Cardiovascular Disease: A Review on various Medicinal Plants

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Abstract

Cardiovascular diseases(CVD) are the one of the most leading cause for death in the all countries, which include diseases of heart and vascular system supplying to the vital organs, caused due to various factors like age, gender, high blood pressure, hyperlipidaemia, diabetes mellitus, tobacco smoking, obesity, psychological factors, various dietary and life style modification, with prevalence of 17.9 million every year. CVD, a group of disorders of the heart and the vasculature, includes high blood pressure(HBP), coronary heart disease(CHD), myocardial infarction, congenital heart defects(CHD), cardiac arrhythmias(CA), heart failure(HF) and stroke Cardio-protection(SCP) include" the mechanism and means that contribute to the preservation of the heart by reducing or preventing Myocardial damage. Among all the cardio vascular diseases, Myocardial infarction(MI) is considered as one of the most dangerous diseases. Cardiovascular diseases are the major health problem of the developing countries of the world. It may present as a typical heart attack, a sudden death or may be detected at an advanced stage and be described as a silent infarct. Cardiovascular disease is one of the number cause disease of death globally and is projected to remain the leading cause of death. There is need for the natural therapy with the help of medicinal plants.

Key words: Cardiovascular disease, Myocardial Infraction, Coronary heart disease, Congestive heart failure, Cardiac arrhythmias, Hyperlipidaemia.

Introduction

Myocardial infarction: It is immovable damage to the heart muscle. "Myo" means muscle, "cardial" refers to the heart, and "infarction" means death of tissue to the lack of blood supply. The myocardial infarction also called as heart attack, which decreases the blood flow or stops to the part of a heart, causing damage to the heart muscle.

Pathophysiology: Cholesterol removal within the wall of the main artery. Then the deposited cholesterol ultimately forms a plaque in the wall of the artery called atherosclerotic plaque. Atherosclerotic plaque formation is a long term process, sometimes this plaque may rupture or destroy, which leads to activate the clotting mechanism so platelet aggregation and fibrin deposition, which leads to formation of an occlusive thrombus in coronary artery. It completely blocks the coronary artery and interrupts blood supply to the part of the myocardium. It also leads to irreversible changes and death of myocardial cells, and ultimately ST-segment elevation of myocardial infarction develops.

Symptoms:

- Pressure, heaviness, tightness, squeezing, (or) pain in your chest
- Discomfort that goes into your back, jaw, arm.
- Fullness, indigestion, sweating, upset stomach
- Severe weakness, anxiety, fatigue.
- Fast (or) uneven heartbeat.

The symptoms are different from person to person. Women are more likely to have the heart attack symptoms.

- Unusual feelings
- Shortness of breath
- Nausea (or) vomiting
- Discomfort in your gut. It may feel like indigestion.
- Discomfort in the neck, shoulder.

The symptoms are more common in people who have diabetics.

Signs:

- Pain in the chest, back, jaw, and other areas of the upper body that lasts more than a few minutes and comes back.
- Vomiting, nausea, shortness of breath, sweating, anxiety.
- Tightness in chest or pressure.

Diagnosis

1. **Electrocardiogram:** The primary use of the electrocardiogram is to detect ischemia. It may be used to follow rapid changes in time. The standard 12 lead ECG is not effective in examining the right ventricle, the posterior basal and lateral walls of the left ventricle. In particular, acute myocardial infarction in the distribution of the circumflex artery which produce a nondiagnostic ECG. The posterior leads V7, V8, and V9 may increase sensitivity for right ventricular and posterior myocardial infraction. The 12 lead ECG is used to classify patients into one of the three groups: those with ST segment elevation or new bundle branch block,

    - These with ST segment depression/T wave inversion and
    - These with a so-called non-diagnostic/normal ECG

2. **Cardiac markers:** Cardiac enzymes are those proteins that leak into the blood stream through the cell membrane that was damaged during the myocardial cell injury. Earlier, the enzymes SGOT and LDH were used to assess cardiac injury. Whereas now the markers are most widely used in the detection of MI. The cardiac troponins T and I are generally released within the first 4-6 hours after myocardial infarction, remain elevated for up to 2 weeks. This gives the complete tissue specificity and act as markers for assessing myocardial damage. Heart-type fatty acid binding protein is other marker, which used in some home test kits. Elevated troponin levels may accurately assess the myocardial infarction. Glycogen phosphorylase BB, a new cardiac marker is considered has potentially useful biomarker in the early detection of ischemia. The diagnosis of MI is based only if any of the two following conditions are satisfied.

    - Onset of Chest pain lasting for more than 30minutes
    - Changes in ECG
    - Levels of cardiac biomarkers

3. **Angiography:** coronary angiography is a technique used to visualize the blood flow. Catheter is a small tube inserted into the artery that moves to the vessels and supply blood to heart. The dye injected in ten catheter helps to detect the blockages in the blood flow. This blocks are cleared by angioplasty which is very sensitive therapy that requires highly skilled and well trained interventional cardiologist.

Treatment

1.Anti-coagulants: Anticoagulants are those drugs that prevent the coagulation, they will make blood clot more slowly or less effective than normal. It may leads to thrombolysis. It is medicines that increases the time to take the blood to clot. They also called as blood thinners. Each type works on different level on the coagulation pathway. It can give by oral route and in some times it can give only by injection.
Example: Heparin

Drugs:

Heparin: It is an anti-coagulant which is used to prevent the formation of blood clots. It is a naturally occurring anticoagulant produced by basophils and mast cells to prevent formation and extension of blood clots. It permits the body's natural clot by lysis mechanism, i.e., fibrinolysis, to work normally to break down previously formed cells. Then it is worked by activating antithrombin 3, which blocks the thrombin from clotting factor.

Mechanism of action: The unfractionated heparin bonds to the inactive antithrombin is binding and divides through the complex change then analyse the active antithrombin to bind and interacts with the factor Xa leading to the respect in the plasma to stop the looking the occlusion of thrombin from prothrombin thereby it inhibit the production of fibrin from fibrinogen and to prevent further blood clotting. It does not affect bleeding time, but it does require the time that blood takes to clot.

Uses:

- Post-operative patients, acute infarction, pulmonary embolism, venous thrombosis.
- It does not cross BBB so safe to use in pregnancy.
- It is used to treat the blood clots.
- It is used for initiate therapy.

Side effects:

More common side effects of heparin include are:

- Irritation, pain, or sores at the injection site.
- Allergic reactions, chills, fever.
- Increased liver enzymes.
- Takes longer time to stop bleeding.
- More easy for bruising.

Adverse effects:

- Abdominal pain
- Stomach pain
- Back pain
- Swelling, headaches.
- Heavy bleeding/wounds
- Joint pain, stiffness
- Bleeding from gums, blood in urine.
- Coughing

Contraindications: If patient does not receive heparin they have some indications like

- The platelet count should be lower.
- The patient should be active, uncontrollable bleed except for disseminated intravascular coagulation.
- The patient should avoid the heparin use because the patient suffered with heparin induced thrombocytopenia.
- The patient should not have routine monitoring test performed or to monitor the therapeutic heparin.

Drug Interactions:

a. Oral anticoagulants: Heparin sodium may prolong the one -stage prothrombin time. Therefore, when heparin sodium is given with dicumarol or warfarin sodium, a period of at least 4-5 hours after the last intravenous dose or one day after the last subcutaneous dose should flow before blood is drawn if a valid prothrombin time is to obtained.

b. Platelet inhibitors: Drugs such as NSAIDS (including salicylic acid, ibuprofen, indomethacin, and celecoxib), dextran, phenylbutazone, thienopyridines, dipyridamole, hydroxychloroquine, glycoprotein IIb/IIIa antagonists (including abciximab, eptifibatide, and tirofiban), and others that interfere with platelet-aggregation reactions (the main hemo-static defence of heparinized patients) may induce bleeding and should be used with caution in patients receiving heparin sodium. To avoid the risk of bleeding, a reduction in the dose of antiplatelet agent or heparin is recommended.

c. Digitalis, tetracyclines, nicotine, antihistamines, or intravenous nitro-glycerine may partially counteract the anticoagulant action of heparin sodium.

d. Heparin sodium in 5% dextrose injection: Intravenous nitro glycerine administered to heparinized patients may result in a decrease of the partial thromboplastin time with subsequent rebound effect upon discontinuation of nitro glycerine. Careful monitoring of partial thromboplastin time and adjustment of heparin dosage are recommended during coadministration of heparin and intravenous nitro glycerine.

e. Antithrombin III (human): The anticoagulant effect of heparin is enhanced by concurrent treatment with antithrombin III (human) in patients with hereditary antithrombin III deficiency. To reduce the risk of bleeding, a reduced dosage of heparin is recommended during the treatment with antithrombin III (human).

2. Vasodilators: Vasodilators are medications that clear the blood vessels. Vasodilator drugs relax the smooth muscle in blood vessel to improve blood flow, thus decreasing blood pressure. Dilation of arterial vessel reduces systemic vascular resistance, which lead to fall in arterial blood pressure. Dilation of venous vessel decreases the venous blood pressure. The vasodilators thus used in treatment of hypertension , pectoris and heart failure.

Example: Nitrate

General mechanism of action: In general, Vasodilators are used to prevent constriction of the blood vessels, which allow greater blood flow to various organs in the body. The mechanism of vasodilators is done in such a way that it binds to receptors on endothelial cells of the blood vessel, which stimulate calcium release. Calcium activates the enzyme nitric oxide synthase and converts L-arginine into nitric oxide. It moves the endothelial cell through diffusion and enters vascular smooth muscle cells. Nitric oxide activates GTP and then converts it into cGMP. cGMP triggers the myosin-light chain phosphatase thus removing a single phosphate from myosin and actin filaments. This dephosphorylation of myosin and actin filaments leads to the relaxation of vascular smooth muscle.

The specific mechanisms of action are the different classes of vasodilators:

- ACE Inhibitors: Prevent the alteration of angiotensin I to angiotensin II, a potent vasoconstrictor.
- Angiotensin Receptor Blockers: Avoid the angiotensin II receptor from binding site of receptor.
- Nitrates: Expansion the amount of Nitric oxide in vascular smooth muscle cells, causing vasodilation. Nitrates dilate veins more than arteries and decrease reload.
- Calcium Channel Blockers: Block calcium channels in the cardiac and smooth muscles, initiate reducing the muscle contractility and vasodilation. They are two classes of CCBs: Dihydropyridines- act on the vascular smooth muscle, and non-dihydropyridines act on the heart.

- Minoxidil: Exactly relaxes the arteriolar smooth muscle, with minimal effect on the vein. Cyclic AMP may mediate its effects.
- Hydralazine: Exact mechanism is still unknown.
- Beta-Blockers: Nebivolol and carvedilol are third-generation β-adrenoreceptor antagonists that have additional endothelium-increased vasodilating properties.

Drug:

Nitrates: Nitrates are converted into nitric oxide**.** This used for the treatment of angina. Angina is an uncomfortable feeling, or pain in chest that can spread to arms, back, jaw, neck or stomach. It is caused by narrowing, blockage or muscle contraction within the coronary arteries, which limits the amount of oxygenated blood reaching part of your heart muscle. By dilating the blood vessels of the heart, nitrates can decrease the stress on the heart by improving blood flow to the heart muscle. This will relieve angina symptoms.

Example: Nitro-glycerine, Isosorbide dinitrate, Isosorbide mononitrate

Mechanism of action: Nitric oxide act as vasodilators. It acts on both coronary arteries as well as systemic arteries. Initially vasodilation of coronary arteries reduce vasospasms and restores coronary blood flow and resolves chest pain. Later the vasodilation of systemic arteries and veins reduces preload and afterload on the heart leading to reduction in myocardial work load.

Uses:

- It is used to treat angina.
- It is used to treat congestive heart failure and stroke.

Side effects:

Headache, Dizziness, Nausea, Light head ness, Restlessness, Stomach discomfort, Skin rash, Dry mouth, Blurring of vision, Hypotension, Arrhythmia.

Adverse effects:

- Headaches (greater than 10%)
- Hypotension (0.1 to 10%)
- Cutaneous flushing (0.1 to 10%)
- Syncope (0.1 to 10%)
- Reflex tachycardia (0.1 to 10%)

Contraindications:

Several contraindications subsist for the use of nitrate. They are the following:

- Allergy to nitrates
- Combined use of phosphodiesterase (PDE) inhibitors such as tadalafil and sildenafil
- Right ventricular infarction
- Hypertrophic cardiomyopathy

These are the following conditions that require care with nitrate administration:

- Patients on chronic diuretic therapy
- Patients with low systolic blood pressure

- Patients with autonomic nervous system dysregulation
- Pregnancy and breastfeeding.

Drug Interactions:
Other medicines that might be taking can increase or decrease the effect of nitrates and these effects are called an interaction. These are the following categories of medicines that can increase or decrease the effects of nitrates.

Viagra (sildenafil), Levitra (vardenafil), or Cialis (tadalafil). Thus taking the nitrates with in 24 hrs do not take Viagra, Levitra, or Cialis. When the barrenness medicines are mixed with nitrates, the combination can lower your blood pressure and make you dizzy, lightheaded, or faint.

- Medicines to treat high blood pressure
- Certain heart medicines
- Retail cough, cold, and flu medicines
- Retail herbal supplements for treating cough, cold, and flu

While taking nitrates, smoking should be avoided, because it can decrease the effect of the medicine. Alcohol should be avoided, because it can increase the effect of the medicine.

3.Beta-blockers: Beta blockers usually known as blocking agents as they reduce blood pressure. They mainly block the effect of epinephrine commonly called as adrenaline. They control the heart beat in such a way that reduces blood pressure. They also help in improving the blood flow by widening the vein and arteries. Thus they effectively act on heart and blood vessels.

Examples : Propranolol (Inderal, InnoPran XL)

General mechanism of action: Beta blockers block the beta adrenoreceptor in the heart, bronchi, pancreas, liver and peripheral vasculature. They slow the heart and decrease the cardiac output.

Drug:

Propranolol: It is a non-selective beta blocker. It is beta-1 and beta-2 receptor. It is a synthetic compound.

Mechanism of action: Propranolol is one of the beta adreno receptor blocker. It blocks beta-1 receptor on cardiac muscle cell which inhibits the function of enzyme adenylate cyclase. This intern ATP inhibits the cyclic AMP synthesis in consequent production of PKA leading to decrease in calcium influx which will be L-type of calcium channels leading to decrease in sympathetic effect on myocardial cell and which is decrease in heart rate and contractility for more propranolol onset renin secretion from the kidney which will reduces angiotensin -2 formation.

Uses:
- It is used to treat the conditions that cause like an irregular heartbeat.
- It helps to prevent the chest pain caused by angina.
- It is used to prevent migraines.

Side effects:
Headache, weak, feeling tried, stomach pain, cold fingers, being sick, diarrhoea, itching, vomiting, nausea, change in blood, toes.

Adverse effects:
Allergic reactions, skin rash, swelling of face, lips, hives, bradycardia, postural hypotension, mood changes, nightmares, dizziness, hallucinations.

Contraindications:

It is contraindicated with an cardiogenic shock, sinus bradycardia and more than first degree block, congestive heart failure unless the failure is secondary to a tachyarrhythmia treatable with propranolol.

Drug Interactions:

- Calcium channel blockers: it has additive effect.
- Prostaglandin Synthase Inhibiting Drugs: Decrease the hypotensive effect of beta blockers.
- Haloperidol: Hypotension and cardiac arrest.
- Theophylline: Clearance is reduced.
- Cimetidine: It increases the blood levels.

4.Calcium channel blockers: Calcium channel blockers are used to treat blood pressure. Calcium make the heart and arteries to squeeze (contract) more strongly. Now these Calcium blockers block the entry of calcium into heart and arteries thus allowing the blood vessels to relax and open. Some calcium channel blockers can slower the heart rate subsequently reducing blood pressure. In addition to this ,they are also prescribed to relieve chest pain (angina) and control an irregular heartbeat. Calcium channel blockers are also popularly known as calcium antagonists.

Example: Nifedipine, Verapamil

General mechanism of action: Calcium channel antagonists, the name itself suggests that it block the calcium by binding to the L-type "long-acting" voltage-gated calcium channels in the heart, vascular smooth muscle, and pancreas. These are categorised into dihydropyridines and non-dihydropyridines. The dihydropyridines act as vasodilators. The non-dihydropyridines reduce the heart rate and is also used in treating hypertension.

Drugs:

Nifedipine: It is a calcium channel blocker in the dihydropyridine subclass. It acts on both antihypertensive and as an anti-anginal medication. FDA-approved indications include chronic stable angina, hypertension. It also has other off-label indications.

MECHANISM OF ACTION: During the depolarization phase of smooth muscle cells, the calcium ions enter the muscle cells through voltage-gated channels. Nifedipine restricts the entry of calcium ions by blocking these voltage-dependent L-type calcium channels in vascular smooth muscle and myocardial cells. Reduced intracellular calcium reduces peripheral arterial vascular resistance and dilatation of coronary arteries thus leading to a reduction in systemic blood pressure and increased myocardial oxygen delivery. Thus it plays a major role in reducing blood pressure and in controlling chest pain.

Uses:

- It is used to control angina.
- It lowers the blood pressure.
- It works by relaxing blood vessels.

Side effects:

- Bloating, swelling of face, arms, hands.
- Labored breathing, light-headedness
- Feeling of warmth, headache, muscle cramps.
- Fast, pounding/ racing heartbeat.

Adverse effects:

Commonly repeated adverse effects are peripheral oedema, headache, flushing, heart burn, nausea, tachycardia, dizziness, palpitations, rash, gum hyperplasia.

Contraindications:

Absolute contraindications:

- Hypersensitivity to nifedipine
- ST-elevation

Relative contraindication:

- Severe aortic stenosis.
- Unstable angina
- Hypotension
- Moderate to severe hepatic impairment.

Drug Interactions:

- Beta blockers: it may increase cardiovascular effects.
- Digoxin: It may increase digoxin serum concentration and monitor digoxin levels if necessary and then reduce digoxin dose.
- Rifampicin: It do not use together as effective levels of nifedipine may not be reached.
- Fentanyl: Severe hypotension has been reported during fentanyl anaesthesia with concomitant use of a beta-blocker and a calcium channel blocker.

5.Opiate analgesic: Opioids are a class of medication used in the treatment of pain. These are the substance that act on opioid receptors to produce morphine like effects. Primarily they used for pain relief, including anaesthesia.

Example: Morphine sulphate

General mechanism of action: Opioids show analgesic effect in both presynaptic terminal and post synaptic terminal. Presynaptically, opioids block calcium channels on afferent nerves thus restricting the release of neurotransmitters namely substance P and glutamate that contribute to nociception. Postsynaptically, opioids hyperpolarize cell membranes by opening the potassium channels thus increasing the potential to generate nociceptive transmission. The mu, kappa, and delta-opioid receptors moderate analgesia spinally and supraspinally. It acts either through weak serotonin reuptake inhibition or by increasing the release of intrasynaptic serotonin by inhibiting gamma-aminobutyric acidergic presynaptic inhibitory neuron on serotonin neurons. These opioids may cause serotonin syndrome and so their usage should be very cautious when combined with other agents with serotonergic activity. Opioids such as methadone also shows analgesic property at the N-methyl-D-aspartate (NMDA) receptor. Methadone binds to the NMDA receptor thus antagonizing the effect of glutamate. This theoretically explains the efficacy of methadone in treating neuropathic pain compare to other opioids.

Drug:

Morphine Sulphate: Morphine sulphate includes moderate to severe pain it may be acute or chronic. It stick to opioid receptor in central nervous system and some of other tissues. They commonly used in pain management, it provides major relief to patients afflicted with pain. In some clinical situations they help by medicating with morphine include management of sedative/end-of-life care, active cancer treatment, and Vaso-occlusive pain during sickle cell crisis. It also has off-label uses for the painful conditions.

Mechanism of Action: Morphine is considered as one of the classic opioid analgesic when compared with other painkillers. Similar to other analgesics, morphine has binding affinity for delta, kappa, and mu-opioid receptors. This drug exhibits its most of the analgesic property by binding to the mu-opioid receptor within the central nervous system (CNS) and the peripheral nervous system (PNS). This results in the activation of descending inhibitory pathways of the CNS as well as inhibition of the nociceptive afferent neurons of the PNS which in turn leads to an overall reduction of the nociceptive transmission.

Uses:

- This medication is used to cure severe ongoing pain (such as due to cancer).
- They commonly belong to the class of drugs known as opioid analgesics.
- It works in the brain to change how they feels and responds to pain.
- The higher strengths of this drug (90 and 120 milligrams per capsule) should be used only if they have been regularly taking the moderate to large amounts of an opioid pain medication.
- Extended-release form of morphine is not for use on an as-needed basis for pain.
- The extended-release of morphine is for around the clock treatment of pain.

Side Effects:

Some side effects can be serious. They include:

- Blue or purple colour to the skin
- Changes in heartbeat, inability to get
- Turbulence, hallucinations, fever, sweating, confusion, fast heartbeat, shivering, severe muscle stiffness or twitching, loss of coordination, nausea, vomiting, or diarrhoea
- Nausea, loss of appetite, or dizziness, decreased sexual desire

Adverse Effects:

Unwanted effect of morphine is constipation. Reduces peristalsis, central nervous system depression, urinary retention, respiratory depression, light-headedness, sedation, weakness. Other effects like euphoria, dysphoria, dry mouth, anorexia, biliary tract spasm, flushing, syncope, pruritis, edema, urticaria.

Contraindications:

Morphine sulphate tablet are contraindicated in patients with:

• Hypersensitivity to the active substance

• Acute chronic obstructive pulmonary disease

• Severe bronchial asthma

• Serious respiratory depression with hypoxia and/or hypercapnia

• Paralytic ileus, acute abdomen

• Head injury, delayed gastric emptying

• Known morphine sensitivity

• Acute hepatic disease

Drug Interactions:

- The concurrent use of opioids with sedative medicines such as benzodiazepines or related drugs can increases the risk of sedation, respiratory depression, coma and death because of additive CNS depressant effect. The dosage and time should be limited.
- Alcohol may improve the pharmacodynamic effects of MST CONTINUS tablets; concomitant use should be avoided.

- Medicinal products that block the action of acetylcholine, for example antihistamines, anti-parkinson's and anti-emetics, may interact with morphine sulfate to enhance anticholinergic adverse events.
- Cimetidine -it inhibits the metabolism of morphine sulfate.
- Plasma concentrations of morphine sulfate may be reduced by the drug of rifampicin.
- Pregnancy: These tablets are not prescribe during pregnancy and labour. Daily use in pregnancy may cause drug use in the foetus, leading to withdrawal symptoms in the child. If opioid use is need for a prolonged period in pregnant women, counsel the patient to the risk of neonatal opioid withdrawal syndrome and ensure that appropriate treatment will be available. Administration during labour may get down respiration in the neonate and an antidote for the child should be readily available.
- Breast feeding: Morphine administration is not recommended for nursing women to secrete in breast milk and may cause respiratory depression in new born baby.
- Fertility : Animal studies have shown that morphine may decrease fertility.

Precautions:

MST CONTINUS tablets should be taken with caution in patients include:

• Impaired respiratory function, Severe cor pulmonale

• Tolerance, Psychological dependence[addiction], abuse profile

• Acute alcoholism, delirium tremens

• Head injury, intracranial lesions or reduced level of consciousness of uncertain origin.

• Hypotension with hypovolaemia, hypothyroidism, adrenocortical insufficiency

6. Thrombolytic drugs: Thrombolysis usually known as thrombolytic therapy, is used to dissolve dangerous clots in blood vessels, improve blood flow, and prevent damage to tissues and organs. They may involve the injection of clot-busting drugs through an intravenous (IV) line or through a long catheter that delivers drugs directly to the site of the blockage. It also may involve the use of a long catheter with a mechanical device attached to the tip that may removes the clot or physically breaks it up.

Example: Streptokinase, Urokinase

General mechanism of action: Thrombosis occurs when blood coagulates in blood vessels. During this process, prothrombin is converted to thrombin by platelets which in turn converts fibrinogen to fibrin thus forming fibrin matrix. Plasmin derived from plasminogen accumulates in fibrin matrix. Tissue plasminogen has good bonding affinity. In this the plasmin binds to the TPA of plasminogen.

This binding helps the conversion. They are subdivided into two categories which are as follows:

1. Fibrin-specific agents: These agents mostly need the presence of fibrin for the conversion, but on a minimal scale can do so in the absence of fibrin too. e.g. alteplase (tPA), tenecteplase
2. Non-fibrin-specific agents: These do not need fibrin presence for conversion, which is why they can do this systemically. e.g., streptokinase

Drugs:

Streptokinase: Streptokinase can be helpful in the management and treatment of acute ST-segment myocardial infarction, deep vein thrombosis, pulmonary embolism, arterial thrombosis or embolism, and arteriovenous cannula occlusion. It is in the thrombolytic class of treatment.

Mechanism of action: Streptokinase is a polypeptide derivative extracted from beta-haemolytic streptococci of group C bacteria. Streptokinase forms a complex with plasmin resulting in a cascade that leads to the breakdown of fibrin clots.

Uses:

- Used immediately after the symptom of heart attack and pulmonary embolism.
- Works by soften the harmful blood clots in the blood vessels.

- It restores the blood flow to the affected tissue.
- Preventing tissue death and improving outcomes.

Side effects:

- Most Common one is Bleeding.
- Allergic Reactions like Fever, shivering, difficulty in breathing, asthma, rash, hives, itching, flushing, nausea and headache.
- Respiratory like Respiratory depression.

Adverse effects:

Allergic reactions, bleeding, fever, shivering, rash, anaphylaxis, bradycardia, hypotension, vomiting, headache, nausea, hypoxia, bronchial asthma, respiratory disorders, sleep apnoea.

Contraindications:

The thrombolytic nature of streptokinase shows its negative effect in patients with active internal bleeding, as it can worsen bleeding in some patients. It is also not suggestable for patients with severe uncontrolled hypertension, intracranial neoplasms, surgery within two months, recent stroke, and intraspinal surgery.

Drug Interactions:

Need of Anticoagulants and Antiplatelet Agents: Streptase, Streptokinase, only or in mix with antiplatelet agents and anticoagulants, may cause bleeding complications.

Aim: The Aim of the present study is to evaluate the cardioprotective effect using various medical plants.

Objectives:

1. Objective of the study is to analyse the cardioprotective activity using various medicinal plant extracts.
2. From the different types of cardiac disease, the main one is myocardial infraction so the objective for this study is to determine a particular plant which is showing cardioprotective activity for myocardial infraction.
3. New therapies are needed to treat myocardial infraction because current treatment has only a limited impact on survival and annual cost.
4. To identify the particular herbal/medicinal plant that can produce cardioprotective effect.
5. To identify the exact pharmacological activity of various medicinal plant extracts and to select the perfect one that can produce (or) treat the disease.

Review literature

- **Mohammad HMF et al., (2020)** studied the cardioprotective effects in clinical studies. They investigated whether clopidogrel and prasugrel could protect against isoproterenol-induced acute MI (A-MI) under hypercholesterolemic conditions in rats. Dietary hypercholesterolemic rats are subjected to acute doses of isoproterenol. Serum lipids, inflammatory markers, aorticendothelin1 and endothelial nitric oxide synthase and immune-expression of BCL2 were determined. Prasugrel and clopidogrel protected against A-MI via anti-aggregatory and anti-inflammatory property. The results are added to the value of these drugs in correcting the cardiovascular dysfunction in patients vulnerable to A-MI after verification by appropriate human studies.

- **Sugiyama A et al., (2020)** determine the canstatin inhibits isoproterenol (ISO)-induced dephosphorylation of nuclear factor of activated T-cells (NFAT)c4, which plays an important role in cardiac hypertrophy, in differentiated H9c2 cardio-myoblasts. They investigated whether invivo canstatin administration prevents ISO-induced cardiac hypertrophy through the inhibition of NFATc4 pathway. Rats were subcutaneously administered with ISO (5 mg/kg) or saline (Cont.) for

7 days. Simultaneously, recombinant mouse canstatin (20 μg/kg) was administered intraperitoneally . Canstatin significantly constrain ISO-induced increase of LVW, left ventricular posterior wall thickness at end-diastole and diameter of cardiomyocytes. Canstatin significantly inhibited ISO-induced stimulation of calcineurin, nuclear translocation of NFATc4, enlarge mRNA expression of β-myosin heavy chain and α-skeletal actin, and intracellular Ca2+ rise in NRCMs. In summary, they for the first time establish the canstatin administration suppressesISO-induced cardiac hypertrophy possibly through the blockade of calcineurin/NFATc4 pathwayin rats.

- **Ghazouani L et al., (2019)** studied the effect of pretreatment and co-treatment with a newly synthesized coumarin hydrazone, (E)-4-hydroxy-N'-(1-(3-oxo-3H-benzo[f]chromen-2-yl) ethylidene) benzo-hydrazide (hereinafter EK6), against isoproterenol-induced myocardialinfarction in rats. The treatment with EK6 or Acenocoumarol a r e studied but changes in biochemistry, cardiac biomarkers, electrocardiography and histopathology etc. Animals are commonly divided into 4 groups: vehicle control (C), isoproterenol + Sintrom (ISO + Sin), isoproterenol + EK6 (ISO + EK6), and isoproterenol (ISO). Myocardial infarction was activated by subcutaneous ISO administration at a dose of 85 mg·kg-1·day-1 with a drug-free interval of 24 h on days 6 and 7. Treatment with ISO led to significant ascent ($p < 0.05$) in serum levels of cardiac injury biomarkers. Pretreatment and co-treatment with newly synthesized coumarin hydrazone reestablish all the ISO-induced biochemical, lipid, cardiac, and histopathological changes in rats with myocardial infarction.

- **Ouyang B et al., (2019)** demonstrate the cardioprotective efficacy by evaluating the biochemical and histopathological changes in isoproterenol (ISO) induced myocardial infarction (MI) rat model. ISO-induced group expose the increased infarct size and cardiac/inflammatory/apoptotic markers. After all the pre-treatment with LU (28 days) rather reduced ($p < 0.01$) the infarct size (14%), lipid peroxidation product (MDA;42%), cardiac markers [(lactate dehydrogenase (LDH) and creatine kinase-MB (CK-MB), cardiac troponin T (cTn T)], inflammatory markers [IL-1β, IL-6, tumor necrosis factor alpha (TNF-α), nuclear factor kappa B p65 subunit (NF-κB p65)] and apoptotic markers (caspase-3 and -9). Also, LU significantly improved ($p < 0.01$) the antioxidants [catalase (CAT), superoxide dismutase (SOD)] as well as markedly upregulated ($p < 0.01$) the protein expression of HO-1 and Nrf2. Moreover, LU considerably reversed all the histopathological changes and thus exhibits its cardioprotective activity. Conclusion: LU exhibits potent cardioprotective activity against ISO-induced cardiotoxicity and might be recommended with standard cardioprotective agents for treating various MI-related complications.

- **Ardjmand A et al., (2019)** consider the cardioprotective effects of cerebrolysin (CLY) onthe severity and inflammatory factors in male rats using isoproterenol (ISO)-induced MI model. Myocardial infarction in rats was induced by infuse ISO (100 mg/kg) subcutaneously (s.c) on the first 2 days, and the CLY (5 ml/kg) was injected intraperitoneally (i.p.) 7 days for post-treatment. On the 3rd day the creatine phosphokinase (CK-MB) and cardiac troponin I (cTnI) elevation in serum and on the 10th day the TNF-α and IL6 levels in serum and heart tissue are consistent by enzyme-linked immunosorbent assay (ELISA). Finally, the heart of each rat was dissected out and disordered for the histopathological examination. Their findings showed only a mild reduction in inflammatory cell infiltration and expansion of edema following treatment in the CLY + ISO group and then also the CLY induced vascular proliferation in the heart tissue. They reported that the severity of pathological changes induced by ISO in MI (e.g., inflammation and edema) can be confined by CLY treatment.

Medicinal plants

1.Rubia cordifolia (Common madder, Indian madder):

The family of Rubia cordifolia is Rubiaceae with genes and species named Rubia cordifolia. It contains chemical constituents which include anthraquinones, alkaloids, steroids, flavones, flavonoids, phenols, saponins, tannins, proteins, and glycosides. This plant has few pharmacological activities like wound healing, antibacterial, antioxidant,

anticancer, anti-inflammatory and analgesic, hepatoprotective, anti-platelet activating factor, anti-acne activity. The extraction of powdered samples of root, stem and leaf of Rubia cordifolia were extracted using petroleum ether, methanol, ethyl acetate and finally with distilled water for 18hrs.The extract was condensed and screened for the preliminary analysis, to check for the presence of bioactive compounds. For the experiment purpose, the animals were reported as adult male wistar rats with weight of 120- 150gms. The animals are divided into 6 groups and maintained at standard libitum. The experimental protocol as follows the group 1 (control rats) has received distilled water (1ml/kg bwt), orally for 10 days. The group 2 rats were injected intraperitoneally with a single drug concentration of CP (100 mg/kg bwt, i.p.) dissolved in saline, on the initial day of the experiment period. Group 3 rats received CP as in Group 2, quickly after that supplement with RC extract (100mg/kg bwt, po) through gavages for 10days continuously. Group 4 rats has received CP as in Group 2, quickly after that supplement with RC extract (200 mg/kg bwt, po) through gavages for 10 days continuously. Group 5 rats received CP as in Group 2, quickly after that supplement with RC extract (400 mg/kg bwt, po) through gavages for 10 days continuously. Group 6 rats received CP as in Group 2, quickly after that supplement with RC extract (100 mg/kg bwt, po) through gavages for 10 days continuously. Here we concluded that the test sample is more effective when compared to the standard sample.

2. Mangifera indica (Mango, Aam):

The family of Mangifera indica is Anacardiaceae with genes and species named with Mangifera indica. Carbohydrates, proteins, amino acids, micronutrients and phytochemicals are the chemical constituents present. It also contains structural carbohydrates such as pectin and cellulose. It also contains amino acids which are as follows lysine, leucine, cysteine, valine, arginine, phenylalanine and methionine. They increase the lipid composition during ripening, particularly the omega-3 and omega-6 fatty acids. The most important pigments of mango fruit include chlorophylls (a & b) and carotenoids. The most important organic acids include malic acid and citric acids. This plant has few pharmacological activities like strong antioxidant, anti-lipid peroxidation, immunomodulation, cardiotonic, hypotensive, wound healing, anti-degenerative and antidiabetic activities. The leaves of Mangifera indica were dried under shade and powdered. The powdered material of Mangifera indica was defatted with petroleum ether (60-80°C). Defatted powdered material was extracted by Soxhlet apparatus with required amount of ethanol for 21-h and concentrated under reduced pressure to yield semisolid mass. Desired amount of powdered sample was dissolved in purified water and the solution can be used for the experiment. For the experiment purpose, the animals were reported as adult male wistar rats with the weight of 200- 250 gms. The animals were divided into four groups and maintained at standard libitum. The experimental protocol as follow as the Group 1 served as normal control and received purified water p.0., for 12 days. Group 2 served as toxic control, in which the animals received a total cumulative dose of 15 mg/kg, i.p. of DOX for 2 weeks in six divided to induce cardiotoxicity. Group 3 received MILE (100 mg/kg body weight, p.o.) for 21 days, suspended in purified water. Group 4 animals received the same treatment as Group 2 along with MILE suspended in purified water (100mg/kg body weight, p.o) for 21 days. Here we concluded that the test sample has more effect when compared to the standard sample.

3. Arachis hypogea (Ground nut, Goober, Pindar & Monkey nut):

The family of Arachis hypogea is Fabaceae with genes and species named with Arachis hypogea. It contains chemical constituents like flavonoids, phenolic acids, phytosterols, alkaloids, and stilbenes. This plant has few pharmacological activities like antimicrobial, antifungal, antiviral, antioxidant, anticancer, antihypertensive, neuroprotective, antimutagenic, antiproliferative, anti-inflammatory. 85 C using menthol as solvent, that may be the best solvent for the extraction of resveratrol from peanut skins . The solvent was filtered and filtrate was allowed to evaporate for one day to obtain dried extract. For the experiment purpose, the animals were reported as male & female wistar rats with the weight of 120-150 grams. The animals were divided into five groups and maintained at standard libitum. The experimental protocol as followed as the Group 1 has served as normal control and received normal diet and water. Group 2 animals were fed with normal diet and isoproterenol injected subcutaneously on 16^{th} & 17^{th} day. Group 3 animals were treated with methanolic extract of arachis hypogea (5 mg/kg of body weight) extract with normal diet. Group 4 animals were treated with methanolic extract of arachis hypogea (5mg/kg of body weight) extract with normal diet. At 16^{th}&17^{th} day isoproterenol was injected subcutaneously. Group 5 animals were treated with Resvita (grape seed resveratrol) along with normal diet for 15 days. At 16^{th}&17^{th} day myocardial infarction was induced by subcutaneous injection of isoproterenol. Here we concluded that the test sample has more effect than the standard sample.

4. Cinnamomum tamala (Indian bark, Indian cassia, Tejpat):

The family of Cinnamomum tamala is Lauraceae with gene and species named as Cinnamomum tamala. It contains chemical constituents like alpha-pinene, camphene, myrcene, limonene, eugenol, p- cymene, methyl eugenol, eugenol acetate, and methyl ether of eugenol. This plants has few pharmacological activities like antibacterial, antifungal, anti-diabetic, hypolipidemic, carminative, sedative, antidepressant and effective for burns and cytoprotective. The coarse powdered material was subjected to sequential soxhlet extraction and then the solvents used are petroleum ether, chloroform and ethanol. The dried powder was defatted by macerating the powder material for 7 days in petroleum ether with continuous stirring. Then the powdered material was subjected to soxhlet extraction with chloroform and ethanol respectively. Finally, the resultant marc of Cinnamomum tamala was subjected to aqueous extraction. The collected extract of Cinnamomum tamala were concentrated by using rotary vacuum evaporator and were air dried at room temperature , weighed and percentage yield was calculated. For the experiment purpose, the animals were reported as adult wistar rats with weight of 150 – 200 grams. The animals were divided into eight groups and maintained at standard libitum. Then the experimental protocol as followed as the Group 1 control rats received 1% Na CMC 2.5 ml/kg/day p.o for a period of 30 days. Group 2 these rats received doxorubicin (2.5 mg/kg body weight, i.p., in six divided doses alternatively for two weeks to a total cumulative dose of 15 mg /kg). Group 3 rats were treated orally for a period of 30 days with EECT (dose-200mg/kg body weight). Group 4 rats were treated orally for a period of 30 days with EECT (dose-400mg/kg body weight). Group 5 rats were treated orally with 200mg/kg EECT for first 15 days followed by doxorubicin treatment (2.5 mg/kg body weight, i.p.) in six divided doses for next 15 days. Group 6 rats were treated orally with 400mg/kg EECT for first 15 days followed by doxorubicin treatment (2.5 mg/kg bodyweight, i.p.) in six divided doses for next 15 days. Group 7 rats received doxorubicin (2.5 mg/kg bodyweight, i.p.) in six divided doses for first 15 days, followed by oral treatment with 200mg/kg EECT for next 15 days. Group 8 rats received doxorubicin (2.5 mg/kg bodyweight, i.p.) in six divided doses for first 15 days, followed by oral treatment with 200 mg/kg EECT for next 15 days. Here we concluded that the test sample is more effective when compared to the standard sample.

5. Lepidium sativum (Garden cress):

The family of Lepidium sativum is Brassicaceae with genes and species named as Lepidium sativum. It also contains chemical constituents like cardiac glycosides, alkaloids, phenolic, flavonoids, cardiotonic glycosides, coumarins, glucosinolates, carbohydrates, proteins, and amino acids, mucilage, resins, saponins, sterols, tannins, volatile oils, triterpene, sinapic acid and uric acid. This plant has few pharmacological activities like antimicrobial, antidiabetic, antioxidant, anticancer, reproductive, gastrointestinal, respiratory, anti-inflammatory, analgesic, antipyretic, cardiovascular, hypolipidemic, diuretic, central nervous, fracture healing, and protective effects. The collected seeds were dried and ground to powder. The extract of Lepidium seed powder was made with a required quantity of distilled water by a dose of 550 mg/kg Bodyweight. The extract was administered through the oral route with the help of a gastric gavage. For the experiment purpose, the animals were reported as male albino sprague-dawley rats with weight of 120- 150 grams. Then the animals were divided into three groups and maintained at standard libitum. Then the experimental protocol as followed as the Group 1 animals in the control group received only saline daily for 8 days by oral gavages. Group 2 animals in FU- treated group received saline orally for 8 days, then were given a single dose of 5-FU (150 mg/kg B.W\I.P injection on the 5^{th} day). Group 3 animals in LS-treated group received a suspension of LS seed powder (550 mg\kg\day0 orally for 8 days and were injected with a single dose of 5-FU (150 mg\kg B. W\I.P) on the 5^{th} day. Here we concluded that the standard sample is less effective when compared to test sample.

6. Tamarindus indica (Tamarind):

The family of Tamarindus indica is Fabaceae with genes and species named as Tamarindus indica. It also contain chemical constituents like beta-amyrin, compesterol, beta-sitosterol, and seven hydrocarbons. The aerial parts of this plant have demonstrated the presence of tartaric acid, acetic acid, succinic acid, gum, pectin, sugar, tannins, alkaloids, flavonoids, sesquiterpenes, and glycosides. This plant has few pharmacological activities like hypolipidemic, weight reducing, antimicrobial, hepatoprotective, anthelmintic, antioxidant, analgesic, & anti-inflammatory etc. The coarse powdered seeds of the Tamarindus indica and the small pieces of the fruit pulp were

then subjected to the extraction process by cold maceration technique separately. After four days the aqueous and alcoholic macerate are decanted and pressed to extract the solvent which was then kept in separate beakers that were covered using cling film to avoid contamination aqueous layer which was then evaporated using steam over a water bath in large petri dishes. The alcoholic macerate was concentrated by distilling out the alcohol and the extract was dried using steam in petri dishes. For the experiment purpose, the animals were reported as adult albino wistar rats with weight of 180- 200 grams. Then the animals were divided into nine groups and maintained at standard libitum. Then the experimental protocol as followed as the Group 1 has normal (Saline 1 ml/rat per oral [p.o]). Group 2 negative control-IPRT (100 mg/kg) 29th & 30th day. Group 3 positive control- vitamin E (100 mg/kg p.o). Group 4 alcoholic seed extract (ALSE) (250 mg/kg p.o). Group 5 ALSE (500 mg/kg p.o). Group 6 aqueous seed extract (AQSE) (250 mg/kg p.o). Group 7 AQSE (500 mg/kg p.0). Group 8 alcoholic fruit extract (ALFE) (250 mg/kg p.o). Group 9 ALEF (500 mg/kg p.o). Here we conclude that the test sample has more effective when compared to the standard sample.

7. Tinospora cordifolia (Guduchi):

The family of Tinospora cordifolia is Menispermaceae with genes and species named as Tinospora cordifolia. It also contains chemical constituents like alkaloids, diterpenoid lactones, glycosides, steroids, sesquiterpenoid, phenolics, aliphatic compounds and polysaccharides. This plant has few pharmacological activities like anti-diabetic, anti-spasmodic, anti-malarial, anti-inflammatory, anti-arthritic, anti-oxidant, anti-allergic, anti-stress, anti-leprotic, hepatoprotective, immunomodulatory, anti-neoplastic activities. Collected leaf material washed under running tap water to remove microbes and dust and then air dried under shade at room temperature for 15 days. The leaves of Tinospora cordifolia was crushed well into fine powder, packed into airtight polythene bags for further use and stored at room temperature. The aqueous extract is prepared by soaking 5 gm of dried powder in 100 ml of distilled water and shaken well. The solution left at room temperature for 72 hours and then the extract was filtered with the help of Whatman No.1 filter paper. The filtrate was kept at low temperature (4 °C) for the experiment use. For the experiment purpose, the animals were reported as male wistar rats with weight of 180 ± 10 g and age of 3months. Then the animals were divided into six groups and maintained at standard libitum. Then the experimental protocol as followed as the Group 1 has received normal saline orally for 14 days. Group 2 has received cisplatin (7.5 mg/kg bodyweight, i.p.) injection on one day. Group 3 has received aqueous leaf extract of Tinospora cordifolia (400 mg/kg body weight) was orally administered for 14 days stating from the day one. Group 4 has received vitamin C (100 mg/kg body weight) was orally administered for 14 days starting from the day one. Group 5 has received cisplatin (7.5 mg/kg, i.p.) injection on day one + aqueous leaf extract of Tinospora cordifolia (400 mg/kg body weight) was orally administered for 14 days starting from the day one. Group 6 has received cisplatin (7.5 mg/kg, i.p.) injection on day one + vitamin C (100 mg/kg bodyweight) was orally administered for 14 days starting from the day one. Here we concluded that the test sample has more potency then the standard sample.

8. Hibiscus sabdariffa (Roselle, Jamica sorrel, Indian sorrel, Red sorrel):

The family of Hibiscus sabdariffa is Malvaceae with genes and species named as Hibiscus sabdariffa. It also contain chemical constituents like protein (3.3g / 100g), fat (0.3g / 100g), carbohydrate (9.2g / 100g), minerals (phosphorus (214mg/100g), iron (4.8 mg/100g), thiamine (0.45 mg/100g), riboflavin (0.45 mg/100g), ascorbic acid (54 mg/100g).This plant few pharmacological activities like antibacterial, antifungal, antiviral, anti-cancer, apoptotic, immunological, antioxidant, hypolipidemic, antidiabetic, smooth muscle relaxant, gastrointestinal anti-inflammatory, analgesic, antipyretic, protective effects, wound healing, and wide range of cardiovascular and CNS effects. The petals of Hibiscus sabdariffa are used as plant material in the present study. The petals of Hibiscus sabdariffa were cut, cleaned, washed thoroughly under running tap water, drained and oven-dried at 55 °C for 12 hrs. The samples were packed in polyethylene bags and stored at 4 °C for laboratory analysis and the reagents, solvents and chemical compounds used for analysis met the quality criteria in accordance with international standards. For the experiment purpose, the animals were reported as wistar rats with weight of 185 ± 15 g. Then the animals were divided into four groups and maintained at standard libitum. Then the experimental protocol as followed as the Group 1 has received 0.5 ml of 0.9% NaCl. Group 2 has received 0.5 ml of 0.9% NaCl + 15 mg/kg bodyweight (BW) of Doxorubicin. Group 3 has received 100 mg/kg bodyweight of AHES + 15 mg/kg body weight of Doxorubicin. Group 4 has received 200 mg/kg body weight of AHES + 15 mg/kg body weight of Doxorubicin. Here we concluded that the test sample has more potency then the standard sample.

9. Momordica charantia (Bitter -melon & Ampalaya):

The family of Momordica charantia is Cucurbitaceae with genes and species named as Momordica charantia. It also contains chemical constituents like triterpenoids, saponins, polypeptides, flavonoids, alkaloids, sterols. This plant has few pharmacological activities like antihyperglycemic, antibacterial, antiviral, antitumor, immunomodulation, antioxidant, antidiabetic, anthelmintic, antimutagenic, antiulcer, antilipolytic, antifertility, hepatoprotective, anticancer, and anti-inflammatory activities. The fresh leaf and stem of Momordica charantia were air dried and ground into fine powder using an electric blender. The powdered extract was apply to the extraction using 50% ethanol and intermittent maceration. Dried ethanol extracts were collected after removing the solvent by evaporation under low pressure using Rotary evaporator. The extract was stored in an air-tight container and kept in the refrigerator at 4 °C for further use. For the experiment purpose, the animals were reported as adult wistar rats weighing between 160-180 grams. Then the animals were divided into nine groups of 8 rats each and maintained at standard libitum. Then the experimental protocol as followed as the Group 1 has (control) received 1 ml distilled water orally daily for a period of sixty (60) days. Group 2 has received freshly prepared cholesterol powder dissolved in coconut oil at a dose of 30 mg/kg body weight orally for a period of sixty (60) days. Group 3 has (standard) received the standard drug Atorvastatin (Lipitor) at a dose of 100 mg/kg body weight orally for a period of sixty (60) days and freshly prepared cholesterol at a dose of 30mg/kg body weight. Group 4 has received 250 mg/kg orally, test drug for a period of sixty (60) days and freshly prepared cholesterol at a dose of 30 mg/kg body weight. Group 5 has received 250 mg/kg orally, test drug for sixty (60) days and freshly prepared cholesterol at a dose of 30 mg/kg b.w. Group 6 has been treated with 250 mg/kg orally, test drug for a period of 60 days. Group 7 has received 500 mg/kg orally, test drug only for a period of 60 days. Group 8 has received 1 ml of coconut oil only for 60 days. Group 9 has received 100 mg/kg of Atorvastatin (Lipitor) only for 60 days. Here we concluded that the test sample have more effect then the standard sample.

10. Syzygium aromaticum (Clove):

The family of Syzygium aromaticum is Myritacaea with genes and species named as Syzygium aromaticum. It also contain chemical constituents like sesquiterpenes, monoterpenes, hydrocarbon and phenolic compounds. This plant has few pharmacological activities like antimicrobial, antioxidant, anti-inflammatory, analgesic, anticancer, and anaesthetic effects. The whole powdered substance of Syzygium aromaticum was weighed and packed in Soxhlet extractor. The solvent used for extraction was the mixture of methanol and distilled water in the ratio of 50:50. Extraction was continued at the temperature of 50 °C till clear solvent was observed in siphon tube. Extract was concentrated in water bath at 40 °C. Concentrated extract was dried at 40 °C in hot air oven. Dried powdered material of extract was packed in an air tight container. For the experiment purpose, the animals were reported as male wistar rats weighing between 150-200 grams. Then the animals were divided into six groups each of 6 animals and maintained at standard libitum. Then the experimental protocol as followed as the Group 1 has received Control 0.9 % Normal Saline. Group 2 has received Negative Control : Isoproterenol 5.25 and 8.5 mg/kg s.c. Group 3 has received Standard (Propranolol 10 mg/kg). Group 4 has received Extract of S.aromaticum 250 mg/kg s.c. Group 5 has received Extract of S.aromaticum 500 mg/kg s.c. Group 6 has received Extract of S.aromaticum 750 mg/kg s.c. Here we concluded that the test sample has more effect when compared to the standard sample.

Conclusion

The present review explains the importance of medicinal plants in preventing and eliminating the cardiovascular diseases and makes an attempt to compile some of the cardio protective plants. Medicinal plants and their healthy food can help in lowering the risk of cardiovascular diseases. Secondary metabolites such as carotenoids, cardiac glycosides, alkaloids, flavonoids, polyphenolic compounds, saponins, terpenoids [triterpenes], fatty acids which are present used in medicinal plants were treated as the responsible agents for potent cardio-protective activity. Thus the review of the disparate herbs which could be effective and reduce the mortality of Cardio Vascular Diseases (CVDs). Cardiac diseases are the one of the main cause of death worldwide. This review is to justify that the Siddha system of medicine with its medicinal plants compile a exact outline of some

Cardioprotective plants. The current review reported that therapeutic and prophylactic potential of plant phytoconstituents for the management of cardiovascular disorders have noted under several ways in chemoprevention, although exact molecular mechanisms are still unclear. It is explain in the review that phytochemicals possess versatile cardioprotective functions. It could not be possible to include all the studies describing cardioprotective effect of medicinal plants or herbal agents in this review because of defined access to research articles and our search strategy. Hence to develop more effective and safe agents from natural herbs is a promising way in excessive and treating cardiovascular abnormalities. The search for new pharmacological-active compounds for drug development is an important issue, as the trend toward using standardized plant extracts of high quality, safety and efficacy will continue. Therefore, all efforts have to be tar gated to reveal the chemical-pharmacological profiles of extracts and fixed combinations and to rationalize their therapeutic application

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