

Diet and Cardiovascular Diseases: Heart Failure

Part I: Plant Based Diet, Beneficial Macronutrients

Abstract

Diet is a major modifiable factor in the prevention and management of cardiovascular diseases. Optimal body weight with a BMI between 20 and 24.9 (18.5 to 24.9 for Asians), and a diet rich in non-starchy vegetables, fruits, whole grains, legumes, moderate in consumption of nuts, seafood, lean meats, low-fat dairy products, and unsaturated vegetable oils, and limited in saturated fats, sodium, red meat, refined carbohydrates, and sugar-sweetened beverages is cardiovascular protective. Trans fats are extremely unhealthy. The benefit of a healthy diet extends to heart failure (HF). Dairy, coffee, tea, and chocolate may also be vascular friendly. Alcohol has a U-shaped relationship, with low to moderate intake cardiovascular healthy, while heavy intake usually causes harm. Some minerals and vitamins are also helpful in the prevention and management of HF. HF is increasing in incidence and prevalence in most world countries. It is associated with severe disability and is a major cause of death globally. The impact of a healthy diet on the development and progression of HF is discussed in this manuscript (in 2 parts).

Keywords: heart failure, plant-based diet, processed red meat, Mediterranean diet, DASH diet

Introduction

Heart failure (HF) is a common global disease¹, affecting between 1% and 2% of all adults². There were 56.2 million prevalent cases of HF in 2019³ with the older population is disproportionately affected⁴. The lifetime risk of developing HF at age 55 is 33% for men and 28% for women⁵. With growing aging population and increasing worldwide obesity, the prevalence of HF is projected to increase by nearly 50% between 2012 and 2030⁶. HF results from an abnormality of cardiac structure or function⁷. The Heart Failure Association (HFA) of the European Society of Cardiology (ESC) defines HF into three subtypes depending on the left ventricular ejection fraction: HF with a reduced EF (HFrEF) [EF is < 40%], HF with a mid-range or mildly reduced EF (HFmrEF) [EF 40–49%], and HF with preserved EF (HFpEF) [EF ≥ 50%]⁸. HFrEF is the most common type in men, whereas HFpEF is the most common type in women⁹. In HFrEF, the systolic function is impaired, while in HFpEF, the diastolic function is abnormal. Several conditions act as etiologies for HF – these include hypertension (HTN)¹⁰, coronary artery disease (CAD)¹¹, dilated cardiomyopathy¹², hormonal dysfunction¹³, infections (such as Chagas' disease)¹⁴, and drug use¹⁵. The pathological processes leading to structural abnormalities and functional problems involved include hemodynamic stress¹⁶, neuro-endocrine activation¹⁷, and inflammation¹⁸.

Despite significant advances in the prevention and treatment of HF having developed in the last decades¹⁹, life continues to be difficult for these patients. Forward failure results in HF patients complaining of excessive fatigue, due to reduced aerobic capacity, decreased muscle strength, and limited exercise intolerance²⁰. With increasing reduction in peripheral circulation, signs of

cardiac cachexia develop. Backward failure due to volume or pressure overload leads to high pulmonary pressure and pulmonary congestion, leading to dyspnea. Liver congestion, ascites, and edema develop in the chronic stage due to volume overload, partly resulting from the permanent activation of compensatory neurohumoral systems²¹. Consequently, HF patients have a poor quality of life²². They are frequently hospitalized²³. It is estimated that 83% of HF patients are hospitalized at least once, and almost 45% are hospitalized four times or more times². HF is also associated with a high frequency of disability²⁴ and increased mortality²⁵. Almost 32.9% of patients with decompensated HF die within one year following hospital discharge²⁶. HF patients also frequently have multiple cardiovascular and non-cardiovascular comorbidities, which contribute to poor outcomes, increased rates of hospitalization, and higher mortality^{27,28}. HF is responsible for extremely high healthcare costs globally^{29,30}. **A healthy diet plays a major role in the prevention and decreased progression of heart failure.**

Discussion

According to estimates by the World Health Organization (WHO), about three-quarters of deaths from cardiovascular disease (CVD) can be prevented by changes in lifestyle³¹. Diet is an established modifiable risk factor involved in the prevention, development, and management of CVDs³². Healthy dietary patterns are associated with lower cardiovascular morbidity³³, lower disability³⁴, and improved quality of life (QOL)³⁵. A healthy diet also lowers CVD mortality rates³⁶. Heart failure patients are no exception. In a prospective cohort study of 20,900 healthy men, healthy lifestyle habits significantly decreased the lifetime risk of heart failure³⁷. Lack of exercise, smoking, excessive alcohol intake, obesity and a poor diet are major modifiable risk factors for HF³⁸⁻⁴⁰. Modification of diet remains an easy, cheap, non-drug, and non-interventional method of beneficially impacting HF. This manuscript discusses the various benefits and harms of different dietary patterns in the development and progression of HF. A healthy diet helps improve many features of HF⁴¹. Diet also plays a significant role in affecting the several risk factors for HF, including hyperlipidemia, HTN, and diabetes mellitus (T2DM)⁴².

Plant-Based Diet

Several meta-analyses have reported that diets rich in fruits, vegetables, whole grains, nuts, seeds, and other plant-based foods reduce the risk of heart disease, including heart failure⁴³⁻⁴⁵. In the Physicians' Health Study of 20,900 healthy male physicians, greater consumption of fruits and vegetables was associated with a decreased risk of HF⁴⁶. In a study of 38,075 Finnish people for a median of 14.1 years, higher consumption of vegetables was associated with a lower incidence of HF in men, but not in women⁴⁷. In a prospective cohort from Sweden of 34,319 women without CVD and cancer at initial assessment, after a follow up period of 12.9 years, greater fruit and vegetable consumption was associated with a lower rate of HF⁴⁸. In the Reasons for Geographic and Racial Differences in Stroke study (cohort of 15,569 people with no CAD or HF diagnosis at enrollment, showed in a subset analysis that after a median follow-up of more than 7 years, patients with closer adherence to plant-based dietary patterns had a lower risk of incident HF⁴⁹. The beneficial role of plant-based diets in improving ejection fraction and positively remodeling the cardiac muscle is now well accepted^{50,51}. Plant-based diets reduce

inflammation⁵², decrease oxidative stress⁵³, reduce blood pressure (BP)⁵⁴, lower HbA1c⁵⁵, produce less trimethylamine-N-oxide (TMAO)⁵⁶, and lower serum lipid levels⁵⁷.

Vegetables and Fruits

Fruits and vegetables contain several micronutrients that help protect against HF. In a cross-sectional study, Polidori et al. found lower plasma concentrations of lutein, zeaxanthin, β -cryptoxanthin, lycopene, α -carotene, and β -carotene in patients with heart failure compared with healthy controls⁵⁸. A retrospective study reported marked decreases in HF incidence with increasing tomato consumption⁵⁹. Tomatoes like papaya and watermelon have high levels of lycopene, and lycopene is cardio protective⁶⁰. Several subsequent studies have confirmed the clinical implications of high fruit and vegetable consumption in HF^{46,47}. In a study of middle-aged and older women followed for >10 years, there was an inverse association between higher fruit and vegetable intake (esp. >5 servings/day) and the rate of HF incidence⁴⁸. Rautanen et al., in a study published in 2015, reported that the consumption of apples, pears, and berries as well as green leafy vegetables, was inversely associated with HF risk in a dose-response manner⁴⁸. Moderate vegetable (about 1 cup of green leafy vegetables (providing about 60 mg/day of vegetable nitrate) intake was associated with a 15% reduction in HF⁶¹. Besides the benefits mentioned before⁵²⁻⁵⁷, fruits and vegetables also increase ejection fraction⁶² and improve functional capacity⁶³. These studies support recommendations of intake of five servings per day of fruit and vegetables, for good HF health.

Chocolate

A meta-analysis showed that chocolate consumption in moderation may be associated with a decreased risk (16% less) of HF⁶⁴. However, higher amounts may lead to weight gain as chocolate, especially milk chocolate, often has a high calorie content⁶⁵. Obesity is a known risk factor for developing HF^{66,67}.

Fish

Several prospective studies have reported that fish consumption is associated with decreased HF risk⁶⁸⁻⁷³. A meta-analytic study reported an inverse association between HF risk and oily fish consumption⁷⁴. Fried fish, however, increases the risk of HF^{68,75}. It is postulated that unfried fish is beneficial due to its high content of omega-3 fatty acids⁷⁶.

Eggs

Prospective studies have reported an increased incidence of HF (28–64 %) with high egg consumption^{77,78}. A 2017 meta-analysis reported a 25 % increase in the risk of incident HF in those with the highest compared to the lowest egg consumption⁷⁹. Another recent study showed that intake of one egg per day was associated with an increased risk of heart failure when compared to no consumption, in US cohorts⁸⁰. The risk increased as the number of eggs consumed per week increased – for 7 eggs [summary relative risk or SRR = 1.15), for 8 eggs per week (SRR = 1.19 and for 9 eggs per week (SRR = 1.23) especially in men⁸⁰. The American Heart Association guidelines consider one egg per week safe for patients who are healthy⁸¹.

Alcohol

Most epidemiologic data is consistent with the potential benefits of moderate drinking on the risk of HF⁸². This relationship has been frequently studied. In the Framingham Heart Study, there was a 59% lower risk of HF in men consuming 8 to 14 drinks per week when compared to abstainers⁸³. The Cardiovascular Health Study reported a 34% lower risk of HF in people who consumed 1 to 6 drinks per week (Hazard Ratio=0.66) when compared with abstainers⁸⁴. Klatsky et al. found that light-to-moderate alcohol consumption was associated with a 40% to 50% lower risk of HF in individuals with antecedent myocardial infarction (MI)⁸⁵. In another large study, compared with abstainers, US male physicians reporting alcohol consumption of 7 or more drinks per week had a 38% lower risk of HF⁸⁶. More recently, Larsson et al., in meta-analysis, (total of 13 prospective studies, with 13,738 HF cases and 355,804 participants), reported that light alcohol drinking (0.1-7 drinks/week) was inversely associated with the risk of HF (Risk Ratio or RR=0.86)⁸⁷. They found that compared with non-drinkers, the RR across levels of alcohol consumption was 0.90 for 3 drinks/week, 0.83 for 7 drinks/week, 0.84 for 10 drinks/week, 0.90 for 14 drinks/week, and 1.07 for 21 drinks/week⁸⁷. This study demonstrated that the risks of HF go up as the amount of alcohol consumed increases. Two other meta-analyses confirmed that high levels of alcohol consumption increase the risk of heart failure^{88,89}. In the United States, one "standard" drink is 12 ounces of regular beer, which is usually about 5% alcohol, 5 ounces of wine, which is typically about 12% alcohol, and 1.5 ounces of distilled spirits, which is about 40% alcohol⁹⁰. The detrimental effects HF were reported in 2005 by Klatsky and colleagues⁸⁵. They determined that the risk of HF without antecedent MI among heavy drinkers was 1.7-fold higher than in abstainers. In the most recent meta-analysis, it was noted that the dose-response relationship between alcohol consumption and HF appeared to be curvilinear⁸⁷. Compared to non-drinkers, the risk for 1-84 g per week, 85-168 g per week, 168-336 g per week, and >336 g per week was RR = 0.86, 0.88, 0.91, and 1.16, respectively. In the United States, a 'standard drink' (as described earlier) contains 14 g of pure alcohol⁹¹. Larsson et al., based on a meta-analysis of eight studies, concluded that former drinkers were at a higher risk for HF compared to lifetime abstainers (RR = 1.22)⁸⁷. Klatsky et al. found no association between beverage types (beer, wine, or spirits) and HF⁸⁵. Moderate alcohol intake also reduces the risk of two major risk factors for HF - MI and T2DM^{92,93}. The beneficial effects are mediated through raising high-density lipoprotein cholesterol⁹⁴, improving insulin sensitivity^{95,96}, raising plasma levels of adiponectin⁹⁷, inhibiting inflammation⁹⁸, improving endothelial function⁹⁹, influencing platelet aggregation¹⁰⁰, coagulation factors¹⁰¹, and fibrinolysis^{102,103}, and increasing the plasma concentration of atrial natriuretic peptide¹⁰⁴.

Whole Grain

Whole grain (kernel) is made up of three key edible parts – the bran, the germ, and the endosperm – covered by an inedible husk. The husk protects the kernel from external assaults such as sunlight, pests, water, and disease. Whole grains contain all three edible parts of the kernel. Whole grain is rich in biologically active compounds, such as fiber, vitamins, minerals, antioxidants, and other beneficial plant compounds including lignans and phytosterols¹⁰⁵⁻¹⁰⁷. These biological compounds help the cardiovascular system by several mechanisms, including

altering glucose homeostasis, lipids, and lipoproteins, and endothelial function¹⁰⁸. Several prospective cohort studies and their meta-analyses have reported a lower risk of CVD with a higher consumption of whole grains¹⁰⁹. Refined grains have the bran and germ removed and, as a result, lack several beneficial ingredients. The benefit of whole grain intake has been reported in HF. A prospective study of individuals consuming seven servings of whole grain breakfast cereal weekly, during a 19.6-year follow-up period, reported a 29 % decrease in HF risk when compared to individuals with no intake of these cereals¹¹⁰. A subsequent prospective study confirmed the beneficial effects and reported a 7 % decrease in HF risk with each additional wholegrain serving¹¹¹.

Dairy

There is a sparsity of studies on the effect of dairy on heart failure. A prospective study done in 2008 reported that dairy intake was associated with an 8 % increase in HF risk^{111,112}. However, a recent systematic review and meta-analysis of prospective studies noted an inverse relationship between biomarkers of dairy intake and HF incidence¹¹³.

Nuts

Nuts (such as almonds, Brazil nuts, cashews, hazelnuts, macadamias, peanuts, pecans, pine nuts, pistachios, and walnuts) are rich sources of mono and poly unsaturated fatty acids, protein, fiber, several minerals (e.g., magnesium, potassium, and zinc), vitamin E, folic acid, and other bioactive micronutrients such as phenolics and phytosterols¹¹⁴. Meta-analyses of prospective studies have shown that nut consumption is beneficial for cardiovascular diseases¹¹⁵⁻¹¹⁷. Data from prospective studies on nut consumption in relation to heart failure is, however, limited^{111,118,119}. Observational studies and clinical trials have shown cardiovascular benefits of nut consumption. Compared to non-consumers, one study reported that walnut consumption was associated with better diastolic function in young to middle-aged adults¹²⁰. Nut consumption is also associated with a decrease in BP¹²¹. HTN is known to increase the risk of HF by 2–3 fold¹²². Nut intake also helps to reduce the risk of T2DM¹²³ which is also a risk factor for HF¹²⁴. There is also a helpful weight loss with nut intake, provided the intake is not high¹²⁵. Two earlier prospective studies found no association between nut consumption and HF incidence^{118,119}. However, in a recent study, nut consumption was inversely related to heart failure. Compared with no nut consumption, for heart failure, the corresponding Hazard Ratio for (frequency of nut consumption: none; 1–3/month; 1–2/week; \geq 3/week) were 0.87, 0.80, and 0.98 respectively¹²⁶. The reduced risk of heart failure associated with moderate but not high nut consumption might be related to the fact that high nut consumption increases rather than decreases body weight.

Nut consumption may favorably influence cardiovascular health by improving blood lipid levels^{127,128}, and its anti-inflammatory¹²⁹, and antioxidant effects¹³⁰. It also helps improve endothelial function¹³¹.

Coffee

It is estimated that 400 million cups of coffee are drunk every day in the US. An 8-ounce cup of coffee contains 80 to 100 mg of caffeine, while an 8-ounce cup of decaf coffee typically has only 2-15 milligrams. The FDA cites four to five cups of coffee (about 400 mgs) per day as a safe amount of consumption¹³². Studies have found that this amount of intake is not associated with any increased risk of heart failure¹³³. Even consumption at higher levels – up to 600 mg/day, although may be associated with some cardiovascular symptoms, these are mild, transient, and reversible, with no lasting adverse effects¹³³. The ARIC study reported that increased coffee consumption also appears to correlate with a reduced risk of developing HF later in life (even after controlling for known risk factors)¹³⁴. Kolb et al. in 2020 even suggested that filtered coffee, without sugar or other additives, may be considered analogous to whole grains and vegetables as a dietary source of phenolic phytochemicals that could offer antioxidant protection in HF¹³⁵. There is some suggestion that increasing decaffeinated coffee consumption may increase HF risk, but the data is limited¹³⁶.

Conclusion

There is growing evidence that nutrition may play a critical role in HF development and progression. **A plant-based dietary pattern is a recognized cardiovascular risk mitigating lifestyle¹³⁷. This diet is high in antioxidants, micronutrients, dietary nitrate, and fiber but low in saturated/trans fats and sodium. Adherence to plant-based diets decrease HF incidence and severity¹³⁸.** They contribute to decreased oxidative stress, lower homocysteine levels, and reduced inflammation as well as to higher nitric oxide bioavailability and a better gut microbiome. Part II of this manuscript covers the relationship between HF and harmful macronutrients, obesity, and its relationship with several special diets.

Acknowledgements: None

Funding: None

Conflict of Interest: None

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