(REVIEW ARTICLE)

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Review on cardiovascular disease and antihypertensive drugs effect on the circulating biomarkers of heart disease

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Abstract

Cardiovascular diseases (CVD), a group of diseases that affect the heart and blood vessels have posed high morbidity rate to humans, with the second-high mortality rate after cancer. These diseases include hypertension, coronary artery disease, cardiac dysrhythmias, cerebrovascular disease, valvular heart disease, cardiomyopathies, peripheral vascular disease, and congenital cardiac abnormalities. These diseases have been observed to affect people of older age and have higher susceptibility in male than the female counterparts. Some circulating biomarkers such as C-reactive protein, cardiac troponin I, myostatin, homocystein, dimethylarginine, e.t.c have been found to be indispensable in the diagnosis and prediction of possible occurrence of CVD. It has been postulated that C-reactive protein rises in acute inflammation and may contribute to further atherosclerosis by stimulating macrophage uptake of low density lipoprotein cholesterol (LDL-C). Antihypertensive drugs are used to combat these diseases and they come in different forms with variable mechanisms of action. Antihypertensive drugs have been postulated to have lowering effect on circulating biomarkers of CVD. However, prophylactic measures are highly recommended.

Keywords: Cardiovascular Diseases; Hypertension; Biomarkers; Antihypertensive Drugs; C - reactive protein

1. Introduction

Cardiovascular diseases (CVD) have been proven to be the largest single cause of death on earth, recording 50% - 75%out-of-hospital death in developed countries but in Nigeria, the distribution of the out-of-hospital death is sparsely studied due to poor patronization of orthodox medical centers [1]. CVD are a group of diseases that affect the heart and the blood vessels (basically the cardiovascular system). These diseases have been seen to affect mostly people of older ages [2]. These disorders include but are not limited to: hypertension, coronary artery disease, cardiac dysrythmias, cerebrovascular diseases, valvular heart diseases, cardiomyopathies, peripheral vascular disease, and congenital cardiac abnormalities. The incidence and prevalence of these disorders vary greatly with culture and country. It had been found to be the leading cause of death in developed and first world countries. It has also been established that age and gender are contributory.

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Cardiovascular diseases can be congenital or acquired. Many etiologies have been tied to these disorders. The effective diagnosis of these diseases are made by targeting specific markers like proteins, nucleic acids and inflammatory products present in the blood circulation at the time of illness [3].

According to Thiriet [3], rare cardiovascular maladies are classified into eight groups:

- Class i (rare afflictions of the systemic circulation)
- Class ii (are afflictions of the pulmonary circulation)
- Class iii (rare cardiomyopathies)
- Class iv (rare congenital cardiovascular disorders)
- Class v (rare cardiac arrhythmias)
- Class vi (cardiac tumors and cardiovascular affections related to cancer)
- Class vii: (cardiovascular sickness in pregnancy)
- Class viii (other types of rare cardiovascular illness)

Researchers have proven that biomarkers of cardiovascular disease are indispensable in the diagnosis of cardiovascular diseases. These biomarkers are found in circulation when the heart is subjected to severe pressure or injury due to an insufficient supply of Oxygen and other insults. These biomarkers can be presented as hormones, enzymes, and proteins. The estimation of these biomarkers in circulation is a breakthrough that has presented better management and prediction of a possible CVD occurrence.

A great fraction of adult human population is on antihypertensive medication for cardiovascular diseases [4]. These medications are classified into diuretics, beta-blockers, calcium channel blockers, rennin-angiotensin system inhibitors, e.t.c. It has been reported that these antihypertensive drugs have a lowering effect on the circulating CVD biomarkers with angiotensin-converting enzyme inhibitors showing the strongest lowering effect by decreasing plasma levels of myostatin [4].

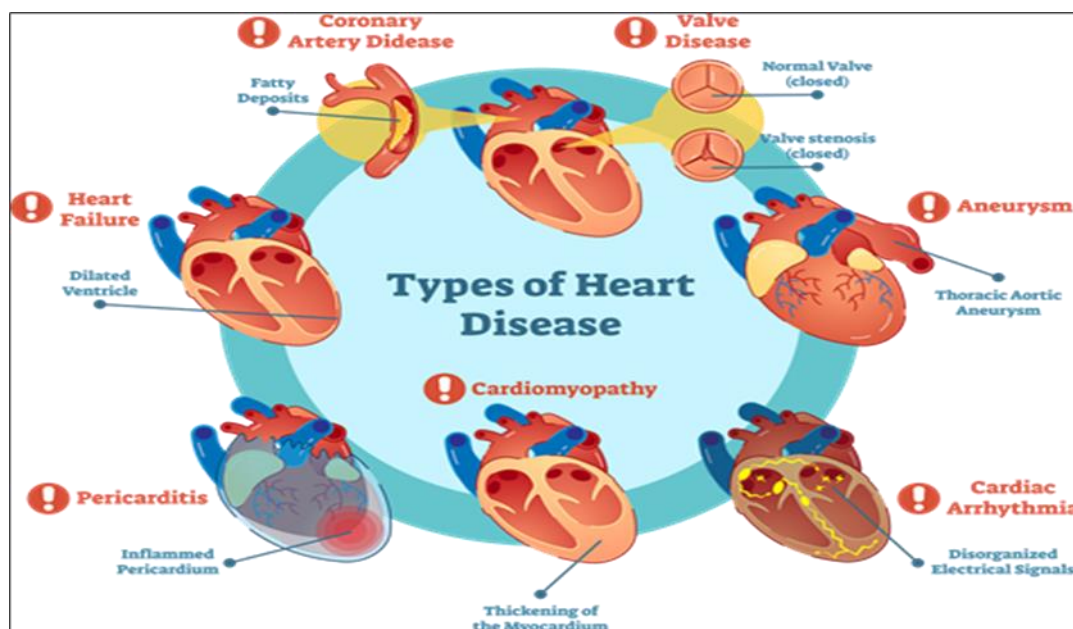## 1.1. Overview of Cardiovascular Diseases

Figure 1 Overview of heart disease

Cardiovascular diseases are defined as a group of disorders that involve the heart and blood vessels [5]. It is made up of many conditions which can develop into a secondary disease [6]. The examples of diseases in this category include: coronary heart disease, stroke, rheumatic heart disease, cardiomyopathy [7], arrhythmia, congenital heart diseases, heart attack, heart failure, mitral regurgitation, mitral valve prolapse, pulmonary stenosis, aortic stenosis, atrial fibrillation, radiation heart disease [6], peripheral atrial disease and deep vein thrombosis [5].

1.1.1. Coronary Heart Disease

This is a heart disease that affects the blood vessels which supplies blood to the heart muscles [5]. This disease affects the larger coronary arteries which are found on the heart's surface in contrast to coronary microvasculature disease which affects the tiny arteries in the heart muscle and is found mainly in women. This disease is caused when cholesterol builds up along the walls of the coronary arteries forming a plaque. This can lead to a total or partial blockage of blood flow in these large arteries cutting the blood supply short or totally [8].

The risk factors for coronary heart disease include the modifiable factors (that is reversible factors like hypertension, hypercholesterolemia, physical inactivity, diabetes, obesity and tobacco smoking) and the non-modifiable ones (age, sex and family history) [9]

1.1.2. Stroke

This affects the blood vessels supplying the brain with blood [5]. It occurs when there is a decrease in blood supply to or blockage in the vessels supplying the brain [10]. There are three forms and they include:

- **Ischemic stroke:** This occurs due to a blockage in the supply of blood to the brain. This is usually caused by a blood clot and it is more common than the others [10]
- **Hemorrhagic stroke:** This happens when there is a reduction in the supply of blood to the brain because of a rupture in the blood vessels. This can be caused by aneurysms, that is, swelling caused by the weakening of the walls of a blood vessel [10]
- **Transient ischemic stroke:** This is not as complicated as the others. The blood flow to the brain is reduced for a short period of time after which it starts flowing normally again and the ensuing symptoms are resolved [10].

Risk factors include: high blood pressure, cigarette smoking, diabetes, abnormal cholesterol levels, high saturated fatty acid diet, low fruit and vegetables diet, physical inactivity and obesity [11].

1.1.3. Rheumatic Heart Disease

This is a damage to the heart muscles and heart valves resulting from rheumatic fever caused by streptococcal bacteria [5]. This is caused by the group A *Streptococcus pharyngitis* which causes throat infection and by extension chronic inflammation. It is the most common in children [12]. If not treated properly, it can cause life-long disabilities or heart failure (death) [13].

The risk factors lie in the constant exposure of oneself to conditions that may result in the contraction of the bacteria, *Streptococcus pharyngitis* which include infrequent hand washing and overcrowding [13].

1.1.4. Cardiomyopathies

These are a group of heart diseases, usually heterogeneous that affect the myocardium and are usually associated with mechanical or electric dysfunction. They usually exhibit inappropriate ventricular hypertrophy or dilatation [14].

Initially, cardiomyopathies were classified into primary and secondary. The primary ones affect the heart while the secondary ones invade the body system affecting multiple organs. But recent studies have come with a more precise classification [14]. The classifications are as follows:

- **Dilated Cardiomyopathy (DCM)**

  This involves the increase in the size of the heart and as result, the heart is unable to pump blood efficiently leading to heart failure [6]. This is as a result of an inherent problem in the myocardium. It can also be secondary to systemic conditions like inflammation, malnutrition and infection, autoimmunity and endocrine diseases. It has been observed that alcohol is a major risk factor [14].

- **Hypertrophic Cardiomyopathy (HCM):**

  In this type of cardiomyopathy, there is a problem with the relaxation of the muscles, flow of blood and electrical stability. This is as a result of the thickening of the heart muscle walls [6]. It is characterized by the hypertrophy of

the left ventricle and also the obstruction of the left ventricle. Genetic mutations have also been observed in a lot of the cases of hypertrophic cardiomyopathy and the mode of inheritance is usually autosomal dominant, although other causes have been found to include metabolic or neuromuscular diseases. Risk factors include: age and family history of sudden cardiac death [14].

- **Restrictive Cardiomyopathy (RCM)**

This occurs when the ventricle is stiff which causes reduced ventricular filling and low diastolic volume during the cardiac cycle. It is usually suspected when there is a normal systolic function and an abnormal diastole in patients [14]. There is enlargement of the atrium due to impaired filling of the ventricle during diastole, though the ventricular activity is normal [15]. RCM can originate from a primary condition like endomyocardial fibrosis or it can be secondary originating from other systemic conditions responsible for infiltration of the myocardium (e.g. amyloidosis) and conditions that give rise to abnormal loading within the myocardial cells [14].

- **Arrhythmogenic cardiomyopathy (ACM)**

The main characteristic of ACM is the replacement of the ventricular myocardium by fibrofatty tissues. It is a chronic condition as the myocardial atrophy occurs slowly over a period of time starting from the epicardium to the endocardium becoming transmural and causing wall thinning [16]. It is a genetic condition that affects either the right or the left ventricle and can cause sudden cardiac death due to electrical instability and also ventricular tachycardia or ventricular fibrillation [14].

- **Arrhythmia**

This is basically an irregular heartbeat. It either comes in form of tachycardia (heart beating too fast) or bradycardia (heart beating too slow). It could happen to a healthy heart or it can happen due to the following: heart disease, electrolyte imbalance, heart injury, healing process after heart surgery, infection or fever, some medications, strong emotions, improper electrical signals, and lifestyle (alcohol, tobacco smoking, caffeine, exercise, etc.) [17].

1.1.5. Congenital Heart Disease

In this case, there is a problem with the heart function or structure that has affected the individual from birth [6]. These problems can affect:

- **The heart walls:** The walls may not develop properly leading to the accumulation of blood in places where it should not be accumulated. This puts pressure on the heart, doubling the workload, thus, leading to high blood pressure.
- **The heart valves:** The valves that direct blood flow may close up or leak, interfering with the pumping of blood.
- **Blood vessels:** There can be a reduction or blockage of blood flow through the veins and arteries leading to other health complications [18].

According to Beckerman [17], congenital heart disease comes in different forms and they are: hole in the heart (septal defect), trial septal defect (hole in between the upper chambers of the heart), ventricular septal defect (hole in between the lower chambers of the heart), complete atrioventricular canal defect (a hole that affects all the four chambers of the heart), valves defects, tetralogy of fallot, large ventricular defect, thickened wall around the lower chamber, aorta above the hole in the ventricular wall, and stiff pulmonary valves.

1.1.6. Heart Attack

Heart attack, also known as myocardial infarction occurs when there is a hindrance to the flow of blood to the heart. It can be total or partial leading to the heart being starved of oxygen and followed by the damage of the heart [19]. This occurs when there is atherosclerosis (buildup of fatty tissues) in the arteries responsible for supplying blood to the heart muscles [20]. When this happens, inflammation occurs and the inflammatory cells released line the surface of the artery and merge with the fatty tissues to form a plaque. This makes the artery walls thicker, making blood flow increasingly difficult [19].

According to the American Heart Association [20], the following symptoms are indications of heart attack: discomforting squeezing, fullness, or pain in the center of the chest with increased pressure, pain in one or both arms, back, neck, jaw,

or stomach, shortness of breath which might be accompanied by chest discomfort, breaking out in cold sweat, light-headedness, and nausea.

Risk factors include; high cholesterol, high blood pressure, smoking, obesity, diabetes, sedentary lifestyle, stress, inherited predisposition and low estrogen levels (females) [19].

1.1.7. Heart Failure

This is a condition in which the heart is unable to pump the amount of blood needed by the body system thereby leading to the disruption of the major bodily functions. This condition weakens or stiffens the heart, thus leading to the reduction and blockage of blood flow. This affects the left or right side of the heart or both. The condition can be acute or chronic but mainly chronic [21]. Shortness of breath, persistent coughing or wheezing, swelling of the ankles and fatigue are considered as the symptoms of heart failure. In addition, jugular venous pressure, pulmonary crackles, increased heart rate and peripheral edema may occur [22].

Risk factors include: cardiomyopathy, congenital heart disease, heart attack, and heart valve disease, arrhythmias, high blood pressure, emphysema, sleep apnea that is left untreated, diabetes, an overactive or underactive thyroid, HIV, severe anemia, chemotherapy and misuse of substances [21].

1.1.8. Mitral Regurgitation

This is the commonest type of heart valve disease. It is a condition in which the valve between the left heart chambers fail to close properly causing the leakage of blood backwards across the valve. The symptoms include fatigue, heart murmur, arrhythmia, dyspnea, palpitations and edema [23].

According to Hanson [24], mitral regurgitation causes three different forms which are:

Acute mitral regurgitation: There is an increase in preloaded and decrease in after-load resulting in increase in end-diastolic volume and a decrease in end-systolic volume. This causes an increase in total stroke volume to extreme levels. But forward stroke volume decreases because much of the total stroke volume regurgitates as the regurgitant stroke volume. These phenomena lead to an increase in left atrial pressure.

Chronic compensated mitral regurgitation: in this form, there is enough time for the left atrium and left ventricle to dilate and accommodate the regurgitant volume. Hence, left atrial pressure is usually normal and only increased slightly. Left ventricular hypertrophy is quite indicative.

Chronic decompensated mitral regurgitation: this involves cardiac dysfunction which impairs both the total stroke volume and forward stroke volume, thus causing a high end systolic volume and end diastolic volume which in turn leads to an increase of pressure in the left ventricle and left atrium. This can proceed to pulmonary edema if left untreated.
Causes of mitral regurgitation are: mitral valve prolapse, rheumatic fever, heart attack, congenital heart defect, cardiomyopathy, damaged tissue cords, endocarditis and radiation therapy. Risk factors include: infections affecting the heart, heart attack, congenital heart defects, history of other heart valve diseases, older age and radiation to the chest [23].

1.1.9. Mitral Valve Prolapse

This is also known as Barlow syndrome. The mitral valve allows the flow of blood from the left atrium to the left ventricle. Mitral valve prolapse occurs when a part of the mitral valve slips backwards into the left atrium. This is as a result of squeezing of the left ventricle during each heartbeat. In most cases, the cause is unknown [25].

1.1.10. Pulmonary Stenosis

This is a congenital heart disease. There is an obstruction of blood flow from the right ventricle to the pulmonary artery and this causes pressure overload in the right ventricle that leads to increase contractility and dilatation. This causes increased wall stress and right ventricular hypertrophy as compensation. It can occur as an isolated valvular (most common), subvalvular or peripheral (supravalvular) [26].

1.1.11. Aortic Stenosis

This is the obstruction of the flow of blood through the aortic valve. When this occurs, there is resistance to systolic ejection and systolic pressure increases between the left ventricle and the aorta. In order to compensate for the increase in pressure in the left ventricle, the wall thickens and diastolic compliance is reduced. This leads to the rise of end diastolic pressure in the left ventricle and an increase in pulmonary artery pressure. Cardiac output decreases because of diastolic and systolic dysfunction. This can develop to heart failure. The condition can be congenital or acquired [27].

1.1.12. Atrial Fibrillation

This is characterized by an irregular heartbeat and predisposes one to stroke. It is often rapid and has a strong connection to the other coronary heart diseases as it is a common secondary development in some cases [6]. It ranges from asymptomatic conditions to serious cerebrovascular occurrences. The risk factors are: age (older age), prior stroke, systemic thromboembolism, hypertension, diabetes, heart failure and arterial disease [28].

1.1.13. Radiation Heart Disease

This is a heart complication that develops with constant exposure to radiation over a long period of time [29]. Any radiation that targets the chest (mediastinum) can cause the damage of the heart valves and blood vessels [6]. Exposing the chest to radiation leads to the development of fibrosis in all the components of the heart and this leads to a significant increase in the risk of cardiovascular heart disease. The different manifestations of radiation heart disease are: coronary artery disease, valvular disease, cardiomyopathy, pericardium disease and conduction abnormalities [29].

1.1.14. Peripheral Arterial Disease

This is a cardiovascular disease that affects the blood vessels found outside the heart and brain [30]. It affects the blood vessels supplying the arms and legs [5]. Peripheral arterial disease is mostly caused by atherosclerosis which is the deposition of fatty material along the walls of the arteries. Also, when blood clots lodge in the arteries, it can lead to peripheral arterial disease. Patients present with hair loss on the limbs, intermittent claudication, leg weakness, coldness of the feet or lower legs, numbness in the legs, brittle toenails, stunted growth of the toenails, foot or leg ulcer that may never heal, pale skin on the legs, difficulty in locating a pulse on the foot and erectile dysfunction in men [30].

1.1.15. Deep Vein Thrombosis

According to Patel [31], deep venous thrombosis is a manifestation of venous thromboembolism. This happens when a blood clot is formed in the deep vein, particularly that of the lower leg, thigh or pelvis. Signs and symptoms include edema, leg pain, tenderness and erythema of the skin over the allocated area [31]. These clots can dislodge and can be carried in the blood stream to other organs of the body where they can lodge and give rise to secondary conditions e.g, pulmonary embolism (where the clot lodges in an artery in the lung interfering with the blood flow to the lungs) [32].

1.2. Risk Factors and Causes of Cardiovascular Diseases

According to Cohen et. al and Eckel and Cornier [33, 34], common risk factors and causes of cardiovascular diseases include: genetics (family based), age (mostly associated with old age), sex (susceptibility is higher in men are than women), cigarette smoking, physical inactivity, diet (high intake of saturated fats, transfats and salt), chronic alcoholism, celiac disease (defective digestion and utilization of fat by the body), insufficient amount of sleep, socioeconomic disadvantages, air pollution, an existing cardiovascular event like heart attack, depression and traumatic stress, occupational exposure (exposure to toxins or extreme temperature), somatic mutations, and radiation therapy.

The traditional risk factors (main causes) are hypertension, diabetes and hypercholesterolemia while the non-traditional risk factors have mostly been observed to be as a result of the production of certain serum markers like homocysteine, plasminogen activator inhibitor-1 (PAI-1), etc which usually lead to inflammation. The inflammation paves way for atherosclerosis [35].

1.3. Complications of Cardiovascular Diseases

Some of the complications of cardiovascular diseases include: angina (chest pain caused by heart disease), atrial fibrillation (abnormal heartbeat), cardiac arrest (the heart stops beating), heart attack (accompanied with chest pain, cold sweats and difficulty in breathing), heart failure (comes with shortness of breath, edema around the ankles, wheezing and fatigue), pulmonary edema (involves extreme shortness of breath, chest pain, pallor of nails and lips, and hemoptysis), stroke (signs are one-sided body weakness, drooping face and a problem with speaking) [36].

2. Laboratory Diagnosis of Cardiovascular Diseases

Cardiovascular diseases can be diagnosed through laboratory testing (blood test) and radiographical analysis.

2.1. Laboratory testing (blood test)

These are the main diagnostic procedures carried out for the detection of heart disease risks. Lipid panel and blood sugar estimation are highly indispensable in the diagnosis of cardiovascular disease. In lipid profile/panel, fats, cholesterol, LDL, HDL, VLDL, and triglycerides are tested while fasting, random, postprandial blood sugar and glycosylated haemoglobin are investigated in diabetes which is one of the risk factors of heart disease. It has also been observed that when the heart muscle cells die during certain heart diseases, there is a release of proteins into the blood stream, thus, a protein test comes in handy in this situation [37].

Furthermore, certain biomarkers play an important role in the diagnosis and prediction of cardiovascular disease. C-reactive protein (CRP), apolipoproteinA1 and B (present in inflammation), cardiac troponin, fibrinogen and PAI-1, elevated levels of homocysteine, asymmetric dimethylarginine and brain natiuretic peptide are the biomarkers associated with heart disease with C-reactive proteins and cardiac troponin as the commonest [37].

2.1.1. C - reactive protein

C-reactive protein (CRP) marks the presence of an inflammatory response in the body system. It is an acute phase protein and it's a major diagnostic factor that is indicative of recent or any early inflammation [38]. Atherosclerosis is an inflammatory disease which arises as a result of injury to the vascular endothelium by the major risk factors of cardiovascular diseases. The inflammatory response comes in form of the fatty streak development. The early stages of this inflammatory response triggers the release of pro inflammatory cytokines which leads to hepatic stimulation and the hepatocytes produce CRP. CRP may further contribute to atherosclerosis by stimulating the macrophage uptake of LDL-C leading to the successful and faster formation of the fatty streak [39]. The following methods are used for the detection of CRP: radioimmunoassay, Enzyme Linked ImmunosorbentAssay (ELISA), chemiluminescence immunoassay and fluorescence immunoassay [40].

2.1.2. Cardiac Troponin I

This is used as a detection factor for myocardial infarction. It is also implicated in cardiac muscle damage and injury [41]. Cardiac troponin is specifically important in myocardial infarction because of its high sensitivity and specificity in myocardial injury. Though small elevations in cardiac troponin levels can also be seen in other cardiovascular diseases like acute coronary syndrome [42]. Different techniques used for the detection of cardiac troponin I are: Enzyme Linked ImmunosorbentAssay (ELISA), optomagnetism, field effect transistor-based analysis, fluorescence immunoassay and electrochemiluminescence (ECL)-based biosensors [43]

Other methods for the diagnosis of cardiovascular diseases include: EKG/ECG (electrocardiogram), stress testing, echocardiography (creates a moving picture of the heart), coronary angiography and cardiac catheterization (for the pictures of blood vessels and heart), chest x-ray (shows shapes and sizes of heart, lungs and major blood vessels), electron-beam computed tomography (detects calcium deposits in the arteries), cardiac MRI (creates pictures of the heart) [37].

2.2. Anti-hypertensives

These are basically drugs used to combat hypertension. This medication is important, so as to prevent the complications associated with blood pressure which can lead to certain heart diseases. These drugs come in different classes, they are: diuretics, angiotensin converting enzyme inhibitors, angiotensin II receptor blocker, calcium channel blocker, renin inhibitors, adrenoceptor antagonists, vasodilators, centrally acting agents and ganglion blockers [44].

Diuretics: Comes in two forms, the thiazide diuretics and the loop diuretics. Diuretics are one of the major therapies for hypertension. Their purpose is to increase natriuresis and diuresis.

- **Angiotensin converting enzyme inhibitors (ACEI):** They are usually the first line of treatment in primary hypertension. They are used in the treatment of some cardiovascular diseases and chronic kidney disease. The inhibition of ACE, tampers with the cleavage of peptide hormone angiotensin I to angiotensin II causing its reduction. This in turn lowers the metabolism of the peptide bradykinin. This phenomenon plays an important role in the therapy of hypertension. Examples are: ramipril, quinapril, benzazepril, imidapril, etc [45].

- **Adrenoceptor antagonists:** They are the B-blockers and alpha blockers. B-blockers fight against catecholamines at the B-adrenoreceptors. When B-receptors are blocked, there is noticeable decrease in heart rate and reduction in contractility. The actions of alpha blockers on the other hand, causes peripheral vasodilation and reduced systemic vascular resistance. It is usually the drug of choice in patient's resistant to other anti-hypertensives. Examples of B-blockers are: metroprolol, esmolol and atenolol. Example of alpha blockers is labetalol [46].
- **Angiotensin II receptor blocker:** This increases the levels of angiotensin II in the body system thereby initiating a negative feedback mechanism. It is a more favorable treatment than ACEI. Examples are: losartan, azilsartan, candesartan, eprosartan, etc [47].
- **Calcium channel blockers:** These prevent the entry of calcium into the heart muscles. They are used in the therapy for tachyarrhythmias and angina. Examples are: amlodipine, barnidipine, clinidipine, clevidipine, felodipine, isradipine, etc [48].
- **Renin inhibitors:** These inhibit the actions of renin, reducing the conversion of polypeptide angiotensinogen to angiotensin I. Example: aliskiren.
- **Vasodilators:** They relax the vascular smooth muscles in resistant vessels. Though the drugs are usually poorly tolerated. Examples: hydralazine and minoxidil [46].
- **Centrally acting agents:** They are usually used in cases that are difficult to treat. Examples: clonidine, methyldopa and moxonidine.
- Ganglion blockers: They antagonize acetylcholine at the nicotinic receptors causing vasodilation. Example: trimetaphan [46].

3. Conclusion

Cardiovascular disease has proven to pose a serious health challenge, especially in the cases of neglect or poor treatment. In such cases, it can progress to secondary ailments affecting the body system. They come in different types and forms, causing different diseases in different parts of the cardiovascular system.

Treatments are available in different forms, however, it's pertinent to take preventive measures to avoid adverse effects and complications. The risk factors vary from age, sex, diet, lifestyle, heredity, presence of certain diseases, e.t.c. CVD predispositions and complications tend to be heightened at old age, thus the importance to keep lifestyle and dieting into check. Saturated fats consumption should be reduced to the barest minimum and replaced with lots of fruits and vegetable; food items that facilitate healthy and effective running of the cardiovascular system. Constant exercise and the elimination of sedentary life style are highly recommended for the maintenance of healthy body mass index (BMI).

Conclusively, cardiovascular disease when properly treated may not be life threatening and can be prevented in most cases with proper and healthy preventive measures.

Compliance with ethical standards

Disclosure of conflict of interest

There is no conflict of interest.

References

[1] Adegoke O, Awolola NO, Ajuluchukwu JN. Prevalence and pattern of cardiovascular-related causes of out-of-hospital deaths in Lagos, Nigeria. African health Science. 2018, 18(4):942-949.

[2] Mensah GA, Brown DW. An overview of cardiovascular disease burden in the United States. Health affairs. 2007, 26(1):38-48.

[3] Thiriet M. Cardiovascular disease: An introduction. Vasculopathies. (2018); 8: 1-90.

[4]   Enroth S, Maturi V, Berggrund M, Enroth SB, Moustakas A, Johansson Å, Gyllensten U. Systemic and specific effects of antihypertensive and lipid-lowering medication on plasma protein biomarkers for cardiovascular diseases. Scientific Reports. 2018, 8(1):1-0.

[5]   Strasser T. Reflections on cardiovascular diseases. Interdisciplinary science reviews. 1978, 3(3):225-30.

[6]   Chilazi M, Duffy EY, Thakkar A, Michos ED. COVID and cardiovascular disease: what we know in 2021. Current atherosclerosis reports. 2021, 23(7):1-2.

[7]   Nason E. An overview of cardiovascular disease and research. RAND Corporation str. 2007:2-4.

[8]   Keys A. Coronary heart disease in seven countries. Circulation. 1970;41(1):186-95.

[9]   Haffner SM. Coronary heart disease in patients with diabetes. New England Journal of Medicine. 2000 Apr 6;342(14):1040-2.

[10]   Ellis C, Egede LE. Ethnic disparities in stroke recognition in individuals with prior stroke. Public Health Reports. 2008,123(4):514-22.

[11]   Mukherjee D, Patil CG. Epidemiology and the global burden of stroke. World neurosurgery. 2011,76(6): S85-90.

[12]   Seventy-First World Health Assembly. Rheumatic fever and rheumatic heart disease. World Health Organization. (2018); A71/25.

[13]   Marijon E, Mirabel M, Celermajer DS, Jouven X. Rheumatic heart disease. The Lancet. 2012, 379(9819): 953-64.

[14]   Jarvis S. Cardiomyopathies1: classification pathophysiology and symptoms. Nursing Times [online]. (2019); 115:7, 38-42.

[15]   Mogensen J, Arbustini E. Restrictive cardiomyopathy. Current Opinion in Cardiology. 2009 May 1; 24(3): 214-20.

[16]   Corrado D, Basso C, Judge DP. Arrhythmogenic cardiomyopathy. Circulation research. (2017); 121: 784-802

[17]   Antzelevitch C, Burashnikov A. Overview of basic mechanisms of cardiac arrhythmia. Cardiac electrophysiology clinics. 2011,3(1):23-45.

[18]   Hoffman JI, Kaplan S. The incidence of congenital heart disease. Journal of the American college of cardiology. 2002, 39(12):1890-900.

[19]   Ridker PM. Evaluating novel cardiovascular risk factors: can we better predict heart attacks? Annals of internal medicine. 1999,130(11): 933-7.

[20]   Currie J, MacLeod WB, Van Parys J. Provider practice style and patient health outcomes: The case of heart attacks. Journal of health economics. 2016, 47:64-80.

[21]   Roger VL. Epidemiology of heart failure. Circulation research. 2013;113(6):646-59.

[22]   Filippatos G, Zannad F. An introduction to acute heart failure syndromes: definition and classification. Heart failure reviews. 2007, 12(2):87-90.

[23]   El Sabbagh A, Reddy YN, Nishimura RA. Mitral valve regurgitation in the contemporary era: insights into diagnosis, management, and future directions. JACC: Cardiovascular Imaging. 2018,11(4):628-43.

[24]   Enriquez-Sarano M, Akins CW, Vahanian A. Mitral regurgitation. The Lancet. 2009, 373(9672):1382-94.

[25]   Guy TS, Hill AC. Mitral valve prolapse. Annual review of medicine. 2012,63: 277-92.

[26]   Fingland RB, Bonagura JD, Myer CW. Pulmonic stenosis in the dog: 29 cases (1975-1984). Journal of the American Veterinary Medical Association. 1986,189(2): 218-26.

[27]   Chambers JB. Aortic stenosis. European Journal of Echocardiography. 2009,10(1): i11-9.

[28]   Nattel S. New ideas about atrial fibrillation 50 years on. Nature. 2002,415(6868):219-26.

[29]   Belzile-Dugas E, Eisenberg MJ. Radiation-induced cardiovascular disease: Review of an under-recognized pathology. Journal of the American Heart Association 2021, 10: eo21686.

[30]   Kullo IJ, Rooke TW. Peripheral artery disease. New England Journal of Medicine. 2016 Mar 3;374(9):861-71.

[31]   Olaf M, Cooney R. Deep venous thrombosis. Emergency Medicine Clinics. 2017 Nov 1;35(4):743-70.

[32]   Agnelli G, Becattini C. Acute pulmonary embolism. New England Journal of Medicine. 2010 Jul 15;363(3):266-74.

[33] Cohen BE, Edmondson D, Kronish IM. State of the art review: Depression, street, anxiety and cardiovascular diseases. American Journal of Hypertension. (2015), 28(11): 1295-302.

[34] Eckel RH, Cornier MA. Update on the NCEP ATP-III emerging cardiometabolic risk factors. BMC Medicine. (2014), 12(1): 115.

[35] Shaima C, Moorthi PV. & Shaheen NK. Cardiovascular diseases: Traditional and non-traditional risk factors. Journal of Medical Allied Sciences. (2016), 6(2): 46-51.

[36] Siu SC, Colman JM. Heart disease and pregnancy. Heart. 2001, 85(6):710-5.

[37] Laslett LJ, Alagona P, Clark BA, Drozda JP, Saldivar F, Wilson SR, Poe C, Hart M. The worldwide environment of cardiovascular disease: prevalence, diagnosis, therapy, and policy issues: a report from the American College of Cardiology. Journal of the American College of Cardiology. 2012,60(25S): S1-49.

[38] Orji KN, Oguji CE, Onyeama UE, Agbo ME, Sunday FC, Makata VC, Nnaji FC, Eze SO, Ike OH, Okonkwo IN. C-reactive protein levels amongst hypertensive patients attending University of Nigeria teaching Hospital, Ituku-Ozalla, Enugu State, Nigeria. GSC Biological and Pharmaceutical Sciences. (2022), 19(03),083-092.

[39] Manzur F, Alvear C, Alayón AN. Role of C-reactive protein in cardiovascular diseases. Revista Colombiana de Cardiología. 2011, 18(5):273-8.

[40] Guoet L, Yang Z, Zhi S, Feng Z, Lei C, Zhou Y. A sensitive and innovative detection method for rapid C-reactive proteins analysis based on a micro-fluxgate sensor system; PLoS ONE. (2018), 13(3): e0194631.

[41] Bayoumy S, Martiskainen I, Heikkilä T, Rautanen C, Hedberg P, Hyytiä H, Wittfooth S, Pettersson K. Sensitive and quantitative detection of cardiac troponin I with upconverting nanoparticle lateral flow test with minimized interference. Scientific reports. 2021,11(1):1-9.

[42] Hoff J, Weiner W, Nambi V. Troponin in cardiovascular disease prevention: Update and Future Direction. Curr Atheroscler Rep. 2016 Mar. 18(3):12.

[43] Spain E, Carvara S, Adamson K, Ma H, O'Kennedy R, De Cola L, Forster RJ. Cardiac troponin I: Ultrasensitive detection using faradaic electrochemical impedance. ASC Omega. (2018), 3(12): 17116-17124.

[44] Law M, Wald N, Morris J. Lowering blood pressure to prevent myocardial infarction and stroke: a new preventive strategy. Health Technology Assessment. (2003), 7(31): 1-94.

[45] Laurent S. Antihypertensive drugs. Pharmacological Research. (2017), 124: 116-125.

[46] Jackson RE, Bellamy MC. Antihypertensive drugs. BJA Education. (2015), 15(6): 280-285.

[47] Reudelhuber TL. The continuing saga of the AT2 receptor: A case of the good, the bad and the innocuous. Hypertension. (2005), 46(6): 1261-2.

[48] Bakris GL, Weir MR, Secic M, Campbell B, Weis-McNulty A. Differential effects of calcium antagonist subclasses on markers of nephropathy progression. Kidney international. (2004), 65(6): 1991-2002.