KAZAN STATE MEDICAL UNIVERSITY

PRESENTATION

IDIOPATHIC THROMBOCYTOPENIC PURPURA

NAME – ABHINAV SUMIT KUMAR

GROUP - 1527

IDIOPATHIC THROMBOCYTOPENIC PURPURA

It's a bleeding disorder

IT IS A DISEASE CHARCTERISED BY SEVER REDUCTION OF PLATELET NUMBERS, CAUSED BY IMMUNE DESTRUCTION OF PLATELETS.

- THE MECHANISM OF I.T.P. APPEARS TO BE IMMUNE COMPLEXES CONTAINING ANTIBODIES, WHICH REACT WITH PLATELETS AND LEADS TO THEIR IMMUNOLOGICAL DESTRUCTION.

IMMUNE THROMBOCYTIC PURPURA (ITP) (IDIOPATHIC THROMBOCYTOPENIC PURPURA)

Autoimmune antibodies against platelets from respone to previous viral infection (ACUTE ITP) or

from underlying dysregulation (CHRONIC ITP)

- **Causes**
- **Immunoglobulin G (IgG) autoantibodies on the platelet surface.**

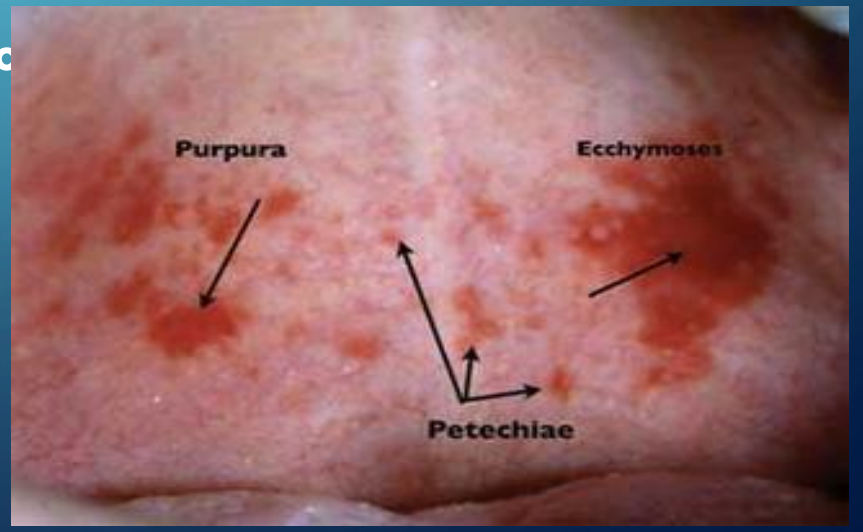
- PLATELET DESTRUCTION
- (THROMBOCYTOPENIA)

CLINICAL MANIFESTATIONS

- Clinical signs and symptoms of ITP are reflective of thrombocytopenia and,
- (bleeding manifestation due to decreased platelet count)

CHARACTERISTIC CLINICAL FEATURES

- Common signs, symptoms, and precipitating factors include the following:
- Petechiae – small 2mm Red or Purple spot on skin caused by bleeing from broken cappillaris and blood vessels. Often located on extremities or on mucous membrane.
- Ecchymosis – bleeding underneath the skin typically caused by bruising >1-2cm
- Purpura – red or purple spot on skin that does't blench on applying pressure >3mm
- Menorrhagia – Menstrual period with abnormally prolo
- Epistaxis
- Gingival bleeding
- Recent live virus immunization (childhood ITP)
- Recent viral illness (childhood ITP)



PATHOGENESIS OF I.T.P.

- Increased platelet destruction caused by anti platelet antibodies antibodies directed against platelet membrane antigen.
- Lack of compensatory response by megakaryocytes due to suppressive effect of anti platelet antibodies
- So, a combination of increased platlet destruction + ineffective megakaryopoiesis.
- Pathogenesis was proved by harrington when he infused hemself with plasma from a women with ITP (Harrington hollisworth Experiment)
- ITP is primarily a disease of increased peripheral platelet destruction, with most patients having antibodies to specific platelet membrane glycoproteins. Relative marrow failure may contribute to this condition, since studies show that most patients have either normal or diminished platelet production.

CLINICAL FEATURES THAT ARE CHARACTERISTRISTICALLY UNUSUAL AND SUGGEST THE POSSIBILITY OF OTHER DIAGNOSIS :-

- Clinical history & Physical examination of Patient
- CBC & Peripheral blood smear
- Testing for autoimmune disorder & exclusion of HIV antiplatelet.

  Frequent bleeding and haemorrhages from larger veins and arteries and bleeding into joints (SUGGEST COAGULATION DISORDER)
- Splenomegaly and lymphadenopathy are the unusual/rare and their presence should lead one to consider other diagnosis.
- Hepatomegaly, splenomegaly and lymphadenopathy are notably absent in ITP and their presence should initiate an investigation for other possible underlying illnesses associated with thrombocytopenia.

DIAGNOSTIC CONSIDERATIONS

OTHER PROBLEMS TO BE CONSIDERED IN THE DIFFERENTIAL DIAGNOSIS INCLUDE THE FOLLOWING:

PSEUDOTHROMBOCYTOPENIA (PLATELET CLUMPING IN THE PRESENCE OF ETHYLENEDIAMINETETRAACETIC ACID [EDTA])

LIVER DISEASE

MYELODYSPLASIA

LYMPHOPROLIFERATIVE, AUTOIMMUNE, OR INFECTIOUS DISEASES

PREGNANCY-ASSOCIATED THROMBOCYTOPENIA

DRUG-INDUCED IMMUNE THROMBOCYTOPENIA (ALCOHOL, HEPARIN, QUININE/QUINIDINE, SULFONAMIDES)

INFECTION/SEPSIS

ACUTE LEUKEMIA

MYELODYSPLASTIC SYNDROME

MALIGNANCY

MEGALOBLASTIC ANEMIA

ISOIMMUNE NEONATAL PURPURA

TRANSFUSION

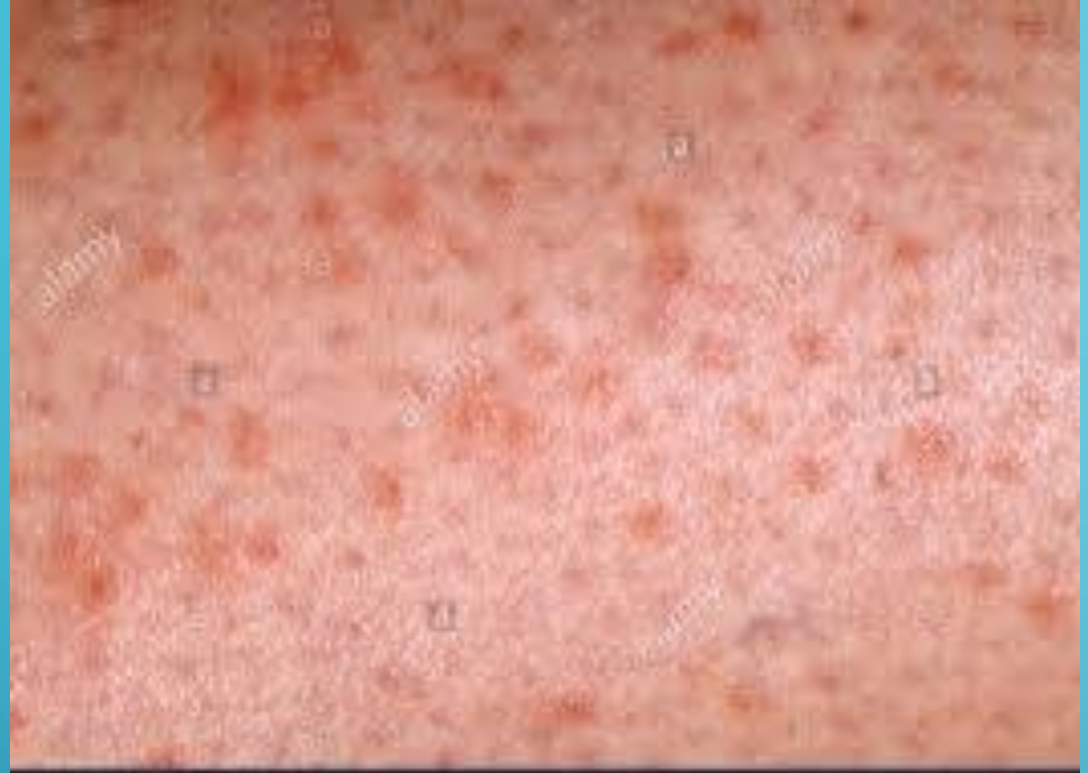
FACTITIOUS

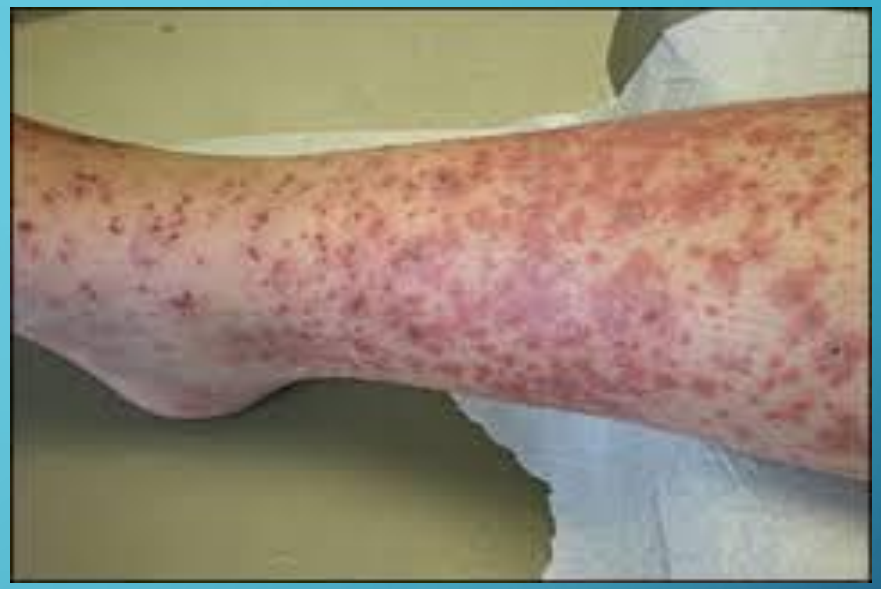
DIFFERENTIAL DIAGNOSES

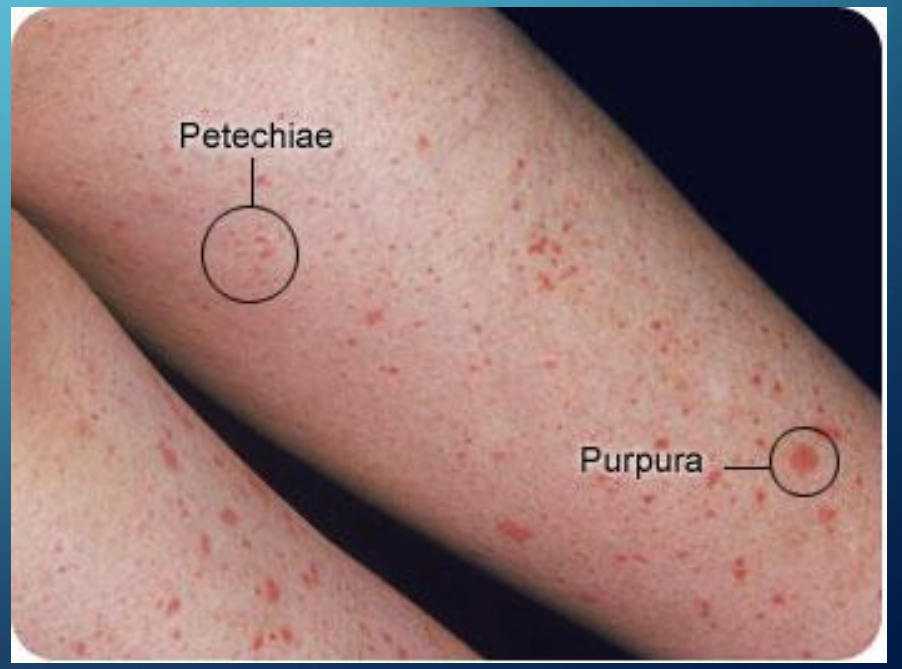
DISSEMINATED INTRAVASCULAR COAGULATION

RAPID TESTING FOR HIV

THROMBOTIC THROMBOCYTOPENIC PURPURA (TTP)






Petechiae
Purpura

LABORATORY DIAGNOSIS

- Isolated thrombocytopenia &
- Platelet antibodies are key diagonostic method for ITP
- Normal or increased number of megakaryocytes on bone marrow examination
- ***FEATURES OF BLEEDING DISORDER***
- Bleeding time is prolonged
- Tests of coagulation are essentially normal
- Normal PT
- Normal APTT
- Normal Fibrinogen Degradation products

IMAGING STUDIES

A CT SCAN OF THE HEAD IS WARRANTED IF CONCERN EXISTS REGARDING INTRACRANIAL HEMORRHAGE.

FEATURES OF ACUTE AND CHRONIC I.T.P.

- I.T.P. Occurs in two forms, namely ACUTE I.T.P. and CHRONIC I.T.P.
- ACUTE I.T.P. and CHRONIC I.T.P. differ in incidence, prognosis and therapy.

CLASSIFICATION OF I.T.P

Features	Acute I.T.P	Chronic I.T.P
Peak age of incidence	Children, 2-6 years	Adults 20-40 years
Sex predilection	None	3:1 female to male
Antecedent infection	Common 1-3 week before	Unusual
Onset of bleeding	Abrupt	Insidious
Hemorrhagic bullae in mouth	Present in severe cases	Usually absent
Platelet count	<20,000/microliter	30,000-80,000/microliter
Eosinophilia	Common	Rare
Duration	2-6 week : rarely longer	Months or years
Spontaneous remission	Occur in 80% of cases	Uncommon

TREATMENT MODALITIES FOR I.T.P.

- Bed rest untill Platelet count increase.
- Diet – Papaya leaf extract, , Pomegranate, milk, vitA, Cod liver oil.
- Common treatment modalities for I.T.P. (platelet count <20,000/microliter or significant mucosal bleeding) :-
- Corticosteteroids - Prednisone (Deltasone, Orasone, Sterapred)

  Methylprednisolone (Solu-Medrol, Depo-Medrol)
- IV Immunoglobulins/gamma globulins Carimune NF 400-800 mg/kg

  Flebogamma 300-600 mg/kg
- Additional immune suppressive agent (rituximab/anti CD 20 antibody)

Complications of ITP

- Bleeding from the GI tract
- Hemarthrosis
- Intracranial hemorrhage
- Prevention may be helped by immunizing all children against the viral diseases of childhood

THANK YOU