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Progress in the Study of Coronary Atherosclerosis and Prevention of Acute Myocardial Infarction

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Abstract

Cardiovascular disease stands as the leading cause of global mortality, with coronary atherosclerosis emerging as the prevailing pathological alteration within this condition. This lesion is distinguished by a notable escalation in oxidative stress. The prevalence of coronary atherosclerosis has exhibited a steady rise over time, leading to a consequential escalation in fatalities resulting from acute myocardial infarction caused by this condition. This alarming trend poses a significant threat to the well-being of our population. To address the imperative issue of preventing acute myocardial infarction caused by coronary atherosclerosis, this study offers a novel theoretical framework elucidating the pathogenesis and progression of acute myocardial infarction. By presenting and consolidating the findings of our research, this project aims to contribute to the prevention of acute myocardial infarction in recent years.

The prevention of atherosclerotic plaque formation, narrowing of blood vessel lumen, obstruction, and myocardial ischemic infarction resulting from heightened oxidative stress in the human body, diminished vitamin C levels, impaired collagen formation, and unregulated cardiac wall repair system necessitates the reduction of oxidative intake, elimination of oxidative accumulation within the body, or augmentation of antioxidant intake.

Furthermore, a substantial body of epidemiological evidence indicates that aberrant lipid metabolism serves as the primary predisposing factor for acute myocardial infarction. Additionally, both intermittent fasting and fat reduction therapy have demonstrated efficacy in preventing acute myocardial infarction. Moreover, it is worth noting that reducing non-high-density lipoprotein cholesterol (nHDL-C) is considered a secondary objective of fat reduction therapy. Moreover, magnesium is a significant dietary component that is frequently disregarded, yet numerous scientific investigations have substantiated the inverse relationship between the presence of magnesium ions in potable water and the prevalence of mortality due to coronary heart disease. However, iron, as an additional constituent, significantly contributes to the susceptibility of individuals to coronary artery disease and myocardial infarction.

Keywords: Vitamin C; Metabolic Syndrome; Magnesium; Iron

Abbreviations: CAT: Lecithin-Cholesterol Acyl Transferase; AMI: Acute Myocardial Infarction; TC: Total Cholesterol; TG: Triglycerides; LDL-C: Low-Density Lipoprotein Cholesterol; Hb: Haemoglobin.

Introduction

Cardiovascular disease, being the primary cause of mortality on a global scale, affects an estimated 300 million individuals presently, resulting in an annual average of approximately 7 million fatalities. While the etiology of cardiovascular disease is multifaceted and diverse, encompassing numerous risk factors, contemporary scholarly literature widely acknowledges coronary atherosclerosis as the prevailing pathological alteration in this condition. This lesion is distinguished by a notable elevation in oxidative stress. The prevalence of coronary atherosclerosis has exhibited a consistent upward trend, leading to a corresponding increase in fatalities resulting from acute myocardial infarction. This alarming situation poses a significant threat to the well-being of our population. To address the issue of preventing acute myocardial infarction caused by coronary atherosclerosis, this project aims to present and consolidate the findings of recent research, offering a novel theoretical framework elucidating the pathogenesis and progression of acute myocardial infarction for the purpose of prevention.

Causes

Approximately half of acute myocardial infarctions can be precipitated by factors such as physical activity involving sudden exertion of force or excessive fatigue, as well as emotional triggers including excessive anxiety, emotional impulses, and stress. Additionally, myocardial infarction can be attributed to various other factors such as consumption of a heavy meal, alcoholism, exposure to cold stimuli, significant blood or fluid loss, shock, arrhythmia, sudden elevation in blood pressure, infection, and post-surgical complications [1]. The occurrence of myocardial infarction in the patient may also be attributed to atherosclerosis. In light of the presence of atherosclerosis, these precipitating factors can also exert a provoking influence, leading to coronary artery spasm, thrombosis, plaque rupture, and ultimately resulting in myocardial infarction [2]. The subsequent factors have the potential to induce myocardial infarction.

Pathogenesis

The occurrence of acute myocardial infarction, a pathological alteration within the realm of cardiovascular disease, is currently under investigation through the development of a novel theory [3,4]. This theory posits that acute myocardial infarction is instigated by a series of underlying processes. This theory posits that acute myocardial infarction is attributed to various processes, (1) an elevation in oxidative stress within the body resulting from an overabundance of oxidants and a decline in dietary antioxidants, particularly vitamin C. Consequently, this leads to an escalation in overall oxidative stress within the body ;(2) The depletion of antioxidants leading to high oxidative stress contributes to the development of vitamin C deficiency. (3) Vitamin C deficiency, in turn, results in impairments in collagen synthesis. (4) Collagen structure plays a crucial role in the maintenance of vascular wall stiffness and integrity, as it is a significant constituent of the capillary wall [5]. Collagen Defects in collagen directly cause defects in the function of the vascular wall and damage to the vascular wall; (5) Cholesterol [6,7], calcium [8], monocytes, etc. enter the vessel wall in an attempt to repair and strengthen the weak vessel wall, due to increased oxidative stress, decreased vitamin C [9], prolonged collagen formation, etc., as well as this unhealthy system of repairing the heart wall that cannot be controlled, it ends up causing atherosclerosis, narrowing of the lumen of the blood vessels, obstruction, and ischaemic infarctions of the myocardium [10].

Ideas for prevention

According to this theory, the concept of mitigating acute myocardial infarction resulting from coronary atherosclerosis can be summarized as follows:

Reducing the intake of oxidants, degrading and removing oxidants accumulated in the body. Therefore, antioxidants (vitamin C, vitamin E, glutathione, anthocyanins and other antioxidants) also help to remove powerful oxidants that are harmful to the body. The water-soluble vitamin C and the less fat-soluble vitamin E work synergistically. They can also play important roles at the same time. In addition, when vitamin E is “disarmed” by various oxidants, vitamin C helps to provide energy for vitamin E. Vitamin C is then regenerated with the help of glutathione, which then uses anthocyanins to complete the regeneration, so they are mutually synergistic when they work together. To supplement these antioxidants, it is necessary to consume the following foods: Fruits - the safest fruits with high levels of heat detoxification, including prunes, apricots, cantaloupe, kiwi, papaya, peach, mango, red grapes, citrus fruits and a variety of nuts. Vegetables - turmeric, green peppers, beetroot, broccoli, purple kale, large carrots, collard greens, baby carrots, kale, squash, spinach, sweet potatoes, tomatoes, beans, bean sprouts, and bean sprouts.

It is possible to increase the intake of antioxidants, particularly vitamin C, which can reduce/stop atherosclerosis [11], and increase the rate of collagen re-synthesis within the wall, restoring wall the strength and integrity.

A low plasma levels of vitamin C in patients with unstable angina alone predict unstable angina syndrome [12]. In myocardial infarction, vitamins C, E, and A prevented the development of major cardiovascular disease and

reduced the risk of developing major complications [13]. The use of vitamins C, E, and A in myocardial infarction may prevent greater cardiovascular disease and reduce the risk of higher complications. Vitamin C (250 mg/kg) given before extracorporeal myocardial surgery has also contributed to myocardial growth and repair [14]. (b) Persistent manifestations of oxidation shown during angioplasty of capillary walls in unstenting coronary thromboplasty [15]. The risk of coronary restenosis in patients in the group given a lower dose (500 mg) of vitamin C was much lower than in control patients not given vitamin C [16]. (c) The risk of coronary restenosis was much lower in the group given the lower dose (500 mg) was much lower than in the control group not given vitamin C; Vitamin C also inhibits the entry of cholesterol into the capillary wall and also accelerates the removal of cholesterol deposited in the capillary wall. Lecithin-Cholesterol acyl transferase (LCAT) is a protease that catalyses the release of cholesterol in the capillary wall. Because vitamin C prevents the rapid breakdown of cholesterol acyltransferase, higher doses of vitamin C allow more cholesterol to be automatically cleared from the capillary walls [17]. The higher the dose, the more cholesterol will be excreted from the capillary walls automatically.

Dietary Habits

Improving the body's antioxidant capacity can be achieved by adopting healthy dietary practices, including the adoption of a suitable low carb/ketogenic diet. Furthermore, it is worth noting that the aforementioned diet also possesses anti-inflammatory properties. Additionally, it is important to acknowledge that ketone bodies themselves exhibit antioxidant properties [18].

Metabolic Syndrome

Numerous epidemiological studies have consistently demonstrated that hypertension, dyslipidemia, smoking, obesity, and hypertension are the primary risk factors associated with coronary atherosclerosis, with dyslipidemia being the most extensively investigated. Perturbations in lipid metabolism represent the foremost predisposing elements for acute myocardial infarction, primarily characterized by elevated levels of total cholesterol, triglycerides, LDL cholesterol, and diminished levels of HDL cholesterol. Consequently, the management of metabolic syndrome and the subsequent prevention of acute myocardial infarction entail the following interventions.

A potential therapeutic approach for metabolic syndrome involves enhancing the condition of the arterial wall through the dissolution of precipitated cholesterol, calcium, and other substances within the wall, subsequently facilitating their discharge into the bloodstream.

Intermittent fasting, through the reduction of caloric intake [19], presents a promising avenue of investigation in the realm of acute myocardial infarction prevention [20], as it effectively mitigates oxidative stress.

Lipid-lowering therapy has been demonstrated as an efficacious approach in the prevention of acute myocardial infarction (AMI) [21]. Historically, the primary objective of lipid-lowering therapy was to actively decrease total cholesterol (TC), triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C). However, even when these lipid markers reached the desired target levels, certain patients still experienced cardiovascular disease events [22].

Nevertheless, despite achieving target levels of TC, TG, and LDL-C, certain patients continue to experience cardiovascular events, suggesting that relying solely on theoretical indices for assessing the risk of acute myocardial infarction is incomplete. Consequently, in recent years, there has been a growing interest in investigating novel lipid indices and their association with acute myocardial infarction. Liu H, et al. [23] conducted a prospective study examining the association between nHDL-C3 levels and the risk of coronary atherosclerosis in a substantial patient cohort. The results indicated that the impact of nHDL-C levels on acute myocardial infarction was marginally greater than that of LDL-C. Consequently, the U.S. National Cholesterol Research Program's Adult Drug Guidelines (NCEPATPIII) recommended considering non-high-density lipoprotein cholesterol (nHDL-C) as a secondary target for lipid-lowering therapy [24].

Magnesium

Magnesium, a vital nutrient, is frequently overlooked, yet numerous scientific studies have substantiated the inverse relationship between the concentration of magnesium ions in drinking water and the mortality rate associated with coronary heart disease. In regions with limited water resources, such as the coastal areas of China, desalinated water is commonly utilized as a primary or even sole source of supply. The process of ocean desalination involves the conversion of salt water containing a significant amount of dissolved solids (TDS) [25], exceeding 40,000 mg/L into fresh water with a reduced TDS concentration. Additionally, this process effectively eliminates impurities present in seawater, including organic matter residues and bacteria.

Desalinated water exhibits high purity, low particle concentration, and low hardness, rendering it a suitable form of "soft water" [26]. The primary techniques employed for its production include reverse osmosis, low-pressure multi-effect, and multi-stage flash evaporation. Among these, the low-pressure multi-effect process is particularly

effective in generating high-quality freshwater, with total dissolved solids typically falling within the range of 2 to 50 mg/L. Hence, the utilization of desalinated water as a water treatment solution proves to be a favorable option for the desalination of seawater [27]. Consequently, desalinated water is devoid of any germs and organic bacteria, while exhibiting a heavy metal element concentration similar to that of conventional drinking water, rendering it the optimal choice [28]. Moreover, the reverse osmosis process employed in the preparation of desalinated seawater ensures a higher degree of purity [28].

The water quality is significantly improved through the removal of salts and minerals, with a removal rate exceeding 99%. However, this process also eliminates beneficial elements such as calcium, magnesium, fluorine, iron, manganese, zinc, cadmium, among others. Consequently, desalinated water exhibits minimal overall hardness and nutrient content, with general hardness levels below 7Smg/L, magnesium content below 7mg/L, and calcium content below 6mg/L [29,30]. The predominant deficiency in magnesium and calcium content is observed in our daily drinking water, which consequently hinders the availability of magnesium. Given the significant role magnesium plays in the human body, this deficiency bears substantial implications.

Magnesium is absorbed through the digestive system and predominantly eliminated in fecal matter. It serves as a significant cofactor for approximately 350 proteins, primarily engaged in energy metabolism. Additionally, magnesium plays a crucial role in facilitating ion and nutrient transfer across the body, contributing to the structural composition of proteins and nucleic acids. Furthermore, it is indispensable for maintaining optimal insulin sensitivity, as well as supporting neuromuscular excitability and tendon contraction. Adequate magnesium consumption additionally serves to mitigate the risk of atherosclerosis, convulsions, and insulin resistance, while safeguarding bone density and averting the onset of osteoporosis.

In a comprehensive analysis of 4,678 patients diagnosed with acute coronary syndromes, Israeli researchers have demonstrated a noteworthy association between the utilization of seawater desalination water and diminished survival rates at both the 30-day and one-year marks. Additionally, these patients exhibited a substantially elevated risk of all-cause mortality within the same timeframes when compared to individuals consuming freshwater. Conversely, prior to the introduction of seawater desalination water, no statistically significant disparities in survival rates or mortality risks were observed between the two geographical regions under investigation. Prior to the implementation of desalinated water supply, there existed no statistically significant disparity in survival rates and mortality risks associated with acute coronary syndromes between the aforementioned regions [31]. There was no significant statistical difference observed in the risk of acute coronary syndrome between the two regions.

Consuming non-desalinated water and augmenting magnesium intake have been found to be beneficial in mitigating mortality rates associated with acute myocardial infarction [32].

Iron

Elevated levels of iron, particularly in the vessel wall, play a significant role in the progression of atherosclerosis, as unbound iron, devoid of storage or binding to large proteins, serves as the archetypal oxidizing agent [33]. Additionally, ferritin, a substantial source of iron, acts as a prominent iron reservoir. Heightened serum ferritin concentrations have been linked to an increased susceptibility to coronary artery disease and myocardial infarction [34]. The range of serum ferritin levels considered normal is typically broad (20-400 ng/ml), encompassing a wide range of abnormalities. However, the actual range is generally narrow, ranging from 15 to 30 ng/ml. In cases where serum ferritin surpasses 30 ng/ml or when there is no conclusive evidence of iron deficiency anemia, it is not recommended for individuals, including women, to supplement with iron [35]. Iron deficiency anemia is characterized by low levels of hemoglobin (Hb) in females (<12g/dl) and males (<13g/dl), as well as transferrin levels below 20%, ferritin levels below 30ug/L, and mean corpuscular hemoglobin (MCH) below 30g/l. Individuals with hemochromatosis who do not meet these criteria, even if they have small molecule hypochromic chronic anemia, should not be administered iron supplementation. Incorporating iron-related assessments, particularly serum ferritin, into routine blood examinations is imperative. Consequently, individuals afflicted with atherosclerosis ought to moderate their consumption of animal liver, animal blood, and other edibles abundant in iron.

Regular Movement

Athletes, specifically joggers, exhibited notably elevated levels of vitamin C compared to non-athletes who received an equivalent dosage of vitamin C. The degree of physical activity positively correlated with the vitamin C levels, with joggers covering 10 miles demonstrating the highest levels, followed by those covering 5 miles, and finally sedentary individuals [36]. This phenomenon could potentially be attributed to the substantial excretion or depletion of iron that occurs during perspiration. The findings of this study demonstrated a notable reduction of approximately fifty percent Shen S, et al. [36] in ferritin levels among the male participants following a four-week exercise regimen. Consequently, this decline

significantly impeded the development of atherosclerosis. Hence, to mitigate the initiation and advancement of atherosclerosis, it is advisable to engage in regular exercise at an intensity equivalent to a ten-mile jog.

Conclusion

With the global prevalence of coronary atherosclerosis steadily rising, there is a pressing need to incorporate strategies for preventing acute myocardial infarction, a distinctive ailment, into the daily routines of the general population. The aforementioned compounds, namely vitamins C, E, A, glutathione, anthocyanins, and magnesium, were synthesized by our research group. Based on our synthesis, we postulate that patients would benefit from increasing their consumption of foods rich in these compounds, while also emphasizing the intake of non-desalinated water. Conversely, it is advisable for patients to limit their consumption of iron-rich foods, such as animal liver and animal blood. In terms of lifestyle, clinical intervention for acute myocardial infarction with coronary atherosclerotic lesions by means of lipid-lowering therapy through appropriate low-carbon ketogenic diets or intermittent fasting, as well as regular physical exercise, should yield good clinical outcomes.

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