Acute Inflammation

27/5/21

2.1 c) Step 6, 7 of Neutrophil phase & Macrophage phase.

Step ⑥: Destruction of phagocytosed material

② ways

✗ O_2 - dependent killing

* most effective.
* used by "Neutrophils"

Clinical: CGD (Chronic granulo-matous Ds.)

O_2 - indep. (enzymatic) killing

* less effective
* Used by "Macrophages"

SOD (Super Oxide Dismutase)

H_2O_2

MPO (MyeloPeroxidase)

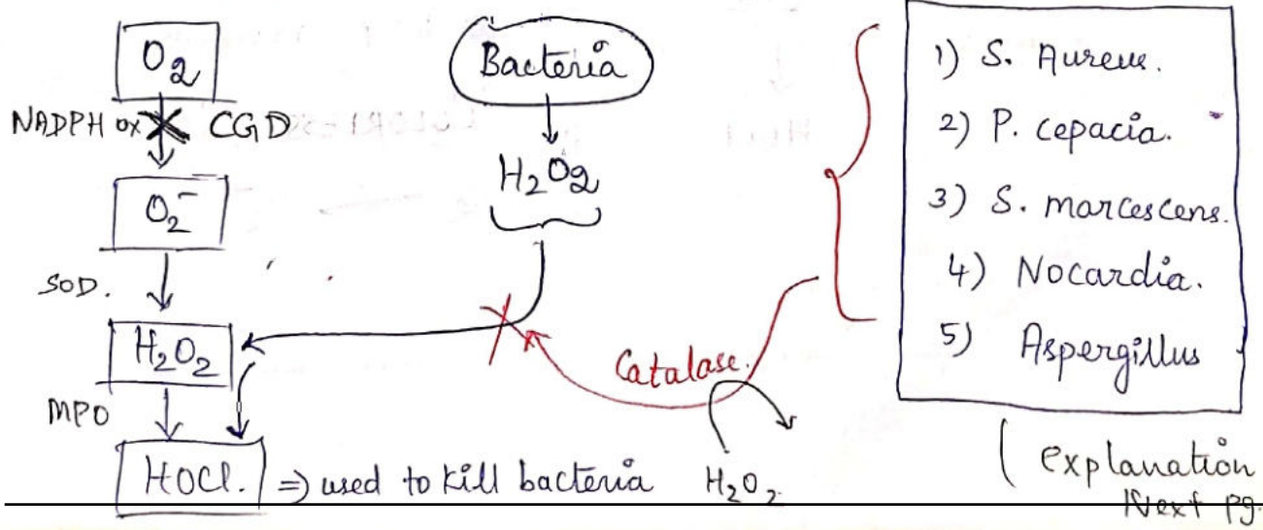
HOCl. (bleach)

used to destroy phagosome.

Clinical: CGD - Chronic Granulomatous Ds.

* Poor - O_2 dependent killing due to -- "NADPH oxidase" defect (X-linked / A.R).

Why Infec^n & Granuloma (formation) occurs in Catalase +ve orgs.? but (mostly) not in Catalase -ve orgs?



(5).

1) S. Aureus.
2) P. cepacia.
3) S. marcescens.
4) Nocardia.
5) Aspergillus

(Explanation Next pg.)

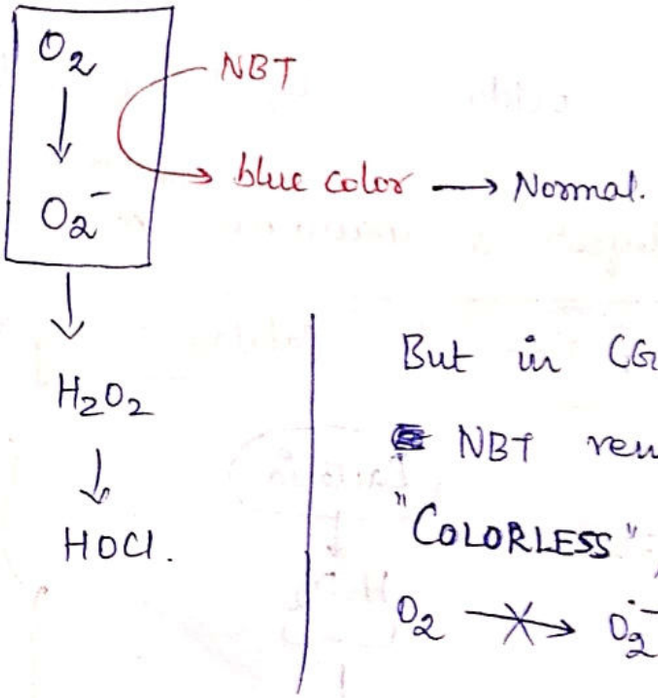
org which destroy H_2O_2.

Note:

Silly thing about Catalase ⊖ve org. is that, in CGD H_2O_2 is obtained from bacteria & that same H_2O_2 → HOCl (bleach) is used to kill the same bacteria ☺

NBT - Nitroblue Tetrazolium Test

(used to Δ CGD)



But in CGD pt. ☞ NBT remains "COLORLESS", bcoz $O_2 \not\rightarrow O_2^-$

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* asymptomatic but ↑ risk of candida infections.

O_2 independent killing: less effective

* occurs via "ENZYMES" present in Leuco-cyte secondary (2°) granules. (eg: lysozyme & Major basic protein (eosinophils).)

Step-⑦ RESOLUTION.

* Neutrophils undergo Apoptosis. → "pus"
* disappear c in 24 hrs after Resolution of inflamm. Stimulus.

* peak 2-3 days after inflamm^n begins.
* predominate after Neutrophils.
* derived from monocytes in blood.
  (monocytes (blood) ⟶ macrophages (Tissues))
* same ⑦ steps.
  ↳ marginal^n, Rolling, adhesion, Transmig. & chemotaxis, phagocytosis, Destruc^n. Resolut^n/continu^n.
  phagocytosis ↓ enzymes in 2° granules. (mostly not by O_2 dep. rxn.)

* Macrophages are the "managers" of A.I. ⇓ step ⑦ of macrophage phase is variable. ⇓

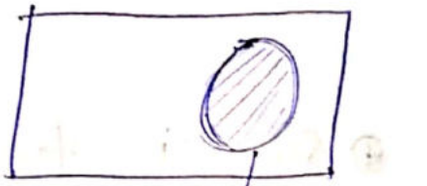
Case (i) : If neutrophils have done a good job then macrophages end A.I by releasing IL-10 & TGF-β (shut down inflamm. proc.) → also involved in Healing.

Case (ii) : If neutrophils are in need of help then, macrophages release IL-8, thereby CONTINUING A.I. ↓ more Neutrophils are called in.

Still we have Neutrophilic response.

Case (iii) : ABSCESS

If Macrophages sense that the infecn can spread it can prevent the spread by creating a "walled off fibrosis around inf. area" This is called Abscess. It's good & prevents spread of infecn


Abscess.

Case (iv) : Chronic Inflammn.

* If the infecn is by Virus which cant be well handled by Neutrophils then macrophages call in for Chronic inflam'

* They ingest some of the Ag. & express it on their surface to ⊕ T-Helper cells. (MHC-II).

⟶ Chronic inflammn