Review Article

Systemic Review On Angina Pectoris

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ABSTRACT

Angina pectoris is a manifestation of coronary heart disease. Patients with angina Pectoris have a reduced quality of life because of their symptoms, impaired activity and anxiety. The evaluation of outcome of management of patients with angina Pectoris is now inextricably linked with an assessment of quality of life. In this review we discuss several drugs treatment like β-blockers, nitrates, calcium channel blockers, zinc. Other therapies like Revascularization, angioplasty and coronary bypass surgery are also discussed in the management of angina pectoris.[1]

Key words: Beta Blockers, Calcium Channel Blockers, Coronary Heart Disease, Angina Statins.

INTRODUCTION

Angina pectoris is caused by inadequate supply of oxygen supplying to the heart muscle.. It is a generally symptoms of coronary artery disease. Typically, angina is described as a “Pressure” or “Squeezing” pain that starts in the center of the chest and may increase to the shoulders or neck, jaw or back. Angina usually occurs an exertion and is relieved by rest. In the most severe cases, it may occur with minimal effort or at rest.

Some people occurrence of angina while sleeping or at rest. The two major cause of angina is coronary artery spasm, and atherosclerotic plaque climax which causes dangerous blockage of the coronary artery. [1]

This type of angina may be caused by a spasm in a coronary artery, which most normally occurs at the site of atherosclerotic plaque in a diseased vessel.

The common risk factors include: smoking high blood pressure or hypertension, high blood fats or cholesterol, hypercholesterolemia, diabetes men are at higher risk than women. [1]

The distress or pain of angina pectoris and of acute myocardial infarction is consequent to ischemia. The actual stimulus at the nerve end-organs that give rise to the pain has not been identified with certainty. In angina pectoris the ischemia is transient because of provisional imbalance between the blood supply and the myocardial necessities in acute myocardial infarction the ischemia is expanded and leads to the irreparable changes of necrosis. It has certain characteristics in common with lactic acid: it is acid, is destroyed by alkali and by oxidation, and develops most rapidly under oxygen deprivation and carbon dioxide accumulation.[5]

Angina pectoris is defined as cardiac-induced pain arising from a lack of myocardial oxygen. not only do 10.2 million Americans have this condition and approximately 500,000 new cases of angina occur each year, but ischemic heart disease is the leading cause of death in the United states.1 Moreover, the lifetime risk of developing coronary artery disease after 40 years of age is estimated at 49% for men and 32% for women. Given the prevalence of this disease, it is important for clinicians to be familiar with the presenting symptoms, as well as current evidence-based treatment options.[4]

Angina pectoris, Latin expression meaning “quinsy of the chest”, is the clinical manifestation of cardiac ischemia, resulting from an imbalance between coronary oxygen supply and myocardial oxygen demand. From a pathophysiological point of view, the supply/demand mismatch is most commonly due to coronary atherosclerosis, whose presence makes inadequate the perfusion of heart under conditions of increased myocardial oxygen demand, such as exertion. Other causes of this mismatch not related to coronary stenosis can be either an increased myocardial demand in course of chronic conditions (such hypertension, aortic stenosis or hypertrophic cardiomyopathy) or a reduced supply, due to anemia.[8]

The incidence of myocardial infarction and angina in women aged less than 50 years has been low: 0-03/1000 woman-years at ages 25-34 rising to 0-67/1000 woman-years at ages 45-49. However, the overall incidence in women who were smokers at entry to the study is more than three times that in women who were non-smokers, the increase in individual risk being proportional to the number of cigarettes smoked.[3]

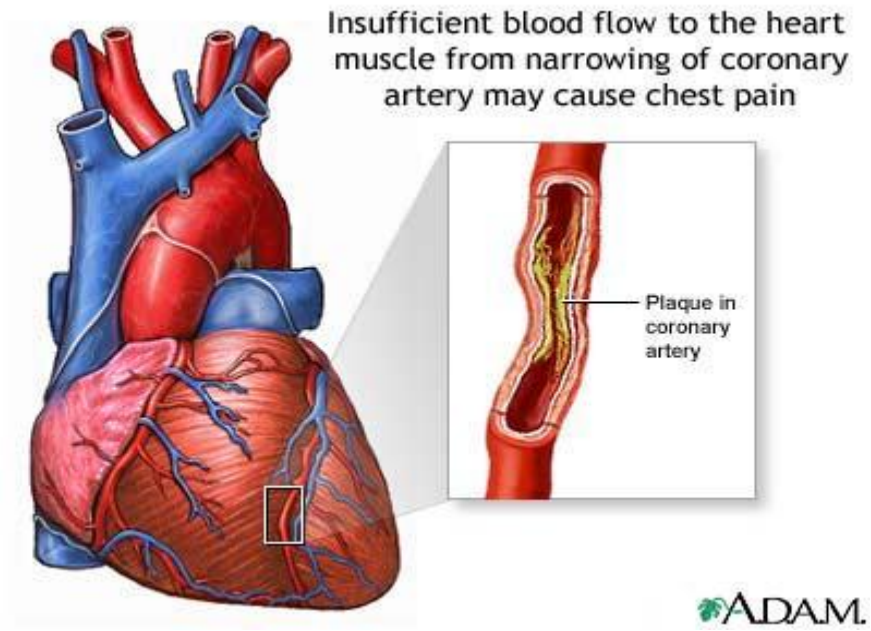

Fig. 1: Heart and plaque in coronary artery

Some patients with coronary artery disease and myocardial &hernia have a marked lack of angina pectoris pain. The reasons for this absence of pain remain unclear. A series of investigations have shown that these patients have increased pain thresholds, which suggest that the endogenous pain modification system may play a role. [10]

Angina is not a disease. It is a sign of an causal heart problem, generally coronary heart disease (CHD).There are a lot of types of angina, including micro vascular angina, Prinzmetal's angina, stable angina, This frequently happens because one or more of the coronary arteries is narrowed or blocked, also called ischemia.

Angina can also be a indication of coronary microvascular disease (MVD). This is heart disease that affects the heart's smallest coronary arteries and is more likely to affect women than men..

Depending on the type of angina you have, there are many factors that can trigger angina pain There is a weak connection between harshness of pain and degree of oxygen deficiency in the heart muscle (i.e. there can be severe pain with little or no risk of a heart attack, and a heart attack can occur without pain).[15] Coronary artery disease, the foremost cause of angina, is due to atherosclerosis of the cardiac arteries.[15]

Angina pectoris, normally known as angina, is harsh chest pain due to ischemia (A be short of o blood, hence a lack of oxygen supply) of the heart muscle, generally due to hindrance or contraction of the coronary arteries (the heart's blood vessels).

PATHOPHYSIOLOGY

The main dysfunction in angina pectoris is decreased oxygen liberation to myocardial muscle cells. The two chief mechanisms by which liberation is impaired materialize to be coronary artery narrowing and endothelial dysfunction. Any other mechanism that affects oxygen delivery can also impulsive symptoms. The end result is a shift to anaerobic metabolism in the myocardial cell. This is followed by a stimulation of pain receptors that innervate the heart. Extra cardiac causes of angina comprise but are by no means limited to anemia, hypoxia, hypotension, bradycardia, carbon monoxide revelation and inflammatory disease. These pain receptors ultimately are referred to afferent pathways, which are carried in multiple nerve roots from c7 through T4.[1]

Extrinsic factors can also play a role in exact circumstances. The oxygen transport capability of blood is based on a number of factors.[1]

Angina treatment involves a number of strategies. In general angina organization includes assessing patient risk factors, such as smoking, hypertension, dyslipidemia, diabetes mellitus, obesity, and physical inactivity.

While modifications in these hazard factors may recover symptoms and decrease cardiac proceedings the majority of patients with

chronic stable angina need specific antianginal medications. The referred emission pain of angina pectoris is hypothetical to occurs because these afferent pathways also bear pain fibers from other regions (i.e. the arm neck and shoulders).[1]

It is optional that beta-adrenergic overcrowding drugs be used as first-line treatment for patients with angina because these agents have been shown to have cardio defensive effect.[1]

The causes of angina pectoris are shown in cause. The most common cause of angina pectoris is coronary atherosclerosis, with a severe stenosis obstructing blood flow leading to myocardial ischemia (1.1). Other causes of angina due to obstruction are rare and include vasospasm, microvascular disease, coronary embolism, and anomalous coronary arteries. Angina can also be pragmatic in the lack of coronary hindrance from decreased oxygen supply (anemia, hypoxemia, or profound hypotension) or from circumstances causing increased oxygen insist (left ventricular hypertrophy, hypertensive crisis, or marked tachycardia).[6]

CAUSES

Angina itself is a indication (or set of symptoms), not a disease. Any of the following may signal angina:

- pain may be of any intensity from An uncomfortable pressure, fullness, squeezing, or pain in the center of the chest
- It may also feel like tightness, burning, or a heavy weight.
- The pain may spread to the shoulders, neck, or arms.
- It may be located in the upper abdomen, back, or jaw.
- The mild to severe.

Extra symptoms may come about with an angina harass, as follows

- Shortness of breath
- Lightheadedness
- Fainting
- Anxiety or nervousness
- Sweating or cold, sweaty skin
- Nausea
- Rapid or irregular heart beat
- Pallor (pale skin)
- Feeling of impending doom

These symptoms are equal to the signs of an imminent heart attack described by the American Heart Association. It is not always easy to tell the distinction between angina and a heart attack apart from angina only lasts a little minutes and heart attack does not go away.

- If you boast never had symptoms like this before, rest. If you are capable, call your healthcare provider, call 911, or go to the nearby hospital emergency department.
- If you boast have angina attacks facing and this attack is parallel to folks, relax for a few minutes. seize your sublingual nitroglycerin. Your angina should be totally calmed in five minutes. If not, you may replicate the nitroglycerin dose and wait an extra five minutes. A third dose may be tried but if you still have no reprieve call 911 or go to the nearest hospital emergency department.
- **Ischemia due to decreased oxygen supply**
  Anemia, hypoxia, hypotension
- **Ischemia due to increased oxygen demand**
  left ventricular hypertrophy, hypertension, tachycardia

What is angina pectoris?

Angina pectoris is the medical term for trunk pain or embarrassment due to coronary heart disease. Angina is a indication of a clause called myocardial ischemia. It occurs when the heart muscle (myocardium) doesn't get as a lot of blood (hence as much oxygen) as it needs. This frequently happens because one or more of the heart's arteries (blood vessels that supply blood to the heart muscle) is tapering or barren inadequate blood supply is called ischemia.[5]

Angina also can occur in public with valvular heart disease, hypertrophic cardiomyopathy (this is an enlarged heart due to disease) or unrestrained high blood pressure. These cases are rare.[5]

When does angina pectoris occur?

Angina frequently occurs when the heart desires more blood. For example, running to grab a bus could prompt an attack of angina while walking valor not. Angina may happen during exercise, strong emotions or tremendous temperatures. Some people, such as those with a coronary artery spasm, may have angina when they're resting. (See below, unstable angina, Prinzmetal's or variant angina pectoris.[5]

Angina is a symptom that someone is at increased risk of heart attack, cardiac arrest and unexpected cardiac death. See the related entries in this Encyclopedia, listed below. Also see "Risk Factors and Coronary Heart

Disease," "Heart Attack Symptoms / Warning Signs" and "Angina Pectoris Treatments." [5]

SIGNS AND SYMPTOMS

- Tightness, squeezing, pressure or ache in the chest.
- Sudden breathing difficulty.
- Chest pain similar to indigestion.
- Chest pain that radiates to the jaw, teeth or earlobes.
- Heaviness, numbness, tingling or ache in the chest, arm, shoulder, elbow or hand usually on the left side. Pain between the shoulder blades.[1]

RISK FACTOR INCREASES WITH

Identifying and treating risk factors for further coronary heart disease is a priority in patients with angina.[1]

The following risk factors increase the risk of coronary heart disease and angina[1]

- Smoking, obesity, diabetes mellitus.
- High blood pressure, high blood cholesterol levels.
- Excess intake of fact or salt.
- Fatigue over work or stress.
- Family history of coronary artery disease.
- Exposure to cold and wind
- Obesity
- Alcohol[1]

PREVENTIVE MEASURES

- Lose weight if patient are overweight
- Get medical treatment for underlying cause or risks.
- Eat a diet that is low in fat and low in salt
- Stop smoking.
- Avoid the stressful physical or emotional factors that trigger angina attacks.
- Exercise resulting after consulting Doctor.[1]

TYPES

A) Stable angina

People with stable angina (or chronic stable angina) have episodes of trunk embarrassment That are usually unsurprising They occur on physical exertion (such as running to catch a bus) or under mental or emotional stress. Normally the trunk embarrassment is calmed with rest, nitroglycerin or both.[5]

People with episodes of chest discomfort should see their physician for an evaluation. The doctor will evaluate the person's medical history and risk factors, conduct a physical exam, order a chest X-ray and take an electrocardiogram (ECG). Some people will also need an exercise ECG (stress test), an echocardiogram or other tests to complete the diagnosis.[5]

B) Unstable angina

In people with unstable angina, the trunk pain is unpredicted and frequently occurs while at rest. The distress may be more harsh and protracted than typical angina or be the first time a person has angina. The most common cause is reduced blood flow to the heart muscle because the coronary arteries are tapering by fatty buildups (atherosclerosis). An artery may be abnormally thin or partially blocked by a blood clot. Swelling infection and secondary causes also can lead to unstable angina. In a form of unstable angina called variant or Prinzmetal's angina, the cause is coronary artery spasm.[5]

Unstable angina is an acute coronary syndrome and should be treated as an Emergency.

People with new, aggravation or constant chest embarrassment should be evaluated in a hospital emergency department or "chest pain unit" and monitored carefully. They're at increased risk for acute myocardial infarction (heart attack).Severe cardiac arrhythmias. These may include ventricular tachycardia and fibrillation. Cardiac arrest leading to sudden death. [5]

C) Variant angina pectoris (Prinzmetal's angina)

Variant angina pectoris is also called Prinzmetal's angina. It frequently occurs impulsively and dissimilar typical angina, it almost occurs when a person is at rest. It doesn't chase physical exertion or emotional stress, either. Attacks can be very painful and usually occur between midnight and 8 a.m.[5]

Variant angina is due to ephemeral coronary artery spasm. About two thirds of people with it have severe coronary atherosclerosis in at least one major vessel. The spasm frequently occurs very close to the blockage.[5]

Diagnosis of Angina Pectoris

1. Diagnosis and assessment of angina involve clinical assessment, laboratory tests and specific cardiac investigations.
2. In the majority of cases, it is possible to make a sure diagnosis based on the history alone, although physical examination and further tests are necessary to confirm the diagnosis and to evaluate the severity of underlying disease.[1]

3. If the history and physical examination imply the presence of angina and CAD, patients are auxiliary evaluated by noninvasive tests such as exercise treadmill testing or coronary angiography.[12]
4. The evaluation of patients with chest pain should take into account symptom characteristics and cardiovascular risk factors, as these may indicate the probability of angina and coronary artery disease (CAD).

Laboratory Tests

1) Complete blood count.

2) Comprehensive metabolic panel—Includes assessment of the patient's kidneys, liver, electrolyte, acid/base balance, blood sugar, lipid profile and blood proteins.

3) Cardiac biomarkers— these are proteins that are released when myocardium cells are damaged. They help to differentiate angina from a heart attack. If the cardiac biomarkers are normal, then the chest pain is more likely to be due to angina and much less likely due to heart muscle damage.[2]

The commonly investigated markers include:

a) Troponin—A cardiac-specific marker. It will be elevated within a few hours of heart damage and remain elevated for up to 2 weeks.

b) CK-MB— A form of the enzyme Creatine kinase found mostly in heart muscle and rises, when the heart muscle cells are damaged.[12]

Non-Laboratory Tests

- An electrocardiography (ECG)—Evaluates the heart's electrical activity and rhythm. During chest pain, depression or elevation of the ST segment may be recorded.
- An exercise stress test.
- Echocardiography—Ultrasound imaging of the heart.
- Radionuclide imaging—A radioactive compound injected into the blood to evaluate blood flow. This shows images of narrowed blood vessels around the heart.
- Coronary angiography—X-rays of arteries using a radiopaque dye, performed during coronary catheterization.[12]
- Coronary catheterization—A thin stretchy tube is inserted into an artery in the leg and threaded up to the coronary arteries to evaluate blood surge and pressure in the heart and the grade of the arteries in the heart.

Pharmacotherphy of Angina Pectoris

CLASSIFICATION OF ANGINAL DRUG[7]

A) Beta blockers

- Metoprolol tartrate
- Metoprolol succinate
- Atenolol

B) CALCIUM CHANAL BLOCKERS

- Amlodipine
- Nifedipine
- Verapamil
- Diltiazem

C) NITRATES

- Isosorbide mononitrate
- Nitroglycerin

D) OTHER ANTIANGINAL DRUG

- Flunarizine
- Trimetazidine

SYNTHETIC TREATMENT

BETA BLOCKERS

Beta-blockers can be non-selective, such as propranolol, selective, such as metoprolol, and atenolol.

Non selective Beta -blockers inhibit both Beta1 and Beta 2 receptors; while selective Beta –blockers would be more specific for Beta 1 receptors. While Beta 1 receptors are present in the heart and induce, upon stimulation, an increase in heart rate and cardiac contractility, Beta 2 receptors are present in the smooth muscle cells of bronchi and vessels, and their activation induce bronco- and vasodilatation. Hence, in patients with asthma, the use of selective Beta –blocker would be recommendable.

Beta1 receptor blockages induce a decrease in heart rate and cardiac contractility, with subsequent reduction of myocardial oxygen consumption. These effects, being induced by catecholamine stimulation, are minimal at rest, but evident in all those conditions associated with sympathetic stimulation, such as exertion, and emotions. This represents the rationale for the clinical use of Beta blockers in patients with angina.

In the treatment of stable angina, it is conventional to adjust the dose of these drugs to reduce the rest heart rate to 55 to 60 beats per minute. Therapy with Beta -blockers limits the increase in heart rate during exercise, which should not exceed 75% of the heart rate response associated with the onset of ischemia. This can be properly evaluated during the execution of an exercise tolerance test (ETT).[8]. In patients with harsher angina, the heart rate can be bargain below 50 beats per minute, provided that there are no

symptoms associated with bradycardia and that heart block does not develop.

ROLE OF BETA BLOCKERS IN ANGINA PECTORIS

Beta blocks which will help to relieve the Symptoms of angina. Beta blocks work by blocking the actions of hormones called adrenaline and nor adrenaline which make the heart beat faster. Beta blockers slow down the heart and lower the blood pressure.

Propranolol was the first medication to be used clinically as a treatment for angina. It is known as a non-selective beta blocker because it blocks both types of viz- beta receptors beta 1 and beta 2. Blocking the beta-1 receptors decreases the amount of oxygen the heart needs in several ways; this results in an improvement in angina symptoms.

The therapeutic effects of beta blockers in angina pectoris can be attributed to an inhibition of beta receptor medicated stimulation of heart rate and myocardial contractility, resulting in an improved oxygen demand and supply balance in the myocardium. If the coronary arteries are narrowed atherosclerosis, the increased burden on the heart can cause inadequate oxygen delivery to the heart muscle (myocardium) itself, leading to the chest pain and other symptoms of angina pectoris.

Beta blockers are familiar class of remedy drugs that frustrate the stimulatory effects of adrenaline (epinephrine) on what are called the beta receptors. Beta blockers act by suppressing these stimulatory impulses and escort to a slowing of the pulse rate and a reduction in blood pressure by reducing the work load of the heart they can also reduce symptoms of angina pectoris.[1]

β-Blockers: Side Effects

1. Drowsiness and fatigue along with dizziness and weakness.
2. Dryness of the mouth, eyes and skin cold hands and feet Sleep disturbances and a decreased sex drive are less common but possible side effects.[1]

Calcium Channel Blockers

Are commonly used to treat Angina. They open up arteries and also reduce the work load of heart. There are many different types of these drugs and they differ in their actions. Some are better at slowing heart rates, and others work better in reducing blood pressure.

When calcium-blockers are contraindicated or not tolerated, calcium antagonists and/or nitrates are recommended as initial therapy.

Calcium antagonists, including dihydropyridine agents, such as nifedipine and amlodipine, and non dihydropyridine agents, such as diltiazem and verapamil, dilate the epicardial coronary vessels and decrease coronary vascular resistance, enhancing coronary blood flow. Dilatation of the epicardial coronary arteries is the principal mechanism by means of which calcium antagonists are used for the treatment of variant (vasospastic) angina.[8]

Role of Calcium Channel Blockers In Angina Pectoris

Calcium channel blockers have become valuable agents for the management of angina pectoris and hypertension. Calcium channel blockers are a heterogeneous group of compounds used in a variety of cardiovascular disorders such as stable angina pectoris, hypertension. Calcium influx into the myocyte. Initiate s a series of events essential for contractility.

Calcium entry into the myocyte first triggers intracellular calcium release; the released calcium then binds the regulatory protein troponin resulting in a calcium troponin complex which allows actin and myosin to interact and contract. The chain of events is the same in vascular smooth muscle cells, excluding that a calcium calmodulin complex instead of calcium troponin permits the contact between action and myosin. The net effect is vasodilatation; the resultant fall in blood pressure decreases cardiac work and may contribute to the efficacy of these drugs in the patient with angina.

The presented calcium channel blocking agents block receptors on the L-type calcium channel which gives climb to a slowly inactivating high threshold current in cardiac cells. (ca2+) through calcium channels. The most ample spread clinical usage of calcium channel blockers is to decreased blood pressure in patients with hypertension, with particular worth in treating elderly patients.

Pihydropyridine is one of the important class of Calcium channel blokers; these include isradipine, lacidipine, lercanidipine, amlodipine, felodipine, nicardipine, nifedipine, nimodipine and nisoldipine. These have more effects of relaxing blood vessels, and less effects of relaxing the heart muscle than verapamil or diltiazem. Most are used to treat high blood pressure and angina. Nifedipine is used to treat Raynaud's phenomenon. As they do not affect heart muscle much they are not useful for arrhythmia, and are unlikely to make heart failure worse. But isradipine, lacidipine and lercanidipine are only used to delight high blood pressure. Infact a dihydropyridine calcium-channel blocker in addition to a beta

blocker medicine is commonly used to prevent angina pains if either does not work enough alone.[1]

Calcium Channel Blockers Side Effect

1. AV node block and PR Prolongation
2. Blood Pressure Reduction
3. Exacerbation of chronic and acute heart failure
4. Extreme Bradycordia[1]

Nitrates

Nitrates are together with beta-blockers the drugs of choice for the treatment of chronic angina. Nitrates are usually given *per os*, even though at a much higher dose, as compared to the sublingual administration, in order to counteract the first-pass metabolism of the liver. Isosorbide dinitrate is given usually at the dose of 40 mg three times a day (tris in die: t.i.d.); alternatively, isosorbide mononitrate (active metabolite of isosorbide dinitrate), which has a longer half-life also because is not undergoing the first pass hepatic metabolism, is usually given at the dose of 20-40 mg twice a day (bis in die: b.i.d.) or, in its slow-release formulation, at the dose of 50-80 mg once a day.

Nitroglycerine can also be given locally in the form of ointment or transdermal patches; in this case, nitroglycerine is absorbed into the bloodstream without receiving hepatic degradation.

All nitrates, above all nitroglycerine, can induce tolerance, which can develop within 24 hours and is characterized by a progressive reduction of the pharmacological effects of nitrates given uninterruptedly at a constant dose.[8]

Role of Nitrates in Angina Pectoris

Nitrates are extensively used for the treatment of Angina Pectoris. However continuous therapy with either oral nitrates or nitroglycerin patches leads to rapid development of tolerance, with loss or diminution of antinational and anti-ischemic effects. Nitroglycerin patches applied for 10-12 hours during day increase exercise duration for 8- 12 hours, but a rebound increase in angina attacks during the nitrate free interval may occur.

Oral isosorbide 5-mononitrate, 20mg twice a day with the first dose administered in the morning and the second does 7 hours later, increase exercise duration for at least 12 hours, without the development of tolerance to either the morning or afternoon does. This dosing regimen has been shown not to produce a rebound phenomenon during the periods of low nitrates levels at night and early hours of the morning ISO-Sorbide dinitrate (30mg) prescribe at 7 AM and 1 PM does not produce tolerance to the 7 AM does, but effects of the afternoon dose have not been evaluated. Recent date suggest that isosorbide dinitrate given 3 or 4 times daily produce tolerance and this dosing schedule is inadequate for anti-angina prophylaxis.It should be recognized that intermittent oral or patch therapy with nitrates during the day leaves the patient unprotected at night and early hours of the morning.

Therapeutic doses of nitroglycerin may reduce systolic diastolic and blood pressure. Efficient coronary perfusion pressure is frequently maintained, but can be compromised if blood pressure cataract excessively or increased heart rate decreased diastolic filling time.

The foremost pharmacological action of nitroglycerin is respite of vascular smooth muscle. while venous effects prevail nitroglycerin create, in dose associated manner, dilation of both arterial and venous beds.Qilation of post capillary vessels, together with large veins promotes peripheral pooling of blood, decreased venous return to the heart and reduces left ventricular end diastolic pressure (Preload) nitroglycerin also produces arteriolar relaxation. There by plummeting peripheral vascular resistence and arterial pressure(after load) and dilates large epicardial coronary arteries; however the amount to which this latter effect contributes to the reprieve of exertional angina is unclear.

Side Effect

1. Headache
2. Orthostatic hypotension, reflex tachycardia
3. Methemoglobinemia (presence in red cells of methemoglobin, which is no longer able to bind oxygen and cannot be converted back to hemoglobin)

Calcium channel blockers are appropriate if beta blockers are contraindicated or not tolerated[7]

Calcium channel blockers can be painstaking second-line for treating the symptoms of angina if a beta-blocker is contraindicated or not tolerated. Calcium channel blockers have been revealed to be similarly effective as beta-blockers in the management of stable angina, i.e. studies have revealed no distinction in nitroglycerin use or exercise time and no substantiation of a difference in total or cardiovascular mortality, or in risk of myocardial infarction or stroke. The recently updated National Institute for Health and Clinical Excellence (NICE) guideline for the management of stable angina considers either

a beta-blocker or a calcium channel blocker as appropriate first-line treatment.

Calcium channel blockers minimize symptoms of angina by dilating coronary and other arteries and increasing coronary blood flow. Non-dihydropyridine calcium channel blockers (verapamil and diltiazem) also reduce myocardial contractility and heart rate and decrease myocardial oxygen demand.

All calcium channel blockers are valuable in the treatment of stable angina. Long-acting calcium channel blockers, e.g. amlodipine, or sustained released formulations of short-acting calcium channel blockers, e.g. felodipine, nifedipine, verapamil and diltiazem, are preferred. Short acting calcium channel blockers, mostly nifedipine, are not optional because they cause reflex tachycardia which may worsen ischemia and have been associated with an increased risk of cardiovascular events.

A rate restraining calcium channel blocker such as verapamil or diltiazem is a appropriate substitute for patients who have had a preceding MI who do not endure beta-blockers or have a contraindication to their use.12 However, verapamil is not appropriate in patients with heart failure.

If combining beta-blockers and calcium channel blockers, it is suitable to use a non-rate restraining (dihydropyridine) Diltiazem may be vigilantly used in combination with a beta-blocker when heart rate remains above 60 beats per minute despite maximum tolerated doses of beta-blocker. Verapamil is not suitable in combination with beta-blockers because severe bradycardia and heart failure can occur.[7]

A long-acting nitrate can be used if a beta-blocker or calcium channel blocker are not tolerated or contraindicated Long acting nitrates, e.g. isosorbide mononitrate, are a suitable choice as monotherapy for people who are intolerant of beta-blockers or calcium channel blockers or if those medicines are contraindicated. They may also be used in grouping with a beta-blocker or calcium channel blocker. Nitrates construct venous and arterial dilatation, tumbling ventricular pre-load and after-load which lowers myocardial oxygen demand and improves subendocardial blood flow.[7]

Nitrate forbearance is a major problem with long-term use, and needs to be avoided because it diminishes the response to short acting nitrates. “nitrate-free” interval of 12–14 hours each day is obligatory to avoid nitrate tolerance. This is achieved with once daily dosing of modified release tablets, e.g. Corangin, Duride.[7]

High-dose zinc to terminate angina pectoris

Zinc deficiency is found in elderly hospitalized patients, and higher proportions of respiratory infections, cardiac failure, and depression were observed among zinc deficient patients as compared with the group of patients with normal zinc status. Today, the elderly tend to avoid meat and other high zinc-content foods due to fears of cholesterol. Rather, they increase consumption of refined wheat products. Wheat was a main source of zinc in the historical diet, but today zinc, magnesium and other critical nutrients have been depleted by the refining process .Therefore, zinc deficiency may play a major, perhaps the dominant role, in causing most adverse cardiac events.

High-dose zinc (50–300 mg/day) significantly decreases serum high-density lipoprotein concentration and increases low-density lipoprotein. while lower doses or biologically unavailable zinc compounds such as zinc oxide do not . It is hypothesized that transient increases in low density serum cholesterol result from release of tissue bound low density lipoprotein by action of zinc. We coincidentally found that zinc could benefit angina pectoris during a zinc gluconate lozenge for common cold clinical trial.[11]

Revascularization to treat symptoms of angina

Revascularization involves either percutaneous coronary intervention (PCI) or coronary-artery bypass surgery (CABG). Revascularization is most frequently performed for symptom relief but a small percentage of patients also have a prognostic benefit (usually those who are at high risk). Stress testing or similar further risk stratification is required in all patients with stable angina unless co-morbidities would prohibit revascularization. Patients who may benefit from revascularization include:

Patients who may benefit from revascularization include

Those at high-risk, e.g. patients with symptomatic multi-vessel disease, proximal left anterior descending or left main artery disease, left ventricular systolic dysfunction, diabetes or a large ischaemic burden (referring to all angina episodes, including silent angina).

Those who have unsuccessful to respond to pharmacological treatment, i.e. patient is immobile experiencing symptoms while on two antianginal drugs.

Optimal Medical Treatment or Revascularization

Clinical trial verification suggests that revascularization firstly provides healthier symptom control than pharmacological treatment, 21 but in the long-term, it appears that there is little difference between the two approaches to angina Symptom control.

Two large, recent clinical trials have compared the effectiveness of pharmacological treatment to revascularization in the management of chronic stable angina. Earlier trials may no longer be relevant to modern clinical practice due to advances in PCI techniques (e.g. the use of stents) and improvements.In the optimal use of medicines for both symptom control and risk factor reduction.[7]

Angioplasty to treat symptoms of angina

Angioplasty actually opens the conduit of diseased arterial segments, relieves the recurrence of chest pain, increases the worth of living and reduces other complications of the disease. [1]Angioplasty is a method used to expand an area of arterial blockage with the help of a catheter that has an inflatable small sausage formed balloon at its tip. Since the balloon catheter is introduced through the skin of the groin, and sometimes the arm is placed within a blood vessel and is useful in the treatment of coronary arteries, the technique is called PTCA or percutaneous transluminal coronary angioplasty

Since it is performed through a little needle hole in the groin (or sometimes the arm), it is much less in persistent than surgery and can be repeated more often should the patient develop disease in the same or another artery in the future. Angioplasties are safer than bypass surgery and according to statistics less than 1% of people die from complications after this procedure.[1]

CORONARY ARTERY BYPASS SURGERY

Coronary artery bypass surgery also coronary artery bypass graft (CABG Pronounced Cabbage) surgery and colloquially heart bypass or bypass surgery is a surgical procedure performed to relieve angina and reduce the risk of death from CAD.

Arteries or veins from elsewhere in the patient's body are grafted to the coronary arteries to bypass atherosclerotic narrowing and import the blood supply to the coronary circulation supplying the myocardium. This surgery is usually performed with the heart stopped necessitating the usage of cardiopulmonary bypass, techniques are available to perform CABG on a beating heart so calted "off pump" surgery.

Both PCI and CABG are more effective than medical management at releasing symptoms. (e.g. angina, dyspnea, fatigue). CABG is superior to PCI for some patients with multivessel CAD.

The prediction following CABG depends on a variety of factors but successful grafts typically last around 10-15 years. In general CABG improves the chances of endurance of patients who are at high risk (meaning those presenting with angina pain shown to be due to ischemic heart disease), but statistically after about five years the difference in endurance rate between those who have had surgery and those treated by drug therapy diminishes.

Age at the time of CABG is critical to the prediction, younger patients with no complicating disease having a high probability of greater long life. The older patient can frequently be expected to suffer further blockage of the coronary arteries.[1]

8) HERBAL TREATMENT

Home Remedies for Angina Pectoris

Many people use home remedies, which have been in use for many centuries. Some of these remedies are ideal for the treatment of angina as well as common heart problems. Angina is very serious problem and you need to visit your doctor for treatment but you can follow these home remedies to support the treatment.

Lemon

many people find that lemon juice is an effective treatment of angina. This is because lemon juice eliminates and stops cholesterol accumulation in the blood vessels.

Garlic

This is a beneficial well being food, which helps in effective treatment of a variety of health problems including angina. This food also minimizes the effect of an angina attack on a patient.

Grapefruit

This natural tonic improves the functions of the heart. Many people include grapefruits in their diet to help in curing angina.

Basil leaves

Many home remedies have basil leaves as a major ingredient. Basil leaves can also be used to make a remedy for angina pectoris. These leaves are chewable and may be taken in the morning. This may help an angina sufferer to minimize the effects of the disorder.

Lemon with Honey
Take a glass of warm water and squeeze half cut slice of lemon and add one teaspoon of honey. Mix it together and drink it before first thing in the morning.

Onion
Onion juice is also very effective for angina suffering person. Take onion juice in the morning. It reduces bad cholesterol in the blood and helps to deliver proper blood supply to the heart.

Parsley tea
Taking parsley tea or beet root juice two times in a day is very effective in treatment of angina.

Diet Change
Increase fruits and vegetables in your daily diet as they are very essential to avoid any type of cardiovascular disease.[13]

Think to avoid angina pectoris

- You should quit smoking if you are an angina patient.
- Reduce excess weight as excess weight is one of the main contributors of health problems.
- Avoid sedentary life and do regular exercise. This is necessary for proper blood circulation in the body.
- Avoid food that contains high cholesterol.
- Avoid high sugar things like soft drinks.[14]

Angina or Chest Pain Treatment at Home:

- When you feel pain in your chest, stop all the work that you are doing.
- Lie down on a comfortable position keeping your head up.
- You can take one aspirin or equivalent but not more than that as it may cause some other unwanted side effects. [14]

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