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Cardioprotective effect of herbs on HTN induced LVH: A Mini Review

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Abstract

The significant risk factor for cardiovascular disease (CVD) related mortality and morbidity is hypertension. And long-term damage caused by primary HTN is LVH. Up-till now there are a lot of antihypertensive therapies available to lower the risk of HTN and LVH but none of them completely eliminate the likelihood of cardiac damage. So, it is need of time to find new drug therapies having cardioprotective effects. Recently herbal traditional medicines have gained popularity and a number of researches have been conducted to evaluate cardioprotective potential of herbs and isolated metabolites. This mini review summarizes some recent researches in this field.

Keywords: Hypertension; Left Ventricular hypertrophy; Cardioprotective, Herbal traditional medicines; Frank-Starling; Flavonoids; Phytoestrogens; Anti-inflammatory; Hemodynamic; Antihypertensive therapies

Introduction

A significant, controllable risk factor for the morbidity and mortality of cardiovascular disease (CVD) is hypertension (HTN) and the primary end-organ affected by HTN is the left ventricle (LV) [1]. Direct hemodynamic stress from a sustained rise in blood pressure (hypertension) results in ventricular hypertrophy [2]. More than 35% of hypertensive people develop left ventricular hypertrophy (LVH) [3]. Due to its increased impact on mortality and disability rates across many nations, hypertension is poised to become a crucial component of global health [4]. The major treatment for people with hypertension is the using of drugs such ACE inhibitors, angiotensin II receptor blockers, and -blockers to effectively regulate blood pressure; nonetheless, the total mortality rate from this condition is still very high [5]. Presently antihypertensive medications lower the likelihood of disease and LVH, although they do not completely eliminate them [6]. In order to create new pharmacological therapy for HTN it is crucial to find drugs that are more successful than currently available medications. Recently, herbal traditional medicine has gained popularity and is expanding quickly in many nations. Natural medicine has been endorsed by WHO as a potential course of therapy [7].

Discussion

When heart is facing some hemodynamic changes, the following actions can be taken by the heart to make up for a hemodynamic burden: (1) enhance crossbridge production by using the Frank-Starling method; (2) build up your muscle to handle the total load; and (3) deploy neurohormonal systems to enhance muscle contraction. The first mechanism's application is constrained, while the third's negative effects come from a chronic adjustment. As a result, mass gain plays a significant part in compensating for hemodynamic overload. Owing to the quick terminal differentiation of cardiomyocytes after birth, this mass gain is not the result of hyperplasia but rather the hypertrophy of preexisting myocytes. The simultaneous acquisition of sarcomeres generates a widening of the myocyte, which then causes a rise in wall thickness in diseases like aortic stenosis or hypertension, which are characterized by pressure overload. Concentric hypertrophy (a higher wall thickness to ratio/chamber diameter) is the result of this remodeling [8,9].

The increase in left ventricular mass known as left ventricular hypertrophy (LVH) is brought on by larger cardiomyocytes. LVH can be a pathological condition that is either hereditary or subse-

quent to LV overload or it can be a physiological adaptation to vigorous exercise, as in athletics [10]. Commonly benign, physiological LVH goes away when physical activity is reduced or stopped. Pathological LVH is a compensating condition that could ultimately develop into a dysfunctional state and lead to increasing LV dysfunction and sudden cardiac death (HF) [10]. Increased myocardial oxygen demand during left ventricular hypertrophy leads to diminished coronary blood flow reserve, which in turn increases the risk of angina, heart attack, and even death Figure 1 [11]. Myocyte growth must be followed by synchronised expansions of the capillary and nerve networks, as well as the connective tissue and ground material, in order for it to support an increased biomechanical load. Collagen makes up the majority of the connective tissue, with minor levels of elastin, laminin, and fibronectin also present. The myocardium contains collagen types I, III, and V, although type I makes up the majority - about 85%-of the tissue's collagen. The intricate collagen web prevents interstitial edoema from developing and accounts for a large portion of the passive diastolic stiffness of the ventricle. It also gives a method for translating force produced by individual myocytes into ventricular contraction [8].

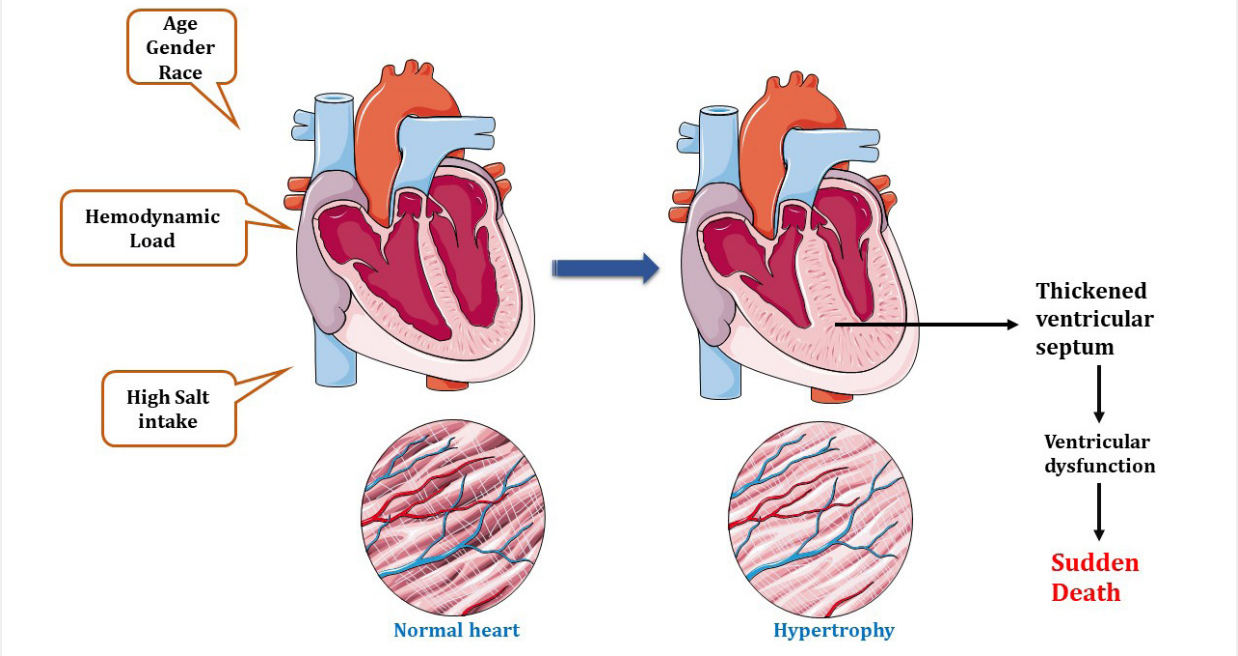

Figure 1: Factors inducing left ventricular hypertrophy.

The prevailing consensus is that a mechanical signal sets off a series of biological processes that cause synchronized heart development. If this is the case, the indications for volume overload and pressure overload are either quite distinct, or they produce patterns and mechanisms of development that are notably different. Myosin heavy chains production rises by around 35% within hours of a pressure overload occurring in the ventricle in vivo; this increase is first mediated by an increase in translational efficiency [12]. Distinct geometric patterns are frequently used to classify HTN-induced remodeling of the LV: concentric LV hypertrophy (LVH; cLVH), and eccentric LVH (eLVH) (Figure 2) [13].

Herbs and HTN induced LVH

Around the world, more than 80% of people use herbal treatments for a variety of illnesses, including arterial hypertension [14]. In animal studies of cardiovascular illnesses, a number of prior research found positive results from employing medicinal herbs as natural remedies. Many of these plants have antihypertensive qualities that enhance vascular and cardiac health [15]. Flavonoids, natural phenolic compounds, and phytoestrogens, which are potent antioxidants, are just a few of the bioactive substances that have been found in plants [16]. Consuming fruits and vegetables with increased flavonoid content helps to avoid the onset of cardiovascular disease [17]. The effectiveness of these plants against cardiovascular illnesses may be attributed to active substances such coumarins and/or polyphenols with antioxidant and anti-inflammatory characteristics, according to bio-guided fractionation of these plant extracts [18]. Table 1 summarizes the antihypertensive potential of some plants and plant based bioactive substances. .

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Table 1: Studies showing protective effect of herbal treatment and non-traditional approaches to treat HTN induced LVH.

Sr no.1	Treatment	Animal model used	Outcome	Publication year	Reference
1	Epigallocatechin-3 gallate (EGCG), the major catechin derived from green tea	Male Sprague Dawley rats	EGCG stops the onset of pressure over-load-induced left ventricular concentric hypertrophy.	2007	[19]
2	Quercetin	Male SHRs and male Wis-tar–Kyoto (WKY) rats H9C2 rat cardiomyocyte cells	Decreased blood pressure and significantly decreased the left ventricle to body weight ratio Considerably reduced Ang II-induced hypertrophy of H9C2 cells,	2013	[16]
3	*Ulmus wallichiana* Planchon	Wistar rats.	Both the BP and HR considerably de-creased.NO and cGMP levels increased, while plasma renin, Ang II, and ACE activity decreased. Decreased expression of ANP, BNP, TNF-, IL-6, MMP9, 1-AR, and TGF-1 and upregula-tion of NOS3, ACE2, and Mas, respectively.	2016	[20]
4	*Moringa oleifera* (MOI) seed powder	male SHRs WKY rats	The positive effects of MOI on cardiac shape and function in SHR are accompa-nied by an increase in PPAR- and PPAR-sig-naling.	2016	[21]
5	Indonesian herbal medicine *Centella asiatica*, *Justicia gendarussa* and *Imperata cylindrica* decoction (CJID)	SHRs and WKY rats	SBP and HR significantly decreased, while LV morphology and function improved including increased activity of SOD but a lower level of MDA CJID improved hypertension mediated LVH via decreasing the formation of ROS through the NOXs-dependent pathway.	2017	[4]
6	*Gardenia jasminoides*	Male ICR mice	Systolic, diastolic, and mean blood pressure in treated mice were lower than those in experimental control mice.	2017	[22]
7	Gallic acid	WKY rats SHR rats	GA therapy reduced SHRs' high systolic blood pressure and lowered cardiac-spe-cific transcription factor expression and inhibited left ventricular hypertrophy.	2017	[23]
8	Thymol	Rats	Through increasing serum antioxidant ca-pacity, it has cardioprotective benefits and also reduces left ventricular hypertrophy.	2017	[24]
9	*Galium verum L.*	SHRs	Significantly enhanced in vivo cardiac function and reduced left ventricular hy-pertrophy. After ischemia, G. verum extract preserved coronary vasodilatory response, systolic function, and cardiac contractility.	2019	[25]
10	*Anogeissus acuminata* (Roxb. ex DC.)	Sprague Dawley rats	A reduction in the size of cardiac cells and fibrosis, as well as the lack of inflammatory cells, along with decreased levels of ACE and renin and increased levels of nitric oxide (NO) and cyclic guanosine mono-phosphate (cGMP)	2020	[26]
11	Metformin	SHRs and WKY rats	Blood pressure was lowered, myocardial glucose uptake was restored, left ventricu-lar hypertrophy was avoided, and cardiac function was enhanced in SHR.	2020	[27]
13	*Sanoshashinto*	male WKY/Izm rats male SHRs	reduced left ventricular hypertrophy vasorelaxant effects	2020	[28]

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17	*Vitex cienkowskii*	SHRs	preventive activity in L-NAME-induced hypertension and improved blood pressure of spontaneously hypertensive rats y reduced the blood pressure in LNHR. Body weight, food and water intake, left ventricular hypertrophy, antioxidant level, renal and hepatic markers, and lipid profile were improved by the treatment with MMVC.	2021	[29]
18	*Rudgea viburnoides* leaves	Male and female Wistar rats	AERV treatment that lasted a long time preserved the levels of elimination of urine and electrolytes (Na+, K+, Ca2+ and Cl−), lowered blood pressure and heart rate, reversed electrocardiographic alterations, left ventricular hypertrophy, changes in vascular reactivity brought on by hyper-tension.	2021	[30]
2	*Curcuma longa* curcumin	Dahl salt-sensitive (DS) and salt-resistant (DR) rats.	Reduced the LV mass index and posterior wall thickening caused by hypertension without impairing systolic function. Additionally, it dramatically decreased the transcriptions of the hypertrophy-response gene, perivascular fibrosis, and increases in myocardial cell diameter brought on by hypertension.	2021	[5]
7	*Vitex cienkowskii* methanol/methylene chloride stem-bark extract (MMVC)	male Wistar rats	Treatment with MMVC reduced left ventric-ular hypertrophy, antioxidant levels, renal and hepatic indicators, and lipid profiles.	2021	[31]
8	*Populus ciliata* Wall ex. Royle	Sprague Dawley rats	Pc. Cr had beneficial effects on LVH by reducing angiotensin II, renin, and cGMP while boosting nitric oxide (NO) and reducing cardiac fibrosis, necrosis, and size of cells.	2021	[32]
14	*Callisia fragrans*	Rats	*C. fragrans* extract can mitigate hypertension and alleviate ventricular hypertrophy and renal dysfunction in reno-vascular hypertensive rats	2022	[33]

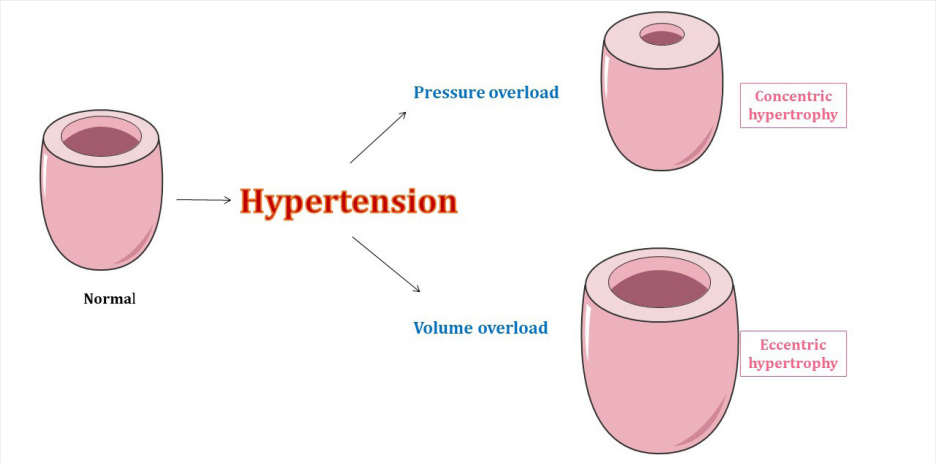

Figure 2: Ventricular Hypertrophy.

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