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Cardiac Troponin Role in Coronary Artery Disease: A Comprehensive Review

Susheela Sharma

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I. INTRODUCTION:

Lipid accumulation and hypertension were thought to be the main risk factors for coronary artery disease (CAD) decades ago, and it was believed that CAD could be successfully cured by antihypertensive and cholesterol-lowering medications (Hansson, 2005) [(1)].

When atherosclerosis develops in the coronary arteries, it can sometimes be asymptomatic [(2)]. This is a main sign of coronary artery disease (CAD). Conditions like silent myocardial ischemia, acute coronary syndrome (ACS), and stable angina are all included in coronary heart disease (CHD), often referred to as ischemic heart disease (IHD) [(3)]. An insufficient flow of blood and oxygen to the myocardium is a main sign of CAD [(4)]. The disorder is brought on by coronary artery blockage, which causes an imbalance between oxygen supply and demand [(5)]. Plaques that block blood flow in the coronary artery lumen are a common feature of CAD [(6)].

The development of numerous new biomarkers linked to cardiovascular risks, such as lipoprotein-associated proteins, C-reactive protein (CRP), troponin, N-terminal prohormone BNP (NT-proBNP), B-type natriuretic peptide (BNP), etc. P For the diagnosis and risk assessment of patients suspected of having acute coronary syndrome (ACS), troponin has been employed as a cardiac biomarker. High sensitivity CRP (hs CRP) blood levels have been used to evaluate heart attack, stroke, and CVD risk. TMAO, fibrinogen, cystatin C, hospholipase A2, and coronary heart disease risks have all been predicted using circulating levels of MPO[(]).Numerous aspects of health or illness can be indicated by biomarkers, such as the quantity or form of exposure to environmental factors, hereditary predisposition, genetic reactions to exposures, markers of subclinical or clinical disease, or signs of therapeutic response( ).

Patients with systolic hypertension, angina pectoris, atherosclerosis, cerebral insufficiency, venous insufficiency, arrhythmia, and congestive heart failure have all been treated with medicinal herbs.

According to Mashour et al. (1998), herbal medications can be grouped according to the main problems they address, even though they often have various cardiovascular effects that overlap. Strong cardioactive glycosides found in several plants have beneficial inotropic effects on the heart. In this review ,we introduced CVDs and their hazard components. At that point we overview the use of herbs for disease treatment in general and CVDs in particular. Advance information on the ethnopharmacology helpful possibilities and therapeutic properties against CVDs of widely utilized plants, specifically ginseng, guggul, hawthorn ashwagandha etc. are accumulated and reviewed.

Hawthorn, (Crataegusspp) is particularly well-known for its strong procyanidins and flavonoids. These phytochemicals have shown significant impacts in the management of cardiovascular health. Patients with coronary artery disease and angina are also frequently treated with it. In traditional Chinese medicine, Salvia miltiorrhiza root is used as a cooling, sedative, and circulatory stimulant. Because S. miltiorrhiza has been demonstrated to widen coronary arteries at all doses( ).

In clinical and experimental research, several herbal remedies or their constituents—including ginseng, garlic,hawthorn, Ginkgo biloba, turmeric,green tea, arjuna ,guggul, and ginsenoside—have been used to treat cardiovascular diseases (CVDs). Similar bioactive compounds of different components, such as flavones, triterpenic acids, and phenol carboxylic acids, are present in several herbal remedies and have indicated beneficial benefits against CVDs( ).
The different types of cardiovascular disease includes the following:

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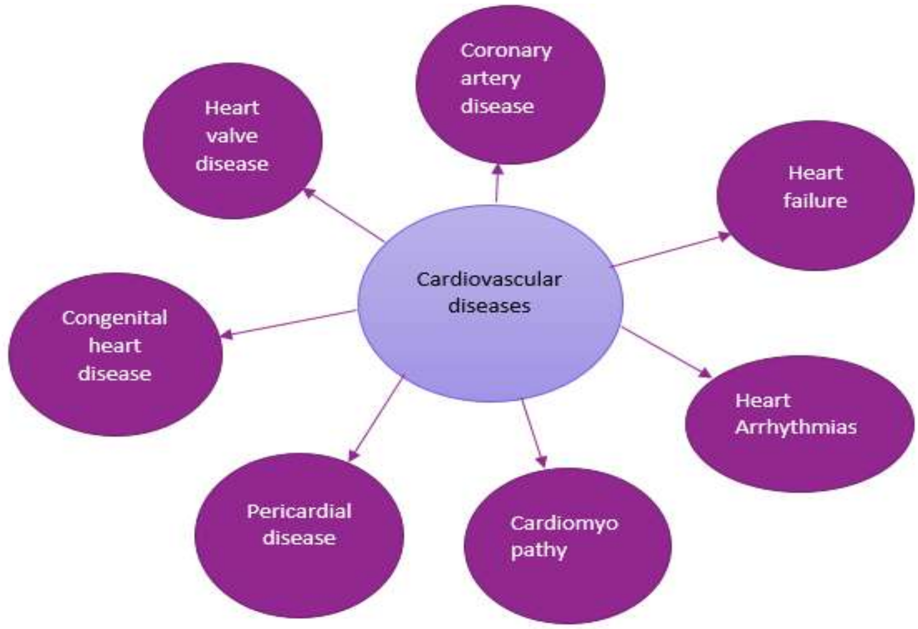

Fig. 1.1 Different types of coronary artery disease

II. REVIEW OF LITERATURE:

Coronary disease artery is the main leading cause of death worldwide [7]. Coronary artery disease is a condition in which the is an insufficient supply of blood and oxygen to the myocardium of the heart[8]. A buildup of fats, cholesterol and other substances in and on the artery walls, then a condition called atherosclerosis, usually causes coronary artery disease [9]. The buildup called plaque makes the arteries narrow [10] . Symptoms are from the lack of blood flow to the heart. They may include chest pain and shortness of breath[11]. A complete blockage of blood flow causes a heart attack[12].The main cause of coronary artery diseases is angina pectoris[13].Is chest pain of discomfort that occurs when a portion of myocardium (heart mucles)of your heart does not get enough blood and oxygen[14]. Not enough blood supply is know is ischemia[15]. Angina can be a symptom of coronary arterydisease this is when the arteries that carry blood to your heart becomes narrowed and blocked. This can happen because ofHardening of arteries (artherosclerosis),A blood clot, Plaque in an arterty that can rupture (unstable plaque) [17].Poor blood flow through a narrowed heart valve,Lessened pumping of the heart muscle,Coronary artery spasm. There are two types of angina pectoris [18].

1. Stable
2. Unstable

Stable angina- It is also known as classical angina (common form)[19]. This occurs when a patient is involved in some kind of physical exercise,eating,emotions[20]. During physical activity, the heart becomes overactivated and often leading to pain in the chest[21].

Unstable angina – It is also known as variant /prinzmetal /Vasospastic angina(uncommon form)[22].This occurs anytime, when a person is at rest or during sleep[23]. Variant angina is less common then the classical angina but get worsen over time and leads to heart attack[24].

Major risk factors :

- Age(>55years old for men ,>then 65 for women).
- Cigarette smoking[25].
- Diabetes mellitus(DM).
- Family history of premature cardiovascular disease(men<55year,female<65years old).
- Hypertension.
- Kidney disease(microalbuminuria or GFR<60ml/min). Obesity (BMI>30kg/m2).
- Physical inactivity[26].
- High cholesterol.
- Unhealthy diet.
- Stress[27].

Condition that exacerbate or provoke angina:

- Excessive thyroid replacement.
- Medications[28].

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- Polycythemia which thickness the blood causing it to slow its flow through the heart muscle[29].

Other medical problems:

- Hyperthyroidism
- Hypoxemia
- Profound anemia[30].

Other cardiac problems:

- Bradyarrhythmia
- Tachyarrhythmia
- Valvular heart disease[31]
- Hypertrophic cardiomyopathy.

A Major cause : Coronary artery disease

Coronary disease artery is the main leading cause of death worldwide .Coronary arteries supply oxygenated blood to the heart muscles[32] .

Coronary artery disease is a condition in which the is an insufficient supply of blood and oxygen to the myocardium of the heart[33].

A buildup of fat cholesterol and other substances in the artery walls, causing the formation of hard ,thick substances called cholesterol plaque[34].

The buildup called plaque makes the arteries narrow( ). Symptoms are from the lack of blood to heart. They may include chest pain and shortness of breath[35]

When coronary artery become narrowed by more then 50%to70%,they may no longer be able to meet the increased blood oxygen demand by the heart muscles[36].Lack of oxygen to the heart muscles causes chest pain (angina)[37].

Treatment of coronary artery disease may include medicines (allopathic and Ayurveda) and surgery[38] .By eating the nutrition rich diet, not smoking ,doing regular exercise ,low fat diet will reduce coronary heart disease and the conditions that can cause it[39].

Medicines for angina pectoris:

Antianginal drugs are those drugs that are used to prevent, abort or terminate the attacks of angina pectoris.

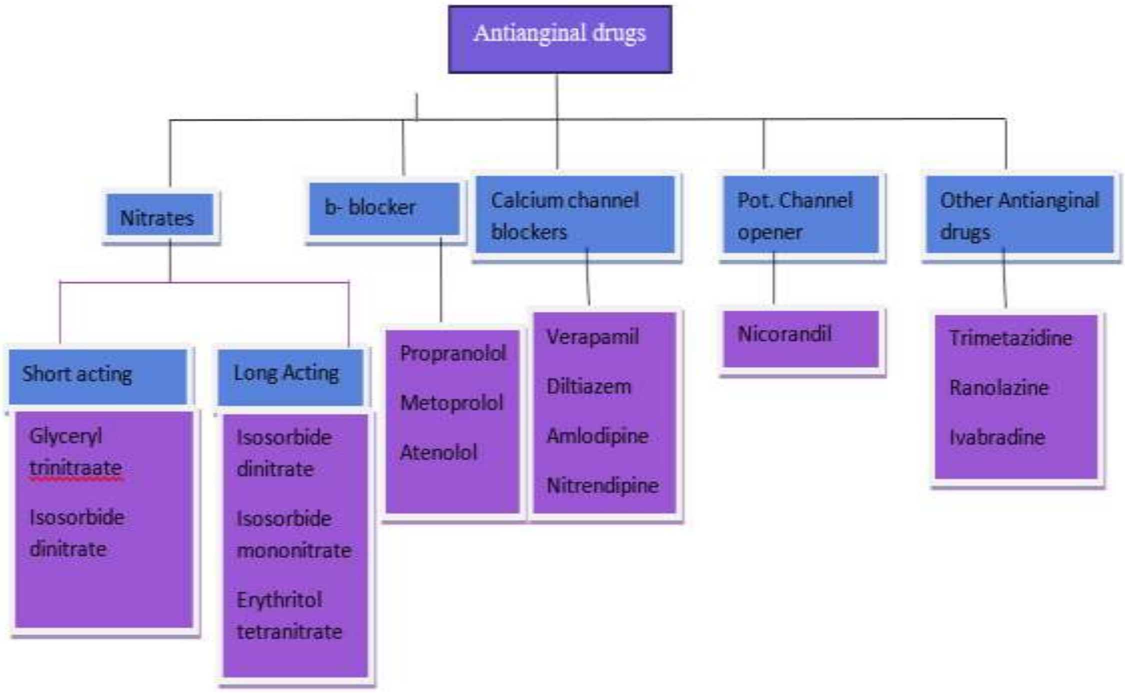

Fig 2.1 Classification of Antianginal drugs

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Table 1.1 Summary of antianginal drugs supported by NICE guidance.

Antianginal drugs	Therapeutic target	Mechanism of action	Prognostic benefits	Hemodynamic side effects	Notes
Beta-blockers	• Block b1-adrenergic channels	• Reduce HR,BP and contractibility • Prolonged diastolic filling time	• Proven prognostic benefit for patient with previous MI or HF.	Yes	Contraindicated in patient with reactive airways disease, severs bradycardia, hypotension and acute HF
Calcium channel blockers	• Block L-type Ca^{2+} channels	• Act as systemic and coronary vasodilators • Rate-limiting agents also reduce HR,BP, and contractility, and prolong diastole	• Previous prognostic benefit for patient with previous MI seen wirh rate-limiting calcium channel blocker.	Yes	Rate- limiting calcium channel blocker are contraindicated in patient with severe bradycardia, acute heart failure and impaired left ventricular function.
Nitrates	• No donor	• Act as systemic and coronary vasodilators.	• No proven prognostic benefits.	Yes	Not to be used with PDE inhibitors(eg.sildenafil)
Nicorandil	• No donor • K_{ATP} channel	• Act as systemic and coronary vasodilator.	• Proven prognostic benefit seen in patients with stable angina.	Yes	Metformin may reduce efficacy.
Ivabradine	• Block I_f channels	• Cause pure reduction in heart rate.	• Proven prognostic benefit seen in patients with heart failure.	No	Effective for patients with sinus rhythm and resting HR>70bpm. Contraindicated in patient with severe bradycardia, acute MI or HF.
Ranolazine	• Blocks late inward Na^{+} current	• Reduces diastolic wall tension and improves myocardial metabolic performance.	• No currently proven prognostic benefit.	No	Improve HbA1c May prolong QT-interval.

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III. BIOMARKERS OF CORONARY ARTERY DISEASE :

Biomarkers are measurable indicators of a biological process or disease.In the context of coronary artery disease,biomarkers can help diagnose,monitor, and predict disease progression[40].

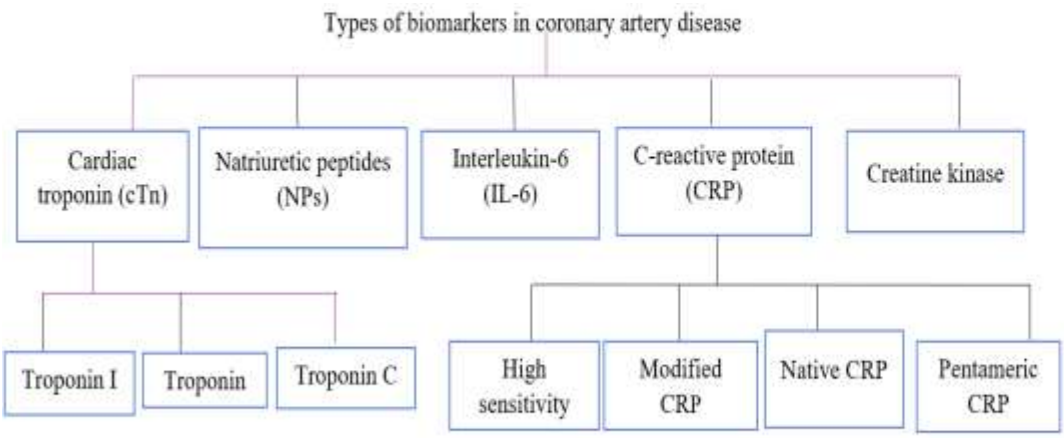

Fig 3.1 types of biomarker coronary artery disease

Cardiac troponin:

Troponin is a protein complex that happens both in myocardial cells and skeletal muscle cells, which interacts with tropomyosin[41]. It comprises of three subunits:

(a) Troponin I
(b) Troponin T
(c) Troponin C

Troponin is the essential basic component responsible for the relationship between an increment within the calcium particle concentration within the muscle cell cytosol and activation of the contractile framework of the cell . Each subunit has accurately defined functions. Troponin T is responsible for the interaction with tropomyosin; troponin I is an inhibitory component that inhibits the hydrolysis of adenosine triphosphate within the nonappearance of calcium particles and, hence, the interaction of actin and myosin in the diastolic stage; and troponin C is responsible for binding calcium particles . It is worth noticing that troponin C isn't a marker specific to the heart muscle, due to the similarity of the amino acid arrangement to troponin C present in skeletal muscles . Within the case of troponin T and troponin I, the difference within the amino acid arrangement between the atoms present in myocardial cells and skeletal muscle cells permits them to be utilized in clinical practice as particular markers for myocardial injury[41] .

Numerous biomarkers have been studied as potential predictors of coronary artery disease and its consequences, such as heart failure, myocardial infarction, and mortality. Nevertheless, international consensus recommendations only advocate two particular cardiac biomarkers for widespread clinical use, despite decades of research in this field: cardiac troponin (cTn) and natriuretic peptides (NP) [42].

One of the most important aspects of diagnosing patients with both acute and chronic cardiovascular illness is measuring the amount of cardiac troponin in systemic venous blood[43] . Troponin T and I are highly specific and sensitive cardiac troponin biomarker for coronary

muscle artery disease but troponin T is more high in acute coronary syndrome[44] .

Cardiac troponin (troponin T and I) are cardiac regulatory protiens those released into the blood stream when heart muscles are damaged[45].

Elevated troponin and CAD :

Cardiac troponin levels in the coronary artery disease are elevated due to different reasons[46]:

1. Cardiac ischemia: Decreased flow of blood to the heart muscles. This will prevents the heart from

getting enough oxygen and results in cardiac cell damage, leading to cTnrelease[47].
2. Myocardial infraction: this also results in increased level of ctn due to cardiac cell damage[48].
3. Cardiac strain: Increased heart workload can also lead to cardiac cell damage and elevated cTnlevel[49]

Table.3.1: Overview of cardiac biomarkers and their utility and potential for identification and prediction of coronary heart disease:

	Mechanism of action	Cardiac specific	Established role in clinical practice(CVD)	Merits	Demerits	Prognostic value in observational studies	Future needs
cTn	Released in reaction to myocardial harm and necrosis [50].	Yes	-Analyse myocardial harm -Included in the definition of myocardial violation –Utilized in hazard stratification devices in patient displaying with suspected or Confirmed CAD.	– Strong prognostic esteem – Recognize subclinical myocardial injury Exceedingly particular and delicate –Profoundly available	-Not particular for myocardial ischemic damage – Increased in additional cardiac disorders like renal failure – Frequently quick normalization after ACS occasions	Solid indicator of repetitive MACE and mortality in ACS patients – Predictor of CV death and CV occasions within the strengthening stage after MI – Solid indicator of passing and MACE in patients with CCS	– Forecast in essential prevention and in patients with suspected or established CHD – Utilized in machine learning calculations nearby CCTA discoveries and clinical biomarkers aimed at progressing chance stratification – Utilized in conjunction with clinical biomarkers to guide CCTA utilize in patients with Suspected coronary artery disease [51].

Herbal remedies for the treatment of coronary artery disease :

Herbal products have been utilized as routine medications for thousands of a long time, especially in Eastern countries. Thousands of clinical and test examinations have focused on the impacts and mechanisms-of-action of herbal medication in the treatment of coronary artery disease[52].

In traditional Chinese medication, the root of Salvia miltiorrhiza is utilized as a circulatory stimulant, narcotic, and cooling drug. S. miltiorrhiza may be valuable as an antianginal drug since it has been appeared to expand coronary arteries in all concentrations, comparable to P. notoginseng[53]. Garlic is one of the herbal medicines that have been inspected more closely by the scientific community. In later decades, research has focused on garlic's utilize in preventing atherosclerosis[54].

1. **Turmeric :**

Turmeric, the rhizome of Curcuma longa plant belonging to the ginger family Zingiberaceae, incorporates a history in Ayurveda and traditional Chinese medicine for treatment of chronic infections[55]. The use of natural bioactive compounds to avoid and improve health is broadly accepted. Traditional medication has continuously found in nature an important source of cures for various pathologies. This is the basis for the use of nutraceuticals as adjuvants in these conditions[56].

Curcuma aromaticaSalisb. Rhizome (CASR) is obtained from plants of the genus Curcuma of the Zingiberaceae family. These plants are basically found in the tropics and subtropics of South and Southeast Asia and to a great extent developed in Bengal, China, and Sri Lanka (Sun et al., 2017). In spite of the fact that over 100 species reportedly exist around the world, Curcuma aromaticaSalisb. is basically developed in China. It promotes blood circulation and reduces pain by regulating and relieving blood stasis[57]. It is also used clinically in the treatment of CHD and other conditions. CASR basically contains two categories of components:terpenoids (mainly sesquiterpenes) and curcuminoids . Modern pharmacological studies have appeared that botanical drugs that advance blood circulation and remove blood stasis have numerous pharmacological effects, such as the direction of lipid digestion system. Curcuma oil has been appeared to significantly control blood lipid records such as total cholesterol (TC) and triglyceride (TG) and inflammatory variables such as interleukin-2 in atherosclerotic rats, which also gives a clinical premise for CASR in the treatment of CHD caused by atherosclerosis, widening of coronary courses, change of microcirculation, and control of hemodynamics[58].

2. **Ginger:**

Ginger, the rhizome of Zingiber officinale Rosc., is a rich source of bioactive compounds that relate to the Zingiberaceae family and are developed basically in India, China, and Southeast Asia[59]. Ginger is broadly developed in the southeast locale of Asia Ginger (Zingiber officinale) is among the medicinal plants with advantageous wellbeing impacts that has been broadly utilized in pharmaceutical products and food. Its unrefined extract is cardioprotective due to its antihypertensive, antiplatelet, and cardiotonic effects[60]. Z. officinale is a broadly consumed spice universally, and the rhizome has been traditionally utilized medicinally for thousands of a long time , especially noticeable in Ayurvedic, Unani-Tibb and Traditional Chinese Medicine systems . Oleoresin from the Z. officinale rhizome contains various bioactive compounds, counting gingerols, shogaols, diarylheptanoids, phenylbutenoids, flavanoids, diterpenoids and sesquiterpenoids . These have been recommended to have safe modulating properties diminishing irritation, improve dyslipidaemia, and diminish blood pressure[61].

Ginger contains bioactive compounds like gingerol and shogaol that have potent anti-inflammatory effects. Chronic inflammation is one of the key contributors to the development and progression of coronary artery disease. By reducing inflammation, ginger may help in slowing the progression of CAD. The antioxidants in ginger can help neutralize harmful free radicals in the body. These free radicals contribute to oxidative stress, which damages blood vessels and promotes the buildup of plaque in the arteries. Ginger's antioxidant effects may help protect the vascular system from damage.

Ginger may also lower cholesterol levels, especially low-density lipoprotein (LDL) cholesterol, which could be a risk factor for CAD. Reducing LDL levels can help prevent the accumulation of fatty plaques within the arteries[62]

3. **Garlic :**

Garlic, especially in the form of aged garlic extricate (AGE), has been considered for its potential helpful impacts in managing coronary course disease (CAD). Research indicates that AGE may offer a few cardiovascular benefit[63].

• **Enhancement of Endothelial Function**: A study including 15 men with CAD illustrated that supplementation with AGE for two weeks essentially enhanced flow-mediated dilation (FMD), a degree of endothelial function. This enhancement was most notable in people with lower pattern FMD levels. However, markers of oxidative stress and systemic inflammation did not appear significant changes during the study period[64].

• **Reduction in Left Ventricular Myocardial Mass**: In a randomized controlled trial, 65 patients with diabetes mellitus consumed 2,400 mg of AGE every day for one year. The results demonstrated a drift toward a significant decrease in left ventricular myocardial mass within the AGE group, recommending potential benefits for cardiac structure in CAD patients[65].

• **Balance of Lipid Profiles:** A meta-analysis of randomized clinical trials surveyed the impact of garlic extract on lipid profiles in CAD patients. The discoveries revealed that garlic supplementation significantly diminished total cholesterol levels. In any case, it did not have a noteworthy impact on triglycerides, low-density lipoprotein cholesterol, or high-density lipoprotein cholesterol concentrations[66].

These studies recommend that garlic, particularly in its aged extract form, may contribute to improving certain cardiovascular parameters in CAD patients. Nonetheless, further investigate is fundamental to completely explain its viability and build up comprehensive treatment guidelines.

4. **Cinnamon** :

Cinnamon, a common spice, is an herbal medication and an anti-inflammatory dietary supplement suggested for the primary and secondary prevention of coronary artery disease(67). The genus Cinnamomum, too known as cinnamon , is a small tropical tree of the lauraceae family. Cinnamon bark contains phenolic compounds such as cinnamaldehyde and cinnamic acid with defensive properties which can decrease the chance of cardiovascular infections, cardiac ischemia and hypertrophy, and myocardial infarction. Moreover, cinnamon has antioxidant and anti-inflammatory properties and shows advantageous effects on the complications of diabetes, obesity, hypercholesterolemia, and hypertension which cause CVDs(68).

Mechanism of action :

- **Lipid Profile Improvement**: Studies recommend that cinnamon can diminish total cholesterol, low-density lipoprotein (LDL), and triglycerides whereas expanding high-density lipoprotein (HDL) levels(69).
- **Anti-Inflammatory Effects**: Chronic inflammation plays a key role in CAD. Cinnamon contains cinnamaldehyde and polyphenols, which diminish inflammatory markers such as C-reactive protein (CRP)(70).
- **Blood Weight Control**: Cinnamon has vasodilatory impacts which will help lower blood pressure, diminishing strain on coronary supply routes(71).
- **Antioxidant Properties**: Cinnamon is rich in polyphenols and flavonoids, which secure endothelial cells from oxidative harm, a major supporter to atherosclerosis(72).
- **Blood Sugar Control**: Insulin resistance may be a hazard factor for CAD, and cinnamon improves glucose digestion system by improving insulin sensitivity(73).

5. **Green tea:((Epigallocatechin Gallate - EGCG)**

Epigallocatechin gallate (EGCG), a prominent catechin found in green tea, has been examined for its potential therapeutic impacts in coronary artery disease (CAD). Research demonstrates that EGCG may offer cardiovascular benefits through several mechanisms(74).

- **Endothelial Function Change**:A double-blind, placebo-controlled, crossover study including 42 subjects with CAD illustrated that a single 300 mg dosage of EGCG altogether improved brachial artery flow-mediated dilation (FMD) from 7.1% ± 4.1% to 8.6% ± 4.7% ($P = 0.01$). However, this useful impact was not observed after two weeks of continued EGCG supplementation, recommending that the intense organization of EGCG can improve endothelial work in CAD patients(75).
- **Cardioprotection in Ischemia-Reperfusion Damage**: A meta-analysis of 25 preclinical animal studies, enveloping 443 animals, surveyed the efficacy of EGCG in myocardial ischemia-reperfusion injury (MIRI). The discoveries shown that EGCG essentially diminished myocardial infarct size (standardized mean distinction = −4.06; 95% confidence interval:−5.17, −2.94; $P < 0.01$) compared to controls. Moreover, EGCG moved forward cardiac function, diminished serum myocardial harm chemicals, and diminished oxidative stretch levels in MIRI animal models(76).

6. **Flaxseed**:

Flaxseed is utilized within the treatment of coronary artery disease (CAD) due to its high content of alpha-linolenic acid (ALA), lignans, and fiber, which offer assistance diminish cholesterol, lower blood pressure, and diminish irritation. It has been studied for its cardiovascular benefits, including improving lipid profiles and reducing atherosclerosis(77). Flaxseed (Linum usitatissimum) contains a few bioactive compounds useful for cardiovascular health, especially in the treatment of coronary artery disease (CAD). The essential phytoconstituents include :

- **Alpha-linolenic acid (ALA)**: A plant-based omega-3 fatty acid, ALA has been related with a decreased rate of cardiovascular events and sudden cardiac death. Dietary intake of ALA from flaxseed has illustrated defensive impacts against ventricular arrhythmias and cleared out ventricular expansion following myocardial infarction(78).
- **Secoisolariciresinoldiglucoside (SDG):** SDG is a lignan with antioxidant properties found abundantly in flaxseed. Investigate shows that SDG can prevent and treat atherosclerosis, the essential cause of CAD, by decreasing serum

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cholesterol levels and inhibiting the movement of hypercholesterolemic atherosclerosis[79].

- **Dietary fiber**: Flaxseed is rich in soluble and insoluble fiber, which contributes to lowering blood cholesterol levels and moving forward overall cardiovascular)health[80].

7. **Ginseng** :

The properties of ginseng and ginsenosides include vasorelaxation, antioxidation, anti-inflammatory, and anticancer ginsenosides affect the neurological system. effects. Additionally, it has been demonstrated that cardiovascular diseaes[81].People with cardiovascular risk factors including high blood pressure and high cholesterol are also frequently treated with ginseng[82].Treatment with ginseng returns coronary blood flow to normal in cases of cardiac ischemia, which can result in myocardial damage that produces ROS[83]

- Efficacy of ginseng in improving cardiac functions: Ginsenoside Rb1 has been shown to decrease heart damage in diabetes caused by streptozotocin[84], and ginseng has been shown to reduce unfavorablepostmyocardialremodelling[85].According to certain research, ginseng prevents heart failure and cardiac hypertrophy via reducing calcineurin activation and modulating Nhe-1[86]
- Efficacy of ginseng in adjusting lipid profile: Red ginseng extract treatment improved coronary flow in myocardial ischemia patients[87].This outcome so demonstrates that ginseng's anticoagulant qualities greatly enhanced blood circulation[88]. The bifidus fermentation procedure is known to significantly increase the hypolipidemic and hypoglycemic effects of red ginseng[89].Ginsenoside Rd also was effective in preventing atherosclerosis in Due to these results, P. ginseng may have an antihyperlipidemic effect[90].
- Efficacy of ginseng in abjusting vasomotor functions: Ginsenoside promoted vascular smooth muscle dysfunction and inhibited angiotensin II-induced proliferation[91].In addition, Korean red ginseng helps hypertensive patients with vascular stiffness[92].All things considered, these findings demonstrate how ginseng enhances vasomotor function. Although ginseng was once believed to raise blood pressure to unsafe levels, our findings demonstrate that ginseng improves vasomotor function[93].

8. **Ashwagandha(Withaniasomnifera)**:

The role it plays in animal models of atherosclerosis, hyperlipidemia, myocardial infarction, myocardial ischemia reperfusion injury, cerebral ischemia, cardiomyopathy, cardiac hypertrophy, cardiotoxicity, and congestive heart failure[94].Associated with the cardioprotective action, the hypolipidermic, hypoglycemic, diuretic, antioxidant, antiplatelet, and antiobesity qualities are attributed. The research on withania's involvement in various cardiovascular disorders has been gathered here[95].

- **Cardioprotective effect**: A significant public health concern, withania therapy has been shown to boost antioxidant enzymes, suppress lipid peroxidation, and raise heart rate, contractility, relaxation, and preload in a manner comparable to that of vitamin E, a proven cardioprotective antioxidant[96].Along in antioxidant activity, the anti-apoptotic activity that supports cardio protection was demonstrated by pro-apoptotic protein Bax being reduced and anti-apoptotic protein Bcl-2 being upregulated, as well as by the attenuation of terminal deoxynucleotidyl transferase biotin-dUTP nick end labeling positivity, a sign of apoptosis[97].
- **Anti-inflammatory effect**:Withania and withanolides show potential new agents for the treatment of inflammatory cascade cardiac conditions since they are strong inhibitors of the pro-inflammatory transcription factors NF-kB and AP-1[98].

9. **Arjuna (Terminalia arjuna)**:

Reversal of Cardiac Injury Enzyme &Improved Mitochondrial Uptake :Arjuna has the ability to restore cardiac function and myocardial integrity. Terminalia arjuna was found to fight the elevated levels of serum glutamate pyruvate transaminase, creatine phosphokinase, γ-glutamyl transpeptidase, and glutamate oxaloacetate transaminase in cardiac necrosis[99].

Additionally, in terms of recovering myocardial integrity and cardiac function, the heart was significantly protected against the lowered levels of γ-glutamyl transpeptidase, succinate dehydrogenase, glycogen, and mitochondrial oxygen uptake. It was noted that elevated blood levels of creatine, glutamate oxaloacetate transaminase,Terminalia arjuna reversed glutamate pyruvate transaminase, phosphokinase, and γ-glutamyl transpeptidase in myocardial necrosis.Additionally, there was a notable defense against the heart's lowered levels of glycogen, succinate dehydrogenase, γ-glutamyl transpeptidase, and mitochondrial oxygen absorption(100).

- **Cardiac Protection Against Oxidative stress**:In terms of cellular metabolite levels, lipid peroxidation end products, glutathione levels, protein carbonyl contents, and other antioxidant enzyme activities, Terminalia arjuna improved the cardiac intracellular antioxidant activity and cardioprotective function. It lowers serum CKMB levels, lowers lipid peroxidation, and raises cardiac antioxidant enzymes(101).
- **Reduction in TC, TG & Increase in HDL**:By inhibiting the liver's ability to produce cholesterol, increasing the excretion of bile acids in the feces, activating the enzyme plasma lecithin:cholesterol acyltransferase, and promoting the breakdown of low-density lipoprotein through receptor-mediated catabolism, Terminalia arjuna reduces lipid levels(102). In rats given cholesterol and triton, Terminalia arjuna bark powder was shown to enhance the levels of high-density lipoprotein-cholesterol while decreasing lipid and βlipoprotein protein levels. It alters the lipolytic activity of hyperlipaemic liver, heart, plasma, and adipose tissues(103).The down-regulation of lipogenic enzymes, inhibition of HMG-CoA reductase, and enhanced hepatic clearance of cholesterol are thought to be the factors leading the hypolipidemic effect(104).

10. **Hawthorn (Crataegus spp.):**

The medicinal potential of hawthorn is supported by its rich phytochemical composition, especially with regard to cardiovascular health(105).Flavonoids, oligomeric procyanidins, and triterpene acids are the main bioactive substances found in hawthorn, and they all have beneficial effects on heart and vascular health(106).Among the polyphenols, flavonoids are known for their strong antioxidant qualities(107).The hawthorn plant contains flavonoids called vitexin, hyperoside, and rutin, which have been linked to anti-arrhythmic and vasodilatory effects as well as lipid profile changes that lower lipid levels(108).They protect myocardial cells from oxidative damage by scavenging free radicals and lowering oxidative stress, which has an impact on the cardiovascular system. They also relax blood vessels, which lowers blood pressure and increases coronary artery blood flow(109).The triterpene acids ursolicand oleanolic acids are present in hawthorn fruit. These antioxidants protect against atherosclerosis and myocardial ischemia while decrease inflammation, which benefits the cardiovascular system. Additionally, they show a protective function against myocardial ischemia by reducing apoptotic pathways(110).Sterols, amines, and vitamins are abundant in the hawthorn plant in addition to these main bioactive substances, and they may work in concert to increase the plant's overall therapeutic effectiveness. All things considered, the different bioactive substances found in hawthorn provide a multi-target strategy for both preventing and treating CVDs. This all-encompassing mode of action highlights hawthorn's potential as a natural remedy for cardiovascular health(111).

11. **Guggul(Commiphora mukul):**

Given its important role as a risk factor, dyslipidemia must be discussed in the context of managing cardiovascular disease (CVD)(112). Modulating lipid metabolism is the main goal of Ayurvedic treatments like the use of KaishorGuggulu, a well-known lipid-lowering agent, and Tapyadi Loha, a formulation with iron and other herbs. These therapies are believed to effectively lower triglycerides, serum cholesterol, and LDL (low-density lipoprotein) levels while also raising HDL (high-density lipoprotein) levels, thereby lowering the risk of atherosclerosis(113).In addition, a number of Ayurvedic formulations have lipid-lowering properties. Herbs like Arjuna in Prabhakar Vati, for example, have been researched for their potential to improve the lipid profile and reduce LDL cholesterol levels, thereby supporting cardiovascular health(114). Similar to this, ingredients like guggulu in KaishoreGuggulu are known to have lipid-lowering qualities that help lower LDL cholesterol levels and may lessen the risk of coronary artery disease and atherosclerosis. Furthermore, according to traditional beliefs, some of the herbs in Arogyavardhini Vati, like Guggulu (Commiphora mukul), have lipid-regulating properties that suggest they may help preserve cardiovascular health and lower the risk of atherosclerosis(116).

The treatment plan includes oral drugs such as Arogyavardini Vati because of their anti-inflammatory, antioxidant, and hepatoprotective qualities. In order to address systemic inflammation

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and oxidative stress, two important factors in the pathophysiology of cardiovascular disease, these qualities are vital[117].

Additionally, Manjistha (Rubia cordifolia), a crucial part of ManjistadiKshara Basti, has strong anti-inflammatory qualities. The management of inflammation-related cardiovascular conditions may benefit from ManjistadiKshara Basti's reduction of inflammation, considering the role that chronic inflammation plays in cardiovascular diseases like atherosclerosis and coronary artery disease[118].

In the same way, KaishoreGuggulu includes herbs known for their strong anti-inflammatory qualities, such as triphala and guggulu (Commiphora mukul). KaishoreGuggulu may help manage a number of conditions indirectly by reducing inflammation. disorders of the heart. The addition of antioxidant-rich herbs like Triphala by KaishoreGuggulu also lessens oxidative damage to the cardiovascular system by removing free radicals[119].

IV. MATERIAL AND METHODS:

A comprehensive literature search was conducted utilizing PubMed, Scopus, and Google Researcher databases. Studies published between 2015 and 2024 were included. The keywords utilized were "Coronary Artery Disease," "Atherosclerosis," "Risk Factors," and "Treatment."(allopathic,ayurvedic).

Exclusion criteria included non-English articles and case reports. Studies were screened based on title and abstract, and full-text reviews were conducted for eligibility. Data on risk factors, the study of disease transmission, and treatment were extracted manually.

To evaluate study quality, the Newcastle-Ottawa Scale (NOS) and Cochrane Chance of Inclination device were used [120,121]. Errors in data choice were resolved through discussion among the reviewers.

Study selection :

Study design: This study aimed to investigate the effect of biomarker (cardiac troponin) on the growth rate of coronary artery disease and their mechanism related to myocardial damage. Here there is a study regarding the effect of natural antagonist to treat coronary artery disease and affecting biomarker.

V. RESULT AND CONCLUSION:

The accumulation of atherosclerotic plaques in the coronary arteries, which results in decreased blood flow to the heart muscle, is the characteristic of coronary artery disease (CAD)[122]. Lipid buildup, inflammatory processes, and endothelial dysfunction are the main causes of the illness, which causes the arteries to narrow and stiffen. These plaques may eventually burst, forming a thrombus (clot) that can totally stop blood flow and result in myocardial infarction (heart attack)[123].

Hypertension, diabetes mellitus, hyperlipidemia, smoking, a lack of physical activity, and a family history of cardiovascular disease are the main risk factors for coronary artery disease (CAD)[124]. Furthermore, it has been established that new variables such oxidative stress and inflammation contribute to the development of CAD[125].Angina, shortness of breath, fatigue, and feeling dizzy are common clinical manifestations of coronary artery disease (CAD). Before a significant cardiac event happens, CAD may occasionally be asymptomatic[126]. Diagnostic techniques consist of: An electrocardiogram (ECG) can detect arrhythmias or ischemia[127].

Stress testing: Used to evaluate how well the heart works when exerted[128].

The gold standard for determining the existence and degree of artery blockages is coronary angiography[129].

Blood biomarkers that aid in determining the probability and severity of CAD include lipid profiles, troponins, and hs-CRP (high-sensitivity C-reactive protein) [130].

VI. CONCLUSION:

With major public health consequences, coronary artery disease continues to rank among the world's most prevalent causes of morbidity and mortality. Numerous risk factors contribute to the development of the disease, and atherosclerosis is a major element in its advancement. To lower the possibility of negative outcomes including heart attacks and strokes, early identification and risk factor management are essential. Patients with CAD now have much greater survival rates and better quality of life due to improvements in diagnostic methods and available treatments. The treatment of CAD has been completely transformed by minimally invasive techniques like drug-eluting stents and percutaneous coronary intervention, particularly for high-risk patients. Changes in lifestyle, however,continueto be the primary component of care and prevention. Even with these improvements, CAD remains a major burden, especially in low-incomewhen there

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is inadequate access to healthcare and preventative measures. Improving long-term results requires more focused treatments and ongoing study into the pathophysiology of CAD. Future developments in regenerative treatments, genetic markers, and customized therapy will probably be the main focuses of CAD management.
In summary, even though there have been significant advancements in the management and treatment of CAD, the most effective strategy of action is still prevention through early intervention and lifestyle change. To continue lowering CAD-related morbidity and death globally, more research and advancements in healthcare are required.

REFERENCE :

[1]. Shanajehan RD, Sharma S, Bhutta BS. Coronary artery disease. In Stat Pearls [Internet] 2024 Oct 9. Stat Pearls Publishing.
[2]. Tohirova J, Shernazarov F. Atherosclerosis: causes, symptoms, diagnosis, treatment and prevention. Science and innovation. 2022;1(D5):7-12.
[3]. Duggan JP, Peters AS, Trachiotis GD, Antevil JL. Epidemiology of coronary artery disease. Surgical Clinics. 2022 Jun 1;102(3):499-516.
[4]. Shao C, Wang J, Tian J, Tang YD. Coronary artery disease: from mechanism to clinical practice. Coronary Artery Disease: Therapeutics and Drug Discovery. 2020:1-36.
[5]. Moffitt EA, Sethna DH. The coronary circulation and myocardial oxygenation in coronary artery disease: effects of anesthesia. Anesthesia& Analgesia. 1986 Apr 1;65(4):395-410.
[6]. Zaman AG, Helft G, Worthley SG, Badimon JJ. The role of plaque rupture and thrombosis in coronary artery disease. Atherosclerosis. 2000 Apr 1;149(2):251-66.
[7]. Sposito AC, Schubach A, de Lima G, et al. Biomarkers in cardiovascular disease. J Clin Pathol. 2004;57(1):1-9.
[8]. Huang Y, Gulshan K, Nguyen T, Wu Y. Biomarkers of cardiovascular disease. Disease markers. 2017 Nov 7;2017:8208609.
[9]. Vasan RS. Biomarkers of cardiovascular disease: molecular basis and practical considerations. Circulation. 2006 May 16;113(19):2335-62.
[10]. Rastogi S, Pandey MM, Rawat AK. Traditional herbs: a remedy for cardiovascular disorders. Phytomedicine. 2016 Oct 15;23(11):1082-9.
[11]. Alizadehsani R, Khosravi A, Roshanzamir M, Abdar M, Sarrafzadegan N, Shafie D, Khozeimeh F, Shoeibi A, Nahavandi S, Panahiazar M, Bishara A. Coronary artery disease detection using artificial intelligence techniques: A survey of trends, geographical differences and diagnostic features 1991–2020. Computers in Biology and Medicine. 2021 Jan 1;128:104095.
[12]. Komilovich EB. Coronary Artery Disease. EUROPEAN JOURNAL OF MODERN MEDICINE AND PRACTICE. 2023 Dec 10;3(12):81-7.
[13]. Tohirova J, Shernazarov F. Atherosclerosis: causes, symptoms, diagnosis, treatment and prevention. Science and innovation. 2022;1(D5):7-12.
[14]. Młynarska E, Czarnik W, Fularski P, Hajdys J, Majchrowicz G, Stabrawa M, Rysz J, Franczyk B. From atherosclerotic plaque to myocardial infarction—The leading cause of coronary artery occlusion. International Journal of Molecular Sciences. 2024 Jul 2;25(13):7295.
[15]. Zambroski CH, Moser DK, Bhat G, Ziegler C. Impact of symptom prevalence and symptom burden on quality of life in patients with heart failure. European journal of cardiovascular nursing. 2005 Sep;4(3):198-206.
[16]. Garje A, Adhav YG, Bodas D. Design and simulation of blocked blood vessel for early detection of heart diseases. In2015 2nd International Symposium on Physics and Technology of Sensors (ISPTS) 2015 Mar 7 (pp. 204-208). IEEE.
[17]. Collet C, Collison D, Mizukami T, McCartney P, Sonck J, Ford T, Munhoz D, Berry C, De Bruyne B, Oldroyd K. Differential improvement in angina and health-related quality of life after PCI in focal and diffuse coronary artery disease. Cardiovascular Interventions. 2022 Dec 26;15(24):2506-18.
[18]. Maurovich-Horvat P, Bosserdt M, Kofoed KF, Rieckmann N, Benedek T, Donnelly

P, Rodriguez-Palomares J, Erglis A, Štěchovský C, Šakalyte G, ČemerlićAdić N. CT or invasive coronary angiography in stable chest pain. New England Journal of Medicine. 2022 Apr 28;386(17):1591-602.
[19]. Faizy TD, Mlynash M, Kabiri R, Christensen S, Kuraitis GM, Mader MM, Flottmann F, Broocks G, Lansberg MG, Albers GW, Marks MP. The cerebral collateral cascade: comprehensive blood flow in ischemic stroke. Neurology. 2022 Jun 7;98(23):e2296-306.
[20]. Moini J, Ferdowsi K. Coronary Artery Disease. InHandbook of Nutritional Disorders (pp. 126-140). CRC Press.
[21]. BUILDER V. Cardiovascular Pathologies and Disorders. Mosby's Pathology for Massage Professionals-E-Book: Mosby's Pathology for Massage Professionals-E-Book.
[22]. .Nguyen TH, Ong GJ, Girolamo OC, De Menezes Caceres' V, Muminovic A, Chirkov YY, Horowitz JD. Angina due to coronary artery spasm (variant angina): Diagnosis and intervention strategies. Expert review of cardiovascular therapy.
[23]. Dugani SB, Moorthy MV, Li C, Demler OV, Alsheikh-Ali AA, Ridker PM, Glynn RJ, Mora S. Association of lipid, inflammatory, and metabolic biomarkers with age at onset for incident coronary heart disease in women. JAMA cardiology.
[24]. Bhatia SK, Bhatia SK. Coronary artery disease. Biomaterials for Clinical Applications. 2010:23-49.
[25]. Lehman W, Galińska-Rakoczy A, Hatch V, Tobacman LS, Craig R. Structural basis for the activation of muscle contraction by troponin and tropomyosin. Journal of Molecular Biology. 2009 May 15;388(4):673-814.
[26]. Jakubiak GK. Cardiac troponin serum concentration measurement is useful not only in the diagnosis of acute cardiovascular events. Journal of Personalized Medicine. 2024 Feb 21;14(3):230. Jacob R, Khan M. Cardiac biomarkers: what is and what can be. Indian journal of cardiovascular disease in women WINCARS. 2018 Dec;3(4):240
[27]. Myrmel GM, Wereski R, Karaji I, Saeed N, Aakre KM, Mills NL, Pedersen ER. Cardiac biomarkers and CT coronary angiography for the assessment of coronary heart disease. Clinical Biochemistry. 2024 Nov 23:110857.
[28]. Kaier TE, Alaour B, Marber M. Cardiac troponin and defining myocardial infarction. Cardiovascular research. 2021 Sep 1;117(10):2203-15.
[29]. sJacob R, Khan M. Cardiac biomarkers: what is and what can be. Indian journal of cardiovascular disease in women WINCARS. 2018 Dec;3(4):240.
[30]. Mair J. Cardiac troponin I and troponin T: are enzymes still relevant as cardiac markers?. Clinica Chimica Acta. 1997 Jan 3;257(1):99-115.
[31]. Giannitsis E, Katus HA. Cardiac troponin level elevations not related to acute coronary syndromes. Nature reviews cardiology. 2013 Nov;10(11):623-34.
[32]. Collinson PO, Gaze DC. Biomarkers of cardiovascular damage and dysfunction—an overview. Heart, Lung and Circulation. 2007 Jan 1;16:S71-82.
[33]. Mair J, Lindahl B, Hammarsten O, Müller C, Giannitsis E, Huber K, Möckel M, Plebani M, Thygesen K, Jaffe AS. How is cardiac troponin released from injured myocardium?. European heart journal: acute cardiovascular care. 2018 Sep;7(6):553-60
[34]. Costache AD, Leon-Constantin MM, Roca M, Maştaleru A, Anghel RC, Zota IM, Drugescu A, Costache II, Chetran A, Moisă ȘM, Huzum B. Cardiac biomarkers in sports cardiology. Journal of Cardiovascular Development and Disease. 2022 Dec 11;9(12):453.
[35]. Chaulin AM. Cardiac troponins metabolism: From biochemical mechanisms to clinical practice (literature review). International Journal of Molecular Sciences. 2021 Oct 10;22(20):10928.
[36]. Myrmel GM, Wereski R, Karaji I, Saeed N, Aakre KM, Mills NL, Pedersen ER. Cardiac biomarkers and CT coronary angiography for the assessment of coronary heart disease. Clinical Biocheistry. 2024 Nov 23:110857.
[37]. Li L, Zhou X, Li N, Sun M, Lv J, Xu Z. Herbal drugs against cardiovascular disease: traditional medicine and modern

development. Drug Discovery Today. 2015 Sep 1;20(9):1074-86.
[38]. Rastogi S, Pandey MM, Rawat AK. Traditional herbs: a remedy for cardiovascular disorders. Phytomedicine. 2016 Oct 15;23(11):1082-9.
[39]. Rastogi S, Pandey MM, Rawat AK. Traditional herbs: a remedy for cardiovascular disorders. Phytomedicine. 2016 Oct 15;23(11):1082-9.
[40]. Zhang HA, Kitts DD. Turmeric and its bioactive constituents trigger cell signaling mechanisms that protect against diabetes and cardiovascular diseases. Molecular and cellular biochemistry. 2021 Oct;476(10):3785-814.
[41]. Ballester P, Cerdá B, Arcusa R, García-Muñoz AM, Marhuenda J, Zafrilla P. Antioxidant activity in extracts from Zingiberaceae family: Cardamom, turmeric, and ginger. Molecules. 2023 May 11;28(10):4024.
[42]. Fei C, Ji D, Tong H, Li Y, Su L, Qin Y, Bian Z, Zhang W, Mao C, Li L, Lu T. Therapeutic mechanism of Curcuma aromaticaSalisb. rhizome against coronary heart disease based on integrated network pharmacology, pharmacological evaluation and lipidomics. Frontiers in Pharmacology. 2022 Aug 9;13:950749.
[43]. Fei C, Ji D, Tong H, Li Y, Su L, Qin Y, Bian Z, Zhang W, Mao C, Li L, Lu T. Therapeutic mechanism of Curcuma aromaticaSalisb. rhizome against coronary heart disease based on integrated network pharmacology, pharmacological evaluation and lipidomics. Frontiers in Pharmacology. 2022 Aug 9;13:950749.
[44]. Mohammadabadi T, Ayad AE, Maheshwari A, BenAyad A. Ginger: A Nutraceutical Supplement for Protection Against Various Cardiovascular Diseases in Clinical Trials. Cureus. 2025 Mar 19;17(3).
[45]. Cerdá B, Marhuenda J, Arcusa R, Villaño D, Ballester P, Zafrilla P. Ginger in the prevention of cardiovascular diseases. Current Topics in Functional Food. 2022 Apr 21.
[46]. Daniels CC, Isaacs Z, Finelli R, Leisegang K. The efficacy of Zingiber officinale on dyslipidaemia, blood pressure, and inflammation as cardiovascular risk factors: A systematic review. Clinical Nutrition ESPEN. 2022 Oct 1;51:72-82.
[47]. Ali B, Al-Wabel N, Al-Meshal I, et al. Ginger and its medicinal properties: A review of literature. Journal of Ethnopharmacology. 2015;175:433-440. doi:10.1016/j.jep.2015.09.004.
[48]. Hutchins E, Shaikh K, Kinninger A, Cherukuri L, Birudaraju D, Mao SS, Nakanishi R, Almeida S, Jayawardena E, Shekar C, Flores F. Aged garlic extract reduces left ventricular myocardial mass in patients with diabetes: A prospective randomized controlled double-blind study. Experimental and therapeutic medicine. 2020 Feb;19(2):1468-71.
[49]. Williams MJ, Sutherland WH, McCormick MP, Yeoman DJ, de Jong SA. Aged garlic extract improves endothelial function in men with coronary artery disease. Phytotherapy Research: An International Journal Devoted to Pharmacological and Toxicological Evaluation of Natural Product Derivatives. 2005 Apr;19(4):314-9.
[50]. Hutchins E, Shaikh K, Kinninger A, Cherukuri L, Birudaraju D, Mao SS, Nakanishi R, Almeida S, Jayawardena E, Shekar C, Flores F, Hamal S, Sheikh S, Johanis A, Cu B, Trad G, Budoff MJ. Aged garlic extract reduces left ventricular myocardial mass in patients with diabetes: A prospective randomized controlled double-blind study. Exp Ther Med. 2020 Feb;19(2):1468-1471. doi: 10.3892/etm.2019.8373. Epub 2019 Dec 27. PMID: 32010324; PMCID: PMC6966199.
[51]. Kheirmandparizi M, Keshavarz P, Nowrouzi-Sohrabi P, Hosseini-Bensenjan M, Rezaei S, Kashani SM, Zeidi N, Tabrizi R, Alkamel A. Effects of garlic extract on lipid profile in patients with coronary artery disease: A systematic review and meta-analysis of randomised clinical trials. International Journal of Clinical Practice. 2021 Dec;75(12):e14974.
[52]. Shang C, Lin H, Fang X, Wang Y, Jiang Z, Qu Y, Xiang M, Shen Z, Xin L, Lu Y, Gao J. Beneficial effects of cinnamon and its extracts in the management of cardiovascular diseases and diabetes. Food & function. 2021;12(24):12194-220.

[53]. Mohammadabadi T, Jain R. Cinnamon: a nutraceutical supplement for the cardiovascular system. Archives of Medical sciences. Atherosclerotic Diseases. 2024 Mar 30;9:e72.

[54]. Khan A, Safdar M, Ali Khan MM, Khattak KN, Anderson RA. Cinnamon improves glucose and lipids of people with type 2 diabetes. Diabetes Care. 2003;26(12):3215-8.

[55]. Gruenwald J, Freder J, Armbruester N. Cinnamon and health. Crit Rev Food Sci Nutr. 2010;50(9):822-34.

[56]. Roussel AM, Hininger I, Benaraba R, Ziegenfuss TN, Anderson RA. Antioxidant effects of a cinnamon extract in people with impaired fasting glucose. J Am Coll Nutr. 2009;28(1):16-21.

[57]. Anderson RA, Broadhurst CL, Polansky MM, Schmidt WF, Khan A, Flanagan VP, et al. Isolation and characterization of polyphenol type-A polymers from cinnamon with insulin-like biological activity. J Agric Food Chem. 2004;52(1):65-70.

[58]. Davis PA, Yokoyama W. Cinnamon intake lowers fasting blood glucose: Meta-analysis. J Med Food. 2011;14(9):884-9.

[59]. Aneja, R., Hake, P.W., Burroughs, T.J. et al. Epigallocatechin, a Green Tea Polyphenol, Attenuates Myocardial Ischemia Reperfusion Injury in Rats. Mol Med 10, 55–62 (2004). https://doi.org/10.2119/2004-00032.Aneja.

[60]. Widlansky ME, Hamburg NM, Anter E, Holbrook M, Kahn DF, Elliott JG, Keaney JF Jr, Vita JA. Acute EGCG supplementation reverses endothelial dysfunction in patients with coronary artery disease. J Am Coll Nutr. 2007 Apr;26(2):95-102. doi: 10.1080/07315724.2007.10719590. PMID: 17536120; PMCID: PMC3773609.Wei, XY., Zeng, YF., Guo, QH. et al. Cardioprotective effect of epigallocatechin gallate in myocardial ischemia/reperfusion injury and myocardial infarction: a meta-analysis in preclinical animal studies. Sci Rep 13, 14050 (2023).

[61]. Patade A, Devare S, Kashyap R. Flaxseed: A health boon. J Nutr Food Sci. 2015;5(4):1000389. doi:10.4172/2155-9600.1000389.

[62]. Parikh M, Raj P, Austria JA, Yu L, Garg B, Netticadan T, Pierce GN. Dietary flaxseed protects against ventricular arrhythmias and left ventricular dilation after a myocardial infarction. The Journal of Nutritional Biochemistry. 2019 Sep 1;71:63-71.

[63]. Prasad K, Jadhav A. Prevention and treatment of atherosclerosis with flaxseed-derived compound secoisolariciresinoldiglucoside. Current pharmaceutical design. 2016 Jan 1;22(2):214-20.

[64]. Bernacchia R, Preti R, Vinci G. Chemical composition and health benefits of flaxseed. Austin J Nutri Food Sci. 2014 Oct;2(8):1045.

[65]. Lee CH, Kim JH. A review on the medicinal potentials of ginseng and ginsenosides on cardiovascular diseases. Journal of ginseng research. 2014 Jul 1;38(3):161-6.

[66]. W. Zhou, H. Chai, P.H. Lin, A.B. Lumsden, Q. Yao, C.J. Chen Molecular mechanisms and clinical applications of ginseng root for cardiovascular disease

[67]. R. Bolli Superoxide dismutase 10 years later: a drug in search of a use J Am Coll Cardiol, 18 (1991), pp. 231-233

[68]. Y. Wu, Z.Y. Xia, J. Dou, L. Zhang, J.J. Xu, B. Zhao, S. Lei, H.M. Liu Protective effect of ginsenoside Rb1 against myocardial ischemia/reperfusion injury in streptozotocin-induced diabetic rats Mol Biol Rep, 38 (2011), pp. 4327-4335

[69]. S.Bodiga, W. Wang, G.Y. Oudit Use of ginseng to reduce post-myocardial adverse myocardial remodeling: applying scientific principles to the use of herbal therapies.J Mol Med (Berl), 89 (2011), pp. 317-32089

[70]. J. Guo, X.T. Gan, J.V. Haist, V. Rajapurohitam A. Zeidan, N.S. Faruq, M. Karmazyn Ginseng inhibits cardiomyocyte hypertrophy and heart failure via NHE-1 inhibition and attenuation of calcineurin activation Circ Heart Fail, 4 (2011), pp. 79-88.

[71]. C.M.Ahn, S.J. Hong, S.C. Choi, J.H. Park, J.S. Kim, D.S. Lim Red ginseng extract improves coronary flow reserve and increases absolute numbers of various circulating angiogenic cells in patients with first ST-segment elevation acute

myocardial infarction Phytother Res, 25 (2011), pp. 239-249.
[72]. J.G. Lee, Y.Y. Lee, B. Wu, S.Y. Kim, Y.J. Lee, H.S. Yun-Choi, J.H. Park Inhibitory activity of ginsenosides isolated from processed ginseng on platelet aggregation Pharmazie, 65 (2010), pp. 520-522
[73]. H.T. Trinh, S.J. Han, S.W. Kim, Y.C. Lee, D.H. Kim Bifidus fermentation increases hypolipidemic and hypoglycemic effects of red ginseng J MicrobiolBiotechnol, 17 (2007), pp. 1127-1133.
[74]. J. Li, Z.Z. Xie, Y.B. Tang, J.G. Zhou, Y.Y. Guan Ginsenoside-Rd, a purified component from Panax notoginseng saponins, prevents atherosclerosis in apoE knockout mice Eur J Pharmacol, 652 (2011), pp. 104-110.
[75]. J.Y. Lee, K.M. Lim, S.Y. Kim, O.N. Bae, J.Y. Noh, S.M. Chung, K. Kim, Y.S. Shin, M.Y. Lee, J.H. Chung Vascular smooth muscle dysfunction and remodeling induced by ginsenoside, a bioactive component of ginseng Toxicol Sci, 117 (2010), pp. 505-514.
[76]. M.Y. Rhee, Y.S. Kim, J.H. Bae, D.Y. Nah, Y.K. Kim, M.M. Lee, H.Y. Kim Effect of Korean red ginseng on arterial stiffness in subjects with hypertension J Altern Complement Med, 17 (2011), pp. 45-49.
[77]. B.H. Jeon, C.S. Kim, K.S. Park, J.W. Lee, J.B. Park, K.J. Kim, S.H. Kim, S.J. Chang, K.Y. Nam Effect of Korea red ginseng on the blood pressure in conscious hypertensive Gen Pharmacol, 35 (2000), pp. 135-141
[78]. Mishra, L.C., B.B. Singh and S. Dagenais, 2000.Dagenais, 2000.Scientific basis for the therapeutic use of Withania has been somnifera (Ashwagandha): a review. Alt Med. Rev.,reported to reduce blood pressure due to autonomic 5: 334-346.
[79]. Matsuda, H., T. Murakami, A. Kishi and 12 M. Yoshikawa, 2001. Structures of withanosides I II III IV V VI and VII the roots of Indian WithaniasomniferaDunal inhibitory activity for tachyphylaxis to clonidine in isolated guinea-pig ileum. Bioorg. Med. Chem., 9: 1499-1507.
[80]. Mohanty, I., D.S. Arya, A. Dinda, K.K. Talwar, S. Joshi and S.K. Gupta, 2004. Mechanisms of cardioprotective effect of Withaniasomnifera in diuretic experimentally induced myocardial infarction. Basic Clin. Pharmacol. Toxicol., 94: 184-90. Exp. Biol., 38: 607-9.
[81]. Mohanty, I.R., D.S. Arya and S.K. Gupta, 2008. Withaniasomnifera provides cardioprotection and attenuates ischemia-reperfusion induced apoptosis Clin. Nutr., 27: 635-42.
[82]. Kaileh, M., W. Vanden Berghe, A. Heyerick, J. H orion, J. PietteT. Essawi and G. Haegeman, 2007. Withaferin A 20. Hamza, A., A. Amin and S. Daoud, 2008. Thestrongly elicits IkappaB kinase beta hyper-phosphorylation concomitant with potent inhibition of its kinase activity. J. Biol. Chem., 282: 4253-64.
[83]. Dwivedi S, Chansouria JP N somini PN, Udupa KN. Influence of certain indigenous drugs on the PGE like activity in the ischaemic rabbit aorta. Indian Drugs. 1987; 2: 378–382.
[84]. Tandon S, Rastogi R, Kapoor NK. Protection by abana, a herbomineral preparation, against myocardial necrosis in rats induced by isoproterenol. Phytother Res. 1995; 9: 263–266.
[85]. Sinha M, Manna P, Sil PC. Terminalia arjuna protects mouse hearts against sodium fluorideinduced oxidative stress. J Med Food. 2008; 4: 733-740.
[86]. Khaliq F, Parveen A, Singh S, Hussain ME, Fahim M. Terminalia arjuna Improves Cardiovascular Autonomic Neuropathy in Streptozotocin-Induced Diabetic Rats. Cardiovascular Toxicol. 2013; 13: 68–76.
[87]. Khanna AK, Chander C, Kapoor NK. Terminalia arjuna: An Ayurvedic cardiotonic regulates lipid metabolism in hyperlipidemic rats. Phytother Res.1996; 10: 663–665.
[88]. Sharma S, Sharma D, Agarwal N. Diminishing effect of arjuna tree (Terminalia arjuna) bark on the lipid and oxidative stress status of high fat high cholesterol fed rats and development of certain dietary recipes containing the tree bark for human consumption. Research in Pharmacy. 2012; 2(4): 22-30.
[89]. Walker, A.F.; Marakis, G.; Morris, A.P.; Robinson, P.A. Promising hypotensive effect of hawthorn extract: A randomized

double-blind pilot study of mild, essential hypertension. Phytother. Res. PTR 2002, 16, 48–54.
[90]. Furey, A.; Tassell, M.C.; Kingston, R.; Gilroy, D.; Lehane, M. Hawthorn (Crataegus spp.) in the treatment of cardiovascular disease. Pharmacogn. Rev. 2010, 4, 32–41.
[91]. Pietta, P.-G. Flavonoids as Antioxidants. J. Nat. Prod. 2000, 63, 1035–1042.
[92]. Zhang, Z.; Chang, Q.; Zhu, M.; Huang, Y.; Ho, W.K.; Chen, Z.-Y. Characterization of antioxidants present in hawthorn fruits. J. Nutr. Biochem. 2001, 12, 144–152.
[93]. 110. Shanthi, S.; Parasakthy, K.; Deepalakshmi, P.D.; Devaraj, S.N. Hypolipidemic activity of tincture of Crataegus in rats. Indian J. Biochem. Biophys. 1994, 31, 143–146.
[94]. Xu, H.; Xu, H.-E.; Ryan, D. A Study of the Comparative Effects of Hawthorn Fruit Compound and Simvastatin on Lowering Blood Lipid Levels. Am. J. Chin. Med. 2009, 37, 903–908.
[95]. Verma, S.K.; Jain, V.; Verma, D.; Khamesra, R. Crataegus oxycantha—A cardioprotective herb. J. Herb. Med. Toxicol. 2007 1, 65–71.
[96]. Judy M. Muller-Delp, "The Coronary Microcirculation in Health and Disease", International Scholarly Research Notices, vol. 2013, Article ID 238979, 24 pages, 2013.
[97]. .Szapary PO, Wolfe ML, Bloedon LT, et al. Guggulipid for the treatment of hypercholesterolemia a randomized controlled trial. JAMA. 2003;290(6):765-772. doi:10.1001/jama.290.6.765 .
[98]. Ramesh P, Palanippan A. Terminalia arjuna, a Cardioprotective Herbal Medicine-Relevancy in the Modern Era of Pharmaceuticals and Green Nanomedicine-A Review. Pharmaceuticals (Basel). 2023;16(1):126. Published 2023 Jan 13. doi:10.3390/ph16010126.
[99]. Kumar G, Srivastava A, Sharma SK, Gupta YK. The hypolipidemic activity of Ayurvedic medicine, Arogyavardhinivati in Triton WR-1339-induced hyperlipidemic rats: A comparison with fenofibrate. J Ayurveda Integr Med. 2013;4(3):165-170. doi:10.4103/0975-9476.118707.
[100]. Parekar RR, Bolegave SS, Marathe PA, Rege NN. Experimental evaluation of analgesic, antiinflammatory and anti-platelet potential of Dashamoola. J Ayurveda Integr Med. 2015;6(1):11-18.doi:10.4103/0975-9476.146565 .
[101]. Wells GA, Shea B, O'Connell D, Peterson J, Welch V, Losos M, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomized studies in meta-analyses [Internet]. Ottawa Hospital Research Institute; 2013 [cited 2025 Mar 31]. Available from: http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp
[102]. Higgins JPT, Thomas J, Chandler J, Cumpston M, Li T, Page MJ, et al. Cochrane Handbook for Systematic Reviews of Interventions. 2nd ed. Chichester (UK): John Wiley & Sons; 2019.
[103]. Libby P, Ridker PM, Hansson GK. Inflammation and atherosclerosis. Circulation. 2002;105(9):1135-1143.
[104]. Ross R. Atherosclerosis—an inflammatory disease. N Engl J Med. 1999;340(2):115-126.
[105]. Ridker PM, Hennekens CH, Buring JE, et al. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. N Engl J Med. 2000;342(12):836-843.
[106]. Blankenberg S, Balling R, Bickel C, et al. The role of oxidative stress and inflammation in coronary artery disease. Eur Heart J. 2005;26(7):660-667.
[107]. Fihn SD, Gardin JM, Abrams J, et al. 2012 ACCF/AHA/ACP/AATS/PCNA/SCAI/STS guideline for the diagnosis and management of patients with stable ischemic heart disease: executive summary. Circulation. 2012;126(25):3097-3137.
[108]. Alpert JS, Thygesen K, Antman E, et al. Myocardial infarction redefined—a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. Eur Heart J. 2000;21(18):1502-1513.
[109]. Gibbons RJ, Balady GJ, Bricker JT, et al. American College of

Cardiology/American Heart Association 2002 guideline update for exercise testing: summary article. Circulation. 2002;106(14):1883-1892.

[110]. . Mancini GB, Moye LA, Ruddy TD. Angiography for the diagnosis of coronary artery disease. In: Fuster V, editor. Hurst's The Heart. 13th ed. New York: McGraw-Hill; 2011. p. 1133-1155.

[111]. Sposito AC, Schubach A, de Lima G, et al. Biomarkers in cardiovascular disease. J Clin Pathol. 2004;57(1):1-9.

[112]. Huang Y, Gulshan K, Nguyen T, Wu Y. Biomarkers of cardiovascular disease. Disease markers. 2017 Nov 7;2017:8208609.

[113]. Vasan RS. Biomarkers of cardiovascular disease: molecular basis and practical considerations. Circulation. 2006 May 16;113(19):2335-62.

[114]. Rastogi S, Pandey MM, Rawat AK. Traditional herbs: a remedy for cardiovascular disorders. Phytomedicine. 2016 Oct 15;23(11):1082-9.