COUNSELING THE DYSLIPIDEMIA POST-MENOPAUSAL WOMAN ON THE LIPID EFFECTS OF HRT

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• NO DISCLOSURES
OBJECTIVES

• Understand changes in lipid levels in women as they progress from perimenopausal to menopausal stages
• Review primary & secondary trials with HRT
• 2012 NAMS Recommendations
• Review the 2011 Update on Effectiveness-based Guidelines on Prevention of Cardiovascular Disease in Women
• Risk Assessment for CVD
• Case presentation incorporating all the above
CASE PRESENTATION #1

52 yo female with hx of abnormal lipids and high blood pressure, IFG, wants to avoid medications and keep working on diet and exercise but is overweight has had no significant results in over a year, She sees her Gyn yearly for her PCP and c/o multi menopause sxes Consult for CVD risk with HRT?
CASE PRESENTATION #1

• 52 yo female
• BMI 32.5
• Waist 36.5
• BP 148/85
• TC 246
• LDL 183
• HDL 49
• TG 144
• BG 109
• hsCRP 0.8

• ASCVD 2.9%
  Lifetime 38.8%
• HgbA1C 5.9%
• EKG normal
• No complications with pregnancy
• Parents with CVD after age 65
MENOPAUSE
MENOPAUSE

- Naturally (spontaneously) average age 51
- Prematurely from medical intervention (e.g., bilateral oophorectomy, chemotherapy, radiation)
- At any time from impaired ovarian function
MENOPAUSE

• About 6,000 US women reach menopause every day (over 2 million per year)
• A woman’s life expectancy in the United States is estimated at 80.5 years

• NAMS Menopause 2012;19:257-71
LIPIDS AT MENOPAUSE
### PROCAM (Munster Heart Study): Menopause and Lipid Risk Factors in 45 to 55 Years Old Women

<table>
<thead>
<tr>
<th></th>
<th>Pre-menopause (n = 1537)</th>
<th>Menopause (n = 2456)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>48.3 ± 2.8</td>
<td>51.0 ± 3.0</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>25.8 ± 4.3</td>
<td>26.4 ± 4.5</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>cholesterol (mg/dl)</td>
<td>221 ± 39</td>
<td>239 ± 41</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>triglycerides (mg/dl)*</td>
<td>88</td>
<td>99</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>LDL-C (mg/dl)</td>
<td>143 ± 36</td>
<td>158 ± 38</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>HDL-C (mg/dl)</td>
<td>59 ± 15</td>
<td>59 ± 16</td>
<td>n.s.</td>
</tr>
<tr>
<td>TC/HDL-C ratio</td>
<td>4.02 ± 1.25</td>
<td>4.31 ± 1.32</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

*: geometric mean,  n.s.: not significant
## PROCAM (Munster Heart Study): Menopause and Hemostatic Risk Factors in 45 to 55 Years Old Women

<table>
<thead>
<tr>
<th></th>
<th>Pre-menopause (n = 229)</th>
<th>Menopause (n = 307)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>fibrinogen (mg/dl)</td>
<td>265 ± 50</td>
<td>276 ± 56</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>D-dimer (ug/l)*</td>
<td>321</td>
<td>345</td>
<td>n.s.</td>
</tr>
<tr>
<td>factor Vllc (mg/dl)</td>
<td>108 ± 26</td>
<td>120 ± 34</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>protein C (%)</td>
<td>111 ± 19</td>
<td>120 ± 24</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>plasminogen (%)</td>
<td>104 ± 14</td>
<td>106 ± 14</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>PAI-1 (U/l)*</td>
<td>2.22</td>
<td>2.48</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>vWF (%)</td>
<td>103 ± 35</td>
<td>96 ± 31</td>
<td>n.s.</td>
</tr>
<tr>
<td>CRP (mg/dl)*</td>
<td>0.32</td>
<td>0.28</td>
<td>&lt; 0.05</td>
</tr>
</tbody>
</table>

*: geometric mean,  n.s.: not significant
CHANGE IN LIPIDS AFTER MENOPAUSE

N=10.
THE MENOPAUSAL METABOLIC SYNDROME

Lipid Triad
• Hypertriglyceridemia
• ↑ LDL cholesterol
• ↓ HDL cholesterol

Abnormalities in Insulin
• Insulin resistance
• ↓ Insulin elimination
• ↓ Insulin secretion
• Hyperinsulinemia

Other Factors
• Endothelial dysfunction
• ↑ Visceral fat
• ↑ Uric acid
• ↓ SHBG
• ↑ Blood pressure
• ↑ PAI-1
MENOPAUSAL HORMONE THERAPY TERMINOLOGY

• **Estrogen therapy (ET):** Unopposed estrogen is prescribed both a) systemically for women who do not have a uterus, and b) locally in very low doses for any woman with vaginal symptoms.

• **Estrogen-progestogen therapy (EPT):** Progestogen is added to ET to protect women with a uterus against endometrial cancer, which can be caused by estrogen alone.

• **Bioidentical hormone therapy (BHT):** Consists of hormones chemically identical or very similar to those made in the body. Available from two sources: 1) FDA-approved and tested; 2) unapproved and untested from compounding pharmacies.
HT AND CHD: META-ANALYSIS OF OBSERVATIONAL STUDIES

• Based on more than 40 observational studies of HT and CHD, the studies suggested cardio-protective effect with HT up to 40-50% among current or ever users of HT compared to never users (p<0.001).

• This was also seen in animal, observational and prospective studies.

REPRODUCTIVE HORMONE PARADOX

• Observational Studies
  – consistently demonstrate a lower risk of CHD in young (premenopausal) women than age-matched men.
  – suggest that CHD risk rises following menopause.
  – demonstrate an increased risk of CHD among young women undergoing premature menopause, compared to age-matched men.

• Yet, randomized trials of HRT in postmenopausal women have not demonstrated a reduction in CHD events.

### Hormones on Mechanisms of CVD

<table>
<thead>
<tr>
<th>Estrogens</th>
<th>Progestins</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓ LDL oxidation</td>
<td>↑ HDL effect* **</td>
</tr>
<tr>
<td>↓ LDL binding</td>
<td>↓ blood pressure**</td>
</tr>
<tr>
<td>↑ lipoprotein* ***</td>
<td>↑ glucose tolerance**</td>
</tr>
<tr>
<td>↑ blood pressure</td>
<td></td>
</tr>
<tr>
<td>↓ oxidation damage</td>
<td></td>
</tr>
<tr>
<td>↓ VSMC proliferation</td>
<td></td>
</tr>
<tr>
<td>↓ glucose tolerance***</td>
<td></td>
</tr>
</tbody>
</table>

**dependent on delivery route of estrogen
**dependent on type of progestin
***dependent on the dose of estrogen

**Cox-2 = cyclooxygenase-2;**
**HDL = high-density lipoprotein;**
**LDL = low density lipoprotein;**
**VSMC = vascular smooth muscle cell**

ESTROGENS ACTION ON ENDOTHELIUM

Early atherogenesis

Established atherosclerosis

Beneficial effects of HRT

- ↑ Vasodilation
- ↑ Nitric oxide
- ↓ Endothelin
- ↑ Cox-2
- ↓ Lesion progression
  - ↑ Nitric oxide
  - ↓ Platelet activation
  - ↓ Inflammatory cell adhesion
  - ↓ LDL oxidation/binding

Altered biology of HRT

- ↓ ER expression, function
- ↓ Vasodilation
- ↑ Inflammatory activation
- ↑ Plaque instability
  - ↑ MMP
  - ↑ Neovascularization

Ouyang et al. J Am Coll Cardiol. 2006 May 2;47(9):1741-53
RANDOMIZED TRIALS OF HT AND CVD

Secondary Prevention Trials
Heart and Estrogen/Progestin Replacement Study (HERS)
Estrogen Replacement and Atherosclerosis Trial (ERA)
Papworth HRT Atherosclerosis Study*
Women’s Estrogen for Stroke Trial (WEST)†
Estrogen in the Prevention of ReInfarction Trial (ESPRIT)†
Women’s Angiographic Vitamin and Estrogen (WAVE) Trial

Primary Prevention Trial
Women's Health Initiative (WHI; E+P, E-Alone)

CEE ± MPA except: *transdermal estradiol ± NETA, †oral estradiol
HEART AND ESTROGEN/PROGESTIN REPLACEMENT STUDY (HERS) TRIAL

• Secondary prevention trial to study HT in women with documented CHD
• Prospective randomized, placebo controlled trial
  – 2,763 women with CAD (MI, previous CABG, PTCA, or angio with >50% occlusion)
  – Age 67 ± 7, 89% white
  – Followed over 4.1 years
HERS TRIAL

• Primary outcome: Nonfatal MI and CHD related deaths

• Results:
  – No difference between HT users and placebo after 4.1 years of follow-up, HR 0.99, 95% CI (0.8 - 1.2)
  – CHD increased in the 8 months of HT (in a post-hoc analysis)

Conclusion: Combined HT should not be used to reduce the risk of CHD events in women with CHD.
HERS Incidence of CHD Events in Treatment and Placebo Groups

- CHD Death + Nonfatal MI
  - RH = 0.99
  - p = 0.91

- Nonfatal MI
  - RH = 0.99
  - p = 0.46

- CHD Death
  - RH = 1.24
  - p = 0.23

- Coronary Revascularisation or Unstable Angina
  - RH = 0.88
  - p = 0.15

Hulley et al. JAMA 1998; 280:605-613
WHI STUDIES

• **WHI E +P trial** (1993-2002):
  • ↑ coronary events, stroke, breast, cancer, VTE
  • ↓ osteoporotic fractures, colon cancer.

• **WHI estrogen alone trial** (1993-2004):
  • ↑ in stroke, VTE
  • ↓ in breast cancer, osteoporotic fractures
Ages 50-79 Hormone Program Design

Women who had no uterus at start of study

- YES
  - n=10,739
  - Conjugated equine estrogen (CEE) 0.625 mg/d
  - Placebo

- CEE 0.625 mg/d + medroxyprogesterone acetate (MPA) 2.5 mg/d
  - Placebo

Women who had a uterus at start of study

- NO
  - n=16,608

WHI is:

3 Controlled Trials

27,347
- Hormone Therapy Trial: Coronary Heart Disease & Fractures. Adverse effect for Breast Cancer?

36,282
- Calcium/Vitamin D Trial: Fractures & Colorectal Cancer

48,835
- Dietary Modification Trial: Breast & Colorectal Cancers & Coronary Heart Disease

1 Observational Study

93,676
- Observational Study

161,808 women total
WHI Estrogen+Progestin Trial
Summary of Disease Rates

<table>
<thead>
<tr>
<th>Disease</th>
<th>Risks</th>
<th>Benefits</th>
<th>Neutral</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breast Cancer</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VTE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Colorectal Cancer</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hip Fracture</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endometrial Cancer Deaths</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

# of cases/yr in 10,000 women

WHI Estrogen + Progestin Trial Findings, July 2002
(N=16,608; mean age 63 yrs; mean follow-up 5.2 yrs)

Risks

- Coronary Heart Disease 29%
- Stroke 41%
- Pulmonary Embolism 113%
- Breast Cancer 26%

Benefits

- Hip Fracture 34%
- Colorectal Cancer 34%

STOPPED Early, Clear Harm

Threshold Level

Stopped 3.3 years early

Adapted from: Writing Group for the Women’s Health Initiative. JAMA 2002;288:321.
WHI Estrogen-Alone and Health Outcomes
(N=10,739; mean age 63.6 yrs; mean follow-up 6.8 yrs)

Risks

- Stroke 39% ↑

Null

- CHD (0.91)
- Pulm Emb (1.34)
- Breast Cancer (0.77)
- Colorectal Cancer (1.08)
- Total Mortality (1.04)
- Global Index (1.01)

Benefits

- Hip Fracture 39% ↓
- Dementia ↑49% (age 65+)

STOPPED Early

Threshold Level

WHI RESULTS

• Over one year, 10,000 women taking estrogen plus progestin compared with a placebo experience:
  • 7 more cases of heart disease
  • 8 more cases of breast cancer
  • 8 more strokes
  • 18 more blood clots

• Over one year, 10,000 women taking estrogen alone (conjugated equine estrogen 0.625mg) compared with a placebo experienced
  • 12 more strokes
  • 6 more blood clots
  • no increased risk of breast cancer or heart disease
**WHI: COMBINED E+P AND CHD**

When CHD results were examined by time since menopause, risk ratio ↑ with ↑ distance from menopause:

<table>
<thead>
<tr>
<th>RR</th>
<th>Time from menopause</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.89</td>
<td>&lt; 10 yrs</td>
</tr>
<tr>
<td>1.22</td>
<td>10-19 yrs</td>
</tr>
<tr>
<td>1.71</td>
<td>&gt; 20 yrs</td>
</tr>
</tbody>
</table>

Manson JE et al. NEJM. 2003; 349:523
THE KEEPS TRIAL:

Is there an optimal time to receive HRT?
KRONOS EARLY ESTROGEN PREVENTION STUDY (KEEPS)

To compare effects of oral vs. transdermal estrogen vs. placebo in early menopausal women on:

- Atherosclerosis progression as assessed by Carotid IMT
- Development/progression of coronary artery calcium (CAC)

TIMING HYPOTHESIS

• Timing Hypothesis: The beneficial effects of HT in preventing atherosclerosis occur only when the therapy is initiated before advanced atherosclerosis develops.

• Predicts that HT is NOT beneficial when given to older women, because the underlying biologic characteristics of the vessel wall and vascular response to HRT are altered in older, more atherosclerotic vessels.
KRONOS EARLY ESTROGEN PREVENTION STUDY (KEEPS)

• N = 727 women aged 42-59 (mean age, 52.7, within 3 yrs of FMP)
• Trial Duration = 48 months
• Multi-center double-blinded placebo-controlled RCT
• Treatment Arms:
  • Oral conjugated equine estrogens (o-CEE) given as Premarin®, 0.45 mg/d (lower dose than WHI)
  • Transdermal Estradiol (t-E2) given by Climara® patch, 50 µg/d
  • Placebo

CHANGES IN IMAGING ENDPOINTS, CIMT
KEEPS CONCLUSIONS

- In healthy women, within 3 years of FMP:
  - O-CEE and t-E2 had *neutral* effects on blood pressure and mainly favorable or neutral effects on CVD biomarkers
  - O-CEE and t-E2 had *no adverse effects* on atherosclerosis progression
  - O-CEE and t-E2 had *no effect* on coronary calcium score

EARLY VS LATE POSTMENOPAUSAL TREATMENT WITH ESTRADIOL (ELITE)

- 643 women
- randomized by two timing groups, <6 yrs or >10 yrs since menopause
- oral 17-beta Estradiol 1 mg per day plus progesterone vaginal gel 10/30 days or placebo

Hadis HN, NEJM, 2016; 374: 1221-1231
EARLY VS LATE POSTMENOPAUSAL TREATMENT WITH ESTRADIOL (ELITE)

• 5yrs follow up
• CIMT baseline and every 6 mo
• CAC baseline and final
• Results:
  – CIMT had less progression with HRT in early timing group with oral estradiol
  – CAC had no change in either group

Hadis HN, NEJM, 2016; 374: 1221-1231
WHAT IS KNOWN?

• After the onset of menopause, women experience an increased CVD risk.
• Menopausal hormone therapy (MHT) in older menopausal women did not find benefit (WHI).
• Women receiving MHT closer to menopause may have lower rate of CVD (KEEPS).
DURATION OF USE

• With ET, potential CAD and CHD benefits with early use

• Initial increase in CHD risk when EPT is initiated further from menopause
NAMS HT SUMMARY

• HT formulation, route of administration, and timing of initiation produce different effects
• Individual benefit-risk profiles are essential
• Absolute risks in healthy women ages 50-59 are low
• Long-term use or HT initiation in older women, however, has greater risks
• Breast cancer risk increases with EPT beyond 3-5 years
• ET can be considered for longer duration of use due to its more favorable safety profile

NAMS Menopause 2012;19:257-71
BENEFIT-RISK CONSIDERATIONS FOR STROKE

- increases risk of stroke.
- No increased risk with < 5 years use in younger postmenopausal women (esp. < age 60)
- Low dose transdermal E - no risk
- Low dose/ultra low dose E+P - fewer side effects
GUIDELINES FOR CVD IN WOMEN
Effectiveness-Based Guidelines for the Prevention of Cardiovascular Disease in Women—2011 Update

A Guideline From the American Heart Association

EXECUTIVE WRITING COMMITTEE
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<table>
<thead>
<tr>
<th>Classification</th>
<th>Strength of Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I</td>
<td>Intervention is useful and effective</td>
</tr>
<tr>
<td>Class IIa</td>
<td>Weight of evidence/opinion is in favor of usefulness/efficacy</td>
</tr>
<tr>
<td>Class IIb</td>
<td>Usefulness/efficacy is less well established by evidence/opinion</td>
</tr>
<tr>
<td>Class III</td>
<td>Intervention is not useful/effective and may be harmful</td>
</tr>
</tbody>
</table>
EVALUATION OF CVD RISK

- Medical history/family history/pregnancy complication history
- Symptoms of CVD
- Depression screening in women with CVD
- Physical examination including blood pressure, body mass index, waist size
- Laboratory tests including fasting lipoproteins and glucose
- Global risk assessment if no CVD or diabetes
“HIGH” CVD RISK CATEGORY

- Known Coronary Artery Disease
- Cerebrovascular disease/Stroke/TIA
- Peripheral arterial disease (PAD)
- Abdominal aortic aneurysm
- Diabetes
- Chronic kidney disease
- Global 10 year risk > 10% on Framingham or Reynolds risk score (now ASCVD Risk)
“AT RISK” WOMEN

At risk (>1 major risk factor[s])

- Cigarette smoking
- SBP >120 mm Hg, DBP >80 mm Hg, or treated hypertension
- Total cholesterol >200 mg/dL, HDL-C <50 mg/dL, or treated for dyslipidemia
- Obesity, particularly central adiposity
- Poor diet
- Physical inactivity
- Family history of premature CVD occurring in first-degree relatives in men <55 y of age or in women <65 y of age
“AT RISK” WOMEN

At risk (>1 major risk factor[s])
• Metabolic syndrome
• Evidence of advanced subclinical atherosclerosis (e.g., coronary calcification, carotid plaque, or thickened IMT)
• Poor exercise capacity on treadmill test and/or abnormal heart rate recovery after stopping exercise
• Systemic autoimmune collagen-vascular disease (e.g., lupus or rheumatoid arthritis)
• History of preeclampsia, gestational diabetes, or pregnancy-induced hypertension
Table 1. Class III Interventions (Not Useful/Effective and May Be Harmful) for the Prevention of CVD in Women

<table>
<thead>
<tr>
<th>Menopausal therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hormone therapy and selective estrogen-receptor modulators (SERMs) should not be used for the primary or secondary prevention of CVD (Class III, Level of Evidence A).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Antioxidant Supplements</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antioxidant vitamin supplements (e.g., vitamin E, C, and beta carotene) should not be used for the primary or secondary prevention of CVD (Class III, Level of Evidence A).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Folic Acid*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Folic Acid, with or without B6 and B12 supplementation, should not be used for the primary or secondary prevention of CVD (Class III, Level of Evidence A).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Aspirin for MI in women &lt;65 years of age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Routine use of aspirin in healthy women &lt;65 years of age is not recommended to prevent MI (Class III, Level of Evidence B).</td>
</tr>
</tbody>
</table>

*CVD indicates cardiovascular disease; MI, myocardial infarction.  
*Folic acid supplementation should be used in the childbearing years to prevent neural tube defects.
NEW GUIDELINES SINCE 2011

• New 2013 ACC Guideline on the Assessment CV Risk
<table>
<thead>
<tr>
<th><strong>Gender</strong></th>
<th><strong>Age</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Race</strong></th>
<th><strong>Total Cholesterol (mg/dL)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Systolic Blood Pressure</strong></th>
<th><strong>HDL - Cholesterol (mg/dL)</strong></th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th><strong>Diabetes</strong></th>
<th><strong>Treatment for Hypertension</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Smoker</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>
Traditional and nontraditional atherosclerotic cardiovascular disease (ASCVD) risk factors in women.

Traditional ASCVD Risk Factors

- Diabetes
- Smoking
- Obesity and overweight
- Physical inactivity
- Hypertension
- Dyslipidemia

Emerging, Nontraditional ASCVD Risk Factors

- Preterm delivery
- Hypertensive disorders of pregnancy
- Gestational diabetes
- Autoimmune disease
- Breast cancer treatment
- Depression

Menopausal hormone therapy timeline.

Observational Studies
1980's Consistently suggested that postmenopausal MHT use may reduce the incidence of CVD.
1990's

HERS and WHI: failed to demonstrate protective vascular effects of MHT.

Prior RCTs

Contemporary RCTs
2000's KEEPS: null effects for CIMT/CAC. ELITE: support for the "timing hypothesis".

2010's Additional research needed to evaluate different doses, formulations and delivery modes currently used.

Future

Upcoming Research

Experimental Studies

HDL • vasodilation of blood vessels

LDL • insulin resistance • lipid peroxidation • intravascular collagen • VSMC proliferation

CASE PRESENTATIONS and Questions
CASE PRESENTATION #1

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- Waist 36.5
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- TC 246
- LDL 183
- HDL 49
- TG 144
- BG 109
- hsCRP 0.8

- ASCVD 2.9%
  Lifetime 38.8%
- HgbA1C 5.9%
- EKG normal
- No complications with pregnancy
- Parents with CVD after age 65
CASE PRESENTATION # 1

- At Risk per Women’s Guidelines
- IFG, Elevated HgbA1C and 4/5 criteria for Metabolic Syndrome
- Obese
- HTN
- High LDL
- Low HDL & high TG
- High Lifetime risk for CVD
CASE PRESENTATION #1- ? HRT

- HRT may worsen HTN
- HRT may benefit low HDL and high LDL but may worsen TG
- HRT may improve Insulin resistance
- Menopause sx's & Quality of life???
RECOMMENDATIONS

1. DASH diet, Low sodium, exercise & weight loss (will help BMI, HTN, PreDM and Lipids)
2. Add HTN medication, preferably ARB/ACE till under control with lifestyle
3. Reeval lipids in 3-6 mo, consider moderate intensity statin
4. Recommend Endocrine or Internal Medicine follow up for IFG & HgbA1C
5. Low dose oral CEE or transdermal estrogen with medroxyprogesterone may be neutral to low risk till age 59

- Lowest dose for the shortest amount of time to control symptoms but NOT for prevention of CVD.
BENEFIT-RISK CONSIDERATIONS FOR CAHD

• **Primary Prevention of CAHD**
  - WHI (Women’s Health Initiative)
  - No Benefit, ACOG 2002

• **Secondary Prevention of CAHD**
  - HERS (Heart and Estrogen/progestin Replacement Study)
  - No Benefit, ACOG 2002
HT & CORONARY HEART DISEASE

• ET may reduce CHD and coronary artery calcification risk when initiated in younger and more recently postmenopausal women without a uterus

• HT is currently not recommended for coronary protection in women of any age

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