Lupus in Pregnancy

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• No financial interests to disclose.
Objectives

• Physiologic changes
• Lupus overview
• Care in pregnancy
• Congenital heart block
  – Hydroxychloroquine
• Lupus flares
Case

- 30 yo P0000
- Lupus nephritis, Cr 1.0 (prepreg)
- CHTN
- What medication is best for CHTN?
- What anomalies are associated with ACE inhibitors / ARBs?
Case

- 30 yo P2002
- Hx of lupus, controlled on hydroxychloroquine
- Hx of child with congenital heart block
- What is recurrence risk?
- What is best prevention at present?
Maternal Physiology

Pathogens, allergens, self-antigens

Maternal Immune System

Fetoplacental tissues/Pregnancy-associated hormones

↑ Anti-inflammatory factors | ↓ Proinflammatory factors
---|---
IL-4 | IL-12
IL-10 | IL-2
TGF-β | IFN-γ
PIBF | TNF-α
Tolerogenic DCs | NK cells
M2 Macrophages | M1 Macrophages
Th2 Cells | Th1 Cells
Regulatory T cells | Th17 Cells
Antibody

↑ Successful Pregnancy
↓ Susceptibility to Inflammatory Diseases
↑ Susceptibility to Infectious Diseases

Robinson, 2012
Maternal Physiology

- Estrogens upregulate T-cell responses, immunoglobulin synthesis, and leukocyte production of IL 1, 2, 6, TNFα.
- Cell-mediated immunity is depressed:
  - Decreased ratio of T cells to B cells
  - Increased ratio of suppressor T cells to helper T cells
  - Decreased ratio of lymphocytes to monocytes
- Inhibition of complement activation in the placenta may be essential for fetal survival.
- Trophoblast may be a target of autoimmunity.

Creasy
Maternal Physiology

• AFP suppresses lymphocyte function
• IL-1, IL-3, TNF-α, IFN γ, Granulocyte-macrophage colony-stimulating factor are critical in sustaining pregnancy.
• IL-3 levels - low in women with RPL
• Pregnancy - Total C3, C4, and hemolytic (CH$_{50}$) complement levels – unchanged / increased
  – Increase in classic pathway complement activation
  – Complement activation can result in excess soluble vascular endothelial growth factor receptor type 1 (sFlt-1), which has implications for placental development and the risk for preeclampsia.
SLE

• Chronic autoimmune d/o with disease flares and remissions

• Can affect all organs
  – Mild cases – skin, musculoskeletal system
  – More severe – kidney, brain
  – Possible manifestations are arthralgias, rashes, renal abnormalities, neurologic complications, thromboemboli, myocarditis, serositis
Epidemiology

- Prevalence of lupus varies with population
- 5-125/100,000 people
- Affects 1% of pregnancies
- Lifetime risk of developing lupus is 1/700
  - peaks at 30 y/o
- Women : Men – 9:1
- Ethnic groups
  - African Americans
  - Hispanics
Etiology

• Unknown

• Genetic linkage
  – 5-12% of affected individuals have an affected relative
  – 25-50% of monozygotic twins are concordant for the disease

• Alterations in HLA system
  – HLA-B8, HLA-DR3, HLA-DR2

• Abnormal B and T cell biology and immune clearance mechanisms
The series of genes shown here is located on which chromosome?

- The long arm of chromosome 6
- The short arm of chromosome 6****
- The long arm of chromosome 16
- The short arm of chromosome 16

Pathophysiology

- Damage due to immune complex deposition, complement activation, inflammation, fibrosis
  - Renal, MSK, hepatic, platelets

- Autoantibodies
  - Antinuclear antibody – most common, ‘lab is open’
    - Increased in pregnancy – 10% of asymptomatic pregnant women without autoimmune disease have ANA ab compared to 2% of nonpregnant controls
    - Screening for lupus b/c of high prevalence in gen pop
  - Antiphospholipid antibodies
    - Anti - dsDNA antibody and anti-Smith Ab - more specific for lupus; dsdna ab correlates with disease activity
    - Anti – Ro/SSA, anti- La/SSB more often associated with sjogrens sd, but seen in 20-40% of females with lupus
      - Associated with neonatal lupus syndrome
Symptom frequency

- Fatigue – 80-100%
- Fever – 80-100%
- Arthritis – 95%
- Myalgia – 70%
- Weight loss – 60%
- Photosensitivity – 60%
- Malar rash – 50%
- Nephritis – 50%
- Pleurisy – 50%
- Lymphadenopathy – 50%
- Pericarditis – 30%
- Neuropsychiatric – 20%
Criteria for Diagnosis

- Per American College of Rheumatology
- Need 4 of 11 (serially or at one time)
  - Malar rash (erythema over malar eminences)
  - Discoid rash (erythematous raised patches)
  - Photosensitivity (unusual rxn to sunlight)
  - Oral ulcers (oral, nasopharyngeal)
  - Arthritis (nonerosive, 2+ peripheral joints)
  - Serositis (pleuritis, pericarditis)
  - Nephritis (>500mg/d proteinuria or cellular casts)
  - Neurologic disorder (seizures, psychosis, stroke with other causes r/o)
  - Hematologic disorder (hemolytic anemia with reticulocytosis, thrombocytopenia <100k, leukopenia <4000 2 occasions, lymphopenia <1500 2 occasions)
  - Immunologic disorder (anti-dsDNA, anti-Sm, positive LAC ACA, false pos RPR or other serologic test for syphilis for 6 months confirmed by treponema pallidum immobilization or fluorescent treponemal ab absorption test)
  - Antinuclear antibodies (without being on drugs associated with drug induced lupus syndrome)
- <4 of 11 = lupus-like syndrome
General Morbidity/Mortality

- Renal and cardiovascular disease
- Thrombosis
- Infection
- Survival rates
  - 5y – 93%
  - 10y – 85%
  - 15y – 79%
  - 20 y – 68%
- Risk factors for death from lupus
  - Lupus nephritis
  - Thrombocytopenia
  - Lung involvement
  - High disease activity at time of diagnosis
Pregnancy Outcomes

• Effect of SLE on pregnancy
  – Increased stillbirth rate 25x (150/1000)
    • Esp with antiphospholipid antibodies
  – Increased preeclampsia rate to 20-30% (7-10%)
  – Increased IUGR rate to 12-32%
  – Increased preterm delivery rate to 50-60% (12-15%)
  – Increased PPROM rate
  – Neonatal lupus only in Ro/La antibody positive patients

• Effect of pregnancy on SLE
  – Worsening renal status if nephropathy (Cr 1.5) present
  – Increased flares if active disease at start of pregnancy
Chronic Renal Insufficiency and Pregnancy Outcomes (vs serum creatinine mg/dL)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Cr&lt;1.5</th>
<th>Cr 1.5-3</th>
<th>Cr &gt;3</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTB</td>
<td>13%</td>
<td>50%</td>
<td>100%</td>
</tr>
<tr>
<td>Perinatal death</td>
<td>5%</td>
<td>17%</td>
<td>33%</td>
</tr>
<tr>
<td>IUGR</td>
<td>10%</td>
<td>20%</td>
<td>100%</td>
</tr>
<tr>
<td>Abortion</td>
<td>11%</td>
<td>21%</td>
<td>25%</td>
</tr>
<tr>
<td>Surviving infants</td>
<td>84%</td>
<td>62%</td>
<td>50%</td>
</tr>
</tbody>
</table>

Queenan 2007
Renal Biopsies in Pregnancy

• Complications -
  – Hematoma formation
  – Bleeding
  – Death

• Usually do not help change management

• If steroids are the change in management, then steroid administration is of lower risk than a renal biopsy
Evaluation & Management
Goals of Management

• Disease control/remission before pregnancy
• Avoid drugs that harm the fetus
• Prompt detection of preeclampsia and placental insufficiency
• Discern between lupus exacerbations and preeclampsia
• Appropriate detection and treatment of lupus flares
Preconception counseling

- Potential complications – preeclampsia, preterm labor, miscarriage, fetal death, fetal growth restriction, and neonatal lupus
- Evaluate lupus activity – delay pregnancy until remission
- Evaluate for nephritis (24 hr urine), hematologic abnormalities (CBC), antiphospholipid abnormalities
- Discontinue NSAIDS and cytotoxic agents
Medications
NSAIDS

- NSAIDS – inhibits cyclooxygenase, lipoxygenase, reduces prostaglandin synthesis
- Class D
- Avoid especially in 3\textsuperscript{rd} trimester
  - Cross placenta, blocks prostaglandin synthesis in fetal tissue
  - Premature closure of ductus arteriosus, fetal pulmonary hypertension, NEC, fetal renal insufficiency
  - Occurs with selective COX-II inhibitors
  - ASA crosses placenta and can affect fetal platelet function and is associated with intracranial fetal hemorrhage in 3\textsuperscript{rd} trimester; avoid in pregnancy
- Used outside of pregnancy – most common anti-inflammatory agent
Hydroxychloroquine

- Hydroxychloroquine (antimalarial/antirheumatic; binds DNA, interferes with vesicle functions, inhibits phospholipid metabolism; immunosuppressive by inhibiting rheumatoid factor, acute phase reactants, enzymes)
  - Stopping this in pregnancy is associated with increased risk of lupus flares, continuing this drug is recommended if needed to control lupus (prospective study by cortes-hernandez showed the increased risk)
  - Large series show no increased risk of anomalies
  - Used in prevention of malaria with increase of fetal anomalies
  - Not associated with increased r/o fetal malformations

- Class C

- Chloroquine possible teratogenic in initial studies
  - Ototoxicity, eye development

Buchanan, 1996; Khamashta 1996
Klinger 2001; Motta 2002
Glucocorticoids

- Glucocorticoids (antiinflammatory, glucocorticoid, mineralocorticoid)
- Preg class C
- Avoid fluorinated glucocorticoids b/c they cross the placenta
  - Hydrocortisone, prednisone, prednisolone inactivated by 11-beta hydroxysteroid dehydrogenase in the placenta allowing <10% of active drug to reach fetus
- High dose associated with maternal/fetal A/E
  - Osteoporosis (tx with vit D, ca2+); glucose intolerance, sodium, h2o retention; hypertension, infection; avascular necrosis
  - Preg complications – GDM, preeclampsia, PPROM, IUGR
  - Incidence of fetal adrenal suppression with maternal tx is low
- Avoid empiric treatment, use at lowest possible dose
- Stress dose steroids (hydrocortisone 100mg IV q8hr in labor and for 24 hr PP)
  - Use if chronic steroids (>5mg/day for >2-4 weeks prior to delivery)
Azathioprine

- Azathioprine (inhibits T lymphocytes)
- Class D
- Teratogenic in animals, appears safe in humans
- Associated with IUGR
- Neonatal immunosuppression
- Indicated in pregnancy if chronic high doses of steroids is not controlling symptoms or to lower steroid dose
Cyclophosphamide

- Cyclophosphamide (alkylates and cross links DNA)

- Preg class D
  - Cleft palate, skeletal abnormalities, abnormal renal function
  - Avoid, esp in first trimester
  - May be needed in cases of severe proliferative nephritis (drug of choice in nonpregnant patients with proliferative lupus nephritis)
  - Crosses placenta
Methotrexate

- Methotrexate (inhibits dihydrofolate reductase; inhibits lymphocyte proliferation) (folate antagonist)
- Preg class X
- Avoid
- Embryolethal, IUFD
- Congenital anomalies
Cyclosporine

• Cyclosporine A (inhibits T lymphocytes)
• Preg class C
• Data comes from use in renal transplant patients, not an animal teratogen, appears safe in humans, long term follow up studies are limited
Tacrolimus

- Tacrolimus (inhibits T lymphocyte activation, immunosuppressant)
- Dose in liver transplant
  - 0.1-0.15mg/kg/d po divide q12 hr
- Preg class C
- Therapeutic drug levels 5-20 ng/ml just before next dose; time to steady state 3 days
- Monitor creatinine, K, fasting blood glucose, serum drug levels
Pregnancy - FDA classes

- **A** – controlled studies show no fetal risk in any trimester, probability of fetal harm is remote
- **B** – animal studies, no risk; if risk in animal studies, controlled human studies do not confirm harm
- **C** – harm in animal studies with no controlled human studies; no available human or animal studies
- **D** – human studies show fetal risk but r/b relative to medical state of mother may support use
- **X** – animal/human studies show fetal risk or abnormalities, use is contraindicated during pregnancy or in women who may become pregnant
Pregnancy Care
Pregnancy

• Labs/Evaluation
  – *CBC, CMP, 24hr UA for TP/CC
  – *Antiphospholipid antibodies
  – *Anti-Ro and anti-La antibodies
  – *Anti-dsDNA antibody
  – Complement (C3 and C4 or CH$_{50}$)
  – Monthly CBC, Platelet count, Complement and anti-dsDNA antibody
  – Maternal echocardiogram if disease present
    >3-5 yrs, cardiac complications, associated CHTN, lupus nephritis
Lupus and Presence of Antiphospholipid Antibodies

- 1/3 of lupus patients
- Risks – thrombosis, fetal loss
- + APA and history of fetal loss or thrombosis = APLS
  - Heparin + LDA is recommended
- Thromboprophylaxis
  - Due to increased thrombosis risk
- Data is lacking that reveals improved outcomes (less SABs, IUFDs, etc) unless APLS is diagnosed
Antenatal care

• Frequent visits to assess lupus status, screen for hypertension
• Continue hydroxychloroquine
  – Depends on control as to whether to initiate it if patient is not medicated
• Monitor for exacerbations/flares
• If chronic hypertension – monitor as such; if lupus nephritis – no ACE inhibitors – initiate CCB !!
• APLS – see above
Antenatal care

• Between 18 and 25 weeks (mothers with anti-ro/la antibodies)
  • *Screening fetal echocardiogram
  • Fetal electrocardiogram through echocardiography
  • Vs. *weekly FHR checks
  • +/- Dexamethasone

• Serial ultrasounds to evaluate fetal growth
• Antenatal surveillance at 32 weeks or earlier if indicated
Neonatal Lupus

- Rash, thrombocytopenia, hepatitis, hemolytic anemia
  - Transient
- Complete heart block – Permanent
- Only if +SSA/B antibodies
  - 25% risk of rash (recurrence risk 25%)
  - <3% risk of heart block (RR 18%)
Congenital Heart Block

- SSA/B + increases risk
- Prior history of child with heart block increases risk (up to 15-20%)
- Dexamethasone – limited data that shows clear benefit
- Hydroxychloroquine Izmirly, 2010
Fetal PR Interval

Figure 1 Four-chamber view and position of the simple Doppler volume in the mitral-aortic continuity. The flow waves through the mitral valve (E- and A-waves) and the aortic valve (V-wave) are shown on the left.

Wojakowski 2009

Figure 2 Mechanical PR interval measured between the onset of atrial contraction (A-wave) and the onset of ventricular contraction (V-wave).
Table 1. Outcomes in 98 Prospectively Followed Pregnancies in 95 Women With Anti-SSA/Ro-SSB/La Antibodies Correlated With the Mothers’ Pregnancy Histories*

<table>
<thead>
<tr>
<th>PRIDE Outcome</th>
<th>No Previous Affected Child (n=74), n</th>
<th>First Pregnancy (n=44)</th>
<th>Previous Children Healthy (n=30)</th>
<th>Previous Child With Rash (n=8), n</th>
<th>Previous Child With CHB (n=16), n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal sinus rhythm</td>
<td>38</td>
<td>27</td>
<td>8</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>First-degree block</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Second-degree block</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Third-degree block</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Rash/normal sinus rhythm</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Isolated cardiomyopathy</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Died (non-CHB)</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Poor outcome unrelated to neonatal lupus</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

*Three of the 95 mothers had 2 evaluable pregnancies during the course of the study; all 3 appear in the First Pregnancy column. The second pregnancies of 2 are tabulated in the Previous Children Healthy column, and the second pregnancy of the third subject is tabulated in the Previous Child With Rash column.
PRIDE Study

- 127 women evaluated, 95 completed course – all had Ro or La Ab
- Fetal echo, weekly 16-26 weeks
- PR >150msec – 1st degree
  - 92 - Normal PR intervals
  - 3 with complete heart block, without prolonged PR interval preceding it
  - Tricuspid regurgitation, atrial echodensity
  - 2 had PR intervals >150msec, 22 weeks
    - Dexamethasone initiated, reported to prevent progression and resolved the 1st degree
- Recurrence – 19% with previous heart block
  - 3% without previous heart block
Evaluation Of The Risk Of Anti-ssa/Ro-ssb/La Antibody-associated Cardiac Manifestations Of Neonatal Lupus In Fetuses Of Mothers With Systemic Lupus Erythematosus Exposed To Hydroxychloroquine

- TLR signaling in pathogenesis of neonatal heart block
- Hydroxychloroquine is a TLR inhibitor
- A TLR inhibitor, might reduce the risk of anti-SSA/Ro/SSB/La antibody associated cardiac manifestations of NL
- Cardiac-NL children (N=50) and controls (N=151) were drawn from the following overlapping pregnancy studies: Research Registry for NL; PR Interval and Dexamethasone Evaluation in Cardiac-NL; and Predictors of Pregnancy Outcomes: Biomarkers in Antiphospholipid Syndrome and SLE
- Ro/La +; SLE dx
- Results Seven (14%) of the cardiac-NL children were exposed to HCQ compared with 56 (37%) of the controls (p=0.002; OR 0.28; 95% CI 0.12 to 0.63).
- Concluded – in mothers with SLE with anti-SSA/Ro/SSB/La antibodies, exposure to HCQ during pregnancy may decrease the risk of fetal development of cardiac-NL

Izmirly, 2010
Maternal Autoantibody Levels In Congenital Heart Block And Potential Prophylaxis With Antiinflammatory Agents

- Retrospective, 2007-2011, Ro/La +
- N =33
- Higher Anti La titers in pregnancies c/b heart block; no difference if Ro Ab
- Did not have to have SLE, etc
- 94% of fetuses maintained normal conduction when the mother was treated with hydroxychloroquine or daily prednisone therapy throughout pregnancy, compared to 59% in the untreated group (odds ratio, 0.1; \( P = .04 \)).
- Maternal treatment with either hydroxychloroquine or daily low-dose prednisone throughout pregnancy may provide a protective effect.

Tunks 2013
Lupus Flares

• **Incidence in pregnancy 15-63%**
  – Studies support and refute that pregnancy increases the incidence of flares

• **Risk factors**
  – Active disease at conception (50% vs 20%)
  – Active nephritis
  – Abrupt discontinuation of hydrochloroquine
Diagnosis Of Flare

• Symptoms
  – Fatigue, fever, arthralgias/myalgias, weight loss, rash, renal deterioration, serositis, LAD, CNS symptoms

• Titers of antibodies

• Rising titers of dsDNA Ab with falling complement levels suggest impending flare
Lupus Vs Preeclampsia

• Lupus flare
  – Arthritis, leukopenia, thrombocytopenia, rashes, pleuritis, fevers
  – Htn, proteinuria, coagulopathy possible
  – Rising antidsdna titer, active urinary sediment, low complement levels suggest lupus flare
  – Complement levels (C3, C4, CH50) generally rise in pregnancy and are unaffected by uncomplicated preeclampsia
  – Normal uric acid
  – Differentiation near term likely not worthwhile, deliver for suspected preeclampsia and initiate tx for lupus flare if patient does not get better
## Lupus Flare vs. Preeclampsia

<table>
<thead>
<tr>
<th>Finding/test</th>
<th>Preeclampsia</th>
<th>SLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased complement</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Increased anti-ds DNA Ab</td>
<td>-</td>
<td>+++</td>
</tr>
<tr>
<td>Antithrombin III decreased</td>
<td>++</td>
<td>+/-</td>
</tr>
<tr>
<td>Microangiopathic hemolytic anemia (Ab screen -)</td>
<td>++</td>
<td>-</td>
</tr>
<tr>
<td>Coombs positive hemolytic anemia (Ab screen +)</td>
<td>-</td>
<td>++</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Leukopenia</td>
<td>-</td>
<td>++</td>
</tr>
<tr>
<td>Hematuria</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Cellular casts</td>
<td>-</td>
<td>++</td>
</tr>
<tr>
<td>Increased serum creatinine</td>
<td>+/-</td>
<td>++</td>
</tr>
<tr>
<td>Hypocalciuria</td>
<td>++</td>
<td>+/-</td>
</tr>
<tr>
<td>Increased liver transaminases</td>
<td>++</td>
<td>+/-</td>
</tr>
</tbody>
</table>
Uterine Artery Notching – Present in Preeclampsia
Renal Biopsies in Pregnancy

• Complications -
  – Hematoma formation
  – Bleeding
  – Death

• Usually do not help change management

• If steroids are the change in management, then steroid administration is of lower risk than a renal biopsy
Renal Biopsy

• Risks in pregnancy
  – Hematoma formation
  – Renal dysfunction

• Lupus Nephritis
  – Increased mesangial matrix and mesangial hypercellularity (increased leukocytes)

• Preeclampsia - Endotheliosis
Renal Biopsy

- **Preeclampsia**
  - A – endothelial swelling
  - B – Glomerular necrosis
  - C – sloughing of tubular epithelial cells
Treatment Of Lupus Exacerbations

- Mild to moderate
  - Hydroxychloroquine
  - Start prednisone 15-20mg/day or increase dose to 20-30mg/day if already on glucocorticoids

- Severe exacerbations without renal/CNS manifestations
  - Rheumatology consult, hospitalize
  - Glucocorticoid treatment (prednisone 1-1.5mg/kg/d; expect improvement in 5-10 days)
  - Taper once significantly improving
  - If patient cannot be tapered off high dose steroids – add cyclosporine or azathioprine

- Severe, life threatening disease – with renal/CNS involvement – Plasmapharesis, IVIG
Lupus Nephritis

- 50% of patients with lupus develop renal disease
- Pathophysiology
  - Immune complex deposition
  - Complement activation
  - Inflammation in kidney
- Retrospective review (burkett, 1985)
  - 242 preg in 156 women with lupus nephritis
    - 59% no change in renal function
    - 30% with transient decrease in function
    - 7% with permanent renal insufficiency
    - Critical creatinine level is 1.5mg/dl
      - Above this - risk is increased of decreased renal function
      - Below this – risk is not increased
- Cyclophosphamide in extreme cases, would try IVIG, plasmapharesis first
Hx of lupus nephritis

• Recommend calcium channel blocker during pregnancy
  – Avoid ACE inhibitor/ARBs (CHD, cranial defects, fetal renal dysfunction)
Delivery

• Consider delivery at 39 weeks to avert ongoing risks of fetal loss, development of preeclampsia
• Cesarean for obstetrical indications
• Stress dose steroids if indicated
• Thromboprophylaxis IP/PP management
Case

- 30 yo P0000
- Lupus nephritis, Cr 1.0 (prepreg)
- CHTN

What medication is best for CHTN?
- CCB – amlodipine, diltiazem

What anomalies are associated with ACE inhibitors /ARBs?
- CHD, microcephaly, fetal renal dysfunction, craniosynostosis
Case

- 30 yo P0000
- Lupus nephritis, Cr 2.5 (prepreg)
- CHTN
- PPC – Should patient get pregnant?
  - Risk of pregnancy complications (preeclampsia, IUGR, premature delivery) >90%
  - Relative CI - due to renal disease prior to pregnancy and risk of progression during pregnancy should complications arise
Case

• 30 yo P2002

• Hx of lupus, controlled on hydroxychloroquine

• Hx of child with congenital heart block

• What is recurrence risk?
  – 15-20% (<3% if neg FHX and +SSA/B)

• What is best prevention at present?
  – Stay on hydrochloroquine periconceptionally even if not needed for lupus treatment
Conclusions

• Goal is control at conception
• Continue hydroxychloroquine
• Rheumatologic evaluation – the Obstetrical version
• APA – LDA daily
  – If APLS – LDA + heparin
• Fetal surveillance
• Hydroxychloroquine 200mg BID may be preventative of fetal heart block in Ro/La patients
End

• ??
References

• Email – dfarley@awhobgyn.com
• Provided on request
• Friedman 2008 PRIDE study
• Izmirly 2010 Hydroxychloroquine study
• Creasy – Resnik Maternal Fetal Medicine, Principles and Practice