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REDUCING RISK OF CARDIOVASCULAR DISEASES AMONG GENERAL POPULATIONS - REVIEW

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Abstract:
Cardiovascular disease (CVD) is a long-term condition with serious and often deadly consequences. In this review, we highlight the main areas targeted for primary CVD prevention in primary care, as well as recommendations to minimize CHD risk. This is accomplished by searching electronic databases (MEDLINE, Embase, SCOPUS, CINAHL,) for published articles prevention measures in lowering the risk of coronary heart disease (CHD). Primary prevention programs aimed at reducing risk behaviors on a population-wide scale, as well as the identification, stratification, and also targeted treatment of high-risk individuals prior to the progression of condition, should be cornerstones of any strategy to reduce the population's fear of CHD and also, prevention strategies must start in childhood.

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INTRODUCTION:

Cardiovascular disease (CVD) is a catch-all phrase covering a variety of connected illnesses such as coronary heart disease (CHD), cerebrovascular disease, peripheral arterial disease, rheumatic and congenital heart disease, and venous thromboembolism. Worldwide, CVD causes 31% of deaths, the majority of which are caused by CHD and cerebrovascular accident [1].

Atherosclerosis is an older condition that has been discovered in Egyptian pharaohs' mummies. It is a pathological entity, and cardiovascular disease is its professional equivalent (CVD). Only recently have its ramifications reached epidemic proportions, mostly in Western affluent society, albeit that epidemic is now spreading to other non-Western people. The clinical symptoms of The symptoms of atherosclerosis are numerous, but they are only the tip of the iceberg [2]. Although the etiology of atherosclerosis is complex, it is estimated that more than 90% of the facts regarding the mechanisms involved are already known, albeit how these realities fit into an overall intelligible framework is less understood1.

CVD accounts for over half of all mortality in the developed world. In some places, death rates have decreased in recent years, and in others, they have increased [2]. When unrefined death rates fall below 15 per 1000 people and life expectancy at birth rises to 55-60 years, the fraction of CVD mortality approaches 20-25%, and non-communicable conditions become a major public health problem [3]. This is the case in the Eastern Mediterranean Region [3]. Given the relatively huge percentage of the total condition problem, both fatal and non-fatal, played by cardiovascular illnesses, it has become critically important to seek to prevent that trend.

CVD is a long-term status with a serious and often fatal results.

METHODOLOGY:

We conducted an electronic search throughout databases (MEDLINE, Embase, SCOPUS, CINAHL,) for published articles including cardiovascular diseases prevnetion all studies which were published till June,2021. Furthermore, we manually searched references list of included studies for more supportive data.

DISCUSSION:

Assessment of risk for CVD:

Forecast graphs provided or released by the WHO and also ACC/AHA can be used to make an accurate prediction of a person's CVD risk. The recommendations are designed to treat key cardiovascular risk components by lifestyle modifications and preventive medication therapy.

The ACC/AHA has established guidelines for the detection, management, and prevention of CVD.

The ACC and AHA released new risk-assessment guidelines for atherosclerotic CVD in November 2013. The following modifications and referrals are included [4-7].

- Stroke is contributed to the list of coronary occasions generally covered by risk prediction equations.

- The guidelines focus mainly on the 10-year risk of atherosclerosis-related occasions; they focus secondarily on the evaluation of life time risk for grownups aged 59 or more youthful without high shorter-term danger.

- The strongest predictors of 10-year threat are identified as age, sex, race, overall cholesterol, HDL-C, blood pressure, blood-pressure therapy status, diabetes, as well as current cigarette smoking status.

- Adjunct formulas for refining danger quotes by sex and race are supplied.

- If risk forecast has to be additional developed after threat forecast equations have been performed, the guidelines indicate that coronary-artery calcium scores, family history, high-sensitivity C-reactive protein, and also the ankle-brachial index can be used.

- The guidelines recommend that statin treatment be thought about in people whose 10-year atherosclerotic cardiovascular disease (ASCVD) occasion danger is 7.5% or greater.

AHA/ACC guidelines advocate using an updated calculation to estimate the 10-year risk of acquiring an initial ASCVD event, which is defined as a nonfatal MI, death from CHD, or stroke (deadly or nonfatally) in an individual who was previously free of ASCVD [6]. The calculator takes into account the following risk factors: gender, age, race, total cholesterol, HDL, systolic blood pressure, therapy for high blood pressure, diabetes mellitus, and cigarette smoking.

The guidelines recommend assessing medical threat variables every 4-6 years for people aged 20 to 79 who do not have current scientific ASCVD. For patients with a low 10-year risk (7.5%), the guidelines recommend assessing 30-year or lifetime risk in adults aged 20 to 59.

According to the guidelines, regardless of the patient's age, doctors must connect risk data to the patient and refer to the AHA/ACC way of life guidelines, which address food and physical exercise. Medical personnel must interact risk information and refer to the AHA/ACC recommendations on blood cholesterol and obesity for individuals with elevated 10-year risk.

Table 1. Risk factors for CVD and recommended European target goals: key points[8],[12].

Non-modifiable	
• Age	Men: ⩾45 years
	Women: >55 years
• Personal history of CHD	
• Family history of CHD	
Modifiable	**Target goals**
• High TC	<5.0 mmol/l (<190 mg/dl)
• High LDL-C	<3.0 mmol/l (<115 mg/dl)
• Low HDL-C	>1.1 mmol/l (>40 mg/dl)
• High triglyceride	<1.2 mmol/l (<150 mg/dl)
• Hypertension	<140/85 mm Hg (140/80 or 130/80 mm Hg in diabetes)
• Diabetes mellitus	Normalise glucose concentrations (HBA$_{1C}$ below 7 mmol/l)
• Current tobacco use	Smoking cessation
• Obesity	Body mass index <25 kg/m^2
• Sedentary lifestyle	Exercise for 30 minutes 3–5 times weekly

HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; TC, total cholesterol.

- **Primary Prevention:**

Physical activity and Diet
Physical activity is widely acknowledged to have a positive influence on the majority of health outcomes, and its effect on CVD is no exception. Death and morbidity directly related to exercise remain relatively low when compared to very high levels of exercise, and in the great majority of cases, the benefits outweigh the risks [9].

NICE recommends 150 minutes of moderate-intensity exercise per week or 75 minutes of vigorous cardio per week. This might be defined subjectively or in terms of relative metabolic rate changes. They also suggest muscle strengthening workouts on two or more days per week [11]. NICE provide just a consensus recommendation relating to the utility of workout as primary prevention, However, standards from the AHA and ESC provide course 1 A recommendations with nearly identical prescriptions, referring to a robust as well as unanimous body of evidence [9],[10].

The guidelines all suggest that any sort of exercise reduces the risk of CVD, with those who begin exercising getting the most benefit and also any type of subsequent increases providing significant but diminishing returns. Despite the obvious benefits, convincing the population to exercise as suggested remains difficult; yet, the research is unequivocal that any increase in physical activity reduces the risk of CVD [9].

Diet is thought to play a substantial role in CVD danger yet the body of evidence regarding its usage is not clear, neither are the guidelines overwhelmingly consensual.

The American Heart Association recommends the Dietary Approaches to Stop Hypertension (DASH) diet, which is low in sweets and saturated fats and high in vegetables, fruits, and whole grains. This has been found to lower blood pressure (BP) and also low-density lipoprotein cholesterol (LDL-C), which are independent risk factors for CVD, but it does not

attempt to establish a direct decrease in CVD danger [10].

NICE recommends reducing saturated fat consumption, increasing monounsaturated fatty acid consumption, and eating 5 portions of fruit and vegetables each day. They also recommend a high-fiber diet and two portions of salmon every week.

They do acknowledge that they do not have proof that these changes will affect directly on CVD risk, however rather that they have advantages on various other areas of health. Significantly, most of the research studies referenced came from pre-1990s when dietary patterns were significantly different, and also nearly all their information were underpowered concerning CVD threat [9].

The ESC recommends switching from saturated to polyunsaturated fatty acids, an increase in fibre, fruit, veggie as well as fish intake as well as abstaining from alcohol as well as adherence to a Mediterranean kind diet. These have all been revealed to offer considerable reductions in CVD risk [9].

In conclusion, there does seem to be good evidence for recommending diet plans high in fibre, vegetables and fruit consumption and reduced in basic sugars and salt. Adherence to a Mediterranean style diet regimen additionally appears to be cardioprotective.

Smoking cessation and Weight loss

Cigarette smoking has long been recognized as a significant risk factor for CVD. According to European data, cigarette smoking doubles the 10-year CVD mortality rate [12], and cigarette smoking is responsible for 30% of CVD deaths [10]. Not only is it unhealthy, but the effect is dose-dependent, with no risk-free lower limit observed. Secondhand smoke is also harmful, as workplace exposure increases CVD risk by 30%, and UK public health initiatives such as cigarette smoking bans have been linked to a significant decrease in CVD events [9].

Stopping smoking is the solitary most affordable treatment in CVD avoidance, and also some benefits are seen within months of cessation [9],[10].All guidelines advise cessation, with brief as well as long-lasting advantages seen irrespective of size or strength of cigarette smoking habit.

Pharmacologically, using nicotine replacement therapy (NRT), buproprion (a norepinephrine dopamine reuptake inhibitor) and particularly varenicline (a partial nicotine receptor agonist) are universally advised. The two previous both improve abstaining prices by 50-- 70%, whilst varenicline increases abstaining [14, 15].

E-Cigarettes are still controversial when it come to CVD danger. Whilst the reduction in poisonous items within cigarette smoke is certainly useful, animal versions of nicotine direct exposure still display CVD results with raised atherosclerotic plaques found in mice versions [16]. Lasting information are waited for to identify the impact after humans.

Having a body mass index (BMI) > 25 is a danger element for CVD with lowest all-cause mortality seen at BMI 20-25 but, because of enhanced all-cause death with BMI< 20, [17] reductions listed below this degree are not routinely suggested. No guidelines prescribe specific weight intervention, but they do recommend maintaining a healthy and balanced weight to reduce CVD risk. BMI is a strong predictor of CVD risk, particularly at higher levels, although there is evidence that, regardless of BMI, visceral adiposity and liver fat are significant risk factors [16], [17]. This helps to explain why the CVD risk profile of the overweight differs depending on the location of adipose deposition. There are actions to suggest that, in addition to BMI reduction, waist circumference reduction as a proxy for visceral fat reduction should become a critical aim for CVD risk reduction.

Table 2. Evidence base for benefit of risk factor modification

Risk factor	Primary prevention of CHD
Smoking	Smoking cigarettes cessation will reduce the danger of death by 50%. Male that stop cigarette smoking have a lowered danger of myocardial infarction and also within 2-3 years the threat resembles those who have actually never smoked.
Diet	Nutritional modifications (decrease in saturated fat, cholesterol and a rise in polyunsaturated fat) can lead to lowered death from CHD. The addition of stanol esters and also plant sterols (which reduce cholesterol absorption) to food, for instance margarine, has been shown to minimize plasma cholesterol focus by around 10%. The impact corresponds with over intake causes variable results.
Cholesterol	Total serum cholesterol of > 6 mmol/l is related to a raised occurrence of CHD risk as well as danger of CHD death.
Exercise	Lack of physical fitness or exercise are associatedwith a raised danger of death from all causes and from cardiovascular disease both in middle-aged and older men.
Alcohol	Mortality from CHD is lowest in those that reported alcohol consumption 8 to 14 units of alcohol a week. Consuming over 21 systems a week boosts total mortality. Distinctions in between types and patterns of alcohol consumption remain vague.
Diabetes mellitus	Mortality from CHD increases concerning 3-fold to 10-fold and 2-fold to 4-fold in patients with kind 1 as well as type 2 diabetes mellitus, respectively.The UKPDS study showed that for every increment of 1% boost in HbA1c there was a 1.11-fold boost in the danger of CHD.
Blood pressure	Chronic hypertension is very closely pertaining to the danger of creating CHD. A decrease of 5 mmHg in diastolic blood pressure is associated with a 21% decline in threat of developing CHD.
Obesity	Although raised body mass index is associated with increased threat of CHD, there are no professional trials of the result of weight reduction on CHD morbidity as well as death.

CHD, coronary heart disease; UKPDS, UK Prospective Diabetes Study; HbA1c, glycated hemoglobin.

CONCLUSION:

CVD is a major cause of disability as well as premature death throughout the globe. The underlying pathology of atherosclerosis establishes over several years and is generally advanced by the time signs and symptoms happen, normally in midlife. Many traditional risk factors for CAD are related to lifestyle; hence, preventive therapy can be adjusted to customizing specific aspects. It is critical to understand these risks in order to reduce disability and also sudden fatalities from CHD, cerebrovascular disease, and peripheral vascular disease in patients at high risk who have not yet suffered a cardiac event. Individuals with established CVD go to extremely high threat of recurring occasions.

Primary prevention programs aimed at reducing risk behaviors on a population-wide scale, as well as the identification, stratification, and also targeted treatment of high-risk individuals prior to the progression of condition, should be cornerstones of any strategy to reduce the population's fear of CHD and also, prevention strategies must start in childhood.

REFERENCES:

1. WHO. Cardiovascular diseases (CVDs). 2016. Available at: http://www.who.int/mediacentre/factsheets/fs317/en/ (accessed 10 October 2016).
2. Lopez AD. Assessing the burden of mortality from cardiovascular diseases. World Health Star. Q. 1993;46:91–96.
3. Dodu SRA. Emergence of cardiovascular disease in developing countries. Cardiology. 1988;75:56–64.
4. Greenland P, Alpert JS, Beller GA, Benjamin EJ, Budoff MJ, Fayad ZA, et al. 2010 ACCF/AHA guideline for assessment of cardiovascular risk in asymptomatic adults: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2010;122:e584–636.
5. Cohen R, Budoff M, McClelland RL, Sillau S, Burke G, Blaha M, et al. Significance of a positive family history for coronary heart disease in patients with a zero coronary artery calcium score (from the multi-ethnic study of atherosclerosis) Am J Cardiol. 2014;114:1210–4.

6. Goff DC, Jr, Lloyd-Jones DM, Bennett G, Coady S, D'Agostino RB, Gibbons R, et al. 2013 ACC/AHA guideline on the assessment of cardiovascular risk: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2014;129:S49–73.

7. World Health Organization. Prevention of Recurrent Heart Attacks and Strokes in Low and Middle Income Populations. Evidence-Based Recommendations for Policy Makers and Health Professionals. Geneva: 2003. [Last accessed on 2017 Jul 25]. Available from: http://www.who.int/bookorders .

8. Hobbs FDR. Cardiovascular disease: different strategies for primary and secondary prevention? *Heart*. 2004;90(10):1217-1223. doi:10.1136/hrt.2003.027680.

9. Piepoli MF, Hoes AW, Agewall S, et al. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of 10 societies and by invited experts) Developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR). Eur Heart J 2016; 37: 2315–2381.

10. Eckel RH, Jakicic JM, Ard JD, et al. 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013. 2014: 63(25_PA).

11. NICE. Cardiovascular disease: risk assessment and reduction, including lipid modification. NICE Guideline CG181, 2016.

12. Perk J, De Backer G, Gohlke H, et al. European Guidelines on cardiovascular disease prevention in clinical practice (version 2012). Eur Heart J 2012; 33: 1635–1701.

13. de Souza RJ, Mente A, Maroleanu A, et al. Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. BMJ 2015; 351: h3978–h3978.

14. Kottke TE, Battista RN, DeFriese GH, et al. Attributes of successful smoking cessation interventions in medical practice: a meta-analysis of 39 controlled trials. JAMA 1988; 259: 2882–2889.

15. Tzivoni D, Keren A, Meyler S, et al. Cardiovascular safety of transdermal nicotine patches in patients with coronary artery disease who try to quit smoking. Cardiovasc Drugs Ther 1998; 12: 239–244.

16. Bhatnagar A. E-Cigarettes and cardiovascular disease risk: evaluation of evidence, policy implications, and recommendations. Curr Cardiovasc Risk Rep 2016; 10: 1–10.

17. Khosravi A, Akhavan Tabib A, Golshadi I, et al. The relationship between weight and CVD risk factors in a sample population from central Iran (based on IHHP). ARYA Atheroscler 2012; 8: 82–89.