Session Number 202

METABOLIC SYNDROME

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Content Description

Discussion will center on metabolic syndrome as a major risk factor for vascular disease. We will evaluate how this group of independent variables relates to the development of disease and its impact on the current population of the United States. Case study approach will be applied both for detection and treatment of the syndrome

Learning Objectives

At the end of this session the participant will be able to:

1. state five (5) components of the metabolic syndrome
2. understand that metabolic syndrome is not considered a cardiac equivalent but that the components are harbingers of vascular disease
3. use case study to create a treatment plan inclusive of education, lifestyle management, and pharmaceutical intervention for reducing components of the syndrome

Summary of Key Points/Outline – See attached slide handout

References

American Journal of Epidemiology 2000; 152(10): 897-907
Sakkinen PA, Wahl P, Cushman M, et al
Inflammation in Atherothrombosis: How to Use High-Sensitivity C-Reactive Protein (hsCRP) in Clinical Paul M. Ridker, MD, MPH. Am Heart Hosp J (2004) 2;4 Suppl 1:4-9
2008 Heart and Stroke Statistical Update. American Heart Association

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Metabolic Syndrome
TRENDS April 2013

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Diagnosis of metabolic syndrome

- Defined as
  - any pathophysiologic dysfunction that results in a loss of metabolic control of homeostasis in the body
- ATP III gives specific criteria for the syndrome but
  - does not go as far as to call it a CAD equivalent
- Components of the syndrome
  - discussed in terms of risk factors and defining levels

ATP III: Risk Is More Than Elevated LDL-C

- Easily measured variables
  - Waist Circumference
  - Low levels of HDL-C
  - Elevated BP
  - Elevated TG
  - Elevated Fasting Glucose

Metabolic Syndrome
Elevated LDL-C
ATP III and Metabolic Syndrome

- Those with metabolic syndrome are at increased risk for development of
  - DM
  - CHD
  - plus increased mortality in general

Adapted from NCEP ATP III. JAMA. 2001;285:2486-2497

Central adiposity + Lack of physical activity + Genes = Metabolic syndrome/insulin resistance

There's no such thing as a sudden heart attack. It requires years of preparation.

Cardiovascular Risk Factors

Diagnosis is established when ≥3 of these risk factors are present:

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Defining Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal obesity (Waist circumference)</td>
<td>&gt; 102 cm (&gt;40 in) &lt; 88 cm (&gt;35 in)</td>
</tr>
<tr>
<td>TG Men &amp; Women</td>
<td>≥150 mg/dL</td>
</tr>
<tr>
<td>HDL-C</td>
<td>&lt;40 mg/dL &lt;50 mg/dL</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>130/85 mmHg</td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>≥110 mg/dL</td>
</tr>
</tbody>
</table>


Features of Patients With the Metabolic Syndrome or Type 2 Diabetes

Atherogenic dyslipidemia is common in patients with metabolic syndrome (MS), including type 2 diabetes (DM).

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No MS/No DM</th>
<th>MS</th>
<th>DM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Waist circumference, cm</td>
<td>89</td>
<td>102*</td>
<td>109*</td>
</tr>
<tr>
<td>LDL-C, mg/dL</td>
<td>124</td>
<td>129*</td>
<td>153*</td>
</tr>
<tr>
<td>HDL-C, mg/dL</td>
<td>54</td>
<td>48*</td>
<td>44*</td>
</tr>
<tr>
<td>TG, mg/dL</td>
<td>105</td>
<td>214*</td>
<td>229*</td>
</tr>
<tr>
<td>Fasting glucose, mg/dL</td>
<td>93</td>
<td>100*</td>
<td>175*</td>
</tr>
<tr>
<td>SBP/DBP, mmHg</td>
<td>118/71</td>
<td>134*/77*</td>
<td>134*/71*</td>
</tr>
<tr>
<td>Prevalent CVD, %</td>
<td>5.2</td>
<td>13.6</td>
<td>26.7</td>
</tr>
</tbody>
</table>

*P < 0.0001 compared with no MS/No DM; †P < 0.01 comparing MS with DM; ‡P < 0.0001 comparing MS with DM.


Metabolic Syndrome: Risk of Death

Risk is Proportional to the Number of ATP III Criteria

CHD=Coronary heart disease, CVD=Cardiovascular disease

Adjusted for age, sex, race or ethnicity, education, smoking status, non-HDL-C, recreational/hemorrhoidal activity, white blood cell count, alcohol-use, prevalent heart disease, and stroke

†Similar adjustments except for prevalent stroke

Case Studies – Does this patient have metabolic syndrome

Case # 1
- 38 y.o. female – no CAD, no Dx. HTN, DM
- Weight 152 lbs, height 5’4” BMI = 26
- Waist circumference 29”
- BP 142/88 – first reading in office
- Labs
  - Glucose 116
  - TG 175, TC 240, HDL 41, LDL 99

Traditional Risk Factors
- Two traditional RFs but only if it is stretched (HDL & BP)
- Framingham - not necessary
- So you would think this woman is at low risk
- Metabolic Syndrome –
  - BP 142/88
  - Glucose 116
  - HDL 41
  - TG 175
- 4 out of 5 for metabolic syndrome
- How does this change your thoughts about this patient?
Role of Insulin Resistance and Compensatory Hyperinsulinemia

Genetics  Environment
Insulin Resistance  Hyperinsulinemia

Glucose Metabolism
- Elevated Glucose levels = diabetes

Uric Acid
- Decreased clearance

Dyslipidemia
- Elevated TG
- Increased PP Lipemia
- Decreased HDL
- Small, dense LDL
- LDL Oxidation

Hemodynamic
- Increased PAI I & fibrinogen

Hemostatic
- Increased Glucose levels = diabetes

Coronary Heart Disease

The Damage from components of metabolic syndrome assert their influence on the endothelium

- Contributing factors in vascular disease:
  - Lifestyle
  - Cholesterol
  - HTN
  - Genetics
  - Diabetes

Endothelial Dysfunction

<table>
<thead>
<tr>
<th>Foam Cells</th>
<th>Fatty Streak</th>
<th>Intermediate Lesion</th>
<th>Atheroma</th>
<th>Fibrous Plaque</th>
<th>Complicated Lesion/Rupture</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Non-obstructive plaque</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

From first decade  From third decade  From fourth decade
- Growth mainly by lipid accumulation
- Smooth muscle & collagen
- Thrombosis, hematomas
Glucose Metabolism

- Insulin resistance caused by abdominal obesity may lead to the development of risk factors for CHD\(^1\)
- Abdominal adiposity and insulin resistance may independently affect hemostatic variables\(^2\)


Natural History of Type 2 Diabetes

- Years from diagnosis
- Insulin resistance, Insulin secretion, Impaired Fasting Glucose, Post-Meal glucose, Fasting glucose
- "Metabolic Syndrome"
- Microvascular complications, Cardiovascular complications, Type 2 diabetes

Inflammation Increased Uric Acid Hs CRP - an acute phase reactant

- High levels associated with increased vascular events, MI, CVA
- Assay precisely measures low levels of CRP
- Most studies demonstrate a 3-4 fold increased risk associated with the highest quartile compared with the lowest levels
- Inflammation is part of the sequence of events for MI
- Stronger prediction when combined with the lipid panel

Incidence of Atherosclerotic Heart Disease and Cerebrovascular Disease in Men and Women - Age 0-84 in the United States, 1988-1994

Data from the National Health Interview Survey and National Health and Nutrition Examination Survey, 1988-1994

Ridker PM. Inflammation in Atherosclerosis. Circulation 1999;99:2291-2296


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Ridker PM. Inflammation in Atherosclerosis. Circulation 2019;139:1128-1137

Ridker PM. Inflammation in Atherosclerosis. Circulation 2020;142:507-516

Ridker PM. Inflammation in Atherosclerosis. Circulation 2021;143:1357-1365

Ridker PM. Inflammation in Atherosclerosis. Circulation 2022;146:920-929
Dyslipidemia

- Dyslipidemia is a major risk factor for CHD, the leading cause of death in the United States.\(^1\)
- The World Health Organization estimates that dyslipidemia is associated with >50% of global ischemic heart disease cases and >4 million deaths per year.\(^2\)
- Two components of dyslipidemia are directly related to risk factors for metabolic syndrome, and one indirectly affects it.

CHD = coronary heart disease.

Causes of Lipid Disorders

- Primary and Secondary Causes:
  - Primary are related to genetics
  - Secondary may be related to medical disorders, and medications, that affect specific parameters of the lipid profile
    - Metabolic endocrine
    - Diabetes
    - Thyroid disease
    - Renal
    - Hepatic
    - drugs

Serum TG Levels: NCEP/ATP III Goals and Cutpoints

<table>
<thead>
<tr>
<th>Classification</th>
<th>Serum TG Level (mg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;150</td>
</tr>
<tr>
<td>Borderline High</td>
<td>150-199</td>
</tr>
<tr>
<td>High</td>
<td>200-499</td>
</tr>
<tr>
<td>Very High</td>
<td>≥500</td>
</tr>
</tbody>
</table>
Features of ATP III - Triglycerides

- Patients with triglycerides ≥200 mg/dL
  - LDL cholesterol: primary target of therapy
  - Non-HDL cholesterol: secondary target of therapy
- Non HDL-C = total cholesterol - HDL cholesterol
  - Example: TC 270 minus HDL 90 = 220 non-HDL-C - Should not be higher than 30 points than LDL goal.
    - If LDL goal 130, non HDL goal = < 130

Atherogenic Changes Associated with Triglycerides

Low HDL-C

- Increased VLDL Remnants
- Small dense LDL particles
- Coagulation changes
- Increased PAI-1
- Increased fibrinogen

HYPERTRIGLYCERIDEMIA

Elevated Triglycerides contributing factors

- Obese and overweight
- Physical inactivity
- Excess ETOH intake
- High carbohydrate diets

- Genetic disorders
  - FCHL
  - Hyperlipidemia
  - FH
  - Familial dysbeta-lipoproteinemia

- Certain diseases
  - Type 2 DM
  - Chronic renal failure
  - Nephrotic syndrome

- Medications
  - Corticosteroids
  - Estrogens
  - Retinoids
  - Higher doses of beta-adrenergic blocking agents
  - Protease inhibitors
  - Tamoxifin
Risk of CHD by Triglyceride Level: The Framingham Heart Study

- Castelli WP. Am J Cardiol. 1992;70:3H-9H.

Other Antitherogenic Actions of HDL
- Reverse Cholesterol Transport
- Cellular Cholesterol Efflux
- Antithrombotic Activity
- Antioxidative Activity
- Antinfectious Activity
- Antiapoptotic Activity
- Antiinflammatory Activity
- Endothelial Repair
- Vasodilatory Activity

Hypertension in Metabolic Syndrome
- Insulin resistance in metabolic syndrome is associated with hypertension
- Increase in sympathetic nervous system (SNS) activity plays a key role
JNC 7 Classification of Blood Pressure for Adults Aged 18 Years or Older

<table>
<thead>
<tr>
<th>BP Classification</th>
<th>Systolic BP</th>
<th>Diastolic BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>80-89</td>
</tr>
<tr>
<td>Stage 1 Hypertension</td>
<td>140-159</td>
<td>90-99</td>
</tr>
<tr>
<td>Stage 2 Hypertension</td>
<td>≥160</td>
<td>≥100</td>
</tr>
</tbody>
</table>

Systolic Goal mm Hg
- Most Patients: <140
- Diabetes: <130
- Chronic Kidney Disease: <130

Diastolic Goal mm Hg
- Most Patients: <90
- Diabetes: <80
- Chronic Kidney Disease: <80

JNC 7: Causes of Secondary Hypertension

<table>
<thead>
<tr>
<th>Medical Conditions</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic kidney disease</td>
<td>NSAIDS</td>
</tr>
<tr>
<td>Primary hyperaldosteronism</td>
<td>Oral contraceptives</td>
</tr>
<tr>
<td>Renovascular disease</td>
<td>Adrenal steroids</td>
</tr>
<tr>
<td>Chronic steroid therapy</td>
<td>Sympathomimetics</td>
</tr>
<tr>
<td>Cushing’s syndrome</td>
<td>Cyclosporine or tacrolimus</td>
</tr>
<tr>
<td>Pheochromocytoma</td>
<td>Erythropoietin</td>
</tr>
<tr>
<td>Aortic coarctation</td>
<td>Ephedra, mu huang, bitter orange</td>
</tr>
<tr>
<td>Thyroid or parathyroid disease</td>
<td>Cocaine or amphetamines</td>
</tr>
<tr>
<td>Sleep apnea</td>
<td>Alcohol</td>
</tr>
</tbody>
</table>

JNC 7 - Features and Key Message

- Persons > age 50
- SBP is a more important cardiovascular risk factor than DBP
- Starting at 115/75
- CVD risk doubles for each increase of 20/10 mmHg
- Normotensive patients at age 50
- 90% lifetime risk of developing hypertension
- Those with SBP 120-139 or DBP 80-89
  - "prehypertensive" and should receive lifestyle modification to prevent CVD
Treatment Approach to Metabolic Syndrome

Risk Reduction Therapy

<table>
<thead>
<tr>
<th>Risk Behavior</th>
<th>% Mortality - 10 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoke Cessation</td>
<td>35 - 45 %</td>
</tr>
<tr>
<td>LDL Reduction to goal</td>
<td>25 - 35 %</td>
</tr>
<tr>
<td>BP management to goal</td>
<td>10 - 15 %</td>
</tr>
<tr>
<td>ASA</td>
<td>10 %</td>
</tr>
<tr>
<td>ACE Inhibitor Use</td>
<td>20 - 30 %</td>
</tr>
<tr>
<td>Weight Loss</td>
<td>20 %</td>
</tr>
<tr>
<td>Exercise</td>
<td>20 %</td>
</tr>
</tbody>
</table>

Grundy 9/2000

Involve the patient

Provider-centered approach may lead to missed diagnoses and poor adherence

Patient-centered approach facilitates identification of risk conditions

Enhanced communication improves patient adherence, outcomes, and satisfaction
**Treatment of Components of Metabolic Syndrome**

<table>
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<tr>
<th>Risk Factor</th>
<th>Defining Level</th>
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<td>Blood pressure</td>
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<tr>
<td>Fasting glucose</td>
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</table>


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**Therapeutic Lifestyle Interventions**

- Weight reduction
  - enhances LDL-C lowering
  - reduces metabolic syndrome risk factors
- Increased physical activity
  - reduces VLDL levels, raises HDL-C, lowers LDL-C levels
  - lowers blood pressure
  - reduces insulin resistance
- Dietary carbohydrate restrictions
  - periodic assessments of dietary changes

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**Health Benefits of Weight Loss**

- Weight loss of 5%–10% in obese individuals with type 2 diabetes, hypertension or dyslipidemia resulted in:
  - Improved glycemic control
  - Reduced blood pressure
  - Improved lipid profile
  - “Several studies demonstrate that small losses...help reduce obesity-related comorbidities and that improvements in these risk factors persist with maintenance of these modest weight losses.”

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Alcohol and CHD

There is a "U-shaped" curve

- One drink lowers CHD risk vs. risk in teetotalers
- Increasing amounts lead to increasing total mortality
- No difference between red and white wine in ecological, epidemiological studies
- Resveratrol in red wine may benefit cardiovascular health via LDL oxidation, nitric acid, or by changes in thrombogenicity, ischemia, or vascular tone
- Observational data
  Alcohol intake may be causally related to lower risk of CHD through changes in lipids (HDL-C, Apo AI, TG) and hemostatic factors

If You Consume Alcohol, Do So in Moderation

Relative risk alcohol consumption and the risk of CHD

One drink equals:
- 12 oz beer
- 4 oz wine
- 1.5 oz 80 proof spirits

10 g alcohol equals to:
- 1 shot liquor
- 1 regular can beer
- 1 glass table wine
- 1 drink/day females
- 2 drinks/day males
- With meals

TLC Teaching Tips: Three P’s for a Healthier Diet

- Fiber: More whole grain products, dietary fiber
- Fruits and vegetables: Dietary sources of antioxidants
- Fish and plant sources of omega-3 fatty acid intake shown to reduce CHD death
  - Secondary prevention studies:
    - Marine omega-3 fatty acids
    - Plant omega-3 fatty acids
  - Primary prevention data is not as consistent
  - Mechanism likely anti-arrhythmic protection
Strategies for Exercise

- Specific counseling advice such as a detailed exercise prescription may help.
  - Frequency
  - Intensity
  - Time (duration)
  - Use acronym FIT with patients
- Suggest incorporating lifestyle activities
  - Climbing stairs
  - Walking
  - Gardening
  - Housework
- View as ongoing process in behavioral change

FIT = Frequency Intensity Time

Atherogenic Dyslipidemia as a Target of Therapy

- Atherogenic dyslipidemia is an important target of therapy for CV risk management, and commonly occurs in patients with the metabolic syndrome and/or diabetes
- A substantial proportion of patients with atherogenic dyslipidemia are not at lipid goals
- Guidelines recommend non-HDL-C as a secondary target in patients with atherogenic dyslipidemia, including combination therapy with a fibrate and statin
**Lipid Therapy Options for Dyslipidemia**

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>LDL-C</th>
<th>HDL-C</th>
<th>TG</th>
<th>Key Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Statins†</td>
<td>↓10%–55%</td>
<td>↑0%–15%</td>
<td>↓7%–30%</td>
<td>Myositis, ↑LFTs</td>
</tr>
<tr>
<td>Bile acid sequestrants†</td>
<td>↓10%–30%</td>
<td>↑3%–5%</td>
<td></td>
<td>Gastrointestinal pain</td>
</tr>
<tr>
<td>Niacin and nicotinic acid derivatives†</td>
<td>↓3%–5%</td>
<td>↑20%–50%</td>
<td></td>
<td>↑LFTs</td>
</tr>
<tr>
<td>Fibrates</td>
<td>↓5%–20%</td>
<td>↑10%–25%</td>
<td>↓20%–30%</td>
<td>↑LFTs</td>
</tr>
<tr>
<td>Cholesterol absorption inhibitors</td>
<td>↓5%</td>
<td>↑1%</td>
<td>↓8%</td>
<td>Upper/lower GI complaints</td>
</tr>
<tr>
<td>Omega-3 fatty acids</td>
<td>↑45%</td>
<td>↑9%</td>
<td>↓40%</td>
<td>↑LGT-C; lack of outcomes data</td>
</tr>
</tbody>
</table>


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**Agents that reduce TGs**

- **Lifestyle management**
  - Exercise - as powerful as any medication if applied appropriately
  - Dietary changes
    - Reduction of carbohydrates
    - Reduced amounts of fruit juice, soda with sugar

- **Medications**
  - Fibrates - Gemfibrozil, Fenofibrate
  - Niacin (Niaspan)
  - HMG Co reductase inhibitors (all)
  - Fish Oil

---

**STATINS: Mechanism of Action**

- Inhibit the rate-limiting enzyme HMG-CoA in cholesterol biosynthesis
- The associated decrease in synthesis stimulates production of LDL receptors
- Also possible increased removal of VLDL and IDL remnants which accounts for some decrease in triglycerides
- Other effects
Current Research theories with HMG Co-reductase inhibitors

- anti-inflammatory effects
- plaque stabilization
- decreased thrombus formation
- endothelial restoration

Dose Efficacy of Statin-Based Therapies for LDL-C Reduction (%)

<table>
<thead>
<tr>
<th>Drug</th>
<th>10 mg</th>
<th>20 mg</th>
<th>40 mg</th>
<th>80 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>CRESTOR® (rosuvastatin calcium)</td>
<td>46</td>
<td>52</td>
<td>55</td>
<td></td>
</tr>
<tr>
<td>Lipitor® (atorvastatin calcium)</td>
<td>37</td>
<td>43</td>
<td>48</td>
<td>51</td>
</tr>
<tr>
<td>Pravachol® (pravastatin sodium)</td>
<td>20</td>
<td>24</td>
<td>30</td>
<td>*</td>
</tr>
<tr>
<td>Zocor® (simvastatin)</td>
<td>28</td>
<td>35</td>
<td>39</td>
<td>46</td>
</tr>
</tbody>
</table>

*Vytorin™ (ezetimibe 10 mg/simvastatin)* reduces LDL-C by 46% to 59%.

Data derived from the prescribing information for Vytorin.

Combination Therapies for Hypertriglyceridemia

- Fibrates & statin therapy
- Niacin & statin therapy
- Prescription omega-3 fatty acids & statin
Combination Therapy: Adding Fibrate to a Statin

**PROS**
- May increase myositis/myopathy risk
- Increase cost and complexity

**CONS**
- May decrease apo B
- May decrease VLDL


Number of Reports of Rhabdomyolysis for Fibrate/Statin Therapies (1998 to 2002)

<table>
<thead>
<tr>
<th>Medication</th>
<th>No. Cases Reported*</th>
<th>No. Prescriptions Dispensed†</th>
<th>No. Cases reported per Million Prescriptions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fenofibrate With cerivastatin</td>
<td>14</td>
<td>150,000</td>
<td>140</td>
</tr>
<tr>
<td>With other statins</td>
<td>2</td>
<td>3,419,000</td>
<td>0.55</td>
</tr>
<tr>
<td>Fenofibrate total</td>
<td>16</td>
<td>3,519,000</td>
<td>4.5</td>
</tr>
<tr>
<td>Gemfibrozil With cerivastatin</td>
<td>533</td>
<td>116,000</td>
<td>4600</td>
</tr>
<tr>
<td>With other statins</td>
<td>597</td>
<td>6,641,000</td>
<td>87</td>
</tr>
<tr>
<td>Gemfibrozil total</td>
<td>1,130</td>
<td>6,757,000</td>
<td>15x increase</td>
</tr>
</tbody>
</table>

† Calculated from data from the National Prescription Audit, IMS Health (January 1, 1998 to March 31, 2002), and a Verispan, LLC Concomitancy Report (January 1, 1998 to March 31, 2002).

JNC 7 Goal Blood Pressures
- Most patients <140/90 mm Hg
- Patients with diabetes or chronic kidney disease <130/80 mm Hg
- Based mostly on observational data, not prospective clinical trials
- Patients with metabolic syndrome
- No specific recommendation

http://www.americanheart.org/presenter.jhtml?identifier=1246168
JNC 7 - Features and Key Message

- Most patients require 2 or more drugs to reach BP goal
- If BP is > 20/10 mmHg above goal
  - initiate therapy with 2 agents either as fixed-dose combination or separately

Chobanian AV, et al. JAMA 2003;289:2560

LIFESTYLE MODIFICATIONS

Not at Goal BP (<140/90 mm Hg or <130/80 mm Hg for patients with diabetes or chronic kidney disease)

Without Compelling Indications

Stage 1 Hypertension
- Thiazide-type diuretics for most; may consider ACEI, ARB, BB, CCB, or combination.

Stage 2 Hypertension
- 2-drug combinations for most (usually thiazide-type diuretics and ACEI, ARB, or BB, or CCB).

Drug(s) for Compelling Indications
- Other antihypertensive drugs (diuretic, ACEI, ARB, BB, CCB) as needed.

If not at goal BP, optimize dosages or add additional drugs until goal BP is achieved. Consider consultation with hypertension specialist.

JNC 7: Algorithm for Hypertension

Controlling Insulin Resistance in Metabolic Syndrome

- 80% of subjects with metabolic syndrome are non-diabetic
- Diabetes mellitus prevention can significantly limit cardiovascular risk complications in these patients
- Lifestyle interventions, including weight loss and physical activity, can significantly reduce the risk of type 2 diabetes mellitus in those with either impaired fasting glucose or impaired glucose tolerance
- Strategies to prevent type 2 diabetes mellitus and reduce the CHD risk might focus on insulin-sensitizing rather than insulin-secretion interventions
Take Home Messages

- The Framingham score may underestimate risk in women, especially those with the metabolic syndrome.
- The risk for CHD and diabetes may be very different in a patient with the metabolic syndrome.
- Avoidance of diabetes is a strong motivator for patients to lose weight.
- Patients with metabolic syndrome but without diabetes or CVD, and ≥2 major CV risk factors need to be treated to goal.
- LDL-C <100 mg/dL, non–HDL-C <130 mg/dL, apo B <90 mg/dL.
- 5%–10% weight-loss can greatly improve lipid profile, BP and markedly reduce the risk of diabetes in a patient with IFG.
- Statin treatment in women > 60 yr with hs-CRP > 2 mg/L can significantly reduce CVD risk.

Take Home Messages

- Metabolic Syndrome (MetSyn) represents a constellation of clinical findings associated with increased risk for diabetes and CHD.
- Increasing obesity, physical inactivity and insulin resistance are associated with increased triglycerides.
- Patients with MetSyn often have normal LDL-C values but elevated levels of apoB containing lipoproteins.
- Mixed dyslipidemia (low HDL-C, high TG and increased numbers of small LDL-P) is common in the metabolic syndrome and diabetes.
- Understanding the pathophysiology of MetSyn helps us to identify treatment targets for the prevention of CVD.

We have our work cut out for us.
Many to work with who need our care and expertise.

ARE YOU UP & READY TO MEET THE CHALLENGE?
References


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