Vasoconcentriction

* fibrin → forms cross-links on the top of the skin

Preventing blood from flowing out and bacteria from getting in

After 3 hrs of this, the skin turns red.

② Inflammation → The body sends WBCs (macrophages) to fight any Pathogens through Phagocytosis and Producing growth factors.

* The constricted BVs now expand Vasodiltation

③ Proliferative Stage → After 2-3 days after wound
↳ fibroblast cells begin to enter the wound, In the process of collegen deposition.

* epidermal cells divided to reform the outer layer of skin, the dermis contracts to close the wound.

④ remodling → The wound matures, the collegen is rearranged and coverted into specific types.

* Through this Process (can take over a year), the strength of new skin is improved, and BVs are strengthed.

* with time the new tissue can reach 50-80% of its original function, depending on the severity of the intial wound

*Lg

*keloid and hypertrophic scar →

↓

is a scar usually first seen behind the ear that grows quit large.

* keloid → grows outside of the are of the incision as opposed to hypertrophic scar which is simply along the same line as the incision.

*keloid most commonly found in African-American.

*Treatment of keloid depends on first trying to decrease the inflammatory response.

* many studies have shown that if you only treat the keloid itself surgically and do nothing else, 100% the keloid will back again.

① Inject it with steriod generally triacinolone or kenalog for (4-6) weeks.

② Excise the keloid surgically, injecting Kenalog at the time of excision and then (2-3) times afterwards.

③ Several thearpies have been tried to Prevent keloids from reforming include 5-flurouricil, chemotherapy agent and Silicone sheeting.

④ Pressure and radiation thearpy generally radiation is reserved for recalcitrant cases (cases recurred in numerous time)

most difficult cases

* Hypertrophic treatment is slightly different, often doesn't need to include surgical excision.

↳ ① Kenalog is often used to decrease the firmness of the scar and decrease some of collegen formation.

② Some types of dermabrasion is used to smooth out the scar and decrease the ability to be seen.

"وَمَا تَوْفِيقِي إِلَّا بِاللَّهِ"

Mays qashou