Causes :- 1) hyperparathyroidism
2) hypervitaminosis D
3) Neoplasma

Gross - Pale chalky white coarse gritty feel.

Micro :- $Ca^{2+}$ salts → blue granules on H & E.

Psammoma body.

Special stain :- Von Kossa - black
Alizarin Red S - Red.

## IMMUNOLOGY AND AMYLOIDOSIS

**Hypersensitivity Rxn :-**

Exaggerated or inappropriate state of normal immune response with onset of adverse effects on the body.

**Type 1: Anaphylactic :-**

Rapidly developing immune response in a previously sensitised person.

Action time :- 15 - 30 minutes

Etiology :- ① Genetic basis
② Environmental pollutants
③ Viral infections

Pathogenesis :-

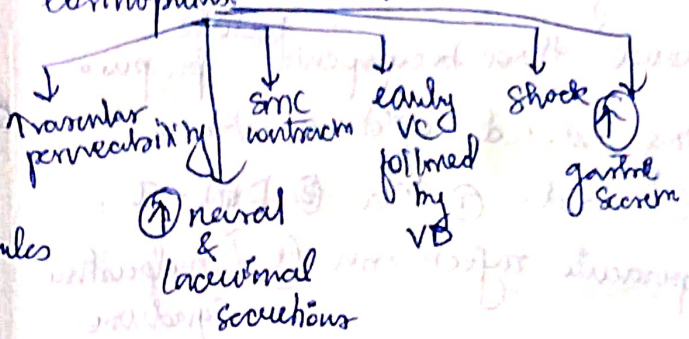
(i) First contact → sensitisation takes place.

Response : Activation of B lymphocytes → IgE secreting plasma cells.

IgE Ab → Fc receptors present on mast cells & basophils.

(ii) 2nd contact :- IgE Ab on mast cells mumb - lysis, Influx $Na^+$ & water ← cell damage, degranulation of mast cells & basophils.

- Released granules → histamine, serotonin, VIP, chemotactic factors of anaphylaxis for neutrophils & eosinophils.




Examples :-

Systemic :- Anti-tetanus serum (ATS)
Administration of drugs - penicillin

Local :- ① Hay fever
② Bronchial Asthma
③ Food Allergy
④ Cutaneous anaphylaxis -
⑤ Angioedema - AD.

**Type II : Antibody - mediated (cytotoxic) Reaction -**

Reaction by humoral Ab that attack cell surface Ag & cause lysis of target cells.

Rxn time :- 15 - 30 min

Pathogenesis :-

① Ag on surface of foreign cell + Fab portion of Ab ⇒ Ag-Ab complex
② Unattached Fc forms link b/w Ag & complement.
③ Ag-Ab binding with Fc → pathway of serum complement

$$C_4 + C_2 \xrightarrow{C_3} C3b$$

④ Activated C3b → acts as opsonin & attracts phagocytes to the site of cell injury
⑤ Ag-Ab complex express MAC

Examples:- ① Auto Immune hemolytic anaemia

② Transfusion Rxn ⑧ Erythroblastosis foetalis

③ Immune thrombocytopenic purpura

④ Drug induced agranulocytic Abx -

⑤ Graves ds ⑥ MG ⑦ DM - I,

⑨ hyperacute rejection Rxn ⑩ Goodpasture Syndrome

## Type III - Immune complex or arthus Rxn :-

Deposition of Ag-Ab complex on tissues which is followed by activation of complement system.

Etiopathogenesis:-

1) Persistence infection - of low grade microbial
2) Extrinsic environmental Ag
3) Auto immune process.

Mechanism:

1) Immune complex formed by interaction of soluble Abs & soluble insoluble Ag.
2) Immune complexes which fail to get removed from body fluids gets deposited onto tissues.
3) Fc component of Ab + complement & activates classical pathway of complement resulting in C3a, C5a & MAC
4) C3a → histamine
C5a → proinflammatory mediators

Examples:

1.) Immune complex GN & Ag → RBM
2.) SLE → Nuclear Ag (DNA, RNA) anti-nuclear & anti DNA auto Abs.
3) Rheumatoid Arthritis
4) Farmer's lung :- Actinomycetes contaminated hay → Ag,
5) Polyarteritis nodosa & Wegner's granuloma with anti-neutrophil cytoplasmic Ag -
6.) Henoch-Schon-lein purpura :- Resp virus → Ag
7.) Drug Produced vasculitis

## Type IV: Delayed hypersensitivity Rxns :-

It is a tissue injury mediated by Tcell mediated immune response w/o formation of Abs.

Etiopathogenesis:-

i) The Ag is recognized by CD8+ Tcells & is processed by APCs.
ii) APC migrate to LN where Ag is presented to helper Tcells -
iii) Helper Tcells release cytokines that stimulate Tcell proliferation.
iv) Activated Tcells - proinflammatory mediators & cause cell destruction

Examples of type IV Rxns :-

1) Rxn against mycobacterium (Mtb (tuberculin rxn, leprosy)
2) Rxn against virally infected cells.
3.) Rxn against malignant cells
4.) Rxn against organ transplantation