



**RhAPP**

RHEUMATOLOGY ADVANCED  
PRACTICE PROVIDERS

**RHAPP NATIONAL CONFERENCE**

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- Rana Aoufe, MPAS
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# Rheumatology Labs RHAPP

Presented By Jackie Fritz RN MSN RN-BC and Rana Aoufe, PA-C  
September 2022

# My First Day On The JOB

Everything in Nursing is named after some Doctor:

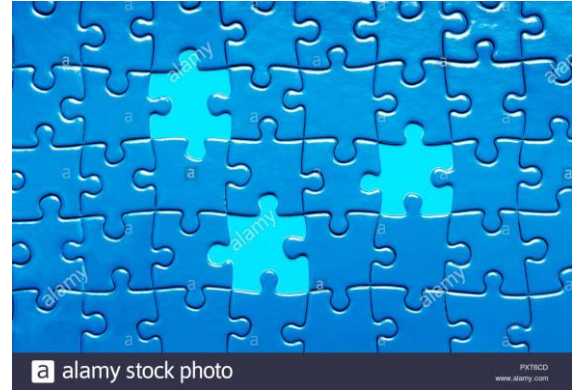
- Swan and Ganz: pulmonary Catheter
- Mobitz: heart Block
- Somogyi: rebound blood sugar
- Wolf Parkinson White: pre- excitation
- Kawasaki: Motorcycle: wait it had a Syndrome attached?
- Wegeners: Bad German Or shorten to help you Understand Granulomatosis with Polyangiitis: GPA? (Much better )
- So SSA and SSB: must have been some labs test for Supercalifragilisticexpialidocious

# Let's Start with the Basics

- Understand the principles of a lab test

- Preview of the Test

- Is it appropriate for the patient?
- Does it guide our diagnosis ?
- What does the test measure
- What are the limitations
- Lab tests are just “ONE PIECE” to a puzzle we are putting together



# ESR Erythrocyte Sedimentation Rate

- Measures Acute Phase reactant
  - Fibrinogen most common
  - Produced by the liver as a part of an inflammatory response : under control of cytokines IL6, IL1 and TNFa
  - RBC are the messengers of the fibrinogen levels
    - Fibrinogen interacts with RBC to make the sediment fall more quickly
    - Acute phase reactant ( CRP) more sensitive

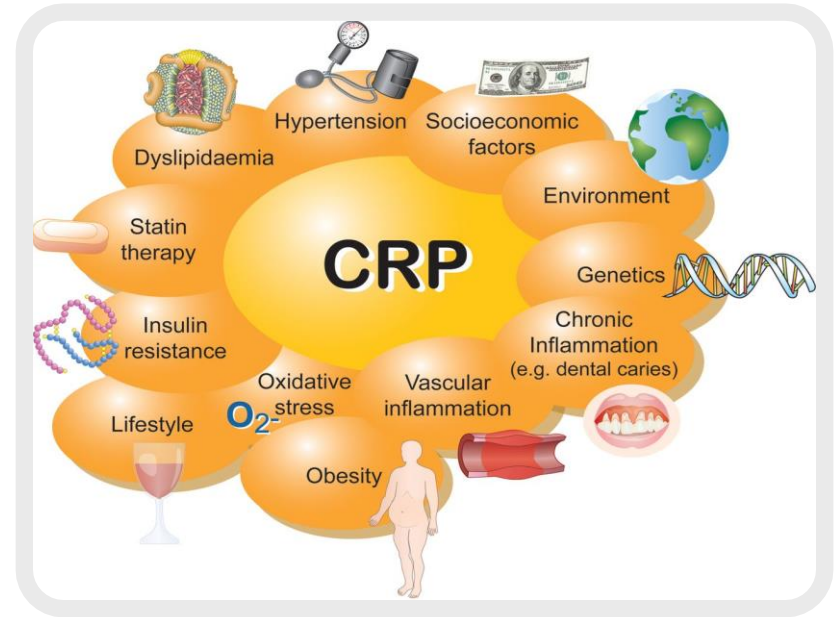
# Deviations of ESR

- Women have higher ESR and men
- ESR rise with age
- ESR above 40 mm/h may be associated w:
  - PMR, RA
- Very non-specific
  - Elevated:
    - Infections
    - Malignancy
    - Anemia
    - And some cholesterol lowering agents



# CRP Capsular Reactive Protein

- Used to help define acute phase reactivity
- Combined with ESR: called acute phase reactants
- Used as a measure of how well disease is being managed
- Also can be responsive to many other things than just inflammatory processes



# ESR/CRP

Important to understand they are NON-specific and NON sensitive.

Ask yourself: With very elevated APR's, have you ruled out **infection** and **malignancy** ?

Always important to have them as a differential !

# Rheumatoid Factor and RA

## Prevalence increases and disease duration

- 30 – 50% positive at disease onset
- 75%+ after one year
- 80% after 18 months
- Prognostic Significance
  - PRESENT 1-5% POPULATION
  - INCREASE W AGE>65 10%
  - **HARDLY EXCLUSIVE TO RA**

# Rheumatoid Factor but NOT RA

- Collagen Vascular diseases
  - Sjogren's Syndrome
  - SLE
  - Cryoglobulinemia
  - Scleroderma
  - Poly/Dermatomyositis
  - **NOT ALL RF+ is Rheumatoid Arthritis**

# RF + Non - Rheumatic Diseases

- Chronic Infections: (Again, are we ruling out infection first?)
  - Elicit Chronic immune response
    - Hep C
    - Endocarditis
    - Osteomyelitis
    - Syphilis
    - Cirrhosis
    - Sarcoidosis

# CASE STUDY

63 y.o male with a highly +RF and negative CCP. Complains of swelling and pain across MCP joints. Rheumatoid arthritis was thought to be the diagnosis and more labs were ordered.

# DIAGNOSIS

- A Red Herring: Hepatitis C
- Check RNA for active disease
- Chronic or acute
- Treat with antivirals: such as Glecoprevir/Pibrentasir
- Brand Name: Mavyret
- Or Sofosbuvir Sovaldi (\$80,000) covered by medical

# Rheumatoid Arthritis

- Anti-CCP: Anticyclic citrullinated peptide
- Above 20 units is +
- Higher CCP the more active the disease
- 97% specific
- Will remain + despite remission
- ETA 14.3.3: good for early RA diagnosis



# Anti-CCP Anti-bodies and RA

- 70-80% Sensitivity
- 90-95% Specificity
- Will remain + even if in remission
- Not used to monitor disease activity
- Diagnosis by ACR criteria
  - 936 patients with early inflammatory arthritis
    - 205 by ACR Criteria
    - 318 Undifferentiated RA
    - 413 Other Diagnoses

# Pyoderma gangrenosum

- This gets better with Infliximab

Painful  
Progressive  
Purple  
Pretibial  
Pathergy

**Associated with** autoimmune disease;

UC  
Crohn's  
RA

## **Treatment:**

Antibiotics  
Steroids  
Biologics



# Case Study

- 45-year-old female with history of tick bite. Joint swelling especially MCP and PIP. Responds well to steroids given in short courses. All Lyme tests are negative. CCP and ETA 14.3.3 also negative. ESR and CRP are quite elevated.
- Parvo virus positive ????
- The Tick bit the dog (which had Parvo) and then the patient!

# Diagnosis

- Reactive RA:
- Treatment : steroids will resolve on it's own

# Immunoglobulins

Immunoglobulins play a key role in the body's immune system.

IgM - produced as body's first response to a new infection (short term production). This decreases as IgG production begins.

IgG - about 70-80% immunoglobulins in the blood are IgG - produced during initial infection, rising a few weeks after it begins and then decreasing and stabilizing. It can be rapidly reproduced when exposed to the same antigen. Form long term protection against microorganisms. (Ex. COVID IgG antibody test)

IgA- about 15% in the blood are IgA. Provides protection in mucosal areas (respiratory and GI tract)

# CASE STUDY

58 y.o with history of hypothyroidism presents with 2-week joint pain. Exam shows swelling across right knee, right ankle, MCP's/PIPs.

No fevers and has malar rash.

2 weeks prior grandson had febrile illness with headaches and body aches. Rash across trunk and extremities. No joint pain.

Granddaughter had similar symptoms. Family members symptoms resolved within 7 days.

Labs showed mild normocytic, normochromic anemia. hgb 11.5, Normal WBC, PLT, CMP, ESR, CRP, RF. ANA + 1:160

Further tests show +parvovirus igM+, CCP/dsDNA negative

# Diagnosis for Case Study

Too early to diagnose lupus?

Yes, too early and diagnosis was consistent with Erythema infectiosum. Symptoms resolved within a week after a prednisone taper.

Take away - Symptoms of viral infection may mimic onset of a systemic autoimmune rheumatological disease

# Lots to Learn ... let's start with basics of ANA

- ANA how referral do you get weekly for + ANA and the poor patient fearful has already been diagnosed with SLE!
- Females: 17.8% +
- Males: 9.6 + peaking both around age forty
- ANA: as you know stands for Antinuclear Antibody which **can** mean that you are allergic to yourself! ...or not
- Popular well “researched” books: invite you to avoid allergy food, gluten, nuts, Lactose and oh yes drink a bottle Guaifenesin daily!



# Let's talk before we go further

- ANA is not nearly as specific for SLE as it is sensitive:
  - Autoimmune Thyroid disease
  - Other Vascular Collagen Vascular Disease (90% SSC)
  - Viral Infections
  - Malignancies
  - Normal people with low titers (ratio 1:80)

# ANA Specificity

- ANA
  - Active SLE 95%
  - MCTD 95%
  - Diffuse SSC 70-90%
    - Systemic Sclerosis
    - CREST: C = Calcinosis, R = Raynauds, E= Esophageal mobility, S = Sclerodactyly, T= Telangiectasia
  - Primary Sjogren's > 20%
  - RA 40-50%
  - Drug induced SLE 100%

# CASE STUDY

27 y.o female with a +ANA 1:160, complains of fatigue, hair loss, joint pain, weight gain, and rash (consistent with dry skin on exam). She was told she had possible lupus and referred to your clinic.

Labs come back with Neg dsDNA, essentially normal CBC/CMP, ESR 34, CRP 0.5, and TSH 24.

Joint pain, fatigue and rash with a +ANA ARE NOT ALL equivalent to LUPUS diagnosis.

# Diagnosis

Diagnosis: Hypothyroid disease

referred to PCP/endo

# ANA Patterns: Homogeneous/Speckled/Nucleolar

- **Homogenous:** very specific to SLE, confirm with:
  - ANTI ds DNA
    - 50-60% sensitive for SLE
    - 90-95% specific
    - **+ = correlates with more renal and systemic disease**
    - Possible implication to glomerulonephritis

## **Speckled:**

- anti-SSA (RO)
- anti-SSB (LA) both SLE and Sjogren's
- anti-SCL 70 (SSC)
  - anti-Smith (SLE)
  - anti-RNP(MCTD)

## **NUCLEOLAR**

- 90% sensitive to Systemic Sclerosis
- Scleroderma
- SCL 70
  - 25 % Sensitive
  - 90% Specificity
  - Risk for more systemic organ involvement

# ANTI<sub>ds</sub> DNA Specificity

- Active SLE 60%
- Can be implicated in SLE  
Nephritis
- 90% TNF<sub>a</sub> AB Induced SLE
- Consider anti-histone

# Scleroderma Antibodies

- There are three main antibodies associated with this disease:
  - anti-Centromere limited to fingers and hands
  - anti-Scl 70 diffuse lung disease ILD
  - anti-RNA III more renal crisis
- Although not exclusive they can be predictors of morbidity and mortality associated with kidney and lung disease

# Anti SCL 70 or Topoisomerase Antibody

- Diffuse SSC 20%
- Primary limited Sjogren's 10 %
- May be associated with ILD
- Usually run with anti-centromere
- Be sure check anti-SSA and anti-SSB
- Polymyositis
- Overlap Syndrome - Inflammatory rheumatic conditions with clinical manifestations of multiple autoimmune diseases. (Had patient with scleroderma/polymyositis features - very difficult to control his disease).

[https://wn.wikipedia.org/wiki/Anti\\_nuclear\\_antibody\\_centromere\\_antibodies](https://wn.wikipedia.org/wiki/Anti_nuclear_antibody_centromere_antibodies)



# Anti-Centromere

- Looks like Spindle
- Limited “CREST” 60-80%
- Diagnostic /prognostic significance
  - Isolated Raynaud's 25%
  - Pulmonary HTN
  - Progressive Systemic Sclerosis

# Anti-Centromere Antibody Specificity

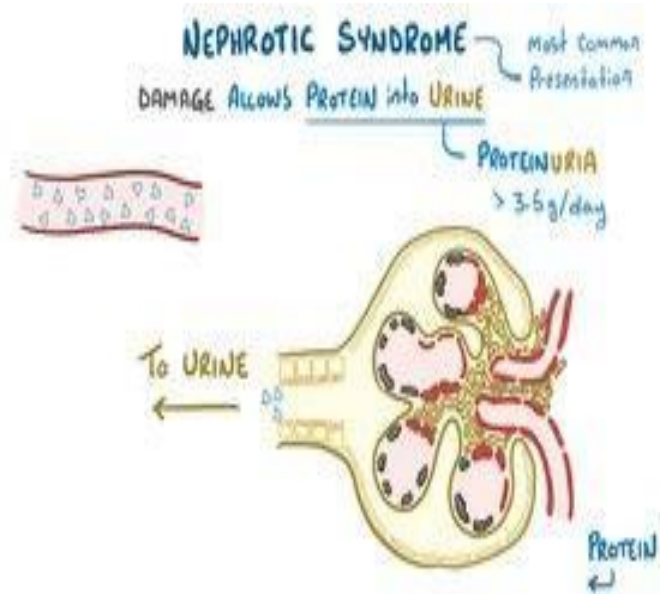
- Limited CREST 10-50 %
- Diffuse SSC 10-15%
- Rare in Active SLE
- Primary Biliary Cirrhosis

## CREST syndrome



# anti-Smith Antibody Specificity

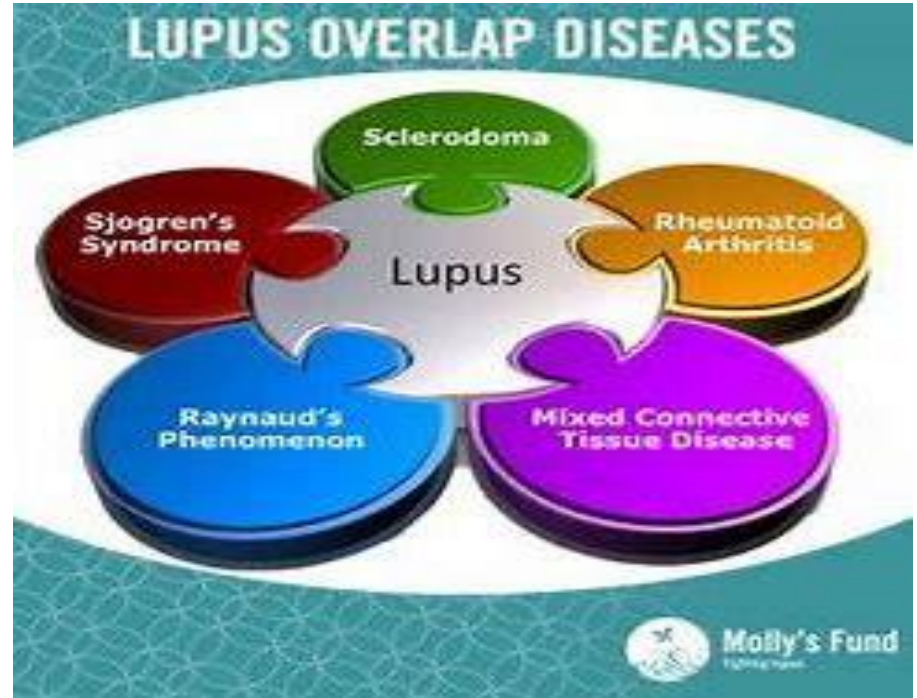
- Only SLE 30%
- Very High Specificity SLE 95%-99%
- More severe disease
- More CNS, Pul HTN
- Pericarditis
- More renal involvement



# anti-RNP antibody usually tested with Smith

- SLE 30% alone
- **MCTD 95% high titer**
- Drug induced SLE 10-20%
- Limited CREST 2%-14%
- Occ Myositis & RA
- Drug induced SLE 10-20%

If Smith & RNP+ Smith rx to  
treatment RNP will remain

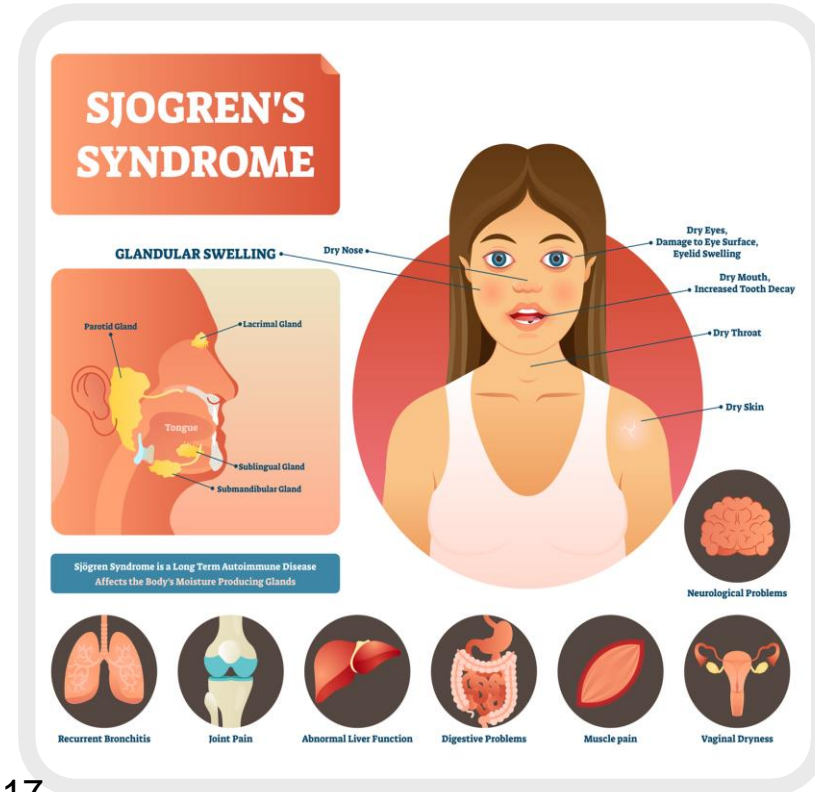


# anti-RNA III vs Anti RNP

- Anti-RNA polymerase III:
  - More cutaneous
  - More scleroderma renal crisis
  - Possible malignancy
  - Myositis
  - Joint contractures
- Anti-RNP: No other antibodies MCTD
  - Digital ischemia
  - Pulmonary Hypertension
  - GERD
  - Sclerodactyly

# anti-Ro SSA and anti-La SSB Antibody Specificity

- SLE 30%
- Primary Sjogren 88%-96%
- Subcutaneous Lupus Erythematosus
  - Papulosquamous usually annular
- **SSB alone percentage 70-80%: Sjogren's**
- EARLY DIAGNOSIS: BEFORE SLE ANTIBODIES
- Neonatal SLE: mom may be asymptomatic, but baby may have Congenital Heart Block/Rash
- If both Ro and La are positive
  - Most likely with positive LUPUS
  - Increased incidence of Vasculitis
  - Lymphoma

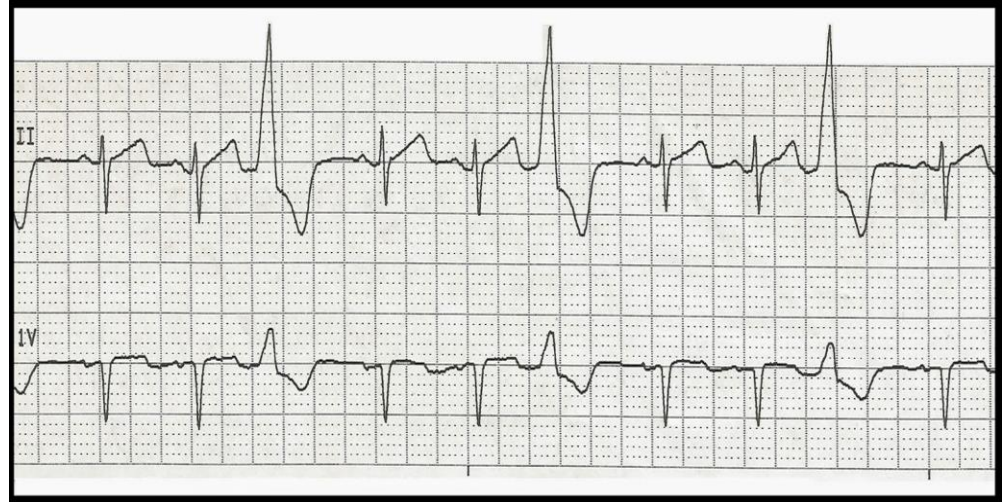


# Anti-Histone Antibody Specificity

- Active SLE- 24-95%
- RA 20%
- Scleroderma
- +++ Drug induced SLE bp meds such as Hydralazine 60-100%
- anti-TNFa Induced

# anti-Chromatin Antibody

- Active SLE 50-90%
- Can be marker for Neuropathy
- Increased sensitivity
  - Drug induced SLE In
  - 50% w Hydralazine
  - 90% w Procainamide





# LUPUS Antibodies Summary

Lupus Antibody	Significance
ANA	- SLE
Anti-dsDNA & anti-Smith	+ usually SLE: with treatment dsDNA will decrease
anti- SSA SSB/Chromatin, RNP	if one + w +ANA increase likelihood of SLE
anti-RNP	+ for SLE but also scleroderma, inflammatory myopathy possible MCTD
anti-SSA&SSB	Only 2 + consider Sjogren's w SLE+ RA+ for sure w symptoms
anti- histone	Sensitivity high for drug induced SLE : if negative not drug induced can be + RA & MCTD and also scleroderma

# Case Study

## Case #1:

54 -year -old man with history of HTN managed with Hydralazine.

He has pain, swelling of MCP and am stiffness.

Labs: ANA, anti-chromatin, anti-RNP and anti-Histone are positive.

## Case #2

24 year old African American with a three-month history of malar rash and MCP swelling, am pain and stiffness that resolves through day

LABS: Positive ANA, anti-Chromatin and anti- Smith. Anti-Ds DNA is negative

# Diagnosis

## **Diagnosis: drug induced SLE**

Warning symptoms may persist for a while  
even if the medications is DCed

**Diagnosis:** SLE but less  
likely renal problems

# Complements

Low C3 (<60) and C4 (<15) levels, (in the usual American measure), occur in active lupus, especially in the setting of kidney disease or immune breakdown of blood cells (autoimmune hemolytic anemia, AIHA).

Certain types of lupus, such as brain disease, do not cause low complement levels.

Make sure to monitor trend of C3/C4. Some patient may have chronic low C3/C4 levels **EVEN** with stable disease.

# CASE STUDY

27 y.o patient feeling well with a 1 - year history of SLE. Doing well on Hydroxychloroquine 300mg QD and denies any complaints. Physical exam shows patchy hair loss along with mild synovitis across MCP's.

Monitoring labs drawn and show low C3/C4, worsening leukopenia, and proteinuria.

New referral to nephrologist was given.

Important to note labs help with disease activity monitoring even when patient says they are doing well

# Elevated LFT ?

- Labs: Certainly, Hepatitis Panel
- Hep B:
  - Surface Antigen + current chronic or acute infection (consider antivirals)
  - Surface Antibody: immunity either vaccine or had the disease and recovered
  - Hep B Core: exposure check for Hep B DNA
- Hep C:
  - Positive IgM acute infection
  - Positive IgG chronic infection
  - past infection: permanent liver damage

# Anti-Smooth antibody

- Chronic immune Hepatitis check for high IgG
- Cirrhosis: is there positive Hep B core+ (exposure)
- Infectious Mono
- Might look at Anti-mitochondrial antibody also:
  - For primary biliary cirrhosis
  - Auto immune hepatitis
  - Cardiomyopathy that can cause cirrhosis (BNP)
  - SLE

# anti-mitochondrial Antibody

- Autoimmune hepatitis
- SLE
- RA graft vs host disease
- Usually run with anti-smooth disease in the presence of elevated LFT
- Primary Biliary Cirrhosis
- Primary Biliary Cholangitis



# Anti-phospholipid Antibody Syndrome

- Autoimmune Clotting disorder against three phospholipid antigens:
- Can be venous or arterial thrombosis
- SLE patient have risk of thrombosis but do not have to have SLE to have lupus anticoagulant!
- Important to correlate thrombotic event or miscarriage
- Clotting dyscrasia
  - Two other antibodies
  - Anti-beta 2 glycoprotein
  - Anti-cardiolipin

# Anti-phospholipid Syndrome

- anti-phospholipids are present in 10% of in healthy individuals
- 30%-50% of patients with anti-phospholipids have SLE
- If Lupus anti-coagulant (LA) is positive the patient may not have to have SLE
- Three components:
  - anti-cardiolipin (ACA)
  - anti-beta 2 glycoprotein (B2GP)
  - anti-phospholipid (APS)
- Test For All Three but only one antibody needs to be positive to correlate with hx of miscarriage or thrombotic event

# Antiphospholipid syndrome

**Important to note:** NOT ALL  
antiphospholipid syndrome case  
EQUAL Lupus!

Do NOT start Hydroxychloroquine on a healthy  
patient that comes into your office with a +ANA and  
newly diagnosed antiphospholipid syndrome.



# ANCA predictive value

- ANCA: revolutionized diagnosis of vasculitis
- High Specificity and Sensitivity
- There is huge problems with interpretation
- **False positives are a problems for Rare diseases**
- General population less than .01 % have vasculitis
- Remember if ANCA is negative : most likely don't need to hunt for further screenings: BUT DO EXAMINE the patient that is what we are so good at !!!

# Vasculitis mostly exam: but a few labs!!

- GCA: Very Elevated inflammatory markers, ESR >50mm/h by Westergren method
- PMR: Very responsive to steroids (15-20mg dosage)
- PAN : Polyarteritis Nodosa (medium arteries):
- CNS vasculitis: Biopsy is definitive diagnosis

# anti-Neutrophil Cytoplasmic Antibodies (ANCA)

- Detects specific antigen in cytoplasm
- P-ANCA if is positive it may correlate with:
  - **Microscopic Polyangiitis**
  - **Eosinophilia granulomatosis with polyangiitis (Churg- Strauss):**
  - **Drugs may cause a positive result, but not be a true vasculitis:**  
Cocaine, Hydralazine( BP) and Propothioracil used for Graves Disease
- C- ANCA
  - Commonly associated with **Granulomatosis with Polyangiitis (GPA)**
  - 90% Sensitive and Specific

# Case Studies

- Case #1 :
- 30- year old female chronic sinusitis for ten months treated with multiple tracheal dilatations. She was being treated at a University Hospital. Now presents with right elbow pain. She denies any rashes, fevers or changes in urination.
- Labs: positive C ANCA and PR3. Chest x-ray, UA and CMP within normal limits.
- Case #2
- 16- year- old African American male drummer for high school, presents extreme fatigue, 16 lbs weight loss, recent SOB, hematuria, periorbital edema and 2+ pitting edema in the feet (my neighbor!!!).
- Labs: Positive C ANCA, MPO and hematuria. Low H&H, O2 saturation 88%.

# Diagnosis of Case Studies

- Case #1
- GPA Can affect anywhere w small vessels:
  - GI bleed and/ or SOB
  
- Case #2
- MPA Do pulmonary assessment



# Creatinine Kinase

- Most important lab value for:
  - Polymyositis hint if CK negative check **Jo-1**
    - LFT elevated due to leakage of damaged muscles
    - Check TSH make sure it is not Hashimoto's Thyroiditis
    - HMG-COA (anti-3 hydroxy-3 methylglutaryl coenzyme)
      - Statin induced inflammatory necrotizing myopathy – symptoms may persist even after statins are stopped. BIOPSY helpful in diagnosis.
      - Stop statin to avoid rhabdomyolysis
      - Important to always check med list and inquire about statins if new complaints of weakness present along with elevated CK.
  - Dermatomyositis: **check for underlying malignancy CEA (Have had a case of polymyositis and patient was diagnosed with lymphoma one year later). Important to have them up to date with cancer screens (coloscopy, mammograms, heme check-up if indicated per labs.)**
  - Many dermatological assessments:
    - Heliotrope rash
    - Shawl sign
    - Gottron's papules
    - Holster sign

# Case Study

- Patient's husband called because his wife was so weak, she could not get off the toilet:
  - She has progressive muscle weakness
  - Purplish color on her eyelids
  - Reported to me that it might be eye shadow
  - No respiratory distress
  - Labs: CK was 16,000 and CEA was negative

# Diagnosis

- Dermatomyositis
- Remember sometimes only JO-1 will be +

# Case Study

- 63 y.o female presents with progressive weakness in the past two years. Statins were stopped 3 months ago. Motor strength 2/5 U/LE's.
- Labs drawn – LFT's elevated, CK in 3000's, aldolase 30. +anti-HMG-COA ab.

# Diagnosis

- Statin induced necrotizing myositis.
- Biopsy consistent with diagnosis.
- Her symptoms continued even after d/c statin
- Started prednisone 60mg QD
- And now.....

# Case Study

39 y.o African American male presents for referral from PCP for elevated CK of 899 with no complaints of weakness. Motor strength on exam 5/5. Patient works out 5-6 days a week. He completed a recent competition for crossfit training. PCP concerned for possibly polymyositis.

NEXT STEP?

# Case Study Cont.

Important to note that NOT ALL elevated CK levels are of clinical significance.

At this time, reassure patient that CK likely of no clinical significance given his normal exam. Have patient stop working out for 2-3 days and then repeat CK/aldolase levels along with CMP.

Serum creatine kinase levels are reported to be around 70% higher in healthy African Americans, as compared to Caucasian population.

Intense exercise often injures muscle tissue, causing increase in CK levels. Repeat CK levels were in the 300's for this patient. Asked to FU PRN.

# Anemia of Chronic Disease is not a new concept...but

- Types of Anemia:
  - Aplastic anemia: bone marrow failure
  - Iron Deficiency: caused by bleeding or heavy menses
  - Hemolytic: RBC destroyed shorten life span
  - Pernicious : B12 deficiency
  - Anemia is not new to us in Rheumatology: anemia of Chronic Disease but when to intervene?
  - Obtaining CBC ALWAYS helpful when making a diagnoses of any inflammatory disease. Anemia VERY common in RA, lupus, vasculitis, etc.
- Medicare does not let us infuse until HgB is below 7 (Only send to ER for hemoglobin less than 7)!!!



# Anemias

## — PERNICIOUS ANEMIA

- Lack of B12
- Lack of Intrinsic Factor
- Labs:
  - Haptoglobin, HCT and Hgb decreased, Bilirubin elevated
- Who is ask risk?
  - Bariatric patient
  - Crohn's
  - Ulcerative Colitis
  - Remember the Gut produces 60% of our immune microbiome

## APLASTIC ANEMIA

- Bone Marrow failure: rare
- Labs: low H&H
- Low Reticulocyte count
- Check LFT
- Low platelets
- Low folate levels

## HEMOLYTIC ANEMIA

- Red cells do not have normal life span
- Decreased erythropoietin
- Increased acidosis: reduces Bicarb, red cells die in 5 days instead of 120 days
- Meds that can cause it: PCN, Dapsone, Cephalosporins
- Anemia of Chronic Disease: NSAIDS, Steroids
- SLE and RA Cytokines increase Hepcidin and increase acute phase reactants, which can mimic Fe deficiency
- Labs:
  - Low H&H
  - Increased bilirubin
  - G6PD deficiency
  - Low reticulocyte
  - Coombs antibodies destroy RBC (Lupus)

## IRON DEFICIENCY ANEMIA

- Caused by:
  - Heavy menses
  - Bleeding
  - SLE changes hemostasis of Fe (anemia of Chronic disease)
  - Poor absorption: again Bariatric surgery, Crohn's, Ulcerative Colitis
- Labs: TIBC elevated due to cells trying to make more transferrin
  - Low FE
  - Low Ferritin
  - Low Reticulocyte Count

# Anemia continued

DO NOT FORGET CERTAIN DRUGS CAN LEAD TO ANEMIA:

MTX

LEFLUNOMIDE

SSZ

# Let's work it backwards

## Which lab would be helpful for diagnosis ?

- Ankylosing Spondylitis
- JRA
- Ulcerative colitis, Crohn's
- Psoriatic arthritis
- Reactive RA or Reiter's Syndrome
- Uveitis

# ANSWER

- **Answer: HLA B27.** Note 10-20% of the population may have positive HLA B27 but will have no inflammatory disease

# Deductive thinking

- Overlap Syndrome
- Raynaud's
- Inflammatory myopathy
- Fevers
- Mechanic Hands

# ANSWER

- **Answer: anti-Jo-1**

# Deductive thinking

- Skin tightening from finger- tips to wrist only
- Telangiectasia ( spider vessels in the face)
- Esophageal dysmotility
- Painful nodules in the hand
- Pulmonary hypertension

# ANSWER

- **Answer: anti-centromere**



# More clinical deductions which lab would you order?

- SLE
- MCTD
- Inflammatory Arthritis
- Scleroderma
- Polymyositis( progressive muscle weakening)
- Raynaud's

# ANSWER

- **Answer: anti-Ribonucleoprotein (RNP antibody)**

# Positive ANA can be all: back to the beginning!!

SLE

SCERODERMA

RA

Hashimoto Thyroiditis

Autoimmune Hepatitis

Inflammatory Myopathy

Hep B or Hep C

Medications: Anti TB, Antihypertensive, Anti-hyperthyroid, TNFa

# ANSWER

- **Answer: 10-20% positive ana with low titer it's Probably NOT LUPUS!!**

# Questions

- Which lab test most specific for SLE of this group:
  - anti-Smith
  - anti-Histone
  - anti-Chromatin
  - anti-Jo-1

# ANSWER

- Answer: of this group: **anti-Smith. Especially for drug induced SLE**
  - **anti-Chromatin is not specific as anti-Smith**

# More Questions

- Elevated WBC
- Elevated acute phase reactants
- Polyarthropathy (rapidly progressing joint pain)
- Sexually active: that gives it away!

# ANSWER

- **Answer Gonorrhoea**



# Most important

- Clinical diagnosis is not just labs for sure
- Good interviewing techniques are critical
- Telemedicine is here to stay so ask guiding questions
- Sometimes we order labs before the patient's first appointment
- Some of my favorites in no particular order:

Lupus Panel	CBC	RA Panel
CMP	Hep Panel	ESR
TSH	CRP	SPEP
ANA	QuantiFERON	UA
Complements	Specific antibodies based on symptomatology	

- May favorite first visit is when they say: **I did not have time to do the LABS!!!!**

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