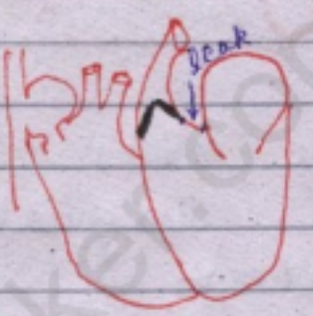
83-88 Extrinsic Hemolytic Anemia

Non 9-Mediated

Mechanical Cause

- Macroangiopathic H·A
- Micro " H·A

Macro Angiopathies H·A



① Mech Cardiac Value
(Eg. If there is prosthetic aortic value. & there is a leak at its side while RBC passes through this leak it is destroyed)
② Calcific Aortic Stenosis
③ March hemoglobinuria
④ Bongo Drummer

Micro Angiopathies H·A

DIC ← fibrin Thrombi
platelate Thrombi → T·T·P (Thrombotic Thrombocytopenic purpura) / HUS

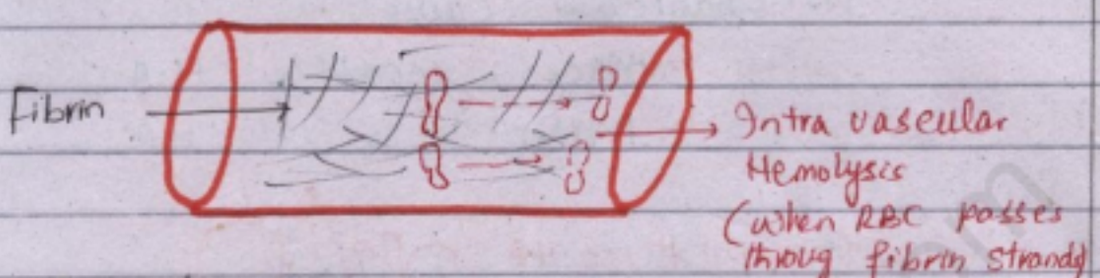
In such cases RBC are segmented known as Schistocyte, Triangular cells Helmet cells

It is an example of Intravascular hemolysis

Examples

DIC
T·T·P
HUS

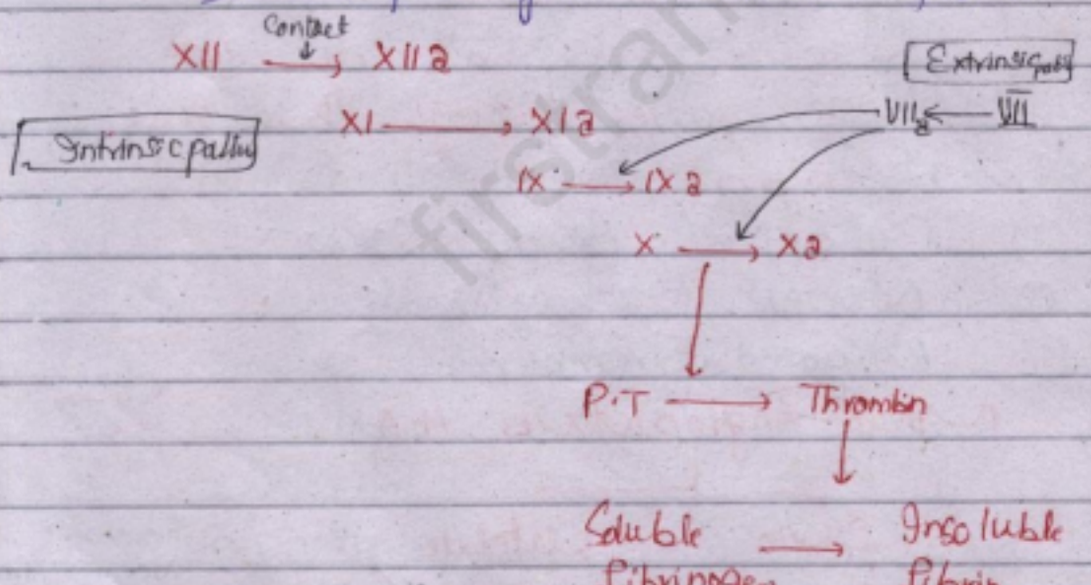
DIC :



Primary Problem is Wide spread activation of Coagulation System.

Coagulation :-

Soluble fibrinogen → Insoluble fibrin



+tion of Coagulation System

- AML
- Septecemia (Endotoxin)
- Obstetric Complications (Amniotic fluid)
- Cancer
- Massive tissue damage.

Not Only Anemia occurs but later on platelate also used & ↓ platelate No

As in this process all clotting factors platelets are consumed

It is known as

Consumption Coagulopathy

& pt start bleeding

e.g

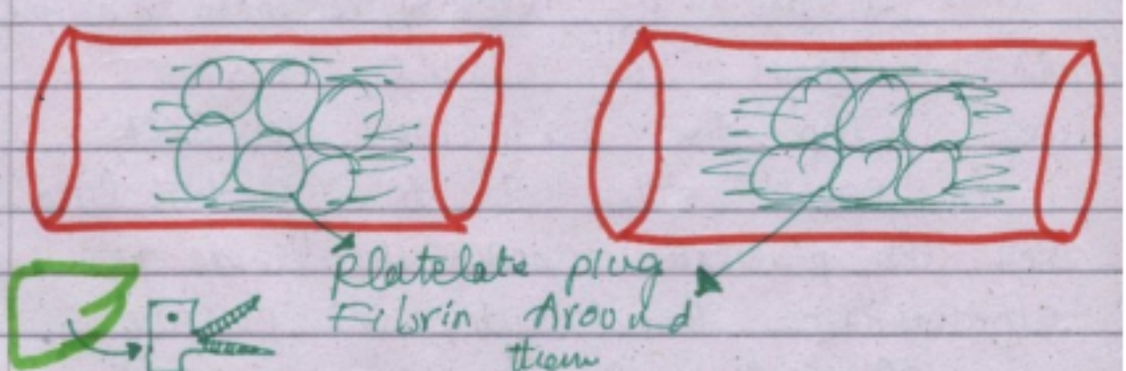
PPH (Post partum Heamorrhage)

Steps

1. wide spread Endo damage
2. Activation of Coagulation factor
3. Lot of fibrin strands
4. RBC fragmentation
5. Activation of fibrinolytic System
6. Bleeding tendencies

Platelate Thrombi

T.T.P HUS

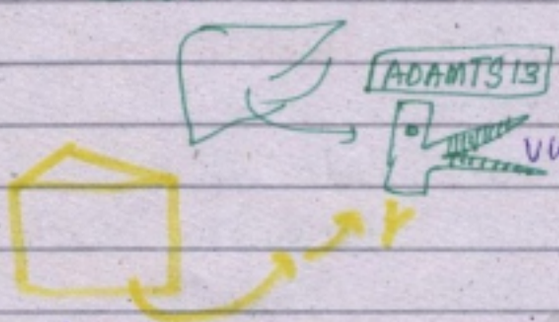


Primary Problem is wide Spread platelate activation & Aggregation.

T.T.P :-

Some many platelate thrombi are formed that many many platelate are used & leads to small Hearmorhage ie purpura

Cause



This pr Normaly cuts VWF & prevent Extra accomulat

Any defect in this pr leads to T.T.P.

① Inherited defect
② Acquired defect (auto antibodies)

This is one of Metaloproteinase Enzyme Ten level of VWF Tes they help the platelate to stick to each other & to Endothelium So all platelate consomed leads to Thrombotic thrombocytopenic purpura

Clinically:

- Fever
- Ischemia & damage → Renal sys, CNS
- Microangiopathic H.A due to blockage of Microcirculation

- Transient Neurological deficit
- Renal failure (Nephropathy)
- Thrombocytopenia

HUS :

They have eaten undercooked food with special type of Ecoli

$O_{157}H_7$

Pt in childhood developed Ecoli induced Gastroenteritis

Shiga toxin is also produced by Ecoli

It damages → Endo → platelets stick to Endothelium & platelets fragment → Intravascular Hemolysis.

In this case Renal damage is more than those in T.T.P such severe damage to glomeruli leads to Uremia

CNS does not suffer.

	TTP	HUS
1)	Microangiopathic H.A	Microangiopathic H.A
2)	Platelete def	platelete def
3)	CNS Involument	No CNS Involvement
4)	~~Less Renal damage~~	More Renal damage
5)	Normal P.T & PTT	PT & PTT > Normal
6)	dense platelete ↓	dense platelete ↓

DIC

(prothrombin times) P·T = ↑↑

(partial Thromboplastin time) P·T·T = ↑↑

d dimer = (activated fibrin)

Immune Mediated

Iso Ab — ABO Rh Incompatibility

Auto Ab — Warm agglutinin, Cold agglutinin, Cold Hemolysis