# AUTOIMMUNE HEMOLYTIC ANEMIAS

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# IMMUNE HEMOLYSIS

- Increased destruction of RBC's by antibody against antigens on RBC

-Hemolytic disease of newborn

-Drug induced

-Hemolytic transfusion reactions

-Autoimmune hemolytic anemias- Patient makes antibodies to antigens on own RBC's

- Divided into Warm, Cold and PCH

# COLD TYPE

- Acute or insidious onset
- Mild to severe anemia
- Female preponderance
- IgM antibodies
- Agglutination of red cells at low temperatures
- Complement activation
- Intravascular and extravascular hemolysis
- Antibodies commonly against I antigen, rarely HI and i

# COLD TYPE

- Acute postinfectious

  Acute, self limited, younger patients
- Chronic idiopathic

  Insidious, older patients
- Cold agglutinin disease (CAD)

  Insidious, elderly women, associated with lymphoproliferative disorders

# WARM TYPE

- Abrupt onset
- Severe anemia
- Slight female preponderance
- IgG antibodies
- Extravascular hemolysis
- Antibodies commonly against broad Rh antigens

# PAROXYSMAL COLD HEMOGLOBINURIA

Rare

Sudden onset of hemolysis

Follows viral infection in children

IgG antibody directed to P antigen

Binds to red cells at low temperatures- when warmed, activates complement causing hemolysis

Donath Landsteiner test- pt's serum + RBC, incubated at low followed by high temp: hemolysis occurs if antibody is present

# ORIGIN OF ANTIBODY

| | WARM (70%) | COLD (30%) |
|---|---|---|
| Idiopathic | 50 – 60 % | 30 – 40 % |
| Drug induced | 25 – 30 % | 1 – 5 % |
| Lymphoproliferative disorders | 10 – 15 % | 15 – 20 % |
| Infections | 0 % | 25 – 35 % |
| Others | 5 – 10 % | 5 – 10 % |

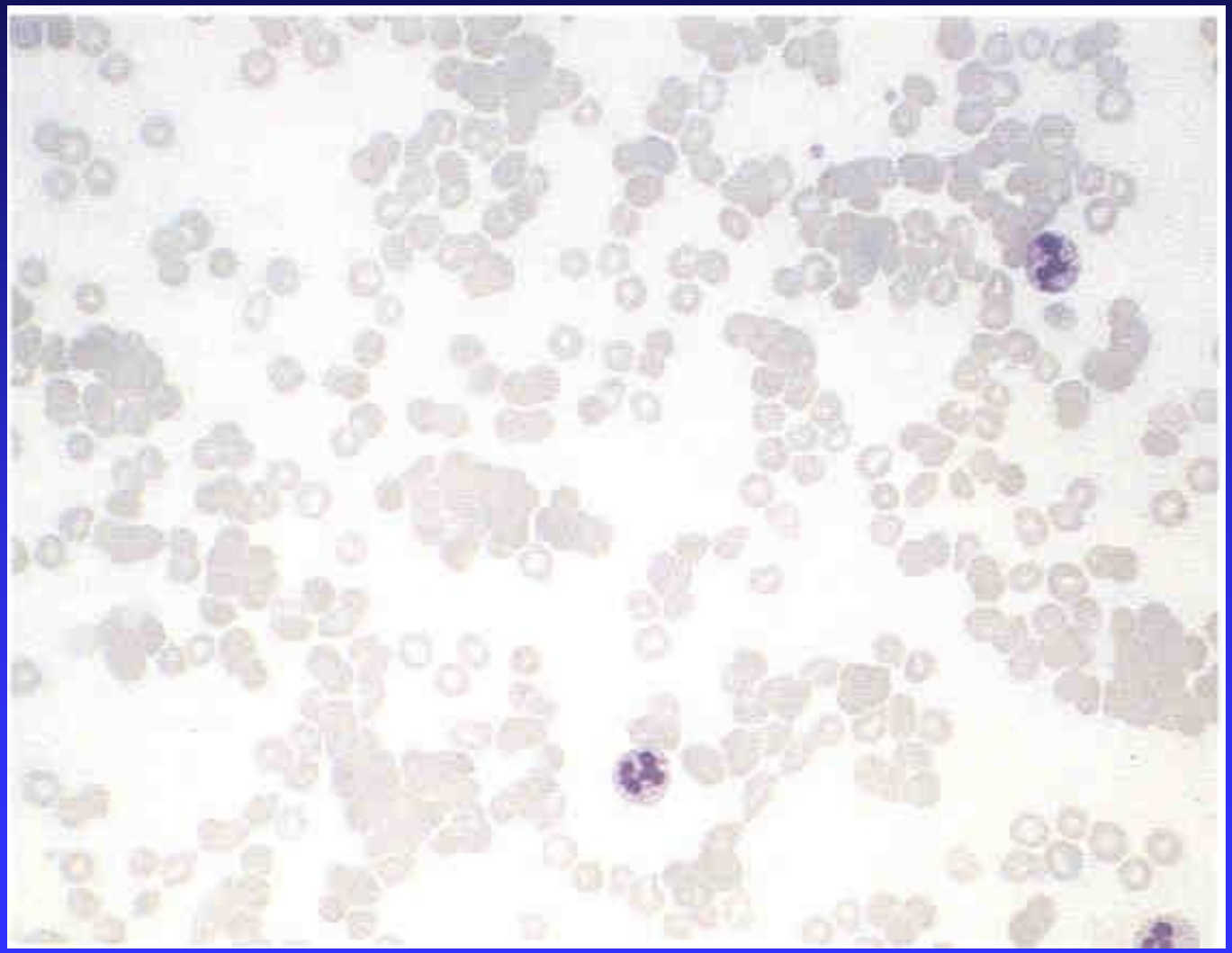
# LAB PARAMETERS

| | WARM | COLD |
|---|---|---|
| DAT | 2+ TO 4+ | 2+ TO 4+ |
| ANTI IgG | 1+ | 0 |
| ANTI IgG + C3 | 1+ | 0 |
| ANTI C3 | RARE | 1+ |

| | WARM | COLD |
|---|---|---|
| COMPLEMENT ACTIVATION | Little to none | Present |
| SERUM COMPLEMENT | Normal to low | Low |
| OSMOTIC FRAGILITY | Increased | Normal |
| PERIPHERAL BLOOD | Spherocytes, Nucleated RBCs | RBC agglutination |

# WARM AIHA

# COLD AIHA

- 

# TREATMENT

- WARM AIHA

-Corticosteroid therapy

-Splenectomy

-Intravenous IVIG

-Immunosuppressants

- COLD AIHA

-Immunosuppressants and splenectomy: no role

-Plasmapheresis

-Keeping patient warm

-Treatment of underlying disease

# BLOOD BANK IMPLICATIONS

- Autoantibodies react with all cells due to broad specificity (Rh null cells rare)
- Determination of patient's extended rbc phenotype (helps in future transfusions)
- Identification of underlying alloantibodies
- Removal of autoantibody reactivity: essential
- Adsorption techniques- two types: autologous and allogenic

# Autologous adsorption

No h/o transfusion or pregnancy

Pt's serum + own RBC's

Autoantibodies adsorbed onto cells

Alloantibodies left in serum

Further testing for specificity

# Allogenic adsorption

H/o transfusion or pregnancy

Pt's serum + panel of RBC's of known phenotype (use pt's red cell phenotypes if known)

Autoantibody adsorbed onto all cells

Alloantibody also removed if corresponding antigen present and left in serum if antigen absent

Further testing for specificity

# Cold AIHA

- Avoid interference by pre-warming techniques
- Cold autoadsorption useful

# TRANSFUSION IN AIHA

-Avoid blood transfusions if possible – shortened survival and alloantibody induction

-“Completely” compatible blood unavailable

-Instead, give “least incompatible blood”

-Leukoreduced RBC’s preferred (reduces FNHTR)

-Transfusion of smaller volume at a time

-Blood warmers for cold type

# NEWER DEVELOPMENTS

- Use of flow cytometry to detect and quantify RBC bound antibodies- precise, reliable and more sensitive method for detecting antibodies
- Gel centrifugation test – less expensive and easily available
- Rituximab, anti CD20 monoclonal antibody, as a therapy for refractive AIHA