classmate
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PLASMA PROTEINS

Properties -

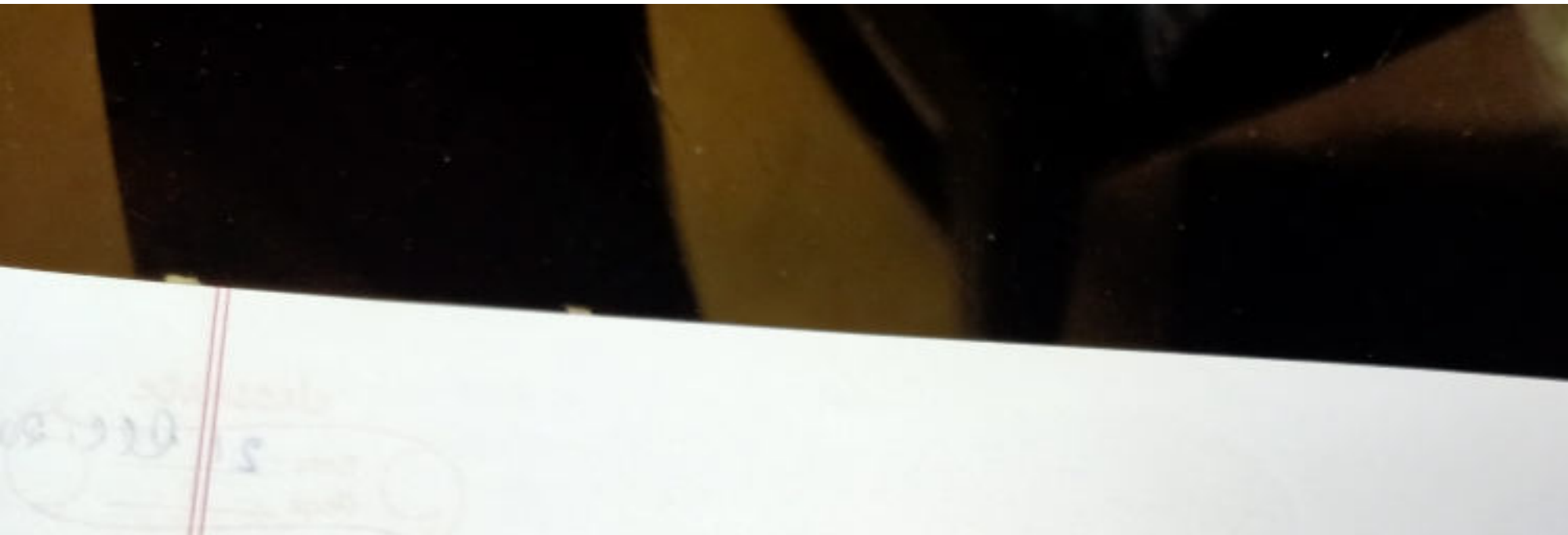
① MW — Fibrinogen > Globulin > Albumin.

② Oncotic P / colloidal osmotic P - ~25 mmHg.

③ Buffering Action - acceptance of H^+.

Func -

① Coagulation: Fibrinogen
② Osmotic / Oncotic P - mainly Albumin (max).
③ Defence Mechanism - Globulin.
Ig
④ Transport of various substances.
⑤ Buffering Act^n - mainly Albumin. (Acid-Base balance)
⑥ Viscosity of Blood - " "
⑦ ESR - {Fibrinogen, Globulin} accelerate Rouleux formation
responsible for ESR.
imp. diagnostic & prognostic tool.
⑧ Suspension stability of RBCs.
⑨ Reserve proteins: fasting / inadequate food intake.



Hyperproteinemia | Hypoprote

↑ se ni pr | ↓ se ni pr

RBC formed from ← Area vascularis ← Mesoderm of Yolk Sac

① Mesoblastic Stage: [Early embryo upto 3 months of foetal life]

② Hepatic Stage: [After (3) months of foetal life]
└ liver & spleen.

③ Myeloid Stage: [Middle of foetal life]
└ bone marrow.

(B) IN CHILDREN:
- All bones having red bone marrow.
- Liver.
- Spleen.

(C) Adults: red bone marrow.
After 18-20 yrs.

If Red bone marrow removed in adults, then
RBC is formed from
[Liver & Spleen] Reticulo

ERYTHROPOIETIN

INTERMEDIATE NORMOBLAST

↓

LATE NORMOBLAST

↓

RETICULOCYTE

↓ ③ Erythropoiesis → Release into blood

ERYTHROCYTE / RBC

CHANGES DURING ERYTHROPOIESES:

① cell size ↓

② cytoplasm ↑ in amount

③ Nucleus ↓ in size

④ staining rxn of cytoplasm changes.
[due to gradual production of RNA amount)

+ BABA

Deep basophilic - initial stage

↓

polychromophilic (both acidophilic + basophilic)

↓

Acidophilic (RBC) - FINALLY
↳ stains with basic dye

⑤ Nucleus.

initially → size - very big
+ contain many nucleoli.

↓ MATURITY of cell.

Finally → Nucleus & Nucleoli - disappear.

Hypoxia.

↓ (+)

Peritubular capillaries of Kidneys.

↓

Erythropoietin.

→ takes 4-5 days for Action.

→ also secreted in ↓ amount by [liver
brain

Function - (3) - PTB.

(2) **Thyroxine** - accelerates the process of erythropoiesis.

(3) **Haemopoetic Growth Factors:**

- stem cell factors (steel factors)
- interleukins (IL) [glycoproteins belong to cytokines family

(+) ↓

IL-3
IL-6
IL-11

] involved in erythropoiesis

proliferation of PHSC.

+ Other disorder.

(I) Vit B. - def. causes Anaemia
+ Pellagra

(II) Vit C. - Anaemia
+ Scurvy

(III) Vit D. - Anaemia + Rickets

(IV) Vit. E - Anaemia + malnutrition

~~Vit A~~

I ... Fibrinogen
II Party - Prothrombin
III Today - Thromboplastin
IV Come on - Ca++
V Lets - Labile Factor
VI - Ans not approved.
Sing VII ~~Sing~~ - Stable Factor
And VIII ~~Carrots~~ - Anti Haemophilic Factor - A
Call 6 IX ~~Ftzz~~ - Christmas Factor / PTC / AHF - B
Senior X ~~Laure~~ Stuart - Prower Factor.
Alzz XI [AHF-C] PTA (Plasma Thromboplastin Antecedent)
Laure XII - Hageman Factor — Anti haemophilic Factor
Tim XIII - Fibrin stabilising factor.

(8) VIII - AHF-A, AHG (Anti Haemophilic Globulin)

(9) IX - [AHF-B, PTC (Plasma Thromboplastin Component). [Christmas factor

(10) X - AHF-C

(11) XI - AHF-C - PTA - Plasma Thromboplastin Antecedent.

KEY RXN. with calcium ... activate X

BLOOD CLOTTING MECHANISM

conversion of blood changes

INTRINSIS SYSTEM

start by → change in blood components

12 → XII a

11 → XI a

9 → IX a

VIII a

X → (Ca++) → X a

5 → Prothrombin 2 → (Ca++) → II a Thrombin

13 → 1 → (Ca++) → I a

Fibrinogen (soluble)

fibrin (insoluble) ↓ Clot.

KEY RXN: with calcium ions (Ca++) activate

EXTRINSIS SYSTEM

* start by body tissue damage

3 (III) → VII a → 7 (VII) in active

V → (Ca++) → X a

4 (Ca++)

body tissue gives

	BLEEDING TIME. (BT)	
(i)	- 3 - 6 min	- 3 - 8 min
(ii)	- time interval b/w oozing out of blood from cut / injury till stoppage of bleeding	- time interval b/w the oozing out of blood from cut / injury till formation of clot.
(iii)	- BT prolonged in purpura	- prolonged in haemophilia
(iv)	- determined by Duke's method.	- determined by capillary tube method.

3- ♀ - carrier

4- CT - prolonged.
5- BT - normal

6- Cause: lack of formation of prothrombin activator
↳ deficiency of factor VIII, IX, XI

7# Types of Haemophilia:

Haemophilia A / Classic Haemophilia	Haemophilia B / Christmas Disease	Haemophilia C
— 85%	15%	very rare
— deficiency of factor		
⊕ Factor VIII (ANFA)	IX (ANF-B) ↳ PTC ~~thromboplastin factor~~	XI (ANF-C) ↳ PTA
—		

8# SYMPTOMS:

1. even small cut / injury leads to great loss of blood from the body
2. Haemorrhage
3. prolonged bleeding.
4. bleeding in joints
5. Blood in Urine
6. spontaneous bleeding.

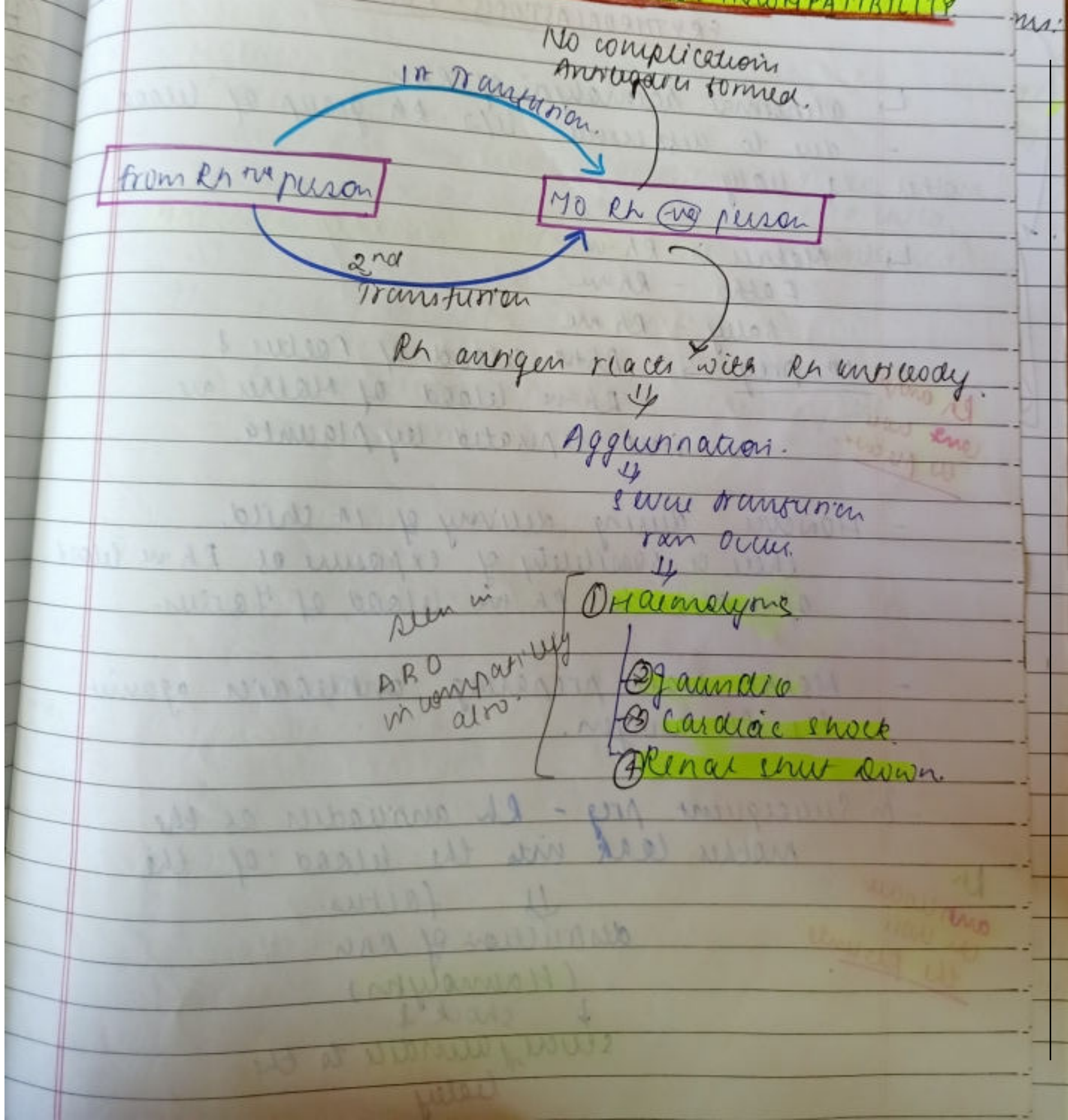
① - prolonged bleeding time.
② - CT. normal.

SYMPTOMS ① spontaneous bleeding under skin from ruptured capillaries.

② small tiny hemorrhagic spots under skin **Purpuric spots** (purple coloured patch like appearance)

Types :-

THROMBOCYTOPENIC PURPURA	IDOPATHIC THROMBOCYTOPENIC PURPURA.	THROMBOSTHENIC PURPURA
↳ due to Thrombocytopenia (platelets count ↓)	↳ unknow cause ↳ Thrombocytopenia (↓ platelets count)	↳ Platelet count - Normal.
↑ Bone marrow disease	↑ Antibodies against platelets	↳ structural & functional abnormality in platelets.
	Blood Transfusion	↳ C.T = N B.T - N / prolong - defective clot retraction.



mother & baby

↳ Mother is - Rh -ve
Father - Rh +ve.
Baby - Rh +ve

Rh antigen cannot cross the placenta.

1st preg. → Rh+ve blood of foetus & Rh -ve blood of Mother are well seperated by placenta.

- However, during delivery of 1st child, there is possibility of exposure of Rh -ve blood of mother to Rh +ve blood of foetus.

- Mother starts preparing antibodies against the Rh antigen.

- In Subsequent preg - Rh antibodies of the mother leak into the blood of the foetus.

Rh antibodies can cross the placenta

↓
destruction of RBC
(Haemolysis)
↓ shock &
severe jaundice to the baby.
may be fatal.

FETALIS.

① Mother - Rh -ve.
Father - Rh +ve.
— anti Rh antibodies administered to mother immediately after delivery of 1st child.
- Anti D antibodies - within 48 hrs administered.

② If baby is born with E.P.
Rh -ve blood is transfused into Rh +ve blood of ~~father~~ infant replacing its own Rh +ve blood.

- Life saving procedure.

Precautions ① Donor must be healthy without any STDs like AIDs, Hepatitis B, etc.
② Rh compatible
③ Matching & Cross Matching.
④ Only compatible blood must be transfused.

HAZARDS. Of BLOOD Transfusion:

⇒ are of ~~Due to~~ 4 types:—

① Reaction due to Mismatched Blood Transfusion - Transfusion Reactions:
due to ① ABO incompatibility.
② Rh incompatibility.

→ leads to :-
volume.

(i) circulatory shock.
(ii) Hypocalcemia - leading to Tetany.
(iii) Hemosiderosis
↳ The deposition of Fe in the form of ~~Tetany~~ hemosiderin.

(3) Reactions due to faulty Techniques during Blood Transfusion:
leads to:
(i) entrance of air into blood stream. (embolism)
(ii) Inflammation of veins.

(4) Transmission of infections:
(i) STDs - AIDs.
Hepatitis B.
Herpes
Bacterial infections.

① = Rate at which erythrocyte settle down.
② - Normally, the RBCs remain suspended uniformly in blood during circulation. This is called Suspension stability of RBCs.

- (i) easy
- (ii) inexpensive
- (iii) non-specific test
- (iv) useful in [Diagnosis, Prognosis.

- Factors Affecting ESR :-

(i) Rouleaux Formation accelerates ESR.
(ii) specific gravity of RBCs & ESR
(iii) ↑ in RBCs size (Macrocytes) - ESR ↑.

Non-specific Immunity

↳ Present at the time of birth.

↳ 1st line of Defence.

(1) Skin - barrier for the entry of pathogens.

(2) Mucous membrane of GIT, Respiratory & Urogenital Tract. — Traps microbes.

(3) Macrophages, Neutrophils & NK - Type of lymphocytes present in body. — phago cytes kills

(4) Interferons - are secreted by virus infected cells which protect the non infected cell from further viral infections.

Acquired Immunity/ Specific Immunity.

↳ most powerful immune mechanism

↳ resistance developed by the body against specific pathogen which may be bacteria, viruses, toxin, etc.

↳ governed by the immune system

Humoral Immunity

Cell-Mediated Immunity

produces an army of proteins in our blood in response to antigen ~~Pathogens~~ to fight with them

↓

Antibodies.

② ↓ maturation
processed in
Thymus.
& produced in
bone marrow
stored in lymphoid tissues.

① Helper T cells - helps in the killing of pathogen & in AIDs.

⊕

② Cytotoxic T cells : kills the cell by making perforin lined pores

③ Suppressor T lymphocytes suppresses the immune system & prevents cells harm to the body

④ Memory T cells - remembers the pathogen. Which on subsequent encounters with the pathogen produces a highly intensified/ anamnestic immune response.

immune response in our body

Auto Antigens / Self Antigens
antigens present in own body's cells
Ex. RBCs contains Antigen A & Antigen B.

Foreign Antigen.
(i) Allergens - induce allergy
Ex- pollen, mites, animal dander.
(ii) Toxins from microbes.
(iii) Materials from transplanted organs.

↳ proteins produced in response to antigens by B-lymphocytes
↳ released into blood.

Ig G
Ig A
Ig M
Ig D
Ig E.

during the time of an organism indicating that it is not a congenital disease.

↳ virus- HIV (Human immunodeficiency virus)
↳ enters the body.
↓
enters macrophages.
↓
Using the enzyme Reverse transcriptase.
HIV converts its viral RNA → DNA
Reverse transcription
viral DNA gets incorporated into host cell DNA.
The host cell treats viral DNA as if its own & subsequently manufactures more virus particles-
↓
Released HIV attacks
helper T lymphocytes & kills them
↓
progressive fall in the no. of
Helper T lymphocytes in the body.

∴ patient becomes so immunodeficient that the he starts suffering from diseases that could otherwise have been avoided.
- leads to wt. loss.

Mode of Transmission

(i) from infected mother to foetus through placenta.
(ii) blood transfusion
(iii) intravenous drug abusers.
(iv) unprotected sexual intercourse.
(v) use of infected needles
syringes & surgical instruments.

- Time lapse b/w infection & the appearance of AIDS symptoms range from few months to as long as 5-10 yrs.

platelet is responsible for Intrinsic Prothrombin Activator.

↓

responsible for onset of blood clotting.

(II) Role in Clot Retraction: blood cells including platelets are entrapped in fibrin threads.

(III) Role in prevention of blood loss (HAEMOSTASIS):
Platelets accelerate haemostasis by
(I) formation of temporary plug
(II) adhesive property - stick to the wall of blood vessels.

(IV) Role in Repair of Ruptured Blood vessels.
Platelet Derived Growth factor (PDGF) helps for repair of endothelium

(V) Role in Defense mechanism:
platelets encircle foreign bodies by the property of Agglutination
↓
Kills them