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Board Review

Serology
Blood group antigens vs. systems

General rule of thumbs

Antigen: On RBCs; targeted by alloantibody

Warm Reactive Antibody, IgG, exposure, HDN

Cold Reactive Antibody, IgM, Natural, No HDN, No HTRs, Insignificant

System: group of genetically linked blood group antigens
ABO is built on:

N-acetyl glucosamine
B: Galactose
H: Fucose
A: N-acetyl galactosamine

Type II

On RBC *membranes glycolipids*, but also *glycoproteins*.
Lewis Antigens are built on

Type I or Type II?

Type I

Secretions, primarily **glycoproteins** or glycolipids?
Secretor (FUT2): Adds Fucose to which position? Terminal galactose
Le (FUT3): Adds Fucose to which position? Subterminal N-acetyl glucosamine
Racial aspects of ABO

1. Who has most type O? Whites, Asians, Blacks or NA?

2. Who has least AB?

3. In which group is the percentage of A and B about equal?

4. A1 has _______ fold more antigens than A2.

5. What antibodies are in Bombay phenotype of a person with AB genotype

6. Which antibodies are in paraBombay with AB genotype
1. Antigens decrease during pregnancy

2. Will Le (a-b+) make anti Lea?

3. Infants Can detect Le (a+b-), Le (a+b+) before transitioning

4. H pylori and Norwalk attach via Leb

5. Le (a-b-) kids may get Ecoli UTIs
I system

1. Type II chains

2. I in adults; i in babies

3. Older people have greater branching

4. Auto anti I can be seen in?

5. What about anti i?
P system

1. ABO related chains

2. P1 is the antigen

3. P, Pk antigens help define the P type

4. Most are P1 (P+, P1+Pk-)
5. Acute HTRs and spont abortions if lack all three
6. P is receptor for parvovirus
7. Anti P1 is cold, insignificant, IgM
   - Increased in bird handlers and hydatid cyst
8. PCH association/Donath Landsteinter-IgG to P
What are the three phases of testing

1. Immediate spin; 2-5% solution, serum, spin
2. 37C ---potentiator, incubate spin
3. IAT (AHG), wash, add AHG spin
# Proteolytic Enzyme

1. **Ficin/papain:** can increase or decrease the ability to detect specific antigens on RBCs
2. Enhanced | Decreased | Unaffected
   | ABO, Le, I, P, Rh, Kidd | MNS, Fy, Lu | Kell, Diego, Colton

1. **Inhibitory substances:** Neutralization

   - ABO
   - Le
   - P1
   - Sd
   - Chido/Rodgers
Lectins

Dolichos biflorus  A1
Ulex Europaeus  H
Vicea Graminea  N
Arachis hypogea  T
Rh

1. Two genes, five antigens, D, E, e, C, c

2. Rh is inherited as a group, haplotype
   Ce  cE  ce  CE

R1:
R2:
R0:
Rz:
r':
r'”:
r:
ry:
Rh

1. Exposure requiring warm IgG
2. Recent data 20-30% will make ab if given a single unit D+ RBCs
3. HTRs primarily extravascular
4. HDFN; also severe with anti c but mild with others
5. Weak D vs. partial D
   1. Quantitative
   2. Qualitative
1. Jka > Jkb (unusual)

2. 50% fix complement

3. Marked dosage

4. Variable expression with time
1. Glycophorin A (MN)
2. Glycophorin B (S U)
3. M=N
4. s>S
5. 2% Blacks lack S, s and U (lack glycophorin B)
Duffy

1. Fy (a-b-) in ~70% blacks; rare in caucasions

2. Their genotype **FyFy**

3. Exposure require warm IgG

4. FyFy people are resistant to P vivax and P knowlesi infection
Lutheran

1. Lu a is high frequency
2. Antibodies IgG mild HTRs, no HDFN
Kell

1. K (K1); k (cellano, K2)
2. K2 in 99.6% (high freq)
3. Other kell antigens also high freq:
4. Kx antigen closely attached to Kell antigens on RBCs
5. K1 is very immunogenic (after ABO and Rh)
6. Exposure required with development of IgG
7. Severe HTRs and HDFN
8. K null
   • No Kell antigens
   • Kx increased
   • Anti Ku
   • McLeod syndrome: absence of Kx; decreased Kell ags, acantholytic hemolytic anemia
   • X-linked CGD (NADPH oxidase)
Diego

- On Band 3 anion exchange
- Di a rare; Di b high freq; Di a is not rare in Asians and native americans
Colton

- Unusual, Co a is very high frequency; Co b is low frequency
- Antibodies are usually IgM
- Anti Co can cause serious HDFN