

Contributory factors and Prevention of Coronary Heart Disease

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ABSTRACT: Coronary heart disease (CHD) has high mortality worldwide, highest in South Asian subcontinent. Malaysian develops CHD at younger age of 58 years, compared to other countries. Contributory factor (risk factor) for CHD includes: smoking, heavy alcohol consumption, unhealthy diet, obesity, genetics, and family history of CHD, hypertension, and sedentary life style. Researchers advocate that atherosclerosis, the precursor of CHD begins in childhood. CHD is life threatening of the diabetic complications. Stroke and heart failure hypotensive heart disease, rheumatic heart disease, and cardiomyopathy are frequent clinical symptoms. ECGs, exercise echocardiography, myocardial perfusion imaging, and cardiac stress are not recommended in those who are not at risk. Primary prevention interventions done through using a variety of scores e.g. Framingham or Reynold scores. Treatment of hypertension, high blood lipids and diabetes. Prevention, healthy lifestyle, avoiding smoking and reduced alcohol consumption.

Keywords: Coronary heart disease (CHD), Contributory factor, Prevention.

Date of Submission: 07-08-2019

Date of acceptance: 23-08-2019

I. INTRODUCTION

Coronary heart disease (CHD) also known as cardiovascular disease (CVD), coronary artery disease (CAD) and ischemic heart disease (IHD), that involve the heart or blood vessels [1]. Coronary heart disease are the leading cause of death worldwide and in all regions except Africa [2]. In 2008, 30% of all global death was attributed to cardiovascular diseases. It is also estimated that by 2030, over 23 million people will die from CHD/CAV diseases each year. It is estimated that 60% of world's cardiovascular disease burden will occur in the South Asian subcontinent despite only accounting for 20% of the world population [3]. Malaysian developing heart disease at younger age of 58 years, compared in Thailand (65 years), Mainland China (63 years), western countries (66 years) and Canada (68 years). WHO estimated in 2014 the Malaysian mortality rate of CHD/CVDs stood at 36 percent. Worldwide, an estimated 17.7 million died from CHD/CVDs in 2015 representing 31 percent of global deaths [4]. The underlying mechanisms (risk factors) vary depending on the disease [1]. CAD, stroke, and peripheral artery disease involve atherosclerosis [1]. This may be caused by high blood pressure, smoking, diabetes, lack of exercise, obesity, high cholesterol, poor diet, and excessive alcohol consumption, among others [1]. Clinical presentation (types of CHD) include, coronary heart diseases, stroke, heart failure, hypotensive heart disease, rheumatic heart disease, and cardiomyopathy [1]. Treating high blood pressure, high blood lipids, and diabetes [1]. Prevention: healthy eating, exercise, avoiding tobacco smoking, and limiting alcohol intake [1]. Papers reviews the risk factor and possible prevention of CHD-CVD.

II. CONTRIBUTORY FACTOR

There are many contributory or risk factors for heart diseases, tobacco use, physical inactivity, excessive alcohol consumption, unhealthy diet, obesity, genetics, predisposition and family history of cardiovascular disease, raised blood pressure (hypertension), raised blood sugar (diabetes mellitus), raised blood cholesterol (hyperlipidemia), undiagnosed celiac disease, psychosocial factors, poverty and low educational status, and air pollution [5,6,7]. While the individual contribution of each risk factor varies between different communities or ethnic groups the overall contribution of these factors is very consistent [8]. Some of these factors, such as age, gender or family history/genetic predisposition, are immutable; however, many important cardiovascular risk factors are modifiable by lifestyle change, social change, drug component (for example prevention of hypertension, hyperlipidemia, and diabetes) [9]. People with obesity are at increased risk of atherosclerosis of coronary arteries [10].

Role of genetics

Genetic factors or family history influence the development of cardiovascular disease in men who are less than 55 years old and in women who are less than 65 years old [9]. Cardiovascular disease in a parent's increases their risk by 3 fold [11]. Multiple single nucleotide polymorphism (SNP) have been found to be associated with cardiovascular disease in genetic association studies [12], but their individual influence is small, and genetic contributions to cardiovascular disease are poorly understood [13].

Age factor

Age is the most important factor in developing CHD/CVD or heart disease, with approximately tripling of risk with each decade of life [14]. Coronary fatty streaks can begin to form in adolescence [15]. It is estimated that 82 percent of people who die of CHD are 65 and older [16]. The risk of stroke doubles every decade after age 55 [17]. Multiple explanations are proposed to explain why age increases the risk of CHD/CVD. One of them relates to serum cholesterol level [18]. In most populations, the serum total cholesterol level increases as age increases. In men, this increase levels off around age 45 to 50 years. In women, the increase continues sharply until age 60 to 65 years [19]. Aging is also associated with changes in the mechanical and structural properties of the vascular wall, which leads to the loss of arterial elasticity and reduced arterial compliance and may subsequently lead to coronary artery disease [19].

Gender

Men are at greater risk of CHD than pre-menopausal women [14,20]. Once past menopause, it has been argued that woman's risk is similar to a man [20], although more recent data from the WHO and UN disputes this [14]. If a female has diabetes, she is more likely to develop CHD than a male with diabetes [21]. Coronary heart diseases are 2 to 5 times more common among middle aged men than women [1]. In a study done by the World Health Organization, gender (sex) contributes to approximately 40% of the variation in sex ratio of coronary heart disease mortality [22]. Another study reports similar results finding that gender differences explain nearly half the risk associated with cardiovascular disease [18]. One of the proposed examinations for gender differences in cardiovascular diseases is hormonal difference [18]. Among women, estrogen is the predominant sex hormone. Estrogen may have protective effects on glucose metabolism and hemostatic system and may have direct effect in improving endothelial cell function [19]. The production of estrogen decreases after menopause, and this may change the female lipid metabolism toward a more atherogenic form by decrease ng the HDL cholesterol while increasing LDL, and total cholesterol [18].

Among men and women, there are notable differences in body weight, height, body fat distribution, heart rate, stroke volume, and arterial compliance [19]. In the very elderly, age-related large artery plasticity and stiffness is more pronounced among women than men [19]. This may be caused by the women's smaller body size and arterial dimensions which are independent of menopause [19].

Smoking

Cigarettes are the major form of smoked tobacco [1]. Risks to health from tobacco use result not only from direct consumption of tobacco, but also from exposure to second-hand smoke [1]. Approximately 10% of cardiovascular disease is attributed to smoking [1], however people who quit smoking by age 30 have almost as low a risk of death as never smokers [23].

Sedentary lifestyle

Lack of exercise and insufficient physical activity (defined as less than 5x30 minutes of moderate activity per week, or less than 3x20 minutes of vigorous activity per week) is currently the fourth leading risk factor for mortality [1]. In 2008, 31.3% of adults aged 15 or older (28.2% men and 34.4% women) were insufficiently physically active [1]. The risk of ischemic heart disease and diabetes mellitus is reduced by almost a third in adults who participate in 150 minutes of moderate physical activity each week (or equivalent) [24]. In addition, physical activity assists weight loss and improve blood glucose control, blood pressure, lipids profile and insulin sensitivity. These effects may, at least in part, explain its cardiovascular benefits [1].

Nutritional factor

High dietary intakes of fat, trans-fat and salt, and low intake of fruits, vegetables and fish are linked to cardiovascular risk, although whether these associations are cause is disputed. The World Health Organization (WHO) attributes approximately 1.7 million deaths worldwide to low fruit and vegetable consumption [1]. The amount of dietary salt consumed is also an important determinant of blood pressure levels and overall cardiovascular risk [1]. Frequent consumption of high energy foods, such as processed foods that are high in fats and sugars, promotes obesity and may increase cardiovascular risk [1]. A Cochrane review found that replacing saturated fat with polyunsaturated fat (plant based oils) reduce cardiovascular disease risk.

Cutting down on saturated fat reduced risk of cardiovascular disease by 17% including heart disease and stroke.[25].High trans-fat intake has adverse effects on blood lipids and circulating inflammatory markers [26], and elimination of trans-fat from diets has widely been advocated [27].In 2008 the WHO estimated that trans-fat were the cause of more than half a million deaths per year [28].

There is evidence that high consumption of sugar is associated with high blood pressure and unfavorable blood lipids [29], and sugar intake also increases the risk of diabetes mellitus [30].High consumption of processed meats is associated with increased risk of cardiovascular disease, possibly in part due to increased dietary salt intake [31].The relationship between alcohol consumption and cardiovascular disease is complex, and may depend on the amount of alcohol consumed. There is a direct relationship between levels of alcohol consumption and risk of cardiovascular disease [1].Drinking a low level without heavy drinking may be associated with a reduced risk of a cardiovascular disease [32].Overall alcohol consumption at population level is associated with multiple health risks that exceeds benefits [1].

Duration of sleep and Celiac disease

Sleep disorders such as sleep disordered breathing and insomnia, as well as particularly short duration or particularly long duration of sleep, have found to be associated with higher cardiovascular disease [33].Untreated celiac disease can cause the development of many types of cardiovascular diseases, most of which improve or resolve with gluten-free diet and intestinal healing. However, delays in recognition and diagnosis of celiac disease can cause irreversible heart disease [7].Some research has indicated there may be an increased risk of heart disease among individuals with celiac disease, and that it may be related to the inflammatory process which is characteristic of the condition—specifically systemic subclinical inflammation. At the same time, researchers have noted that other aspects of celiac disease could also be related to increased risk of atherosclerosis. These include a decrease in nutrients like B vitamins and folic acid which can result from intestinal damage, factors which are generally reversed with strict adherence to gluten-free diet [34].

Low socioeconomic group

Cardiovascular disease affects low-and middle income (low socioeconomic) countries [35].There is relatively little information regarding social patterns of cardiovascular disease within low-and middle income countries[35],but within high income countries low income and low educational status are consistently associated with greater risk of cardiovascular disease[36].Policies that have resulted in increased low socioeconomic (socio-economic) inequalities have been associated with greater subsequent socio—economic differences in cardiovascular disease [35].Implying a cause and effect relationship. Psychological factors, environmental exposures, health behaviors, and health care access and quality contribute to socio-economic differentials in cardiovascular disease [37].The commission on Social Determinants of Health recommended that more equal distribution of power,wealth,education, housing, environmental factors,nutrition, and healthcare were needed to address inequalities in cardiovascular disease and communicable diseases [38].

CHD and air pollution

Particulate matter (PM) has been studied for its short –along term exposure effects on CHD/ CVD.Currently,PM_{2.5}is the major focus, in which gradients are used to determine CHD/ CVD risk. For every 10ug/m³ of PM 2.5 long term exposure, there is an estimated 8-18% CHD/CVD mortality risk [39,wp,49].Women had a higher relative risk (RR) (1.42) for PM 2.3 induced coronary artery disease (CAD) than men (0.90) did [39].In regards to short term exposure (2 hours),every 25 ug/m3 of PM 2.5 resulted in 48% increase of CHD/ CVD mortality risk [40].In addition, after only 5 days of exposure, a rise in systolic (2.8mmHg) and diastolic (2.7 mmHg) blood pressure occurred for every 10.5 ug/m3 of PM2.5 [40].Other research has implicated PM2.5 in irregular heart rhythm, reduced heart rate variability(decreased vagal tone),and most notably failure [41].PM2.5 is also linked to carotid artery thickening and increased risk of acute myocardial infarction [51].

CHD determination

Existing coronary heart disease or previous or cardiovascular event, such as heart attack or stroke, is the strongest predictor of a future CHD event [42],age sex, smoking, blood pressure, blood lipids, and diabetes are important predictors of future CHD in people are not known to have CHD [43].These measures and sometimes others, may be combined into composite risk scores to estimate an individual's future risk of cardiovascular disease [42].Numerous risk scores exist although their merits are debatable [44].Other diagnostic tests and biomarkers remain under evaluation but currently these lack clear-cut evidence to support their routine use. These include family history, coronary artery calcification score, high sensitivity C-reactive protein (hs-CRP),ankle-brachial pressure index, lipoprotein subclasses and particle concentration, lipoprotein(a),apolipoproteins A-1 and B,fibrinogen,white cell count homocysteine-terminal pro B-type natriuretic

peptide(NT- proBNP),and markers of kidney function [45].High blood pressure is also linked to an increased risk [46].

Environmental factors

Little is known about the relationship between work and CHD ,but links have been established between certain toxins, extreme heat and cold, exposure to tobacco smoke, and mental health concerns such as stress and depression[47].A 2015 SBU- report looking at non-chemical factors an association for those [48],includes:.

- a).with mentally stressed work with lack of control over their working situation-with an effort reward imbalance [48].
- b).Individuals experience low social support at work; who experience injustice or experience insufficient opportunities for personal development; or those who experience job insecurity [48].
- c).Those that work night schedules; or have long working weeks [48].
- d).persons exposed to noise [48].

Specifically the risk of stroke was also increased by exposure to ionizing radiation [48].Hypertension develops more often in those who experience job strain and who have shift work [48].Differences between women and men in risk are small, however men risk suffering and dying of heart attacks twice as often as women during working life [48].

Exposure to chemicals and Mutational factors

A 2017 SBU report found evidence that workplaces exposure to silica dust, engine exhaust or welding fumes is associated with heart disease [49].Association also exists for exposure to arsenic, benzo pyrenes ,lead, dynamite, carbondisulphide,carbonmonoxide, metal working fluids, and occupational exposure to tobacco smoke[49].Working with the electrolytic production of aluminum or the production of paper when sulphate pulping process is used is associated with heart disease [49].An association was also found between heart disease and exposure to compounds which are no longer permitted in certain work environments, such as phenoxy acids containing TCDD (dioxin) or asbestos [49].Workplace exposure to silica dust or asbestos is also associated with pulmonary heart disease. There is evidence that workplace exposure to lead, carbondisulphide,phenoxyacids containing TCDD as well as working in environment where aluminum is being electrolytically produced with stroke [49].

As of 2017, evidence suggests that certain leukemia associated mutations in blood cells may also lead to increased risks of cardiovascular disease. Several large scale research projects looking at human genetics data have found a robust link between of these mutations, as condition known as hematopoiesis, and cardiovascular disease related incidents and mortality [50].

III. PATHOPHYSIOLOGY

Population studies show that atherosclerosis, the major precursor of cardiovascular disease, begins in childhood. The path-biological Determinants of Atherosclerosis in Youth (PDAY) study demonstrated that initial lesions appear all the aortas and more than half of the right coronary arteries of youths aged 7-9 years [51].This is extremely important considering that 1 in 3 people die from complications attributable to Atherosclerosis. In order to stem the tide, education and awareness that cardiovascular disease poses the greatest threat, and measures to prevent or reverse this disease must be taken. Obesity and diabetes mellitus are often linked to cardiovascular disease [52],as are a history to chronic kidney disease and hypercholesterolemia [53].In fact, cardiovascular disease is the most life-threatening of the diabetic complications and diabetics are two to four-fold likely to die of cardiovascular related causes than nondiabetics [54].

IV. DIAGNOSTIC -WORKOUT

Screening ECGs (either at rest or with exercise-stress ECG) are not recommended in those without symptoms who at low risk [55].This includes those who are young without risk factors [56].In those at higher risk the evidence for screening with ECG is inconclusive [57].Additional echocardiography, myocardial perfusion imaging, and cardiac stress is not recommended in those who do not have symptoms [58].Some biomarkers may add to conventional risk factors in predicting the risk of future cardiovascular disease; however, the value of some biomarkers is questionable [59].Ankle brachial index (ABI),high sensitivity C-reactive protein(hcCRP) and coronary artery calcium also of unclear benefit in those without symptoms as of 2018 [60].NIH recommends lipid testing in children beginning at age of 2 if there is a family history of heart disease or lipids problems [61].

Screening and selection for primary prevention interventions has traditionally been done through absolute risk using a variety of scores (ex.Framingham or Reynolds risk scores) [62].This stratification has separated people who receive the lifestyle interventions(generally lower and intermediate risk) from medication(higher risk).The number and variety of risk scores available for use has multiplied, but their efficacy

according to a 2016 review was unclear due to lack of external validation or impact analysis [63]. Risk stratification models often lack sensitivity for population groups and do not account for large number of negative events among intermediate and low risk groups [61].

V. PREVENTION AND TREATMENT

Up to 90% of cardiovascular disease may be preventable if established risk factors are avoided [64]. Currently practiced measures to prevent cardiovascular disease include:

- a). Tobacco cessation and avoidance of second-hand smoke [65]. Smoking cessation reduces risk by 35% [66].
- b). A low fat, low sugar, high fiber diet including whole grain and fruit and vegetables [65]. Dietary interventions are effective in reducing CVD risk factors over a year, but long term effects and their impact on CVD events is uncertain [67].
- c). Regular exercise at least 30 minutes, 5 days a week (can be 10 minutes, 3 times per day) reduces risk of CVD events by 26% [68,69].
- d). Reduce alcohol consumption. Moderate alcohol consumption lowers risk of CVD by 25- 30% [65,69].
- e). Lower blood pressure, if elevated. A 10mmHg reduction in blood pressure reduces risk by about 20% [70].
- f). Decrease non-HDL cholesterol [71]. Statin treatment reduces CVD mortality by about 31 % [71].
- g). Decrease body fat if overweight or obese [72]. Weight loss following bariatric surgery is associated with a 46% reduction in CVD [73].
- h). Reduce stress-decrease psychosocial stress [74]. Mental stress-induced myocardial ischemia is associated with an increased risk of heart problems in those with previous heart disease. [75]. Severe emotional physical stress leads to a form of heart dysfunction known as Takotsubo in some people [76].

Treatment and management: CHD-CVD disease is treatable with initial treatment primarily focused on diet and lifestyle interventions [1]. Influenza may make heart attacks and strokes more likely and therefore influenza vaccination may decrease. Blood pressure medication reduces CHD-CVD in people at risk [70]. Statins are effective in preventing further CHD-CVD in people with CHD [77]. Anti-diabetic medication may reduce CHD risk in people with Type 2 diabetes, although evidence not conclusive [78]. Aspirin has been found to be of only modest benefit in those at low risk of heart disease as the risk of serious bleeding is almost equal to benefit with respect to CHS-CVD problems [79]. A systematic review estimated that physical inactivity is responsible for 6 % of the burden of the disease from coronary heart disease worldwide [80]. Proper CHD-CVD necessitates a focus on MI and stroke cases due to their combined high mortality rate, keeping in mind the cost-effectiveness of any intervention, especially in developing countries with low or middle income levels [62].

VI. CONCLUSION

The majority of coronary heart disease (CHD) or cardiovascular disease (CVD) occurs in South Asian countries with high mortality. Risk factors of CHD/CVD e.g., smoking, heavy alcohol consumption, unhealthy diet, obesity, genetics, family history, hypertension and sedentary lifestyle. Understanding of these risk factors is important for the prevention of coronary heart disease.

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