Coronary Artery Disease Risk Factors – an Update

Dr. Fazilatunnesa Malik

Associate Professor

Department of Cardiology

National Heart Foundation Hospital and R I, Dhaka

CVD – Global Trends

- Midst of a true global CVD epidemic
- It is the leading cause of death and disability in both developed and developing countries (80% - CAD and 20% - stroke)
- 30% of all deaths world wide each year
- 80% of all deaths in developing countries

Risk Factors

It is a characteristic or feature of an individual or population that is present early in life and is associated with an increased risk of developing future disease

Primary prevention
Secondary prevention

Classification of Absolute Risk

- High risk

Risk of CHD equals that of established disease
10 year absolute risk for MI > 20%
Needs intensive risk reduction therapy

- Intermediate risk

10 year absolute risk for MI - 10-20%

- Low risk

10 year absolute risk for MI < 10%

Classification of High Risk

1. Clinical CHD	2. Non coronary forms of clinical atherosclerotic disease
H/O ACS	Peripheral arterial disease
H/O stable angina	Abdominal aortic aneurysm
H/O coronary revascularization procedure	Symptomatic carotid artery disease
3. DM	Asymptomatic patients with carotid narrowing > 50%

Dyslipidemia – LDL-C

- Primary atherogenic factor
- Initiate and sustain atherogenesis
- Lowering of it reduces risk of CHD
- Primary target of lipid lowering therapy
- Lowering reduces the risk of CHD but not total mortality

LDL-C, mechanism

- Involved in the process of atherogenesis
  - Deposit cholestyerol in arterial wall
  - Poinflammatory effect
- Endothelial dysfunction
  - Plaque formation and growth
  - Plaque instability and disruption

Diets Raising the LDL-C

- Saturated FA

  diary fat – milk, butter, cream, cheese

  animal fat – fatty cuts of meat

- Trans FA

  margarine

- Dietary cholesterol

  eggs, dairy fat, other animal products

Diets Lowering LDL-C

- Plant stanols
- Dietary fibres
- Unsatured FA

Drug Therapy to Reduce LDL-C

1. Statins – drug of choice
2. Fibrates
3. Niacines
4. Nicotinic acid

Proposed Treatment Plan

Risk Catag.	LDL-C Goal	Initiate TLC	Consider Drug Treatment
HR: CHD or CHD equiv.	<100 mg% OG:<70 mg%	≥100 mg%	≥ 100 mg% (<100 mg% - Dr.Op)
MHR: 2+ RF (risk 10-20%)	< 130 mg% OG:<100 mg%	≥130 mg%	≥ 130 mg% (100-129mg%-Dr.Op)
MR: 2+ RF (risk < 10%)	< 130 mg%	≥130 mg%	≥160 mg%
LR: 0-1 RF	< 160 mg%	≥160 mg%	≥ 190 mg% (160 mg% - Dr.Op)

Secondary Goal After Reaching LDL-C Goal

If TG are > 200 mg%, then non HDL-C are considered secondary goal

Cigarette Smoking

- This increases the risk 2-3 times and interacts with other risk factors to multiply risk
- No form of smoking is safe even environmental or passive smoking

Cigarette Smoking - effects

- Increased vascular reactivity
  - Increased level of oxidation products – oxidized LDL-C
  - Direct effects of CO and nicotine → endothelial damage → increased vascular reactivity

Cigarette Smoking – effects, contd.

- Other mechanism
  - Lowers the threshold for myocardial ischaemia
  - Decreases capacity of O_2 carrying capacity of blood
  - Increased risk of coronary spasm
  - Increases fibrinogen level
  - Increases platelet aggregability

Benefits of Prevention

- Primary prevention
  - Precipitous fall in CHD events
  - Incase of a previous smoker, relative risk declines nearly to that of a non smoker in a year or less
- Secondary prevention
  - Recurrent events in MI surviving patient is strikingly reduced

Prevention Plan

Goal is complete cessation

No exposure to environmental tobacco smoke

- Counseling
- Drug treatment
  - First line – sustained release bupropion, various nicotine preparations
  - Second line - clonidine

Hypertension

Often a silent cardiovascular risk factor with increasing prevalence

Both systolic and diastolic BP as well as wide pulse pressure have a strong, positive, continuous and graded relationship to CHD

Hypertension

- Clusters with insulin resistance, hyperinsulinemia, glucose intolerance, dyslipidemia, LVH, obesity and occasionally in isolation
- BP reduction as small as 4-5 mm Hg results in large and clinically significant reduction in all risk factors in both middle aged and elderly as well as in high risk group

Target Goal of BP Reduction

< 140/90 mm Hg, in general

< 130/85 mm Hg, if renal insufficiency or heart failure is present

< 130/80 mm Hg, if DM is present

< 140 mm Hg, in ISH

Mechanism of coronary events

- Impaired endothelial function
- Increased endothelial permaeability to lipoprotein
- Increased adhesion of leukocytes
- Increased oxidative states
- Haemodynamic stress triggering acute plaque rupture
- Increased myocardial wall stress and O_2 demand

Left Ventricular Hypertrophy

- Doubling the risk of cardiovascular death in both men and women
- Associated with HTN, obesity, excessive salt intake, advanced age and heredity
- Prevention – most antihypertensive are effective, but

ACE – 12%	CCB – 11%
Diuretic – 08%	BB – 05%

Diabetes Mellitus

- ¾ th of the death in DM are due to cardiovascular disease, mostly CHD
- NIDDM who suffers MI, have greater risk of recurrent MI with worse outcome
- DM abolishes CHD protection of a premenopausal women and diabetic women have 2 fold risk of recurrent MI in comparison to DM men

DM – Significant Association

- Diabetic dyslipidemia present in ¼-⅓ patients with NIDDM
- 50% of IDDM and 80% of NIDDM patient have hypertension
- Glycaemic control of both type prevent microvascular but not macrovascular compl.
- Drug treatment for dyslipidemia and HTN reduce CHD events in NIDDM

Metabolic Syndrome

Characterized by multiple metabolic risk factors and increased risk of CHD

- Glucose intolerance
- Hyperinsulinemia
- ↑ TG
- ↓ LDL-C
- Hypofibrinolysis
- HTN
- Microalbuminuria
- ↑ small dense LDC-C
- Central obesity

Metabolic Synd.-Diagnosis

Needs 3 of the following 5 characteristics

01. Abdominal obesity: waist circumferance - > 40 inch in M and > 35 inch in F
02. TG: ≥ 150 mg%
03. HDL-C: < 40 mg% in M and < 50 mg% in F
04. BP: ≥ 130/85 mm Hg
05. Fasting glucose: ≥ 110 mg%

Metabolic Synd.-Management

Very effective in reducing CHD risk in NIDDM

- Intensive life style modification
- Drug therapy in high risk patients for metabolic risk factors
  - Dyslipidemia – statins, fibrates, nicotinic acid
  - HTN – ACE I
  - Prothrombotic state – low dose aspirin
  - Insulin Resistance – glitazones, metformins

Physical Inactivity

- Doubles the risk for developing CHD
- Most beneficial in sedentary individuals who become moderately active
- Advocate 30 min or more of moderate intensity physical activity on most, preferably all days
- Same activity 3-4 times a week for CHD patients
- 200 calories expanded daily in moderate intensity exercise is recommended

Physical activity causes

1. Slows the process of atherogenesis
2. Decreases myocardial O_2 demand
3. Increases myocardial efficiency
4. Increases electrical stability
5. Others
   - ↑ HDL-C
   - ↓TG
   - ↓BP
   - ↓obesity
   - ↑insulin sensitivity
   - ↓platelet aggregation
   - ↑fibrinolysis
   - ↑average size of LDL-C with out change in BP, level of LDL-C

Obesity

They accelerates the progression of coronary atherosclerosis in adolescent and young adult and associated with increased mortality

Obesity-Association

Insulin resistance	↓ HDL-C
Hyperinsulinemia	↑ TG
NIDDM	Small dense LDL-C
HTN	Infl. Thromb. Diast. dysfunction, LVH

Obesity-Diagnosis

BMI is the measure of obesity

BMI = weight in Kg/(height in meter)^2

- Normal = 18.5-24.9
- Overweight = 25-29.9
- Obesity = $\geq$ 30

Waist circumference > 40 inch in M and > 35 inch in F correlates overweight and obesity

RF – Interventions Might Lower Risk of CHD

- Psychological factors
- Hyperhomocysteinemia
- Oxidative states
- No alcohol consumption

Unmodified RF

- Age and sex
- Post menopausal states
- Socioeconomic status
- Family history of early onset CHD

Additional Emerging RF

- Inflammatory markers
  - Fibrinogen
  - CRP
- Infection
- Endogenous fibrinolysis
  - Tissue type Plasminogen Activator
  - PAI-I
  - D-Dimer


ThankYou