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The Impact of Omega-3 Fatty Acids on the Most Common Cardiovascular Diseases – A Literature Review

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ABSTRACT

Background

Epidemiological data suggest a beneficial effect of omega-3 fatty acids on cardiovascular risk.

Aim of the study

The aim of this literature review is to compile currently available studies and examine the mechanisms of action of omega-3 fatty acids and their application in the most common cardiovascular diseases, such as hypertension, coronary artery disease, atrial fibrillation, and heart failure. The article also analyzes the relationship between clinical and therapeutic effects and factors such as dose, the presence of cardiovascular diseases, and the population under study.

Material and methods

Literature available in the PubMed database was reviewed using the following keywords „Omega-3" „fish oil" „hypertension" „heart artery disease" „cardiovascular risk" „ cardiovascular diseases" „ heart diseases" „ cardiovascular outcomes" „heart failure" „anti-inflammatory"

Conclusion

This literature review suggests that omega-3 fatty acids may improve treatment outcomes in certain cardiology patients. Clinical effects are dose-dependent, and influenced by the duration of supplementation as well as the population in which they are applied. Regardless of the dose, omega-3 fatty acids do not reduce cardiovascular risk; however, their addition to therapy in patients already undergoing cardiological treatment may offer benefits. Higher doses may exert hypotensive effects, significantly reduce hypertriglyceridemia, and improve left ventricular ejection fraction, although they are associated with an increased risk of atrial fibrillation.

Keywords

„omega-3 fatty acids" „PUFA" „EPA" „DHA" „arterial hypertension" „coronary heart disease" „atrial fibrillation" „heart failure"

Introduction

Cardiovascular diseases (CVD) are the leading cause of death in developed countries [1].
In the 1970s, researchers observed a lower incidence of coronary artery disease among residents of Denmark, who traditionally followed a seafood-based diet rich in polyunsaturated fatty acids (PUFAs) [2]. Over time, the impact of omega-3 fatty acids on cardiovascular risk has been more thoroughly studied. Omega-3 fatty acids are attributed with functions such as cellular membrane formation, anti-inflammatory, anti-aggregatory, and cardioprotective effects [3]. For this reason, omega-3s are being explored for their potential use in cardiovascular diseases. The aim of this article is to compile available studies and examine how omega-3 fatty acids may currently influence the most common cardiovascular diseases, including hypertension (HT), coronary heart disease (CHD), atrial fibrillation (AF), and heart failure (HF).

- **Arterial hypertension**

Omega-3 fatty acids may reduce blood pressure, likely through an increase in nitric oxide production and improvement in endothelial function by interacting with molecules such as E-selectin, VCAM-1, and ICAM-1 [4]. Other mechanisms through which omega-3 fatty acids may reduce blood pressure include a reduction in heart rate, decreased oxidative stress, as well as inhibition of L-type Ca^{2+} channels, activation of K^{+} channels, activation of the TRPV4 channel, or inhibition of TRPC1/5 channels [5]. The results of studies on the effect of omega-3 fatty acids on blood pressure are not entirely consistent and may depend on factors such as race and genotype. In one study, researchers observed that a greater reduction in blood pressure occurred in patients with the CYP4F2 V433M genotype [6]. Additionally, a different hypotensive response to omega-3 fatty acids has been observed in Asian populations compared to Western populations, likely due to polymorphisms in the FADS2 gene, which provide an advantage in Asian populations [7].
Polyunsaturated omega-3 fatty acids lower blood pressure in patients with primary hypertension, untreated hypertension, and in individuals with normal blood pressure but with hypercholesterolemia. No hypotensive effects were observed in healthy individuals with normal blood pressure. This is likely associated with improved endothelial function in individuals with cardiovascular diseases [8]. A 2014 meta-analysis showed that the administration of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) may be more effective than other lifestyle interventions, such as sodium restriction or increased physical activity, in individuals without antihypertensive therapy [9]. According to more recent studies, the hypotensive effect may be dose-dependent, but this is not a linear relationship; the greatest reduction in systolic blood pressure (SBP) and diastolic blood pressure (DBP) occurs with doses of 2-3g/day [10]. It is worth noting that some meta-analyses did not provide evidence of a significant reduction in blood pressure with increased omega-3 fatty acid intake [11].

- **Coronary Artery Disease**

Atherosclerosis is a vascular disease that is one of the leading causes of death worldwide, characterized by a chronic inflammatory process and lipid metabolism disorders, leading to the accumulation of lipids in the blood vessel wall.

Epidemiological studies indicate that moderate fasting hypertriglyceridemia or postprandial hypertriglyceridemia (175–499 mg/dL) increases the risk of atherosclerotic cardiovascular disease [12]. Polyunsaturated omega-3 fatty acids influence the reduction of triglyceride levels [13]. Another pathogenic factor in coronary artery atherosclerosis is the inflammatory process. Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) have been shown to exhibit anti-inflammatory effects by altering eicosanoid production and reducing the generation of other inflammatory mediators, such as chemokines, cytokines, and adhesion molecules. Additionally, omega-3 fatty acids have antithrombotic effects [14,15]. Beyond lowering triglyceride levels, EPA and DHA, at the cellular level and in animal models, inhibit the deposition of calcium in the arteries; however, it is unclear whether this effect occurs in humans [16]. Thus, omega-3 fatty acids exert their effects on atherosclerotic plaque formation at multiple levels. A meta-analysis of 10 large studies using low doses of EPA and DHA did not show an impact on cardiovascular risk [17]. According to 86 randomized controlled trials (RCTs) involving 162,796 participants, omega-3 supplementation, including higher doses, does not significantly affect cardiovascular events [18]. Other studies in patients with low omega-3 levels, consuming <1.5 fish meals per week, suggested that omega-3 supplementation reduces the risk of adverse cardiovascular events by 19% [19]. Based on research, it is recommended to consider adding omega-3 fatty acids to statin therapy, as such therapy likely more effectively slows the progression of atherosclerotic plaques and, in patients with hyperlipidemia, further reduces cardiovascular risk [20,21].

- **Atrial Fibrillation**

Atrial fibrillation (AF) is the most common arrhythmia worldwide and significantly impacts mortality, making it a major public health concern [22]. Laboratory studies and animal models have demonstrated the antiarrhythmic effects of omega-3 fatty acids [23,24].

The antiarrhythmic benefit may arise from several mechanisms, including modulation of ion channel properties, membrane stabilization, and effects on the sympathetic-parasympathetic balance [25]. Earlier meta-analyses suggested that polyunsaturated fatty acids may reduce the risk of atrial fibrillation [26]. However, in many studies evaluating the impact of omega-3 fatty acids on cardiovascular risk, an increased incidence of AF was observed. The mechanism by which omega-3 supplementation may induce atrial fibrillation remains unclear [27,28]. The risk of atrial fibrillation is notably higher when the omega-3 supplementation dose exceeds 1g/day [29].

- **Heart Failure**

In developed countries, the incidence of heart failure has been increasing in recent years due to the aging population. According to the ESC Long-Term Registry, the most commonly treated outpatient patients for heart failure are those with reduced ejection fraction (HFrEF) [30]. In HFrEF, myocardial injury leads to the activation of inflammatory responses by the immune system [31]. Inflammation is one of the main factors contributing to poor prognosis in heart failure.

Studies suggest that polyunsaturated omega-3 fatty acids, such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), may inhibit inflammation through various mechanisms, including modulation of gene expression in the nuclear factor kappa B (NF-κB) pathway [32]. One meta-analysis suggested that omega-3 fatty acids may reduce inflammatory markers such as IL-6, TNF-α, hs-CRP, and inhibit left ventricular remodeling [33]. Research indicates the potential benefits of omega-3 supplementation in heart failure management [34]. Furthermore, the WMA meta-analysis explored the dose- and time-dependent effects of omega-3 supplementation. High doses of 2-4g/day for at least one year significantly improved left ventricular ejection fraction and peak oxygen uptake (VO2), whereas lower doses or shorter treatment periods did not yield the same effects [35].

- **Conclusion**

Omega-3 fatty acids, due to their actions such as vasodilation, improvement of endothelial function, reduction of triglyceride levels, anti-inflammatory effects, inhibition of atherosclerotic plaque progression, and enhancement of left ventricular ejection fraction, are being explored for their potential use in the treatment of cardiovascular diseases [4,13,14,15,32,33,35]. Some clinical effects of omega-3 supplementation appear to be dependent on factors such as dose, duration, and population [6,7,10,29,35]. According to research, EPA and DHA supplementation, regardless of the dose, does not reduce cardiovascular risk or mortality [17,18]. Some studies suggest that their addition may be beneficial for patients already treated with statins or those with pre-existing cardiovascular diseases, compared to healthy individuals, likely due to impaired endothelial function, which omega-3 fatty acids can improve [8,20,21]. High doses may improve left ventricular ejection fraction and peak oxygen uptake in heart failure [35]. However, it should be noted that higher doses significantly increase the risk of atrial fibrillation [27,28,29].

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