The Egyptian Journal of Hypertension and Cardiovascular Risk
TEJHCR 2024; VOL.15 (1): 1–5
https://doi. 10.21608/tejhcr.2025.386456.1018

https://tejhcr.journals.ekb.eg
Print ISSN 1687-5338
Online ISSN 2974-4873

REVIEW ARTICLE

Prevention of Coronary Heart Disease (CHD) 2025

M. Mohsen Ibrahim, MD

Department of Cardiovascular Medicine, Cairo University, Cairo, Egypt

ABSTRACT

This review address the various risk factor for coronary heart disease (CHD) and approaches for prevention. Risk factors belong to two groups: Non- modification which include advancing age, gender, race and genetic predisposition. Modifiable risk factors are cigarette smoking, visceral obesity, hypertension, dyslipidemia, diabetes mellitus, psychic stress and depression increased CRP, hyperhomoceyteinemia and sedentary lifestyle: Approaches for prevention are directed against these modifiable risk factors and include smoking cessation, weight reduction if obese, physical exercise and a healthy diet low in saturated fat, red meat and whole fat milk and its product and rich in vegetables, fruits and nuts. Drugs used for primary prevention include statins and aspirin. Treatment of anxiety and depression through combined treatment with anti-depression and psychotherapy. **Conclusion:** Prevention of CHD is possible and cost effective. Nationwide awareness programs addressing both public and medical community are needed. These include CV risk factors prevention and treatment. Healthy lifestyle is the core of the prevention program. Cessation of smoking, body weight reduction if obese, physical exercise and a healthy diet are the cornerstone for CHD prevention in addition to treatment of cardiovascular risk factors.

ARTICLE HISTORY Received 18 May 2025; Revised 19 May 2025; Accepted 21 May 2025

KEYWORDS Hypertension, Prevention, Heart disease

Prevention of coronary heart disease in 2025

Coronary heart disease (CHD) includes a range of clinical disorders from asymptomatic atherosclerosis and stable angina to acute coronary syndromes (unstable angina, NSTEMI, STEMI). The World Health Organization (WHO) reported that ischemic heart disease was responsible for approximately nine million deaths in 2016.

In USA and the UK mortality rates due to CHD is decreasing while in developing countries there is an increasing trend in mortality.

Implementation of prevention methods of CHD is responsible for the decline in mortality in developed countries.

Magnitude of the problem

CHD namely angina pectoris and acute coronary syndromes are possibly the main reasons for cardiologist visits and important cause of morbidity and mortality.

Worldwide atherosclerotic CHD is largely preventable and there are largely clinical benefits of both primary and secondary prevention. However, the incidence of CHD is expected to increase over the next decades partly due to increasing aging population and inadequate risk factors control. In a recent international study (INTRASPIRE) of patients interviewed after CHD hospitalization, Obesity, High BP, Smoking, Sedentary lifestyle, increased LDL-cholesterol, diabetes were common risk factors. Achieving risk factor control was noted in only 1% of patients indicating inadequate secondary prevention. Half of adults have at least one cardiovascular risk factor which are mostly undiagnosed namely diabetes, hypertension and dyslipidemia. Healthful life style habits that do not permit the appearance of risk factors is the preferred method for prevention of CHD.

Primary prevention must focus on the control of the risk factors of hyperlipidemia, hypertension, diabetes mellitus, obesity and sedentary lifestyle.

The incidence of CHD can be expected to increase dramatically over the next several decades in part due to inadequate risk factor control and an increasingly aging population with more clinical comorbidities. Furthermore, inadequate secondary prevention remains a significant and challenging worldwide problem.

CHD is the greatest cause of morbidity and mortality in most of the world, including both wealthy as well as poorer countries. What is needed are more effective strategies to reduce cardiovascular risk factors.

There are available well-recognized and effective therapeutic interventions to reduce incident CHD events.

It is unclear whether revascularization is an effective approach to prevent recurrent events. Revascularization may be an effective therapy for angina pectoris in symptomatic patients with severe obstructive disease but it has little role in reducing events in patients with chronic CHD, except in high risk patients with left main CAD and those with reduced LV function who have multivessel disease or diabetes in whom coronary bypass surgery can be beneficial.

Important interventions are needed, more effective strategies to reduce cardiovascular risk factors which

CONTACT M. Mohsen Ibrahim, egyptianhypertensionsociety@gmail.com, Department of Cardiovascular Medicine, Cairo University, Cairo, Egypt
Tel: 01222273816

include lifestyle interventions to decrease sedentary behavior, eliminate cigarette smoking and improving diet. Other therapeutic approaches include the use of lipid lowering and antihypertensive drugs when needed.

Prevention of CHD is possible and cost effective. Unfortunately, there is extremely low rates of achieving treatment guidelines standards in CHD patients.

CHD Risk Factors

Initial evaluation of risk factors is the first step in prevention of CHD

Age and gender

Advancing age is a non-modifiable risk factors for CHD. Aging is associated with progressive stiffening of arteries, increasing prevalence of other atherosclerotic risk factors namely hypertension, impaired glucose tolerance, abdominal obesity, sedentary lifestyle and psychic depression. Atherosclerosis begins in early childhood and advances throughout life. Risk factors in childhood are similar to those in adults.

CHD is more common in males than females before the age of menopause at which point the sex difference begins to diminish. Endogenous estrogen plays a role in protecting younger women from CHD.

Among women with premature ovarian failure higher rates of CHD are seen compared with premenopausal women of the same age. The use of hormone replacement therapy confers substantial reduction of up to 50 percent in the risk of CV disease. Estrogen lower total and LDL cholesterol. Other effects may include beneficial changes in arterial blood flow, coagulation and fibrinolysis, and insulin and glucose metabolism. Improvements in blood flow may be mediated through the binding of estrogen to specific receptor in arterial walls, giving rise to increased production of prostacyclin and subsequent vasodilation. Interaction between estrogens and blood platelets reduce platelet aggregation. Estrogen treatment of post-menopausal women can reduce or prevent symptoms of myocardial ischemia.

Smoking

Smoking contributes to both the atherosclerotic and thrombotic processes through effects on platelets and increased fibrinogen levels. There is an increased incidence of arrhythmias in response to catecholamines. Smoking appears to contribute to the generation of atherosclerotic plaques. Nicotine causes release of catecholamine from the adrenal gland. Cigarette smoke causes arterial endothelial damage. Smokers of filter cigarettes suffer as many heart attacks as smokers of non-filter cigarettes.

Dyslipidemia

There is a continuous relationship between total plasma cholesterol concentration and CHD incidence. Lowering cholesterol levels might be expected to reduce CHD mortality.

There is an inverse relationship between HDL cholesterol concentration and CHD incidence independent of total or LDL cholesterol concentration.

Mean HDL cholesterol concentrations are usually higher in women than men. HDL-C of 40 mg/dl is the cut point below which risk of CHD increases. The evidence for triglycerides as a risk factor for CHD is less convening than for cholesterol. Triglycerides (TGs) were found to be a significant independent risk factor only in women aged over 50 years.

Greatest risk is high triglycerides or low HDL-C. An increase in TGs more than 200 mg/dl with LDL/HDL-C ratio more than 5 increases the risk of CV events. TGs more than 230 mg/dl are associated with decreased survival. TGs-related risk is present in low HDL-C. Cholesterol ester transfer protein (CETP) catalyzes transfer of TGs to HDL and LDL with transfer of cholesterol ester in the opposite direction. HDL-C elevation correlates with decrease in CV events. Low HDL-C (< 35mg/dl) is an independent risk factor for CHD. HDL exerts its antiatherogenic effect by serving as a receptor particle for peripherally derived cholesterol which transports to the liver for excretion from the body.

Diabetes mellitus (DM)

DM increases risk for CHD 2-4 times. Associations with insulin resistance including dyslipidemia, changes in fibrinolytic and coagulation factors.

Increased procoagulant activity, platelet aggregation and decreased fibrinolytic activity are present in DM which predispose to atherothrombosis.

Visceral Obesity

Intra-abdominal fat is a major determinant of fasting and postprandial lipid availability. There is a powerful relation between central abdominal fat and insulin resistance.

Increased visceral fat correlates with hyperinsulinemia, decreased glucose tolerance, dyslipidemia and increased BP. Cortisol secretion is increased in subjects with central obesity. Medications that induce weight gain include antipsychotics, antidepressants and steroid hormones.

Hypertension (HTN)

The risk of stroke or myocardial infarction rises progressively with both systolic or diastolic blood pressures. A common sequel to mild HTN is slowly progressive cardiac failure. Isolated systolic HTN is associated with increased mortality. BP rises steadily with age. BP in youth is a predictor of BP in old age.

Regular screening needs to begin by age 35. CHD events and mortality rise, but stroke doesn't, if DBP is treated to below 85 mmHg.

The incidence of side effects from treatment is important, the threshold for treatment is heavily dependent on side effects.

Being labeled as hypertensive may also cause disability. Weight reduction and low salt intake lower BP. There is clear evidence that treating isolated high systolic BP common in older people is beneficial.

Chronic Anxiety and Depression:

Type A personality has been associated with coronary proneness in both sexes. Increased BP and blood lipid levels occurred in response to stressful stimuli. Acute stress, working primarily through the limbic system, hypothalamus and adrenal medulla, results in increased catecholamine release. This leads to increased BP, heart rate and mobilization of cholesterol and increased platelet stickiness. Chronic stress has effects through central influences on the pituitary adrenocortical axis with concomitant increase in cortisol release.

Chronic anxiety and depression have a minor, but statistically significant association with increased CHD event risk. Masked depression is more prevalent in those with anxiety. To screen for generalized anxiety disorder, the patient is asked if over the last 2 weeks have been bothered by feeling nervous, anxious and not being able to stop or control worrying.

Sedentary lifestyle

Physical inactivity is an independent predictor of CHD and is directly related to CVD mortality rate. Most CVD mortality benefits of physical activity can be attained through moderate intense activity. Exercise induces positive changes in lipoprotein metabolism.

CRP

- Independent risk factor, and an inflammatory marker
- Mediates uptake of ox-LDL by macrophages
- Induces inflammatory cytokines
- Induces MCP-I generation and secretion
- Increase generation of Reactive Oxygen Species (ROs) from WBCs
- Cutoff values are <1.0 mg/L for low risk and >3 mg/L for high risk

Infection

- Chlamydia pneumonia, Influenza A, Helicobacter pylori, hepatitis C
- Mechanisms are increased ROS, Oxidized LDL-C, IL-1, IL-6, TNFa in monocytes and decrease HDL-C.

Approaches to prevention

Primary prevention

Preventing CHD includes measures for the general population before the development of CHD (primary prevention) and measures to prevent recurrence of complications of CHD (secondary prevention).

Approaches to primary prevention

1. Smoking cessation
2. Weight reduction
3. Dietary approaches
4. Physical exercise
5. Coping with psychic stress and depression

1. Smoking cessation

Smoking cessation is particularly important in secondary prevention of arterial disease. CHD and major arterial diseases are smoking related diseases. Stopping smoking confers benefits within 2-3 years. Simple advice has been shown to increase sustained cessation to about 5 percent at one year.

Nicotine replacement therapy roughly doubles smoking cessation rates, compared with placebo, with one year sustained abstinence rates of at least ten percent achievable in primary care.

2. Weight reduction

Over weight (body mass index >25 kg/m^2) should be advised to lose weight through both dietary and lifestyle changes. Weight loss can improve lipid profile, reduce BP and improve glycemic control.

Medications that interfere with weight loss or induce weight gain such as antipsychotics, antidepressants, anti-epileptics and steroid hormones should be discontinued if possible. Waist circumference should be measured at least in overweight persons to better classify obesity. WC ≥ 102 cm in men and ≥ 88 cm in women represent the threshold at which weight reduction should be advised.

A realistic target of weight reduction is 5-10% of original weight and a maximum weekly weight loss of 0.5-1 kg.

For weight reduction, a lifestyle modification program is needed which includes limiting calories intake and physical exercise. High caloric rich foods (sugary beverages, sweets, cakes, biscuits, fried food) should be avoided. Reducing meal size and limiting fat and sugar intake is recommended.

Pharmacologic treatment should be considered only after dietary advice, exercise and behavioral approaches have been started and evaluated. Bariatric surgery should be considered on an individual case basis in patients with BMI ≥ 40 kg/m^2 or BMI > 35 kg/ m^2 with severe comorbidities.

3. Dietary approaches

The vegetarians experienced significantly less ischemic heart disease mortality than the non- vegetarian health food users. CHD death rate in the non-meat eaters was around 25 per cent lower than in the meat eaters. Countries with a high intake of fruit and vegetables have lower than expected rates of CHD.

It is recommended to replace butter and hard margarine with polyunsaturated margarine and full fat with skimmed milk and eat more fish, fiber rich foods, fresh fruit and vegetables.

Coronary angiography showed significant overall regression of coronary atheroma in an experimental group with very low-fat vegetarian diet.

Eating a diet that is high in saturated fat raises blood lipid levels and increases the risk of premature CHD. A diet that is low in saturated fat but high in monounsaturated and polyunsaturated fats in patients who make comprehensive life-style changes often experience significant and sustained reductions in frequency of angina and LDL cholesterol level. Also, coronary artery lesions tend to regress rather than progress. Intake of fresh fruit. and vegetables, whole grain. Cereals and limited intake of dietary cholesterol is likely to provide the best protection against premature CHD. Saturated fats should be less than 7% of the calories and total fat less than about 30%.

4. Physical exercise

There is good evidence that people who exercise adequately have a reduced risk of CHD. The minimum exercise required to improve fitness is brisk walking for at least 20 minutes twice a week. A graduated program of slowly increasing exercise is essential in sedentary individuals especially in those with pre-existing cardiovascular disease.

It appears that low intensity exercise of longer duration is equivalent to more intense exercise of short duration. Benefit can be obtained from less vigorous exercise in older people.

The beneficial effect of exercise is mediated through reduction in BP, increase in HDL cholesterol and reduced platelet aggregation and fibrinogen concentration with long term vigorous exercise. Training reduces myocardial oxygen demand at a given level of exercise which lowers the risk of ischemia. Cardiovascular fitness is based on total treadmill test time which correlates with measured maximal oxygen uptake.

Fitness level is an independent predictor of cardiovascular risk, independent of conventional risk factors.

5. Coping with psychic stress and depression

Increases in BP and blood lipid levels occur in response to stressful stimuli. There is doubling of CHD risk in association of social isolation and phobic anxiety. Depression significantly increases the risk of developing CHD. Physiologic derangements include abnormal platelet activation and endothelial dysfunction.

Treatment of depression includes medication, psychotherapy or both. Type of treatment recommended depends upon the type of symptoms, the severity of symptoms and the patient's personal preferences. Combined treatment with antidepressants and psychotherapy is recommended as first line treatment for patients with severe major depressive disorder.

Selective serotonin reuptake inhibitors (SSRIs) are the most commonly prescribed antidepressants. Use of SSRIs in depressed patients who experience an acute MI might reduce subsequent cardiovascular morbidity and mortality.

Prevention of depression depends upon social support-people surrounding individual, physical exercise and stress management at home and work.

Drugs for primary prevention

Statins

Effective drugs for lowering LDL-C, with clinical benefit for CHD mortality and for all-cause mortality. They are competitive inhibitors of HMG-COA reductase which decrease cholesterol synthesis. More than three-fourth of the body's cholesterol pool is of endogenous origin and two-third is produced in the liver. Reductions in LDL-C range from 15% to 60%. Statin therapy increases HDL-C by 5-10 % with TGs reduction from 10% to 20%. The most important adverse effects of statin therapy are hepatotoxicity and myopathy which both are rare.

Indications of statin for primary prevention

The level of LDL-C and the presence of CV risk factors determine the need for statins in primary prevention. The risk factors include age >65 years, family history of CHD in first degree relatives, diabetes mellitus, CKD (serum creatinine >2.5mg/dl), cigarette smoking, obesity, hypertension, metabolic syndrome, low HDL-C, hypertriglyceridemia, CRP $\geq$ 2 mg/L.

An LDL-C > 190 mg/dl on two estimations even in absence of CV risk factors is an indication for statin therapy. Levels of LDL-C > 160mg/dl in presence of 2-3 CV risk factors or > 130 mg/dl in presence of more than 3 CVRFs, DM or CKD are indications for statins. The target LDL-C with statins depends upon the degree CV risk. In low risk patients (no or one RF it is <140 mg/dl, in high risk patients (>3 RFs) or diabetes or CKD target LDL-C is less than 70 mg/dl.

Aspirin

Aspirin (75-100 mg/day) is indicated in all patients with prior CV event. SBP should be <130 mmHg. It is contraindicated if there is a history of GI bleeding or peptic ulcer. It is recommended for primary prevention in patients 50 years or older and have high risk (10 years atherosclerotic CV disease risk more than 20%) and in patients with diabetes mellitus, 40 years or older and have additional risk factors.

It completely inhibits TXA_2 mediated platelet aggregation without an effect on prostaglandins production. Higher doses >325 mg/ day are used as antipyretic, analgesic and anti-inflammatory.

Aspirin should be used infrequently in the routine primary prevention of CHD and limited to selected high-risk patients with low bleeding risk.

New clinical trials suggest a reduced benefit of aspirin for preventing first CVD events.

Conclusion

The incidence of CHD can be expected to increase dramatically over the next several decades in part due to inadequate risk factor control and an increasingly aging population with more clinical comorbidities. Furthermore, in-adequate secondary prevention remains a significant and challenging worldwide problem.

CHD are the greatest cause of morbidity and mortality in most of the world, including both wealthy as well as poorer countries. What is needed are more effective strategies to reduce cardiovascular risk factors. There are available well-recognized and effective therapeutic interventions to reduce incident CHD events.

References

1. Ambrose JA, Singh M. Pathophysiology of coronary artery disease leading to acute coronary syndromes. F1000Prime Rep. 2015; 7:08
2. Ampofo AG, Boateng EB. Beyond 2020: modelling obesity and diabetes prevalence. Diabetes Res Clin Pract 2020; 167:108362
3. Anderson L, Brown JP, Clark AM, et al. Patient education in the management of coronary heart disease. Cochrane Database Syst Rev. 2017; 28, 6(6): CD008895.
4. Boden WE, O'Rourke RA, Teo KK, et al. Optimal medical therapy with or without PCI for stable coronary disease. N Engl J Med 2007; 356:1503–16.
5. Ford TJ, Corcoran D, Berry C. Stable coronary syndromes: pathophysiology, diagnostic advances and therapeutic need. Heart. 2018;104 (4): 284-292
6. Kaminsky LA, German C, Imboden M, et al. The importance of healthy lifestyle behaviors in the prevention of cardiovascular disease. Prog Cardiovasc Dis 2022; 70: 8-15.

7. Kones R. Primary prevention of coronary heart disease: integration of new data, evolving views, revised goals, and role of rosuvastatin in management. Anderson L, Brown JP, Clark AM, Dalal H, Rossau HK, Bridges C, Taylor RS. Patient education in the management of coronary heart disease. Cochrane Database Syst Rev. 2017; 28; 6(6): CD008895
8. Maron DJ, Hochman JS, Reynolds HR, et al. Initial invasive or conservative strategy for stable coronary disease. N Engl J Med 2020; 382:1395–407.
9. Massberg S, Polzin A. [Update ESC-Guideline 2017: Dual Antiplatelet Therapy. Dtsch Med Wochenschr. 2018 Aug;143(15):1090-93.
10. Ross R. Atherosclerosis, an inflammatory disease. N Engl J Med. 1999; 14;340(2):115-26
11. Shavelle DM. Almanac 2015: coronary artery disease. Heart. 2016;102(7):492-9.
12. Visseren FLJ, Mach F, Smulders YM, et al. 2021 ESC guidelines on cardiovascular disease prevention in clinical practice. Eur Heart J 2021; 42:3227-337.