Explanation of basic cause of cardiovascular disease

Abstract

There are lots of factors which can be a cause for cardiovascular disease including obesity, smoking, genetic factors, drug product, abnormal lipid metabolism and etc. Cardiovascular diseases spread all over the world and the death rating risk of cardiac disease is getting higher. That's why the cause of cardiovascular disease must be known to overcome cardiac disease and for this reason, this review article was considered inevitable and we will be talking about the structure of human cardiac cells and the basic cause of cardiovascular disease.

Keywords: Obesity, smoking, genetic factor, intercalated disc, renin-angiotensin system, MMP

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Introduction

The risk factors of cardiovascular disease are endogen and exogen. Obesity, smoking, abnormal lipid metabolism, genetic factor, and renin-angiotensin system dysfunction belong to the basic risk factors of cardiovascular diseases. Also, cardiovascular disease rating is different for ethnic differentiation. In this review, we will be talking about the explanation of the basic cause of cardiac disease and the structure of cardiac heart cells.

Cardiovascular disease occurs due to the genetic factor and depends on a multiple caused disease. Most of the cardiovascular disease's root are genetic causes. Cardiovascular diseased people are more in the western country. Some analysis which are performed by efficacy method has shown that most of the cause of this kind of diseases are genetic mutations which regulate lipid metabolism, renin-angiotensin system, fibrinolytic proteins. Angiotensin 2 and renin play a big role in the cardiovascular disease, so the activated angiotensin bind to the receptor which are located in the vascular wall and delivers vasoconstriction and it will cause hypertension due to narrowed vascular lumen [1]. That's why for the hypertension disease it is needed to block binding of the angiotensin 2 with the receptor, it prevents the high blood pressure which may cause atherosclerosis of the central artery as coronary artery and peripheral artery. Inhibitor of the angiotensin-converting enzyme also inhibits the formation of angiotensin 2 from angiotensin 1 and during this time the bradykinin level increases in blood serum which this molecule deliver vasodilator. Angiotensin 2 takes to play in the formation of atherosclerosis. Aliskiren is an inhibitor of renin and hydrophilic nonpeptide which has low molecule weight intakes orally [2]. The daily prescriptions dose is 300 mg/day and prevents having atherosclerosis plaque.

The synthesis of renin and angiotensin inhibited by three ways 1) the inhibition of generating angiotensin 1 from angiotensinogen (by inhibiting aspartyl proteases, zinc-dependent specific proteases), 2) inhibition of generating angiotensin 2 from angiotensin 1 (by converting enzyme), 3) the blockade of angiotensin receptor in the vascular wall. Renin synthesis occurs in the different organ by different kind special cell group including retina, scrotum, ovarium, and adrenal. There are two kind receptors of the angiotensin 2 in the vascular wall. Angiotensin 2 could hydrolize by a specific kind of proteases. Amount of the prorenin increases during pregnancy, it is explained by the prorenin synthesis process in ovarium [3].

Lots of gene mutations are responsible for cardiovascular disease, some of them are shown in the table **(TABLE 1).**

TABLE 1. Chromosomal aaation studies in selected populations.

Chromosome loci	Study population	Aaaoclatlon
Chromosome 9p21.3 (17)	Welcome trust case	Coronary artery disease
	Control consortium	
	German myocardial infarction family study	Coronary artery disease
Thrombospondins and plaeminogen activator inhibitor-2 (18)	Gene quest	Coronary artery disease
Thrombospondins-4 (18)	Gene quest	Coronary artery disease
Thrombospondins-2 (18)	Gene quest	Coronary artery disease
Plasminogen activator inhibitor-2 (18)	Gene quest	
15q26 MEF2A (16)		Coronary artery disease
2q21.1-22 (22)	Finns	Premature coronary artery disease
Xq23-26 (22)	Finns	Premature coronary artery disease
16p13.3 (23)	Indo-Mauritians	Coronary artery disease
14o11.2-12 (24)	Western European	
6p21.3 (28)		Coronary artery calcification
10q21.3 (28)		Coronary artery

The gene mutation which is regulating heavy chain of myosin is located in a 14q1 chromosome. Cardiac troponin regulating gene is located on a 1q3 chromosome and alpha-tropomyosin located on a 15q2 chromosome. The mutations in 11q11 and 7q3 genes could be the reason for the hypertrophy of cardiac [4].

The human myocardiocyte contracting 3 billion times average in lifespan, pumping 7000 l blood from the heart every day. There is an intercalated disc in between human cardiac cells and close to Z disc. Intercalated disc forms by three cell junction **(FIGURE 1)**. 1) *fascia adherens* is fixing the terminal part of actin 2) Desmosomes prevent the separation of cardiac cells during the contraction 3) Gap junction propagates the depolarization along with the cardiac cells. The genetic defect of regulator genes of intercalated disc being causes dilatation of cells, hypertrophy, deficiency of depolarizing of cardiac cells [5].

Spreading of cardiovascular disease is also dependent on the ethnic group as every ethnic group has a specially selected genome. For example, Europa originated Caucasian has this risk is higher, except the Southern Asians. African-Americans belong to the risked ethnic group for cardiovascular disease. But African-Carribians and the UK people have a lower risk than other ethnic groups. China and Japan people also belong to a risked group, but in this ethnic group, the coronary heart disease is faced less than others. Performed trial by

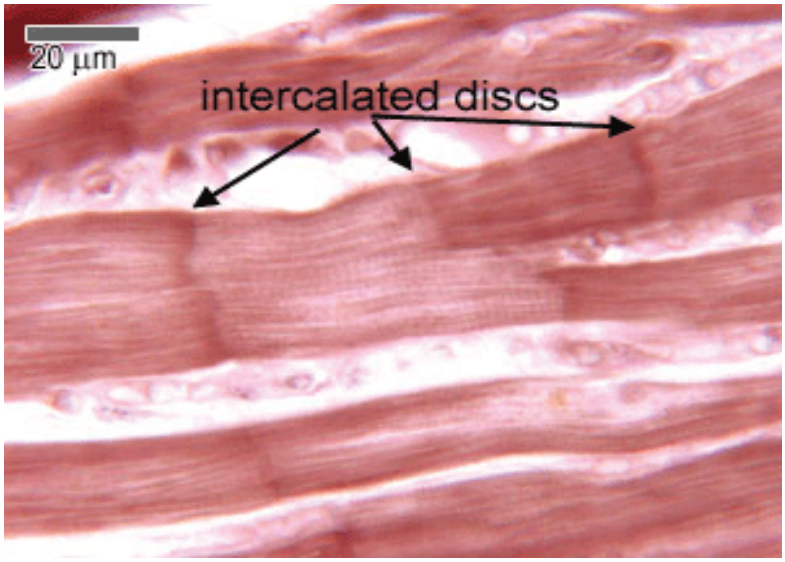

FIGURE 1. The intercalated disc.

angiography, intravascular ultrasound, and computed tomography results have shown that the atherosclerosis process being more in left circumflex coronary artery compared to another part of a coronary artery because of hemodynamic and anatomical localization [6]. There is some kind of enzymes which play the role to form the cardiovascular disease as Matrix Metalloproteinase (MMP), to deliver pathology process of MMP which depends on the relationship between the rate of enzyme and its inhibitors. If there is some inhibitor shortage of MMP in the heart tissue it will deliver degradation of the extracellular matrix of heart, the heart valves will incur degradation due to deficiency of inhibitors of MMP [7].

Obesity is a risk factor for the delivering of cardiovascular disease. For the detection of the obesity rate used Body Mass Index (BMI) **(FIGURE 2).** It means that if we want to detect the rate of obesity in a person we must calculate the person's BMI. The body weight is divided quadrat of height for this purpose. Normal BMI is being between 18.5-24.9 interval, the higher one means obesity which is classified in four groups: 1) 25.0-29.9 is overweight, 2) 30.0-34.9 is 1st-grade obesity, 3) 35.0-39.9 is 2nd-grade obesity, 4) 40.0-49.9 is 3rd-grade obesity. In the United States, 70% of people suffer from obesity. Every year 2.6 million people die because of obesity [8]. Obesity is a risk factor for cardiovascular disease, diabetes, stroke, asthma, liver and bile bladder disease, cancer, sleeping apnoea, osteoarthritis, gynecology disease, insulin resistance. During obesity for each over 10 kg, the arterial pressure become 3 mmHg higher in systolic phase than normal blood pressure and diastolic pressure being higher 2.3 mmHg than normal pressure [9].

Every year 140000 people die due to cigarette smoking in the world. One of every 10 death occurs because of cigarette smoking [10]. Increased risk of having thrombosis, atherosclerosis, and narrowing of the vessels is there in a smoker. Some toxic substances in cigarette could cause to deliver aorta aneurysm. In any case, the normal amount of nicotine in the blood hasn't been higher than 0.7 ng/ml in smokers [10]. There should be a stop on smoking, it should be gradually throw away, not suddenly, because some substances of the cigarette irritate the sympathetic nervous system and blood flow to the heart tissue and demand oxygen, others nutrition for the myocardium, more after this myocardium, begins getting hypertrophy. While the cessation of smoking suddenly can deliver the risk of myocardial infarction due to some substances of the cigarette will not deliver blood flow to the heart tissue and resulted in ischemia in the myocardium and next step of ischemia may cause myocardial infarct. A cigarette causes cardiovascular disease more among smoker women than among men smoker. In ultrasonography, it has shown dilatation of the heart tissue and aneurysm of a coronary artery in a smoker. CO_2, the nicotine, the aromatic hydrocarbons, the oxidant gas amount is present in cigarette smoke. Nicotine also causes lipid abnormalities, insulin resistant and endothelial dysfunction. Cigarette smoke increase epinephrine from 44 to 113 pg/ml within 10 minutes after smoking and norepinephrine from 227 to 315 pg/ml in plasma and make free oxygen radicals. Heart beating and blood pressure is higher in a smoker than nonsmokers. The diagnosis which is getting by positron emission tomography has shown microvascular blood pressure is low in smokers and blood supply is so poor because of the narrowed effect of nicotine in the vascular wall. A total area of endothelial cells is equal to four tennis court area and all endothelial cells weight is equal one kilogram which all makes protein, mediator, lipid and hormone character matter which is very important for human organism and in common a total amount of endothelial cells is bigger than endocrine glandular in a human body. The cigarette could deliver the vascular pathology, endothelial injuring (the inflammation, the proliferation, the thrombosis). Cigarette smoking also increases the activity of endothelial cells and angiogenesis by angiogenic cytokines that some chemical in cigarette can deliver. A cigarette is able to increase the vascularizing of cancer tissue based on performed trial which can be seen in cancer tissue of lungs *In vivo*. Hematocrit was higher than normal because of CO and carboxyhemoglobin, C reactive protein also was higher because of the small inflammation area [11]. Cardiovascular risk in women which are beneath 50 years have a low risk because of protective effect to a vascular vessel of estrogen in women [12].

Some kind of drug product as Amphotericin B could impart toxic effect to the myocardium

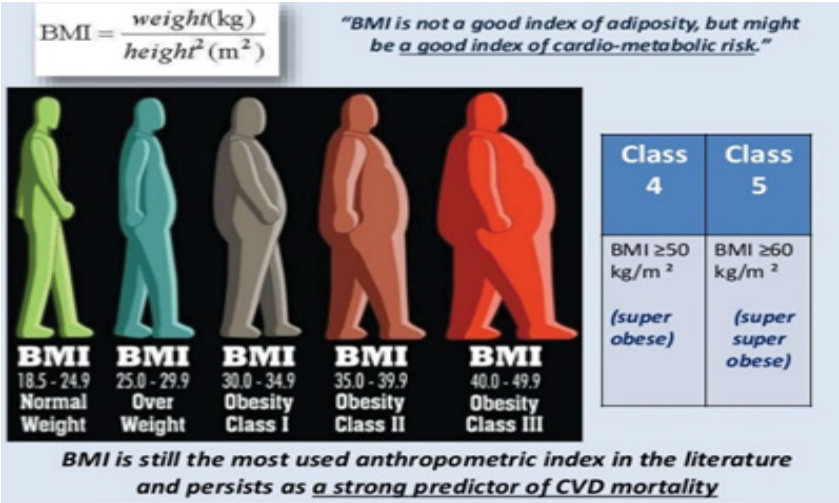

FIGURE 2. The calculation of body mass index.

and consumption of this drug could deliver arrhythmia, fibrillation myocardium infarction [13,14].

Conclusion

There are lots of factors that can cause cardiovascular disease including obesity, smoking, genetic factors, drug product, abnormal lipid metabolism and etc. Endogen factors are genetic, renin-angiotensin system, obesity, MMP enzymes, exogen factors are smoking, some drugs as AmB. In this review, we have some pieces of information about each factor.

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