International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 3 May-June 2022, pp: 2003-2008 www.ijprajournal.com ISSN: 2456-4494

Pathophysiology of Different Types of Cardiovasular Diseases Diseases of the Heart

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Submitted: 05-06-2022 Revised: 18-06-2022 Accepted: 27-06-2022

ABSTRACT: There are many types of heart disease, and each one has its own symptoms and treatment. For some, lifestyle changes and medicine can make a huge difference in improving your health. Heart disease is a huge term that can describe many different conditions. All of these conditions have some effect on the heart or the blood vessels that supply the heart, cardiovascular disease is not a single ailment, but a disorder of the heart and circulatory system. Heart disease can refer to damage in the heart's lining, valves, muscle, arteries, or electrical system.

Key words: Heart disease, pathophysiology, cardiovascular diseases.

Introduction:

Some disease is classified by its effect on the different components of the heart. In broad stroke the heart has four chambers (two ventricles and two atria), muscle, lining, an electrical system, valves, coronary arteries and veins. Heart disease may be defined by what part of the heart it mostly affects.

DISEASES OF THE HEART

Heart disease types affecting the heart chambers

Kinds of heart disease that can affect the heart chamber include:

- Congestive heart failure, also known simply as heart failure, including:
  - Diastolic dysfunction
  - Systolic dysfunction
- Cor pulmonale (also known as pulmonary heart disease)

Types of heart disease that can affect the muscle itself include:

- Cardiomyopathy, such as:
  - Dilated cardiomyopathy
  - Hypertrophic cardiomyopathy
  - Restrictive cardiomyopathy
- Myocarditis, which is an inflammation of the heart muscle.

Heart disease types affecting the valves

Kinds of heart disease that can affect the heart valves (valvular heart disease) include:

- Mitral stenosis
- Mitral valve regurgitation
- Mitral valve prolapsed
- Aortic stenosis
- Tricuspid stenosis
- Tricuspid regurgitation

Types of heart disease within the arteries or veins

- Heart attack, also known as a myocardial infarction or MI
- Coronary artery disease (CAD) also known as ischemic heart disease
- Atherosclerosis, or hardening of the arteries

Other heart Disease types

Other types of disease that can affect any part of the heart include:

- A cardiac tumor, which can be either malignant (cancerous) or benign

(noncancerous). Myxoma is the most common cardiac tumor.

- Sudden cardiac death.

1. Pathophysiology Of Congestive Heart Failure:

The adaptive mechanisms that may be adequate to maintain the overall contractile performance of the heart at relatively normal levels become maladaptive when trying to sustain adequate cardiac performance.

In the initial stages of congestive heart failure, cardiac physiology attempts to adapt via several compensatory mechanisms to maintain cardiac output and meet the systemic demands. These include the Frank-Starling mechanism, changes in myocyte regeneration, myocardial hypertrophy, and myocardial hypercontractility. With increased wall stress, the myocardium attempts to compensate via eccentric remodeling, which further worsens the loading conditions and wall stress[1].

A decrease in cardiac output stimulates the neuroendocrine system with a release of epinephrine, norepinephrine, endothelin-1 (ET-1), and vasopressin. They cause vasoconstriction leading to increased afterload. There is an increase in cyclic adenosine monophosphate (cAMP), which causes an increase in cytosolic calcium in the myocytes. This increases myocardial contractility and further prevents myocardial relaxation.

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An increase in afterload and myocardial contractility with impaired myocardial relaxation leads to increased myocardial oxygen demand. This paradoxical need for increased cardiac output to meet myocardial demand eventually leads to myocardial cell death and apoptosis. As apoptosis continues, a decrease in cardiac output with increased demand leads to a perpetuating cycle of increased neurohumoral stimulation and maladaptive hemodynamic and myocardial responses.

A decrease in cardiac output also stimulates the renin-angiotensin-aldosterone system (RAAS), leading to increased salt and water retention, along with increased vasoconstriction. This further fuels the maladaptive mechanisms in the heart and cause progressive heart failure. In addition to this, the RAAS system releases angiotensin II, which has been shown to increase myocardial cellular hypertrophy and interstitial fibrosis. This maladaptive function of angiotensin II has been shown to increase myocardial remodeling[1].

In HFpEF, there is a decrease in myocardial relaxation and an increase in the stiffness of the ventricle due to an increase in ventricular afterload. This perpetuates a similar maladaptive hemodynamic compensation and leads to progressive heart failure[2].

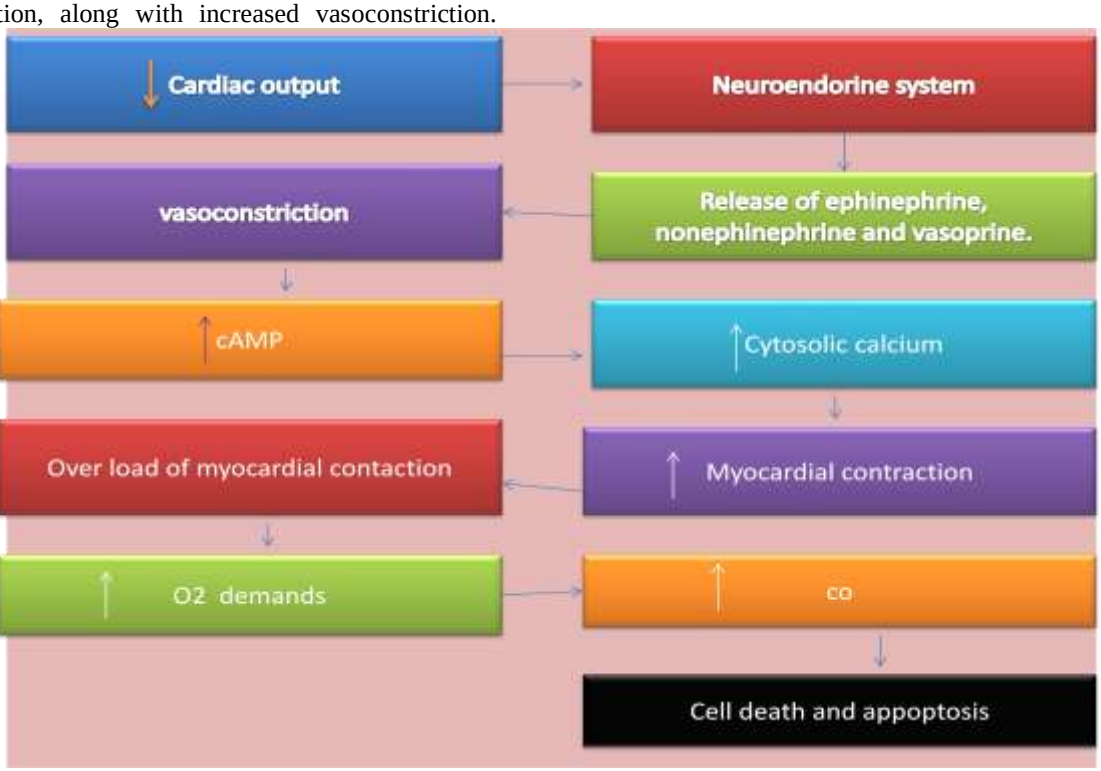

Fig no: 01 flow chart of CHF.

2. Pathophysiology Of Cor Pulmonale:

The pathophysiology of cor pulmonale is a result of increased right-sided filling pressures from pulmonary hypertension that is associated with diseases of the lung[3,4,5].

Under normal physiologic conditions, the right ventricle pumps against a low-resistance circuit.

Normal pulmonary vascular resistance is approximately one-tenth of the resistance of the systemic arteries. Chronic hypoxemia leading to chronic vasoconstriction produces smooth muscle proliferation in small pulmonary arteries. Hypoxemia produces changes in vascular mediators such as Nitric Oxide, Endothelin1 (ET1) and platelet-derived growth factors (PDGF A and B). Nitric oxide is a vasodilator; hypoxemia reduces endothelial cell production of nitric oxide and results in impaired smooth ms relaxation.

The initial pathophysiologic event in the production of cor pulmonale is an elevation of pulmonary vascular resistance. As the resistance increases, the pulmonary arterial pressure rises, and the right ventricular work increases leading to right ventricular enlargement (e.g., thickening, dilation, or both).

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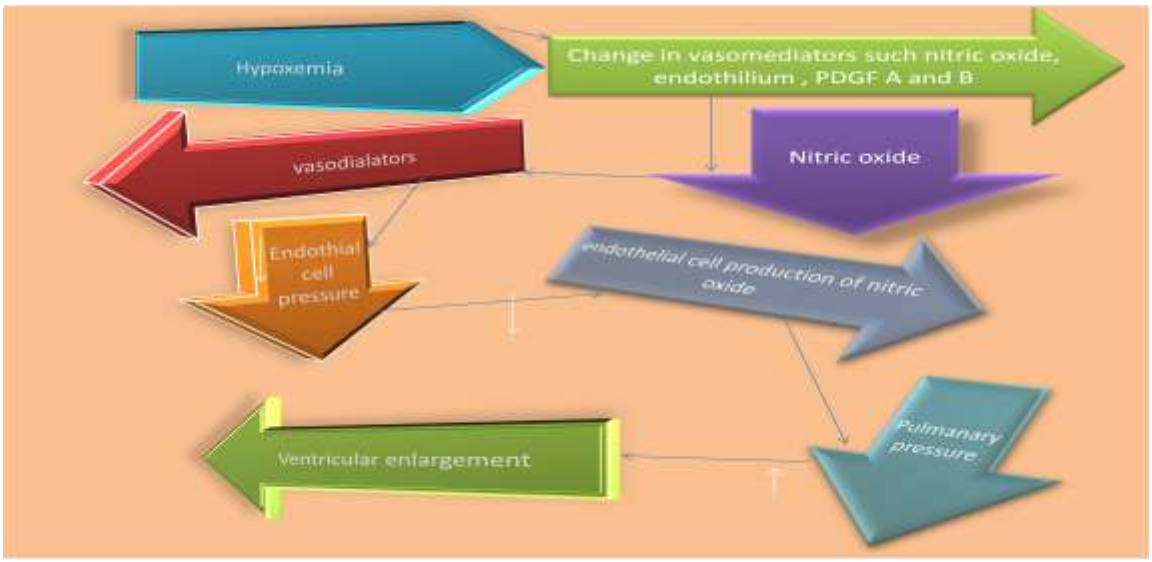


Fig no: 02 flow chart of corpulmonale.

3. PATHOPHYSIOLOGY OF CARDIOMYOPATHY

The left ventricle can be enlarged from two broad underlying conditions: dilation and hypertrophy.

Left Ventricular Dilation

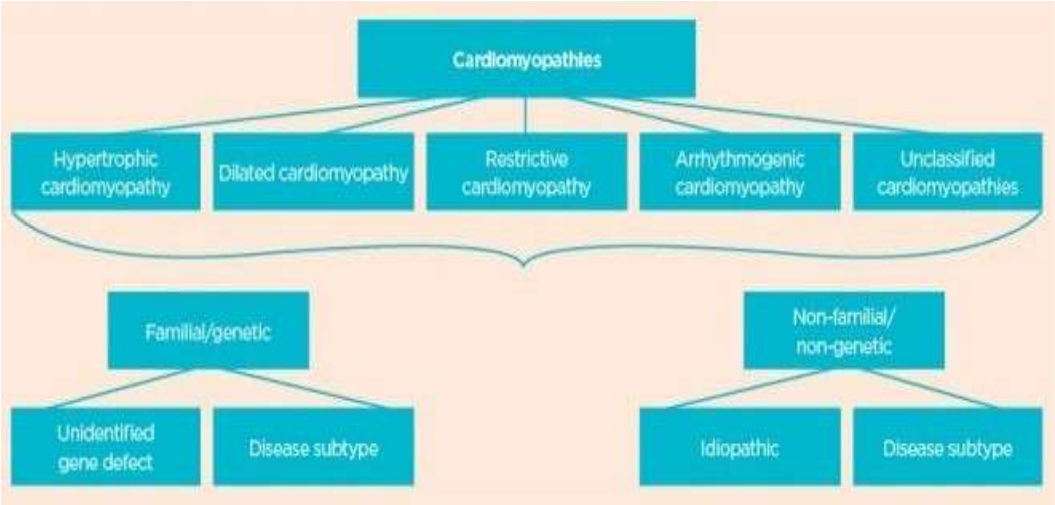
Left ventricular dilation can occurs as a result of volume overload. Conditions that cause volume overload can be further broken down as follows:

- Valvular Disease: More common underlying valvular heart disease conditions would include mitral regurgitation and aortic regurgitation.
- Congenital Heart Disease: Patent ductus arteriosus and a ventricular septal defect.
- High output states resulting in volume overload: Anemia and thyrotoxicosis.
- High stroke volume states: Complete heart block and prolonged severe sinus bradycardia.
- Can occur as a result of ischemia and remodeling.

Left Ventricular Hypertrophy

Left ventricular hypertrophy occur due to factors that can cause the heart to work harder than normal. Cardiac hypertrophy is seen in the following condtions:

- Hypertension
- Aortic valve stenosis
- Hypertrophic cardiomyopathy
- Atheletic training[10]



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4. PATHOPHYSIOLOGY OF VALVULAS HEART DIESEASE

The atrioventricular valves (mitral and tricuspid) and the semilunar valves (aortic and pulmonic) are two types of mature heart valves. These valves consist of an outer layer of valve endothelial cells (VECs) surrounding three layers of extracellular matrix each with specialized function and interspersed with valve interstitial cells (VICs). Changes in the functionality and localization of matrix components potentially lead to VHD, since the proper organization of extracellular matrix (ECM) is essential in maintaining overall valve morphology and normal valve function. The three layers of ECM, consisting of collagens, proteoglycans and elastin, collectively contribute to the biomechanical support for the valves and any derangements in these morphological units can have detrimental effects on the complicated structures of valves that open and close approximately 100,000 times daily in order to maintain proper directionality of blood flow through the heart chambers. The protective endothelium over the surface of the valve leaflets is formed by the VECs, which communicate with VICs in the underlying layer and regulate their response to alterations in the blood flow. Genetic or acquired/environmental causes that disrupt the normal organization and composition of the ECM and communication between VECs and VICs alter valve mechanics and interfere with the valve leaflet function, culminating in heart failure.

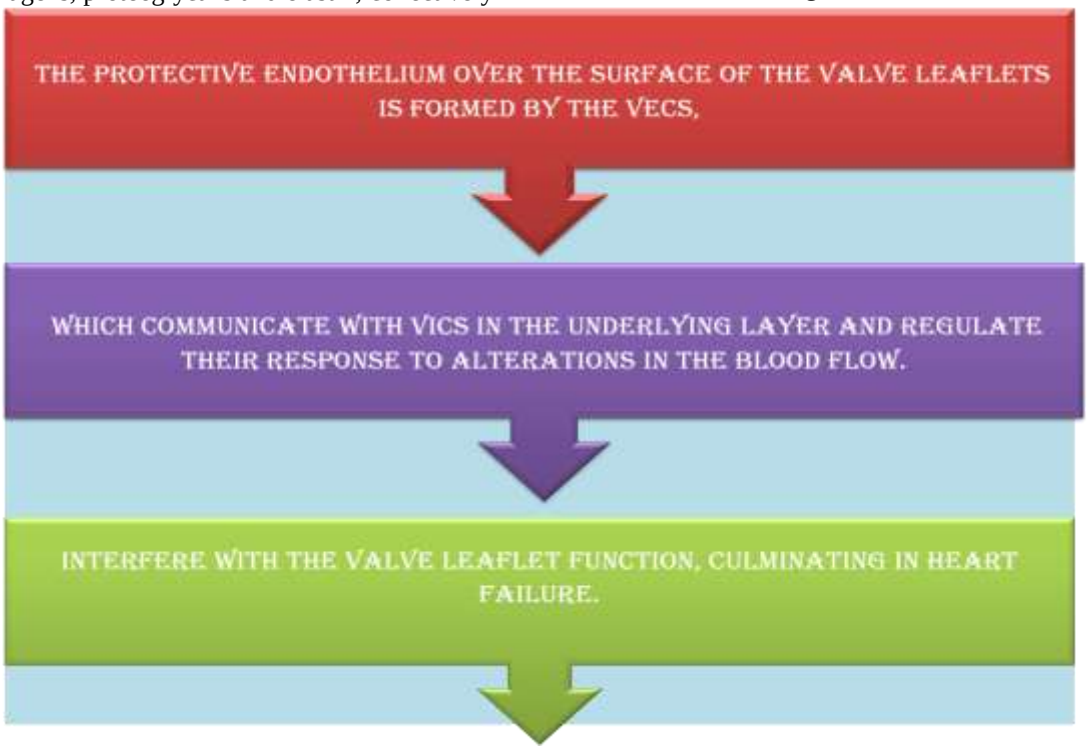

Fig no: 04 Flow chart of valvulas heart disease.

5. Pathophysiology Of Myocardial Infaraction

Myocardial infaraction is the condition which occurs due to imbalance between coronary blood supply and myocardial demand.

The rapidly growing atheromatous plaques are compared of a substantial inner lipid core containing a large mass of thrombogenic lipids and macrophages bearing tissue factors. The lipid core in these plaques is surrounded by a relatively thin fibrous cap, which often exhibits local inflammation, degradation, and repair of its matrix. In these young and rapidly growing plaques. If the matrix removal from the fibrous cap by inflammation exceeds the deposition of the fibrous cap matrix overlying the lipid-rich, necrotic core of the plaque, it become unstable and vulnerable to fissuring and rupture. In contrast, the mature or stable atheromatous plaque is characterized by a small inner lipid core covered by a relatively think and stable fibrous cap.

Cycles of small fissuring and disruptions of the fibrous cap, leading to local platelet aggregation, mild degrees of thrombosis, myocyte migration, and healing of the fibrous matrix, is

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believed to be part of the natural evolution of the growing atheromatous plaque. By this mechanism, relatively slowly accruing epicardial coronary plaques may progress to high-grade stenosis or even to complete occlusion without precipitating acute MI because this slow process is uaually accompanied by the concomitant growth of co-lateral coronary circulation. However, during the same process of atherosclerosis, in those plaque that are lipid laden and with thin and unstable fibrous cap, abrupt rupture of the plaque may occur. The resultant thrombus may occur. Thus, rupture of an unstable or vulnerable coronary plaque is considered to be the common pathophysiologic mechanism of the vast majority of acute coronary syndromes ranging from unstable angina through non-Q-wave acute MI (AMI) and Q- wave AMI [7].

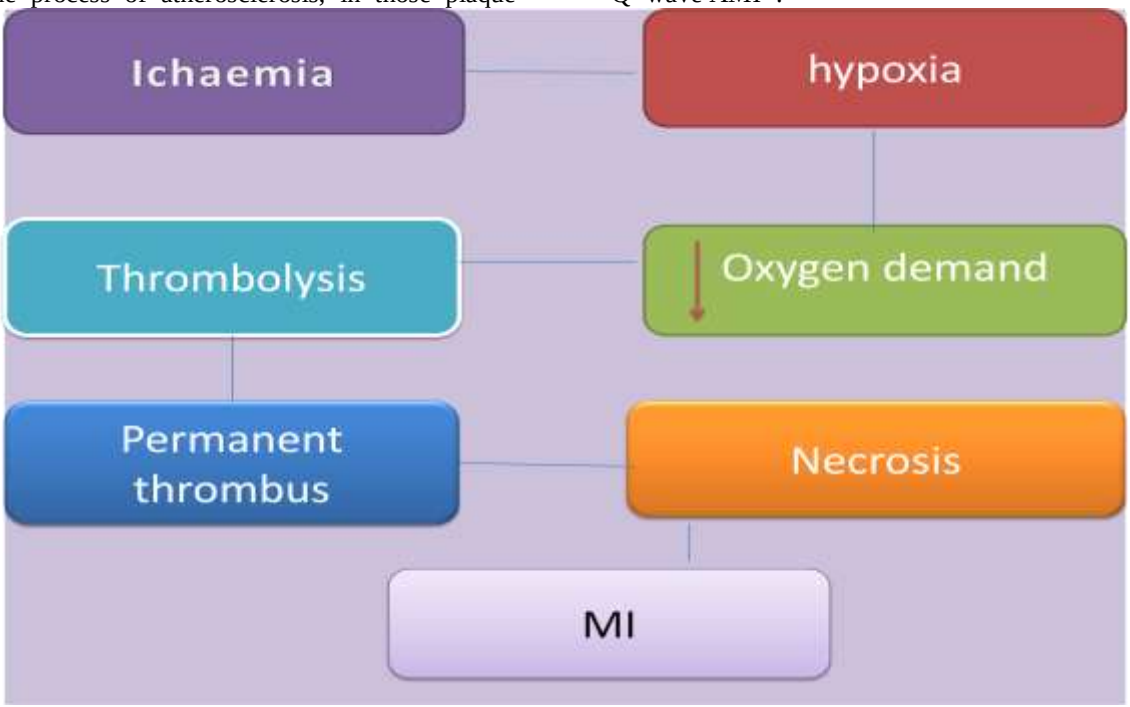

Fig no: 05 flow chart of MI

6. Pathophysiology Of Atherosclerosis:

Hypercholesterolaemia is considered one of the main triggers of atherosclerosis. The increase in plasma cholesterol levels results in changes of the arterial endothelial permeability that allow the migration of lipids, especially LDL-C particles, into the arterial wall. Circulating monocytes adhere to the endothelial cells that express adhesion molecules, such as vascular adhesion molecule-1 (VCAM-1) and selectins, and, consequently, migrate via diapedesis in the subendothelial space [1]. Once in the subendothelial space, the monocytes acquire macrophage characteristics and convert into foamy macrophages. LDL particles in the subendothelial space are oxidised and become strong chemoattractants. These processes only enhance the accumulation of massive intracellular cholesterol through the expression of scavenger receptors (A, B1, CD36, CD68, for phosphatidylserine and oxidised LDL) by macrophages, which bind native and modified lipoproteins and anionic phospholipids. The end result is a cascade of vascular modifications[8].

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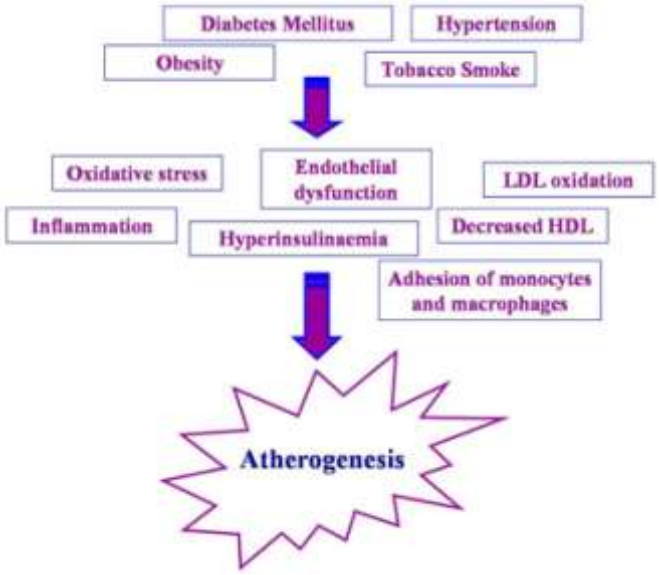

Fig no: 06 flow chart of atherogenesis

7. Pathophysiology Of Myxoma:

Cardiac myxoma arises from remnants of subendocardial vasoformative reserve cells, which are primitive mesenchymal cells that are normally involved in the supportive structure of the endocardium. is The exact pathogenesis of cardiac myxoma is not fully understood. It is thought that cardiac myxoma is produced by the neoplastic theory, dysembryoplastic theory, histopathogenesis of glandular cells in myxoma or the thrombotic theory. The site of tumor attachment, normally the foramen ovale, considered to be consistent with an origin from multipotent mesenchymal cells or from embryonic rests[09.]

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