Herbals based therapy for the management of Myocardial infarction

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Abstract
Myocardial infarction (MI) is a notable health burden with an increasing prevalence. Herbs have been functional for medical treatments since the dawn of civilization and some derivatives (eg, aspirin, and digitalis) have been incorporated in the human pharmacotherapy. Herbal treatments for cardiovascular diseases have been harnessed in patients with congestive heart failure, angina pectoris, systolic hypertension, cerebral insufficiency, atherosclerosis, arrhythmia and venous insufficiency. Nevertheless, meticulous scientific evaluation has not been carried out for many herbal remedies used today that may cause possible toxic effects. Persistent testing is required to illuminate the pharmacological activities of the numerous herbal remedies now being used to treat cardiovascular diseases like MI. This review elucidates the prevalence, available treatments and need for herbal medicines to treat MI.
1. INTRODUCTION

In 2017, ischemic heart disease became the world's most prevalent cause of death [1]. Myocardial infarction (MI) is a permanent necrosis of myocardial cells caused by extreme and persistent myocardial ischaemia [2]. The epicardial coronary artery plaque normally occurs from ruptures or erosions triggering the superimposed thrombosis and coronary artery occlusion. The disequilibrium of supply and demand of myocardial oxygen induces heart failure, arrhythmias, myocardial illness and sudden death [3]. In the case of coronary atherosclerosis myocardial infarction can often arise if unfavorable blood distributes the blood stream away from a stenotic coronary artery-supplied myocardial region [3]. That is a condition of myocardial ischemia at the edge of the continuum or acute coronary syndrome. The occurrence of atherosclerosis relies on the predisposition of risk factors. For patients with acute myocardial infarction, 70% of fatal incidents are attributed to atherosclerotic plaque occlusion. Risk factors include smoking, exercise, high blood pressure, obesity, cholesterol, LDL and triglyceride [4]. In the United States, around 50 per cent of all MIs result in individuals younger than 65 years of age. In the future, though, as the demographic shifts and the population age rises, a greater number of MI patients may be older than 65. Acute myocardial infarction classical signs involve sudden thorn pressure (typically left-hand or left-hand radiating pain), shortness of breath, fatigue, anxiety, palpitation, sweating and anxiousness. (often described as a sense of impending doom). Women may have less common symptoms than males, most frequently shortness of breath, fatigue, indigestion and tiredness [5]. For both men and women around the globe, cardiac problems remain the main cause of death [6]. Relevant risk factors include pre-existing coronary disorder, smoking with cigarettes, elevated lipid blood loads and low lipoprotein levels, asthma, blood pressure, obesity, chronic kidney disease, cardiac attack, medications in heavy alcohol intake and excessively high levels of stress [7-8].
2. **Pathophysiology of myocardial infarction**

The potential pathophysiological pathways underlying the circadian myocardial infarction trend involve abrupt variation in the blood pressure, heart rate, platelet aggregation and fibrinolytic behaviour, which contribute to elevated chances of plaque breakup and intracoronary thrombosis [9]. Vulnerable plaques are typically those that induce only mild to moderate stenosis and have a lipid-rich centre and a small, macrophage-dense, collagen-poor, fibrous cap. Mechanical damage, circadian cycle, inflammation and infection are all variables that cause plaque breakup. Plaque breakup triggered by myocardial infarction may accompany progressive thrombosis and vasospasm [10]. The non-invasive in vivo myocardial tissue characterisation is given by cardiovascular magnetic resonance (CMR). This is accomplished by utilising various pulse patterns, like the reverse recovery series, after gadolinium-chelate contrast media are used. Increased myocardial signal strength revealed by T2-weighted imaging distinguishes regions of increased water content that are distinctive features of acute myocardial infarction, associated with active myocardial inflammation and oedema [11]. Enhanced late myocardial results from T1 imagery following contrast show a permanent myocardial damage (i.e. myocardial necrosis) and the spatial resolution of this methodology will reliably determine harm transmurality [12]. In addition, the existence of microvascular obstruction (poor contrast penetration due to vascular damage) is defined by CMR, which is considered to be an independent left ventricular remodelling indicator of long-term prognostic effect [13]. Acute myocardial infarction is the product of superimposed luminal thrombus coronary arteriosclerosis. Myocardial infarction of regular nonatherosclerotic arteries may be triggered by the following factors: coronary spasm, coronary embolism and thrombosis. During a sustained cardiac arrest with resuscitation, concentric subendocardial necrosis may, on the one side, result from global ischemia and reperfusion. Myocardial ischemia shares characteristics with other forms of myocyte necrosis, such as inflammation, but unique
differences occur from myocyte hypoxia that differ depending on vessel occlusion, time between occlusion and reperfusion, and collateral circulation [14]

3. Prevention of myocardial infarction

The probability of repeated myocardial infarction decreases with strict blood pressure and dietary improvements, primarily avoidance of smoking, daily exercise, a sensible diet for heart attack patients and restriction of alcohol consumption [15]. Patients are typically initiated for multiple long-term post-MI therapies to avoid cardiovascular events such as more myocardial infarctions, cerebrovascular accident (CVA) [16]. Unless contraindicated, the likelihood of collapse of a plaque and repeated myocardial infarction can continue to be minimized by antiplatelet treatment such as aspirin and/or clopidogrel. Due to its low cost and equal effectiveness, aspirin is first rate, with clopidogrel reserved for those with aspirin sensitivity. Clopidogrel and aspirin in combination may further decrease the risk of cardiovascular events, although the risk of bleeding is higher [17]. Beta blocker treatment, such as metoprolol or carvedilol, should be initiated [18]. These were particularly helpful in high-risk conditions, such as left-ventricular failure and/or continued heart disease [19]. Mortality and morbidity were minimized by β-Blockers. ACE inhibitor therapy can begin 24–48 hours after MI in hemodynamically healthy patients, especially in patients with MI background, diabetes mellitus, hypertension, anterior infarction position (as measured by ECG) and/or signs of left ventricular dysfunction. ACE inhibitors decrease death, heart disease progression and post-MI ventricular restructuring [20]. Statin therapy has been found to decrease post-MI mortality and morbidity [21-22]. The benefits of statins could be stronger than their lowering LDL effects. The common opinion is that statins have plaque stabilization and many other effects that in addition to their effects on blood lipids, may avoid myocardial infarction [23]. In patients with cardiac disease and left ventricular impairment when used in combination with traditional
treatments, the aldosterone blocker agent eplerenone has been shown to further decrease the likelihood of cardiovascular mortality after MI [24].

Need of traditional medicines for myocardial infarction

Indigenous system of medicine employs medicinal substances from various sources, like; plant, animal, mineral, etc. The formulations have been used since ages with apparent benefit in various disorders including certain serious chronic ones [25]. They are also claimed to be safe and free of any adverse effects. However, they lack scientific evidences in support to their claims. Traditional medicine incorporates medical information structures that emerged in diverse cultures over centuries before the age of mainstream medicine. Traditional herbal remedies, Ayurveda, Unani, ancient Iranian medicine, islamic, conventional Chinese medicine, acupuncture, traditional African medicine and other medicinal expertise and procedures around the globe are all methods regarded as traditional medicines. Traditional medicine is defined by the World Health Organization (WHO) as: "Health habits, methods, information and values integrating medicines centered on plants, animals and minerals, spiritual therapies, manual strategies and exercises, implemented individually or in combination to cure, diagnose and prevent diseases or sustain well-being” [26].

In certain Asian and African nations, up to 80 per cent of the people depend on conventional medicine for their primary health care needs. Modern medicine is also referred to as complementary and natural medicine when adopted outside the traditional society. Herbal drugs may be very profitable, producing revenues of billions of dollars, but a health threat may also be adulteration or synthetic herbs. However the WHO also states that excessive usage of conventional medicines or practices can have harmful or harmful consequences and that more study is required to evaluate the effectiveness and protection of some of the practices and medicinal plants used by traditional medicine systems [27].
4. Conclusions

Despite the plenitude of knowledge regarding MI, its frequency continues to be on the surge. Therefore, it is a requisite to discover novel safe, potent and relatively economical drug contenders like herbal medicines. However, clear clinical therapeutic benefits have not yet been assured. Per se, herbal medicines cannot be safely recommended as an alternative therapeutic medicine. Superior-designed studies and future clinical trials involving larger sample sizes are need of the hour to examine the role of different medicinal plants and their underlying mechanisms in the context of MI. Safety and toxicity of these herbal remedies ought to be the priorities for all the future clinical trials.

References


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